THESIS

Submitted for the Degree
of
Doctor of Medicine
University of Cape Town

BY

FRED BENJAMIN,
M.B. Ch.B (Cape Town) M.R.C.O.G.

JANUARY, 1960.

GLUCOSE TOLERANCE IN PATIENTS WITH ABNORMAL ENDOMETRIAL BLEEDING DURING AND AFTER THE CLIMACTERIC.

CONTENTS.

CONTENTS.

CONTENTS.

CHAPTER 6.

CONTENTS.

CHAPTER I.

INTRODUCTION AND HISTORY.

CHAPTER I.

INTRODUCTION AND HISTORY.

" All men desire by nature to know "

ARISTOTLE.

Two thousand years elapsed before man could unravel several of the problems and implications of disturbed glucose tolerance. As far back as 30 years B.C. gross manifestations of the disease now known as Diabetes Mellitus were recognised. Despite great advances, however, there is much that is still unknown.

The history of many a malady begins with the observation of the illness in its m ost grotesque form. As medical science gathers more and more knowledge, the less severe symptoms and signs come to be recognised. Gradually special tests are evolved, by means of which the disorder can be diagnosed in its earliest stages. And thereafter, the tendency to develop the condition is seen before it becomes

manifest.

The long history of disturbed glucose tolerance follows this pattern. Many centuries have passed in the gradual evolvement of the various stages. With the passing of the years improved diagnostic methods were discovered, so that mild forms of the disorder could be detected. In the past few decades the concept of mildly impaired glucose and "pre-diabetes" has been formulated. One of the most interesting of recent developments, for example, has been the conclusion that the birth of overlarge babies may be a sign of predisposition to this metabolic disturbance.

This thesis is an attempt to advance a stage further our knowledge of disturbed glucose tolerance in the gynaecological sphere. It represents a study of the association of impaired glucose tolerance with certain pathological states of the endometrium, including cancer; and it brings to light a manifestation that has not been described before, namely, certain benign dysfunctional endometrial bleedings.

As this study evolved, it became more and more apparent that only the fringe of the subject had been touched. The more one delved into the matter the more one realised how much remains to be investigated.

The conclusion is inevitable that, although this thesis has made some advances in our knowledge, its main contribution is in the opening up of wider fields for further research. Many gaps in our knowledge, and many avenues for further exploration, have been exposed. This is but a beginning, a further link in the chain of our understanding of this interesting and challenging subject.

HISTORY:

An ancient Egyptian record - Papyrus Ebers, mentions polyuria, and this is believed by some to be the first mention of diabetes. In the works of Hippocrates there is no clearly defined description of the disease. "Polyuria without pain, but with emaciation and danger" was recorded in the Graeco-Roman period by Celsus (30 B.C.- 50 A.D.).

Aretaeus of Cappadocia (30 - 90 A.D.) noted the "melting down of the flesh and limbs into urine" and gave the disease its name "Diabetes" - a Greek word meaning to "run through a siphon". Galen (131 - 201 A.D.) added little to the knowledge of

the disease; he attributed diabetes to a weakness of the kidneys. Diabetes was described as a "disease of thirst" by Tchang Tchangking (200 A.D.) who observed a patient, suffering from the disease, drink ten quarts of water a day with a relative degree of polyuria. "Excess appetite" was recorded as a symptom of diabetes by Chinese writers about 600 A.D. The "sweetness of the urine" of diabetic patients was first mentioned by Ayew Veda Susruta. Ants were observed to flock around the patients' urine. Diabetes is very common in India, and has probably been so for centuries. As early as the sixth century it was referred to as Madhumeba - honey urine - and its association with dietary indiscretions was then recognised. The Arabians were quite familiar with diabetes. Rhazes (860 - 932) commented upon its treatment, and Avicenna (980 - 1035) noted the association of carbuncle, gangrene and tuberculosis. Crystals were formed on the evaporation of the urine of a diabetic patient by Paracelsus (1493 - 1541), who mistook them for salt, and lipaemia was first associated with diabetes by Helmont (1574 - 1644).

Though Dutch, Portuguese and Italian physicians knew of the condition, little advance was made

in Europe, until Willis, in 1674 rediscovered the sweet taste of the urine "as if imbued with honey and sugar", and differentiated the disease from diabetes insipidus. Dobson (1715) made the next fundamental advance by recognising that the sweet taste was due to a sugar which fermented. However, it was not until 1815 that Cheureul found that the sugar was glucose. Disease of the pancreas was first described as occurring in a patient dying of diabetes by Cawley (1788). The adjective " Mellitus" was added to diabetes by Cullen (1709 - 1790) to distinguish the disorder from diabetes insipidus.

There then followed a period of empirical treatment. Rollo (1796) restricted the diet to animal food and a few green vegetables, fast days, restriction of carbohydrates, exercise, and alcohol! However, the importance of these observations was soon lost sight of.

An experimental era now started. Gregory demonstrated a fermentable sugar in the blood of diabetic patients. Trammer in 1841, and Fehling in 1850, introduced qualitative tests for sugar in

the urine. Claude Bernard (1885) founded the theory of sugar formation from glycogen and postulated that increased blood sugar levels were due to overproduction of sugar by the liver. He, also, first demonstrated the relationship between hyperglycaemia and glycosuria.

Parallel with these observations, a number of fundamental anatomical studies were made. Brunner, in 1682, discovered that removal of the pancreas produced polyuria and polydipsia in experimental animals. In 1788 Cowley noted that the disease may be caused by injury to the pancreas, but the importance of Brunner's observations was apparently over-looked for over 200 years. In 1869, Langerhans discovered the islets in the pancreas; but at that time the part played by this tissue in diabetes was still not suspected. In 1889, Von Meiring and Minkowski definitely established the connection between the pancreas and diabetes; by removing the pancreas, they succeeded in producing a diabetes in dogs, not unlike that in man. It took, however, another decade before Opie (1901)

and Ssobolew (1902) independently established the relationship between the islet tissue of the pancreas and diabetes. Naunyn introduced the term acidosis; he also recognised renal glycosuria.

Important advances included Allen's under-nutrition regime. He presented the hypothesis that diabetes was " a disorder of total metabolism and not of carbohydrate metabolism alone". Joslin and others confirmed the benefits of Allen's under-nutrition regime.

The discovery of insulin in 1921 by Banting and Best dwarfed in importance most other discoveries. Houssay and Negenta (1924) showed that the removal of the pituitary gland of the dog increased the animals' sensitivity to insulin. Diabetes was also attenuated by the removal of the adrenal cortex of depancreatized cats (Long and Lukens, 1936). Then in 1937, Young experimentally produced permanent diabetes by injecting, intraperitoneally, increasing amounts of crude extract of the anterior pituitary gland.

Protamine zinc insulin was discovered by Hagedorn in 1936. The selective destruction of the beta or insulin-producing cells of the islets of Langehans by the intravenous injection of Alloxan (Jacobs, 1937, and others) presented a new approach to the study of diabetes, and possibly to its aetiology. At the time of development and introduction of the suplhonamides as antibacterial agents in the early 1940's, it was noted in studies in animals that certain of the compounds lowered the blood sugar. However, no significant clinical application was made of these findings until 1955. Franke and Fuchs then published clinical observations on the potent hypoglycaemic action of a sulphonamide, Carbutamide. After extensive clinical trials in America, Canada, and the United Kingdom, however, carbutamide has been withdrawn from clinical use, because of serious toxic effects. In the past 2 or 3 years another sulphonylurea, tolbutamide, has been used without serious ill-effect in the treatment of mild diabetes of the adult type. How the drug acts is still uncertain, but its clinical usefulness is well established

and it is full of promise.

LATENT DIABETES AND " PRE-DIABETES ":

Twenty-seven years ago Skipper (1933) first observed that women may have big babies long before they develop overt diabetes. Thus, probably, was born the concept of "pre-diabetes". It was shown by Oakley and Peel (1949) that the peri-natal loss in pre-diabetic years is about 23%. This high foetal loss reaches its peak in the 2 years immediately preceding the diagnosis of diabetes. (Gilbert and Dunlup, 1948).

It is well-known that there is a high incidence of temporarily diminished sugar tolerance and even gross diabetes, in patients suffering from acromegaly, Cushing's syndrome, hyperthyroidism, sever, burns, staphlococcal infections, and other conditions. When investigations have been made, it has been found that there is a high incidence of diabetes in the families of persons so affected (Jackson, 1955: Joslin et al., 1952). It seems

likely that the particular condition in each case simply renders manifest a pre-diabetic state.

Several groups of pqtients may be suspected of being pre-diabetic: The following are suggestive features: 1) A positive family history 2) the birth of overlarge babies, especially if repeated 3) obesity 4) "temporary diabetes" during any acute stress 5) glycosuria during pregnancy-often merely attributed to renal glycosuria without full investigation and follow-up 6) xanthosis and mild carotinodermia (Hoet et al., 1958) 7) repeated attacks of acute pancreatitis 8) certain other conditions known to be frequently associated with mild diabetes e.g. gout (Weiss et al., 1957) 9) renal glycosuria or spontaneous non-insulinomatous hypoglucaemia (Seltzer et al., 1956) 1o) typical diabetic-type vascular disorder (e.g. retinal aneurisms, or angina pectoris in a premenopausal woman).

Evidence will be produced in this thesis to show that many of the dysfunctional endometrial bleedings, especially at the time of the climacteric, may be further manifestations of a

pre-diabetic, unrecognised diabetic, or frankly diabetic state.

CHAPTER 2.

EVOLUTION OF THE PRESENT INVESTIGATION.

CHAPTER 2.

EVOLUTION OF THE PRESENT INVESTIGATION.

"If circumstances lead me, I will find
Where truth is hid, though it were hid
Indeed within the centre"
SHAKESPEARE, Hamlet, Act II, Scene II.

During the past 20 years a controversy has been apparent in the gynaecological literature. Publications that have appeared since 1957 have made this problem even more confusing. The question at issue is whether cancer of the endometrium and diabetes mellitus are associated. Several investigators have found a positive association between these two diseases while others have not. The earliest part of the present investigation was an attempt to resolve this controversy. By carrying out a survey at the Groote Schuur Hospital, Cape Town, the association of these two conditions

was shown to be incontrovertible.

A more detailed and searching study of all the publications on the subject was then made, and thereby the reasons for the apparently contradictory findings were exposed. As part of my own investigation a large series of control subjects were studied, as well as patients suffering from abnormal uterine bleeding due to causes other than cancer of the endometrium. Some interesting and significant findings came to light which had not been previously reported. It was therefore considered advisable to carry out more extensive reasearch into the subject of glucose tolerance in all types of uterine bleeding, of both benign and malignant aetiology, that occur during and after the climacteric.

This thesis presents the research done, the results of the investigation, and an analysis and discussion of these results.

GLUCOSE TOLERANCE IN PATIENTS WITH CANCER OF THE ENDOMETRIUM:

In the ubiquitous search for the aetiology

for cancer, metabolic factors have been widely studied. Much attention has been paid to carbohydrate metabolism and glucose tolerance.
In 1934, Marble reviewed 256 cases of malignant diseases of all types, in which diabetes mellitus was known to be present. After a careful analysis of these cases, he came to the conclusion that diabetes does not predispose to cancer, nor cancer to diabetes, but that "both diseases occur more commonly in the aged". (In his series there were 151 females, 33 of whom had cancer of the cervix or body of the uterus; the number of cancers of the endometrium was therefore small). However, in 1948 Jacobson studied all the cancers that occurred among a very large series of diabetics and concluded that the incidence of all cancers " is higher among diabetics than among non-diabetic individuals".

These two workers based their conclusions on a study of cancers generally, and not on any specific type of cancer. In the two decades that followed, however, increasing evidence appeared that, when it came to a study of cancer of the

endometrium, the association of diabetes mellitus was much more significant. Figures that have received considerable attention in British literature are those of Stanley Way. In 1954 he reported that as many as 29% of 106 such patients had unquestionable diabetes, and a further 43% had a "pre-diabetic type of glucose tolerance curve". This remarkably high incidence of frank diabetes was found by other investigators too - Moss (1947) found 39% of diabetes among 23 cancers of the endometrium, and Garnet (1958) demonstrated that 33% of 50 consecutive cases of carcinoma of the body of the uterus suffered from diabetes; Louw (1958) also found a significantly high incidence. However, other workers were unable to confirm that there was an increased incidence of diabetes mellitus in their cases (Hertig et al., 1949; Smith, 1941; Palmer et al., 1949; Scheffey et al., 1943; Vander, 1958). Why are these reports so contradictory? If we analyse the methods whereby these various writers arrived at their diagnoses of diabetes mellitus, the reason for the discrepancy in the results becomes apparent. The results and the methods of making the diagnosis are

set out in Table I.

TABLE I. INCIDENCE OF DIABETES MELLITUS IN CASES OF CANCER OF THE ENDOMETRIUM.

	No. of Cases.	Impaired Glucose Tolerance			How Diabetes Diagnosed.
		Diabetic	Not Frank Diabetes	Total	
Moss (1947)	23	39%	26%	65%	Glucose Tolerance Tes
Garnet (1958)	50	33%	33%	66%	Glucose Tolerance Tes
Way (1954)	106	29%	43%	72%	Glucose Tolerance Tes
Present Series (1959)	50	28%	24%	52%	Glucose Tolerance Tes
Smith (1941)	307	4%			Clinically diabetic.
Vander (1959)	483	5.6%			Clinically diabetic; and then, only, fasti blood sugar.
Hertig et al. (1949)	500	9%			Clinically Diabetic.
Scheffey et al. (1943)		11%			Clinically Diabetic.

It is obvious, then, that whenever the cases are fully investigated by complete glucose tolerance tests a high incidence of diabetes mellitus is found. In each and every series where the incidence of diabetes mellitus was low, routine glucose tolerance tests had not been done - the disease had only been diagnosed when the patient was an obvious diabetic (eg. where there was clinical evidence of diabetes, glycosuria, or raised fasting blood sugar). In these latter series, therefore, only the severe diabetics were discovered (and then possibly not all of them) and many of the less severe cases, as well as all cases of mildly impaired glucose tolerance were missed. This closer analysis of the literature, as well as my own investigation, (to be presented later) shows that the high incidence of diabetes and of mildly impaired glucose tolerance is incontrovertible.

However, even these proven high figures are not significant unless they are compared with suitable control subjects, i.e. women of the same age groups, and under similar conditions, who do not suffer from cancer of the endometrium.

CONTROL SUBJECTS:

How can suitable controls be obtained? An exploration of the literature reveals 4 studies that are relevant:

1. Spiegelman and Marks (1946) studied 1,300,399 random females over the age of 35 years, by doing a single fasting blood sugar on each person, and found the incidence of diabetes to be as follows:

Age	Total Female Population	% with Diabetes.
Over 35 years.	1,300,399	1.02

2. In 1946, the United States Public Health Service began a survey of the town of Oxford, Massachusetts, which has a population of about 5,000. Seventy per cent of the inhabitants were tested. Blood samples were taken one hour after a meal, and specimens of urine were examined; if the results were thought suspicious, the tests were

repeated, and if still abnormal, a glucose tolerance test was performed. In this way 2,468 of the female population were studied, and the incidence of diabetes was found to be as follows (Wilkerson and Krall, 1947):

Age	Total Female Population	% with Diabetes
All ages	2,468	1.8
Under 15	619	0.0
15 - 24	372	0.3
25 - 34	417	0.2
35 - 44	342	1.3
45 - 54	275	4.0
55 - 64	196	6.9
65 - 74	162	5.2
75 and over	85	6.7

The results of a more recent study in the same town by Wilkerson, Krall and Butler (1959), showed a continuation of the trends already observed, and also demonstrated that both sexes are

equally affected, a finding which is at variance with most other statistics.

3. Joslin et al. (1952) surveyed an urban population of 2,502,391 persons (National Health Survey, 1935 - 1936). There were 9,182 diabetics, and the following data were obtained:

Age	% Diabetics in the Population		
	Total	Males	Females.
All ages	0.367	0.273	0.453
0 - 14	0.038	0.035	0.041
15 - 24	0.059	0.062	0.057
25 - 34	0.100	0.090	0.108
35 - 44	0.261	0.20	0.316
45 - 54	0.656	0.449	0.864
55 - 64	1.425	0.996	1.821
65 and over	1.839	1.458	2.147

4. Walker (1959) reported the results of a diabetes survey initiated by the British Diabetic Association. This survey was made in a large Midland village, Ibstock. Specimens of urine

passed after a high carbohydrate meal were submitted for testing by over 80% of the population of 5,400. When glycosuria was detected, a glucose tolerance test was carried out. There were 33 known diabetics in the village, and another 25 mild symtomless cases were detected in the course of the survey. This suggests that the true incidence of diabetes in Great Britain is about 1.4%, and that for every known case, there is another as yet undetected and untreated.

The following data were obtained:

Age	No. of diabetics among 4105 examined.		
	Total	Males	Females.
All ages	58 (1.4%)	20 (0.9%)	38 (1.9%)
5 - 14	1	1	0
15 - 24	3	3	0
25 - 34	1	1	0
35 - 44	4	1	3
45 - 54	9	3	6
55 - 64	20	5	15
65 - 74	13	3	10
75 - 79	5	3	2
80	2	0	2

It is thus seen that none of these control studies is based on routine glucose tolerance tests. They cannot therefore serve as strict controls for this present investigation. They do, however, in each case, show the rising incidence of diabetes with age, and especially after the age of 45 years. The increased incidence with age has been shown by practically all other workers in the field (Spence, 1921; Deren, 1937; Albanese, et al., 1954; Smith et al., 1948, and 1949; Smith 1948; Schneeberg et al., 1952; Chesrow et al., 1954). This is not a function of absorption, since it is equally evident following the intravenous administration of glucose (Schneeberg et al., 1952).

Since no suitable control series was found in the literature, I carried out full glucose tolerance tests on 100 random women of 45 years of age and over who were not suffering from cancer of the endometrium or benign glandular hyperplasia.

The results were as follows:

TABLE 2. Glucose Tolerance Tests on 100 Control Women of 45 years and over.

No. of Controls	Normal	Impaired Glucose Tolerance.		
		Diabetic	Mildly Impaired Glucose Tol.	Total
100	78%	13%	9%	22%

The percentage of diabetics and of cases with less severely impaired glucose tolerance is therefore remarkably higher than in any of the aforementioned series. This is understandable because each control subject was fully investigated by a glucose tolerance test. This investigation demonstrates, inter alia, that a great many patients with diabetes are not detected unless full glucose tolerance tests are done.

Because of the marked rise in the incidence of diabetes mellitus after the age of 45 years, a control group of 50 women between 35 and 45 years of age were similarly studied. The following data were obtained:

TABLE 3. Glucose Tolerance in 50 Control Women between 35 and 45 years.

No. of Controls.	Normal	Impaired Glucose Tolerance		
		Diabetic	Mildly Impaired Glucose Tol.	Total.
50	90%	2%	8%	10%

GROUPS INVESTIGATED BY GLUCOSE TOLERANCE TESTS:

Having thus obtained proper and suitable control groups, one was now in a position to compare these with the results obtained in cases of carcinoma of the endometrium. I went further and made glucose tolerance studies on all cases of abnormal uterine bleeding, of both benign and malignant aetiology that were encountered round about and after the climateric. In each case the results were correlated with the histological picture of the endometrium, because the latter is a mirror of pituitary-ovarian endocrine activity. Some interesting and significant findings came to light, which will be presented.

The following groups then, were investigated

by carrying out glucose tolerance tests in each case:

1. 100 Random women over the age of 45 years and over who were not suffering from cancer of the endometrium or benign glandular hyperplasia.

2. 50 similar women between 35 and 45 years of age, to serve as further control subjects in this lower age group.

3. 50 cases of carcinoma of the endometrium.

4. 50 cases who were proven by histological examination of the endometrium to have benign glandular hyperplasia.

5. 10 consecutive cases in whom the endometrium was not a hyperplastic one, but where secretory changes were absent.

FURTHER STUDIES:

In certain of the above groups striking abnormalities of glucose tolerance were found.

(These will be described later). In an attempt to determine the cause of these abnormalities of glucose tolerance other investigations were made.

1. Follicle-stimulating hormone was administered to patients and the effect of this on the glucose tolerance test was studied.

2. Oestrogens were administered to post-menopausal women, to see whether this altered the glucose tolerance curves.

3. Patients who had benign glandular hyperplasia with abnormal glucose tolerance, and who had undergone a hysterectomy, were followed up, and the glucose tolerance curves repeated after operation.

4. Many exogenous factors, unrelated to the endogenous control of carbohydrate metabolism, may influence the glucose tolerance curve (See page 31). The same standard conditions were therefore applied when each test was carried out. However, in view of the possibility of unknown exogenous

factors being present, and influencing the curves, a number of cases, where aberrations were found, were submitted to a repeat test at a different time. The fact that all of these showed similar aberrations confirmed that the latter were, in fact, due to true endogenous faults in glucose tolerance, i.e. the findings were not caused by technical errors or extraneous influences.

5. The study of the aforementioned control groups revealed an abrupt rise in the incidence of abnormal glucose tolerance after the age of 45 years. In order to determine whether this coincided with the age of the menopause, it was considered necessary to establish the age of the menopause in Cape Town. This was done by studying a thousand random women in the Western Cape who had already reached the menopause, to determine the average age of the menopause in this group. Likewise, 100 post-menopausal women attending the diabetic clinic of the Groote Schuur Hospital, and 100 post-menopausal women with cancer of the endometrium were investigated along the same lines.

SUMMARY.

Conflicting reports regarding the incidence of diabetes mellitus in cases of cancer of the endometrium have been reported in the literature. This stimulated a study to find the reasons for the controversy. I carried out investigations and was able to show that a raised incidence of impaired glucose tolerance in such cases is incontrovertible. Since no suitable control series was found in the literature, suitable groups were studied. Investigations were then extended to embrace all cases of abnormal uterine bleeding encountered during and after the climateric. The results were correlated with the histological picture of the endometrium. In certain groups striking abnormalities of glucose tolerance were found. In an attempt to detect the cause of these abnormalities, further investigations were necessary, and were therefore made.

CHAPTER 3.

THE GLUCOSE TOLERANCE TEST.

CHAPTER 3.

THE GLUCOSE TOLERANCE TEST.

Since the glucose tolerance test is the main test on which this investigation is based, the subject must be considered in more detail. It was necessary to decide on a standard glucose tolerance test, and on a standard interpretation, and then to apply these identical conditions to the control and other groups.

The rise and fall of the blood sugar concentration following oral or intravenous administration of glucose - "the glucose tolerance curve", has been used for a long time, both clinically and experimentally in animals and man, as an index of the efficiency of the mechanisms regulating the concentration of glucose in the blood. However, many of the published data are conflicting and the variation in the method employed (Moyer and Womack, 1950) by different workers makes comparison of

the results impossible. Different results have often been obtained, even when the same procedure has been used. Lack of agreement about the interpretation of the different blood sugar curves increases the confusion. There are several reasons for this state of affairs:

1. Exogenous factors may influence the curves:

It has been shown that there are many factors, unrelated to the endogenous control of carbohydrate metabolism, that may influence the glucose tolerance curve. Thes factors include 1) the antecedent diet, especially the carbohydrate content (Conn, 1940; Himsworth, 1932, 1933, 1934, 1934; Irving and Wang, 1954; McLellan and Wardlaw, 1932; McCullagh and Johnston, 1938; Srinivasan, 1957, Sweeney et al., 1928; Tolstoi, 1929, Turnbridge et al., 1940; Wayburn and Gray, 1942), 2) the state of nutrition (Conn, 1940; Du Vigneaud and Karr, 1925; Goldblatt, 1925), 3) preceding exercise (Consoatti, 1907; Evenson, 1942; Lennox, 1927) 4) Prolonged inactivity (Blotner, 1945), fear or other emotion (Hinkle and Wolf, 1952; Mirsky, 1946; Ross, 1938) 5) posture during the test (Nielsen, 1928)

6) interference with the bloof supply to muscle masses by a simple mechanical procedure such as elevation of the legs (Cajori et al., 1925) 7) the strength of the glucose solution, and the dose administered (Hale-White and Payne, 1926; Mosenthal and Barry, 1950) 8) the use of capillary or venous blood (Mosenthal and Barry, 1950) 9) the use of a torniquet in obtaining blood sampling (Gilbert et al., 1926) 11) fever, or even the mildest infection such as a "cold" (Laurence and Buckley, 1927; Ross, 1938; Sweeney and Lackey, 1928; Turnbridge and Allibone, 1940), 12) method used for the blood glucose estimation and the accuracy of the procedure (Mosenthal and Barry, 1946, 1950) 13) iggestion of certain drugs, for example large doses of sedatives and hynotics (Hunter and Greenberg, 1954; Merrivale and Hunter, 1954) 14) various anaesthetics (Fenton, 1945), steroids (Ingle, 1954), and salicyclates (Reid et al., 1957).

According to Thompson and King (1957) glucose tolerance can be defined as "the capacity to dispose of administered glucose under standard conditions". Standardisation and control of all the

aforementioned factors is therefore essential, no matter what procedure is used in carrying out the glucose tolerance test, to obtain results of any value.

2. Adequate control groups are necessary:

An exploration of the literature reveals that subjects selected for control observations have often been too few to establish the limits of normal tolerance. Only rarely have such factors as age (Silverstone et al., 1957), sex, weight (Ogilvie, 1935), parity (Lund and Weese, 1953), and previous diet or activity, been taken into consideration.

THE CHOICE OF GLUCOSE TOLERANCE TEST:

Oral glucose tolerance tests:

Most workers have administered the glucose load by mouth. Various doses have been tried, especially 50G. and 100G. and some have considered it necessary to vary the dose according to the subject's weight. Exton and Rose (1934)

suggested giving two doses of glucose (the "one-hour, two-dose, glucose tolerance test"). It has been shown conclusively that glucose is absorbed from the stomach, the proportion varying with the total glucose load. The absorptive capacity of the stomach is, however, readily saturated, and rarely exceeds 100 mgs. per hour, so that at high glucose loads the proportion of glucose absorbed from the stomach will be small. Beeler et al. (1922) showed that one hour after giving 100G. glucose orally to normal and diabetic subjects 22 - 60G. of the administered glucose could be recovered by washing out the stomach, without a consistent and significant variation of the glucose tolerance test. And Leonards and Free (1945) found that in normal adult male subjects undergoing the Exton-Rose test (1934), 38 - 62G. glucose could be recovered from the gastric contents after 1 hour.

Intravenous glucose tolerance tests:

The intravenous glucose tolerance test has been investigated by Bozner et al., (1941), Amatuzio et al., (1953), Bastenie et al., (1953, 1954), and Duncan (1956). Provided adequate control.

groups are used, and the tests performed under rigidly standardised conditions, intravenous tests may be valid in detecting alterations in glucose tolerance. However, they may miss abnormalities of glucose tolerance altogether; and where they do detect an abnormality, they give little information per se about the mechanism or sire of the defect, and may only indicate the need for further investigation. The procedure is rarely employed for the diagnosis of diabetes. The only indication for its use, probably, is in cases where there is dome disturbance of intestinal absorption.

The Insulin-glucose tolerance tests:

Another test is the insulin-glucose tolerance test (Engel and Scott, 1950). The patient receives 0.1 unit of crystalline insulin per kilogram body weight intravenously, and thirty minutes later, or at the onset of hypoglucaemic manifestations (whichever comes first), 0.8G. glucose per kilogram body weight by mouth. Normally, the glucose causes a marked rise in blood sugar, reaching a peak within 60 minutes. In cases of

Addison's disease and of panhypopituitarism, the rise in blood sugar is significantly decreased and delayed.

Workers with considerable experience in the diagnosis and treatment of diabetes have given consideration and thought to all the aforementioned tests, and have come to the various conclusions as to which is the most satisfactory. Duncan (1952) states that the standard oral glucose tolerance test is the most exacting, and when properly assessed, the test of choice for determining, in doubtful cases, the presence of absence of diabetes. The value of this test lies more in its diagnostic aid, and not so much as an indicator of the severity of the diabetes. Moyer and Womack (1950) agree that for most purposes the oral glucose tolerance test is adequate. In fact, in the literature there is fairly general agreement that the "Standard" one-dose (50G.) glucose test is probably the best (Jackson, 1952). Various attempts have been made to devise a more satisfactory test for unmasking the potential diabetic.

There is evidence that the intravenous glucose tolerance test is not very sensitive (Moyer and Womack, 1950). The diabetogenic action of steroids has been used with some success, a large dose of cortisone being given prior to the test (Fajans and Conn, 1954; Duncan, 1956). The cortisone-modified glucose tolerance test, however, was disappointing in that only one among the subjects classified as potential diabetics gave a positive response, i.e. investigating "lag" curves by glactose-tolerance tests (Dische et al., 1958; Goudie et al., 1958; B.M.J. Editorial, 1959).

Accordingly, the following criteria formed the basis of the glucose tolerance tests used in this investigation:

1. An unrestricted carbohydrate diet was allowed for several days before the test, and the subjects were not known to be suffering from any infection or hepatic disease.

2. The blood was taken half-hourly, beginning with a fasting specimen, and continuing at least up to

2 hours. Fasting and hourly urine specimens were also taken.

3. Capillary blood was used. It is easier to obtain, and does not miss the "lag" type of curve" (Jackson, 1952). Capillary blood gives the same fasting reading as venous blood, but after glucose it is higher to a variable degree (up to 50 mgm.).

4. The Hegedorn-Jensen method of estimating glucose was used.

INTERPRETATION OF THE GLUCOSE TOLERANCE TESTS:

According to Duncan (1952) curves are considered indicative of diabetes if the fasting blood sugar level exceeded 120 mgm. per cent or if the 2 hour value exceeded 130 mgm. per cent; when the diabetes is of a mild nature, the fasting blood sugar may be normal, while the 2 hour level is elevated.

Jackson (1952) adduced evidence to show that values above 140 mgm. per cent at 2 hours, and above 130 mgm. per cent at 2½ hours in the oral glucose

tolerance test were "highly suggestive, if not pathognomonic" of pre-diabetes. In a larger series he (Jackson, 1953) later confirmed this view. Similar glucose tolerance curves, and similar conclusions were reported by Hoest and Lukens (1954), Kritzer (1952), and Lund and Weese (1953). At first the British Medical Journal (Editorial. 1952) doubted the significance of such small aberrations, in the glucose tolerance curve, but later (Editorial, 1959) stated that "in Britain, particular attention is paid to the 2 hour value, figures in excess of 100 mgm. "true"glucose per cent being regarded with suspicion." Lawrence (1947) was quoted in support of this statement. This view was re-iterated in a later Editorial in the British Medical Journal (1959), in which it was stated that " the most informative point on the curve is probably the 2 hour figure, and if this is over 120 mgm. per cent it is very suspicious". Peak values above 180 mgm. per cent are attributed to rapid intestinal absorption if the 2 hour figure is near normal; but Joslin et al., (1952) do not recognise the "lag" type of surve as a separate entity, and

regard the height of the peak rather than the 2 hour value as evidence of potential abnormality.

Krätzer (1952) considered a "positive" glucose tolerance curve as one which showed a value of 200 mgm. per cent or greater in the first hour, above 150 mgm. per cent in the second hour, and over 125 mgm. per cent in the third hour.

Jackson and Woolf (1957), having followed up 10 patients with "prediabetes" found that every single patient yielded a more definite abnormality after at least three years. He stated that these results demonstrate clearly the sinificance of the minor aberrations in the glucose tolerance which he labelled "pre-diabetic".

In a later communication (Jackson, W.P.U.; 1959) he makes the following comments: " Most controversy has centred around those glucose tolerance curves which have been just outside the authors accepted normal limits. It has been found that the usual abnormality is a slightly high figure 2 or 2½ hours after the ingestion of glucose. In our own investigations we considered as abnormal a 2 hour level above 140 mgm. per 100 ml., with anything

between 120 and 140 as 'suspicious'. Occasionally the 2 to 2½ hour level was normal, while the fasting level was just over 120 mg., or the 1 hour level above 200 mgs. Such curves were also considered abnormal, but were much less common. Other workers, though using different techniques, have accepted very similar criteria of abnormality".

In view of the conclusions drawn from all these studies, the following interpretation was placed on the glucose tolerance results obtained in this investigation:

1. A normal glucose tolerance curve:

The normal curve was regarded as one where the fasting level is below 120 mgm., the highest level is below 200 mgm., and the two hour level is below 120 mgm. or the most 140 mgm., per cent.

2. Unquestionable diabetes:

This is shown by a curve where the fasting level is above 120 mgm., per cent, and a two-hour level which is usually greatly elevated.

3. Mildly impaired glucose tolerance (possibly mildly diabetic or pre-diabetic).

This third group is the most difficult to classify, and has been termed "pre-diabetic" or "mildly diabetic" by various authors. I have preferred to label this group "mildly impaired glucose tolerance", because although there is much evidence (see "Interpretation of the glucose tolerance tests" above) that such curves may represent a prediabetic or mildly diabetic state, the matter is still under discussion, and investigation. This group included cases where the fasting level is below 120 mgm. per cent, but the two hour level is elevated above 120 mgm. and especially above 140 mgm. per cent; or where the 1 hour level is above 200 mgm. per cent.

SUMMARY.

Since the glucose test is the main test on which this investigation is based, the subject is discussed in detail. It was necessary to decide on a standard glucose tolerance test, and on a standard interpretation, and then to apply these

identical conditions to the control and other groups.

For many reasons that are put forward, the "Standard" (50g.) oral glucose tolerance test was chosen, and was carried out under strictly laid down criteria in each subject investigated.

The interpretation of the glucose tolerance curve is a very difficult subject and is discussed in detail. Glucose tolerance curves can be divided into 3 groups, namely, normal, diabetic, and an intermediate group. The last-mentioned group is the most difficult to interpret and reasons are submitted for labelling this group "mildly impaired glucose tolerance".

CHAPTER 4.

INVESTIGATIONS AND RESULTS.

CHAPTER 4.

INVESTIGATIONS AND RESULTS.

Using the same glucose tolerance test aforementioned on each subject investigated, and placing the same standard of interpretation on the results, the following groups were studied. In view of the known effect of age on glucose tolerance (see page 23) this factor was constantly taken into consideration.

1. WOMEN WHO ACTED AS CONTROL SUBJECTS:

(a) Subjects of 45 years of age and over.

One hundred consecutive women of 45 years of age and over, who were not suffering from cancer of the endometrium or benign glandular hyperplasia were submitted to glucose tolerance tests, under the aforementioned standard conditions. These were in -and out- patients attending the Groote Schuur

Hospital, who were seen because of unrelated diseases, such as prolapse,"stress incontinence", urinary disorders other than infection, leucorrhoea, backache, abdominal pains, cervical erosions, and gynaecological symptons of psycho-somatic origin.

In each case the endometrium was examined histologically, having been obtained by endometrial biopsy, curettage, or hysterectomy. If the endometrium was found to be hyperplastic or carcinomatous, the case was excluded. All these women, therefore, had a normal endometrium - an endometrium which was either proliferative or secretory, or physiologically atrophic due to the menopause.

This group was unselected in that it consisted of random consecutive gynaecological patients of 45 years of age and over. The majority of them, however, did have vaginal bleeding of varying degree, and pelvic pathology. (The cause of the bleeding and the type of pathology is given on each glucose tolerance curve seen in the following pages). It was therefore considered that this was the best type of control group for comparison with the cases of cancer of the endometrium and benign glandular hyperplasia - for here we have a most comparable

group of the same ages and usually with pelvic pathology as well as vaginal bleeding - similar conditions that pertained in the other 2 groups.

Photographs of all these original glucose tolerance curves are seen in the following pages. A summary of the clinical history, and histological appearance of the endometrium in each case is shown on each glucose tolerance curve. The detailed figures of the blood sugar levels and ages of the subjects are listed in Table 4. A summary of the results is given in Table 5, after the presentation of the glucose tolerance curves and table of details.

Age 55 years. Menopause at 52 years. C/O Post-menopausal bleeding: few days. 3/4/58 D&C: Scanty curettings. 2 benign polypi - one endometrial and one endocervical; scanty inactive endometrial glands.

1.

Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

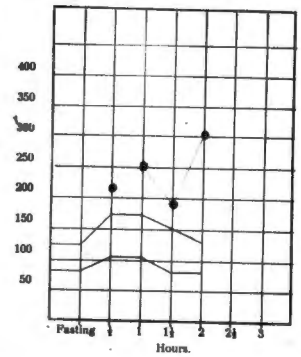
Serial No. 21671 - 72. Date 30.6.59.

Patient's Identification MRS. OLGA TURECKI. (58/04322).

Ward A10. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

	URINE SUGAR
1.	+
2.	+
3.	+



BLOOD SUGAR	
Fasting	No spec. received.
½ hour	215 mgs.%.
1 hour	251 mgs.%.
1½ hour	190 mgs.%.
2 hour	304 mgs.%.
2½ hour	
3 hour	

Signature L. ANSTEY

Age 54 years. C/O Hot flushes - fibroids size 20 weeks. 13/6/59
Hysterectomy: Fibroids - atrophic endometrium.

2.

Report from Pathological Department.
UNIVERSITY OF CAPE TOWN.

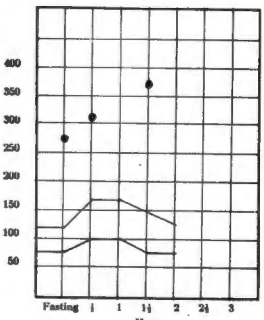
Serial No. 17570 - 1. Date 29.5.59

Patient's Identification CISSY JOHNSON (59/28964).

Ward B7. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

Specimen	URINE SUGAR
1.	++++
2.	++++
3.	++++



BLOOD SUGAR	
Fasting	275 mgs.%.
½ hour	320 mgs.%.
1 hour	Insufficient
1½ hour	373 mgs.%.
2 hour	Clotted.
2½ hour	
3 hour	

A. J. V. D. WALT

Signature

Age 48 years. Pruritis vulvae. Monilia infection. D&C: No curettings.

3.

Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

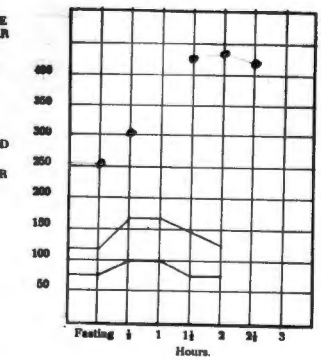
Serial No. 32368 - 69 Date 11.9.59.

Patient's Identification MADGE [illegible] (13484)

Ward C20. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

	URINE SUGAR
1.	+
2.	++
3.	++



BLOOD SUGAR	
Fasting	251 mgs.%.
½ hour	304 mgs.%.
1 hour	Clotted.
1½ hour	432 mgs.%.
2 hour	438 mgs.%.
2½ hour	429 mgs.%.
3 hour	

L. ANSTEY.

Signature

Age 79 years. C/O Post-menopausal bleeding menopause at 50 years. O/E N.A.D. Blood pressure 190/80. 2 D&C's - no curettings.

4.

Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

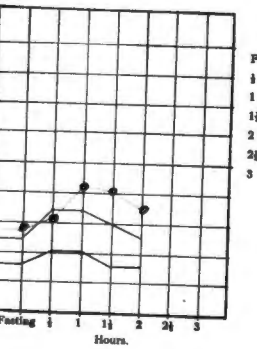
Serial No. 33849 - 66. Date 27.10.59.

Patient's Identification KILLY BAKER. (59/06025)

Ward A10. Physician or Surgeon Dr. Benjamin.

GLUCOSE TOLERANCE TEST.

Specimen	URINE SUGAR
1.	0
2.	0
3.	0



BLOOD SUGAR	
Fasting	197 mgs.%.
½ hour	288 mgs.%.
1 hour	313 mgs.%.
1½ hour	301 mgs.%.
2 hour	176 mgs.%.
2½ hour	Clotted.
3 hour	

Signature G. M. POTGIETER

Age 54 years. Post-menopausal bleeding. D&C : inactive endometrial glands.

5.

Report from Pathological Department.

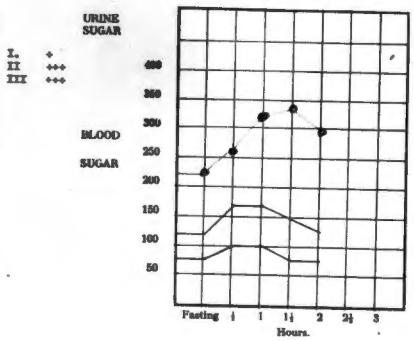
UNIVERSITY OF CAPE TOWN.

Serial No. 8150-53 Date 14.3.58.

Patient's Identification ANNIE CORTIER. (58/02638)

Ward A 8. Physician or Surgeon Dr Buss.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting 233 mgs.%.
½ hour 266 mgs.%.
1 hour 326 mgs.%.
1½ hour 337 mgs.%.
2 hour 294 mgs.%.
2½ hour
3 hour

Signature

Age 57 years. Menopause at 52 years. C/O post-menopausal bleeding: 1 month. O/E Carcinoma cervix stag III. Endometrium atrophic.

6.

Report from Pathology Department.

(Chemical Pathology).

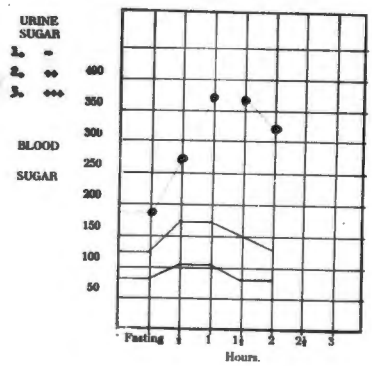
UNIVERSITY OF CAPE TOWN.

Serial No. 21660 - 70 Date 30.6.59.

Patient's Identification MINNIE ABRAHAMS (59/16927)

Ward 27. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting 189 mgs.%.
½ hour 272 mgs.%.
1 hour 334 mgs.%.
1½ hour 308 mgs.%.
2 hour 339 mgs.%.
2½ hour
3 hour

Signature L. ANSTEY

Age 71 years. Post-menopausal bleeding: 1 week. Menopause at 56 years. D&C- fibroids. 1/6/59 hysterectomy: benign endometrial polyp.

7.

Report from Pathological Department.

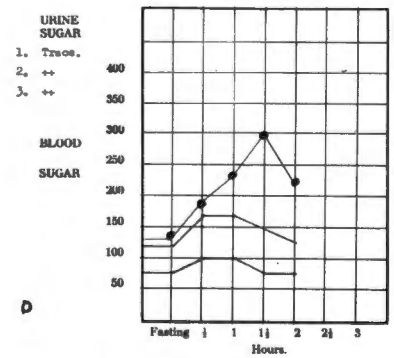
UNIVERSITY OF CAPE TOWN.

Serial No. 16257 - 8. Date 23.6.59

Patient's Identification [illegible] (56/06656)

Ward C1C. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting 137 mgs.%.
½ hour 166 mgs.%.
1 hour 238 mgs.%.
1½ hour 301 mgs.%.
2 hour 219 mgs.%.
2½ hour
3 hour

D

Signature J. J. V. D. WALT

Age 71 years. C/O Post-menopausal bleeding. O/E Blood pressure 240/120. 16/6/59 D&C: no curettings.

8.

Report from Pathology Department.

(Chemical Pathology).

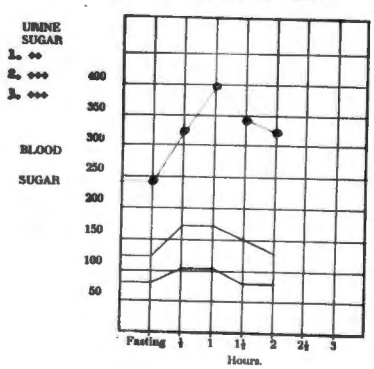
UNIVERSITY OF CAPE TOWN.

Serial No. 19530 - 9 Date 7.6.59

Patient's Identification ETHEL JACKSON (59/304.59)

Ward 4B Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting 243 mgs.%.
½ hour 333 mgs.%.
1 hour 399 mgs.%.
1½ hour 344 mgs.%.
2 hour 333 mgs.%.
2½ hour
3 hour

Signature J. J. V. D. WALT

Age 70 years. Post-menopausal bleeding. Menopause at 60 years. Fibroids (hysterectomy) Atrophic endometrium.

9.

Report from Pathological Department.

UNIVERSITY OF CAPE TOWN.

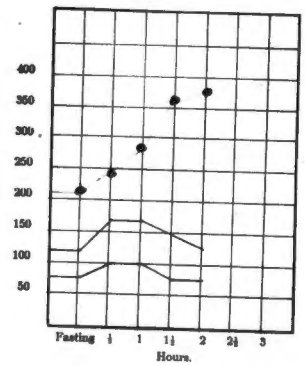
Serial No. CT14 - 15. Date 8.1.57.

Patient's Identification FRANCES JAGER (56/27279).

Ward 19 Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR
1. +
2. Negative.
3. +++



BLOOD SUGAR
Fasting 206 mgs.%.
½ hour 240 mgs.%.
1 hour 236 mgs.%.
1½ hour 362 mgs.%.
2 hour 369 mgs.%.
2½ hour
3 hour

Signature

Age 78 years. Post-menopausal bleeding. Senile vaginitis. Endometrium - no curettings.

10.

Report from Pathological Department.

UNIVERSITY OF CAPE TOWN.

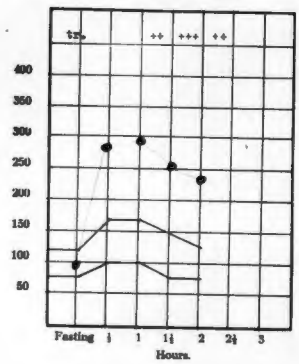
Serial No. 31663-64 Date 25.9.58.

Patient's Identification ANNIE ALICE PARSONS. (64669).

Ward G 10. Physician or Surgeon Dr Hoosey.

GLUCOSE TOLERANCE TEST.

URINE SUGAR
tr. ++ +++ ++



BLOOD SUGAR
Fasting 94mgs.%.
½ hour 280 mgs.%.
1 hour 295 mgs.%.
1½ hour 254 mgs.%.
2 hour 234 mgs.%.
2½ hour
3 hour

Signature

Age 89 years. Menopause at 55 years. C/O post-menopausal bleeding: 2 years. 24/8/59 D&C and removal of polyp: No curettings.

11.

Report from Pathology Department.

(Chemical Pathology).

UNIVERSITY OF CAPE TOWN.

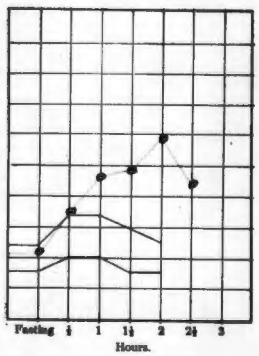
Serial No. [redacted] Date 26.8.59.

Patient's Identification [redacted] (59/08439)

Ward [redacted] Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR
1. Negative.
2. trace.
3. trace.



BLOOD SUGAR
Fasting 209 mgs.%.
½ hour 277 mgs.%.
1 hour 238 mgs.%.
1½ hour 246 mgs.%.
2 hour 296 mgs.%.
2½ hour 227 mgs.%.
3 hour

Signature S. M. POTGIETER

Age 79 years. Menopause at 50 years. C/O Post-menopausal bleeding: 2 weeks.. 26/8/59 D&C - no curettings. Blood pressure 190/80. (son has diabetes).

12.

Report from Pathology Department.

(Chemical Pathology).

UNIVERSITY OF CAPE TOWN.

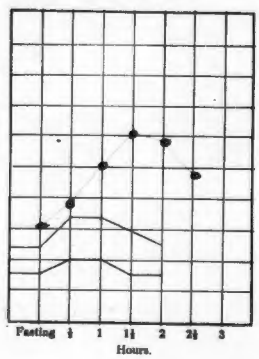
Serial No. 2994 -59. Date 28.8.59.

Patient's Identification KELLY BAKER. (59/06855)

Ward G20. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR
1. +
2. +
3. ++



BLOOD SUGAR
Fasting 157 mgs.%.
½ hour 293 mgs.%.
1 hour 254 mgs.%.
1½ hour 301 mgs.%.
2 hour 286 mgs.%.
2½ hour 240 mgs.%.
3 hour

Signature L. ANSTEY.

Age 49 years. C/O Pain since hysterectomy and sterilisation: 2 years. 28/10/59 D&C: Early secretory endometrium corresponding to the 19th. day which she is. Obese blood pressure 180/120.

17.

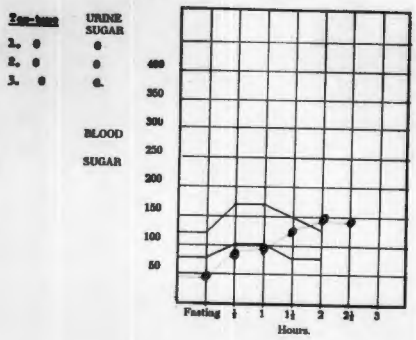
Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

Serial No. 33412 - 3. Date 6.11.59.

Patient's Identification FATIMA BOSHOFF (287)

Ward A39. Physician or Surgeon Dr. Benjamin.

GLUCOSE TOLERANCE TEST.



Signature G. M. POTGIETER

Age 47 years. Prolapse and menorrhagia. D&C: Secretory endometrium.

18.

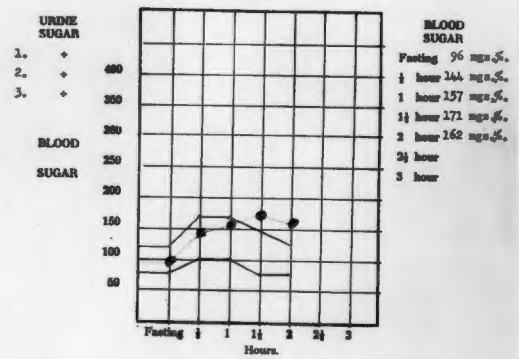
Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

Serial No. 33898 - 99. Date 30.9.59.

Patient's Identification ZIPPORA LE ROUX (

Ward B7. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.



G. M POTGIETER

Age 47 years. Menopause 1 year ago. C/O Post-menopausal bleeding. 25/9/59 D&C: atrophic non-secretory endometrium.

19.

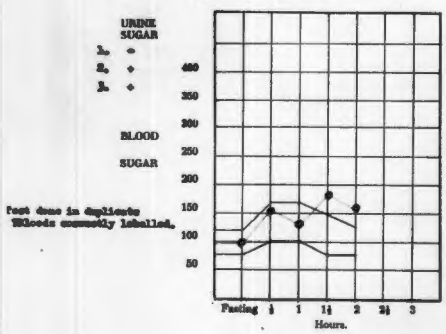
Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

Serial No. 33487 - 496 Date 28.9.59. at 11 a.m.

Patient's Identification STELLA REINE (28896)

Ward 680. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.



Post done in duplicate Bloods correctly labelled.

Signature G. M. POTGIETER

Age 53 years. C/O Post-menopausal bleeding 3 days. Menopause at 50 years. O/E Obese; blood pressure 185/100. 4/9/59 D&C : Inactive endometrium.

20.

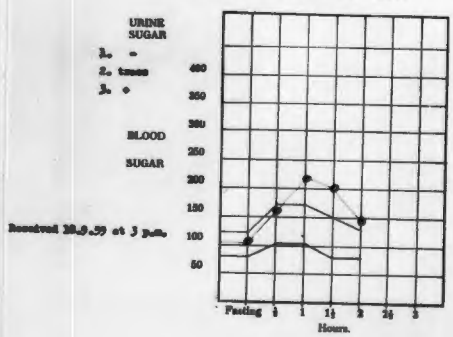
Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

Serial No. 33410 - 411. Date 28.9.59.

Patient's Identification DAPHNE MASTERS (28/0019)

Ward A38. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.



Received 28.9.59 at 3 p.m.

Signature L. ANSTEY.

Age 53 years. C/O menorrhagia: 10 years. O/E Fibroids size 16 weeks. 16/9/59 D&C: inactive stroma and few non-secretory glands. 18/9/59 Hysterectomy: simple follicular cysts, endometrium shows marked atrophy and thinning.

13.

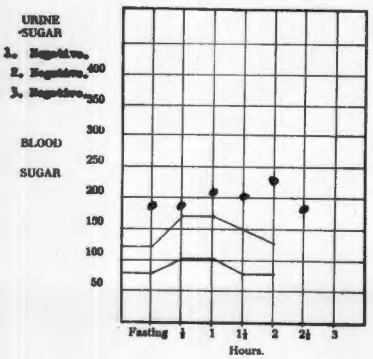
Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

Serial No. 3870 - 59. Date 15.9.59.

Patient's Identification ANNA [illegible] (59/09516.)

Ward G&O. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR	
Fasting	186 mgm.%.
½ hour	186 mgm.%.
1 hour	212 mgm.%.
1½ hour	202 mgm.%.
2 hour	187 mgm.%.
2½ hour	179 mgm.%.
3 hour	

G. M. POTGIETER
Signature

Age 47 years. C/O Menorrhagia: 4 months. 4/28 > 8-12/28 day type. Known diabetic on Rastinon and diet. O/E Obese++, Blood Pressure 160/120. 25/8/59 D&C: Bulky uterus (L.N.P. 1/8/59) Secretory endometrium corresponding to 24th. day of cycle.

14.

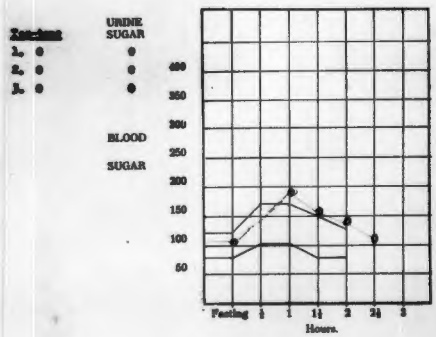
Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

Serial No. 3926 - 59. Date [illegible]

Patient's Identification [illegible] (A42Bo)

Ward A&O. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR	
Fasting	207 mgm.%.
½ hour	[illegible]
1 hour	192 mgm.%.
1½ hour	193 mgm.%.
2 hour	197 mgm.%.
2½ hour	207 mgm.%.
3 hour	

G. M. POTGIETER
Signature

Age 45 years. C/O Menorrhagia: 2 years. O/E Fibroids size 14 weeks. 13/7/59 D&C: Bulky contents (L.N.P. 1/7/59) Secretory endometrium

15.

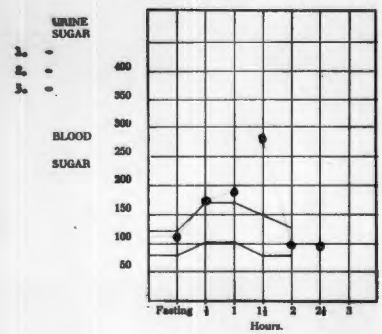
Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

Serial No. 2995 - 59. Date 13.7.59.

Patient's Identification [illegible] (59/04521).

Ward G&O. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR	
Fasting	130 mgm.%.
½ hour	176 mgm.%.
1 hour	188 mgm.%.
1½ hour	156 mgm.%.
2 hour	87 mgm.%.
2½ hour	91 mgm.%.
3 hour	

G. M. POTGIETER
Signature

Age 49 years. C/O Menorrhagia and polymenorrhoea ever since [illegible] lisation 12 years ago. Repeated D&C's, last one on 31/10/57. Normal secretory endometrium each time, secretory endometrium [illegible] corresponding with the stage of the cycle.

16.

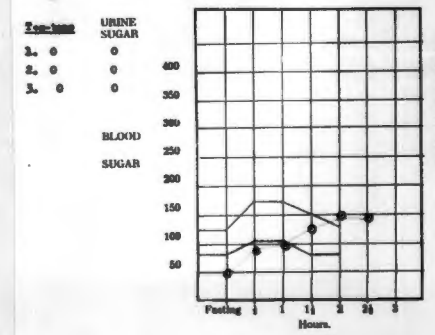
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

Serial No. 3838 - 59. Date 6.11.59.

Patient's Identification [illegible] (B27)

Ward A&O. Physician or Surgeon Dr. [illegible]

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR	
Fasting	68 mgm.%.
½ hour	84 mgm.%.
1 hour	99 mgm.%.
1½ hour	125 mgm.%.
2 hour	143 mgm.%.
2½ hour	141 mgm.%.
3 hour	

G. M. POTGIETER
Signature

Age 68 years. Menopause at 53 years. C/O Prolapse. 3/7/59 Vaginal hysterectomy - histology: atrophic and inactive post-menopausal endometrium.

21.

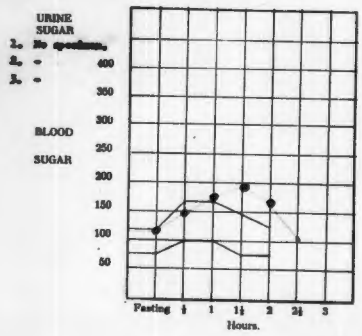
Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

Serial No. [redacted] Date 13.7.59.

Patient's Identification MRS. ANNA KINNOCH (58/07209)

Ward [redacted] Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR
Fasting 110 mgs.%.
½ hour 147 mgs.%.
1 hour 178 mgs.%.
1½ hour 190 mgs.%.
2 hour 160 mgs.%.
2½ hour 103 mgs.%.
3 hour

G. M. POTGIETER
Signature

Age 50 years. Prolapse. Vaginal hysterectomy: Atrophic endometrium.

22.

Report from Pathological Department.
UNIVERSITY OF CAPE TOWN.

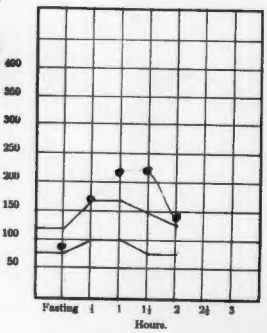
Serial No. 17578 - 59 Date 28.5.59

Patient's Identification MARGARET CLAASSEN. (58/07179)

Ward A9 Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR
1. [redacted]
2. trace Negative.
3. trace Negative.



BLOOD SUGAR
Fasting 89 mgs.%.
½ hour 172 mgs.%.
1 hour 215 mgs.%.
1½ hour 229 mgs.%.
2 hour 128 mgs.%.
2½ hour
3 hour

J. J. V. D. WALT
Signature

Age 51 years. Menorrhagia. Multiple fibroids - normal endometrium.

23.

Report from Pathological Department.
UNIVERSITY OF CAPE TOWN.

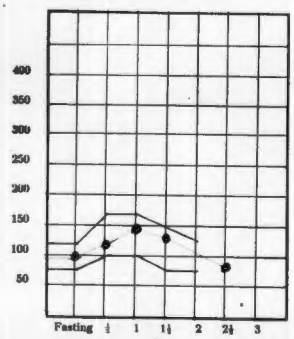
Serial No. 38780/81. Date 11.11.58.

Patient's Identification BARBARA HENDERSON. (17591).

Ward 610. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR
Fasting Absent.
1½ hr. "
2½ hrs. "



BLOOD SUGAR
Fasting 99 mgs.%.
½ hour 121 mgs. %.
1 hour 142 mgs.%.
1½ hour 133 mgs.%.
2 hour 76 mgs.%.
2½ hour
3 hour

Signature

Age 50 years. Menorrhagia: 1 month. Atrophic endometrium on D&C.

24.

Report from Pathological Department.
UNIVERSITY OF CAPE TOWN.

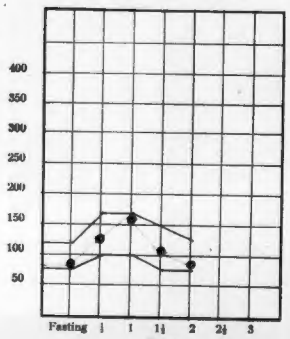
Serial No. 30396-57/57 Date 11.9.57.

Patient's Identification JOHANNA OCTOBER (261939)

Ward A9 Physician or Surgeon Prof. Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR
Nil.
Nil.
Nil.



BLOOD SUGAR
Fasting 79 mgs.%.
½ hour 126 mgs.%.
1 hour 162 mgs.%.
1½ hour 107 mgs.%.
2 hour 88 mgs.%.
2½ hour
3 hour

Signature

Age 52 years. C/C Menorrhagia. Glands show secretory activity.

25.

Report from Pathological Department.

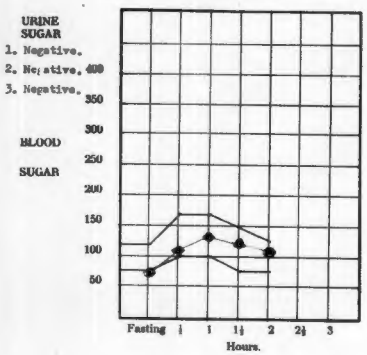
UNIVERSITY OF CAPE TOWN.

Serial No. 14447 - 48 Date 10.5.57

Patient's Identification MRS. LEVINA CORNIA (168492)

Ward CXC. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting	67 mgs.%.
½ hour	105 mgs.%.
1 hour	143 mgs.%.
1½ hour	124 mgs.%.
2 hour	106 mgs.%.
2½ hour	
3 hour	

Signature L. ANSTEY.

Age 53. Menorrhagia: 1½ years. D&C and Hysterectomy: size 24 weeks. Endometrium non-secretory, no hyperplasia.

26.

Report from Pathological Department.

UNIVERSITY OF CAPE TOWN.

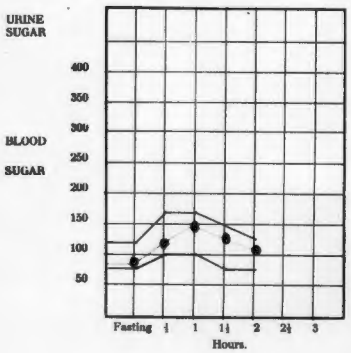
Serial No. 37688-89/57 Date 8.11.57.

Patient's Identification ANN KOETJE (57/ 25436)

Ward 27 Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

o/s
o/s
o/s



BLOOD SUGAR

Fasting	77 mgs.%.
½ hour	119 mgs.%.
1 hour	146 mgs.%.
1½ hour	121 mgs.%.
2 hour	110 mgs.%.
2½ hour	
3 hour	

Signature

Age 48 years. Discharge and irregular menstruation - oligo, hypo - and amenorrhoea. D&C: Atrophic endometrium, chronic cervicitis. Hysterectomy - fibroid.

27.

Report from Pathological Department.

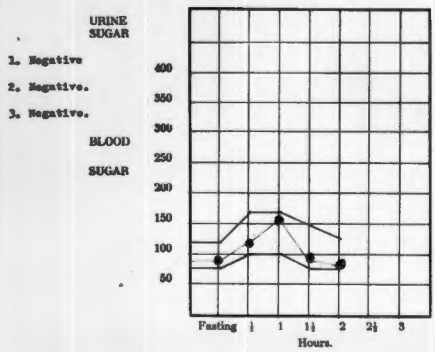
UNIVERSITY OF CAPE TOWN.

Serial No. G 18419 - 20 Date 25.6.56.

Patient's Identification CAROLINE NEUMANN (56/05687).

Ward G 20. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting	87 mgs.%.
½ hour	115 mgs.%.
1 hour	158 mgs.%.
1½ hour	94 mgs.%.
2 hour	80 mgs.%.
2½ hour	
3 hour	

Signature

Age 49 years. Intermenstrual blood-stained discharge : 1 month Menstruation 4/28 day type, but bleeding much more scanty for 5 months. D&C - no curettings; biopsy carcinoma of cervix - treated with radiotherapy.

28.

Report from Pathological Department.

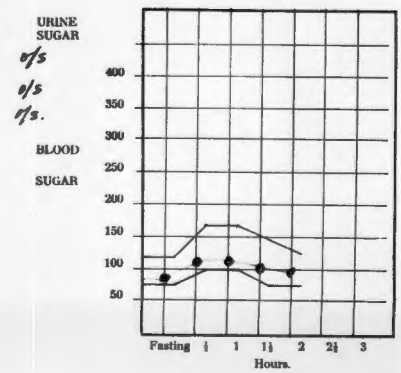
UNIVERSITY OF CAPE TOWN.

Serial No. 36248-49/57. Date 30/10/57.

Patient's Identification ALETTA BOOYSEN. (57/25670)

Ward M. Physician or Surgeon Dr. Buss.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting	79 mgs.%
½ hour	116 mgs.%
1 hour	116 mgs.%
1½ hour	107 mgs.%
2 hour	
2½ hour	
3 hour	

Signature G. Luyker.

Age 48 years. C/O Menorrhagia. Hysterectomy - Numerous fibroids, endometrium secretory.

29.

Report from Pathological Department.

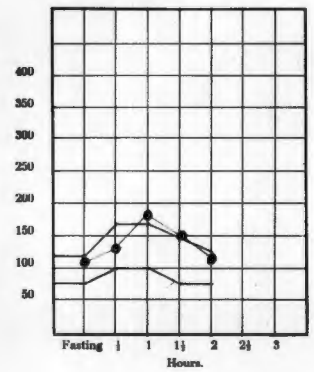
UNIVERSITY OF CAPE TOWN.

Serial No. 15475 - 6. Date 17.5.59

Patient's Identification HAJIRAH BROWN (29/09155)

Ward A9 Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting 108 mgs.%.
½ hour 134 mgs.%.
1 hour 186 mgs.%.
1½ hour 150 mgs.%.
2 hour 125 mgs.%.
2½ hour
3 hour

Signature [illegible]

Age 50 years. C/O Menorrhagia: 9 months. (sterilization and wedge resection 5 years previously). D&C and Hysterectomy: Normal endometrium and uterus.

30.

Report from Pathology Department.

(Chemical Pathology).

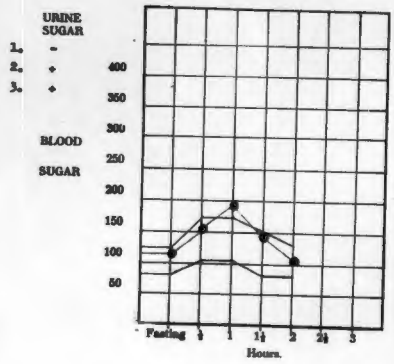
UNIVERSITY OF CAPE TOWN.

Serial No. 19884 - 285. Date 12.6.59.

Patient's Identification MARY COSTHUIZEN (380176).

Ward A.30. Physician or Surgeon Prof. Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting 115 mgs.%.
½ hour 151 mgs.%.
1 hour 197 mgs.%.
1½ hour 142 mgs.%.
2 hour 101 mgs.%.
2½ hour
3 hour

Signature J. J. V. D. WALT

Age 49 years. C/O Prolapse and menorrhagia for 6 months. O/E Prolapse, marked cervicitis. D&C 17/6/59: Secretory endometrium on 32nd day of cycle.

31.

Report from Pathology Department.

(Chemical Pathology).

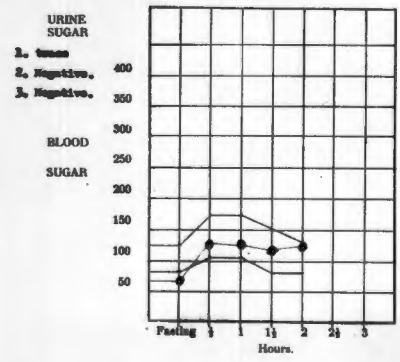
UNIVERSITY OF CAPE TOWN.

Serial No. 20438 - 9. Date 19.6.59.

Patient's Identification ADELAIDE DE WAAL (47866)

Ward G20. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting 62 mgs.%.
½ hour 134 mgs.%.
1 hour 132 mgs.%.
1½ hour 117 mgs.%.
2 hour 127 mgs.%.
2½ hour
3 hour

Signature J. J. V. D. WALT

Age 48 years. C/O Menorrhagia for 2 years. D&C 15/6/59: normal proliferative endometrium and small fibroids (7th. day of cycle).

32.

Report from Pathology Department.

(Chemical Pathology).

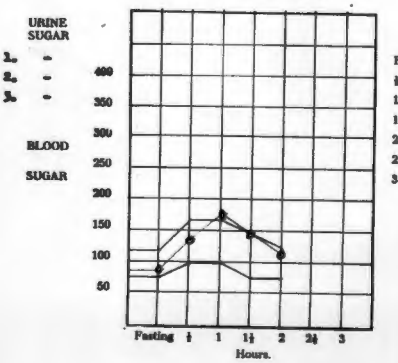
UNIVERSITY OF CAPE TOWN.

Serial No. 19195 - 96. Date 16.6.59.

Patient's Identification AMELIA MEINTJES (29/14020).

Ward A.8. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting 87 mgs.%.
½ hour 141 mgs.%.
1 hour 174 mgs.%.
1½ hour 147 mgs.%.
2 hour 111 mgs.%.
2½ hour
3 hour

Signature J. J. V. D. WALT

Age 48 years. C/O Bouth of amenorrhoea (up to 3 months) followed by bouts of menorrhagia. " Hot flushes". (Had thyroidectomy 3 years ago - B.".R. + 13, serum cholestrol 161 mgs %) 17/6/59 D&C:(about 14th. day of cycle) Normal proliferative endometrium - no evidence of glandular hyperplasia.

33.

Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

Serial No. 16546 - 7. Date 6.6.59

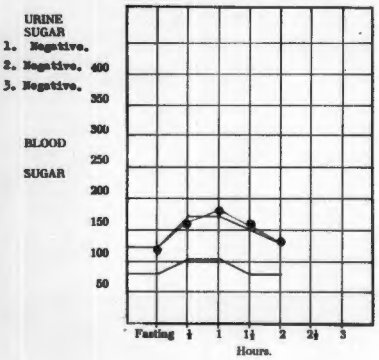
Patient's Identification CHARLOTTE MATTHEE (59/14417)

Ward A2. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR
1. Negative.
2. Negative.
3. Negative.

BLOOD SUGAR



BLOOD SUGAR
Fasting 119 mgs.%.
½ hour 162 mgs.%.
1 hour 180 mgs.%.
1½ hour 156 mgs.%.
2 hour 133 mgs.%.
2½ hour
3 hour

J. J. V. D. WALT
Signature

Age 46 years. C/O Bouts of amenorrhoea followed by bouts of menorrhagia. 17/7/59 D&C during amenorrhoea 35 days - proliferative endometrium - not hyperplastic. 24/7/59 Hysterectomy: Proliferative endometrium - tendency to hyperplasia. Follicular cysts ovaries.

34 F.S.H. 12 rat units.

Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

Serial No. 24334 - 35. Date 23.7.59.

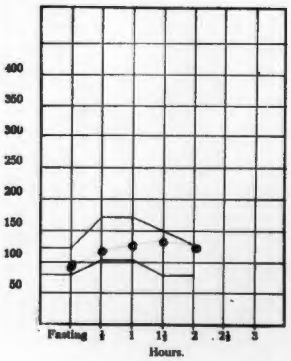
Patient's Identification MRS. ZOE TRIEGAARDT (59/20438)

Ward G2B. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR
1. -
2. -
3. -

BLOOD SUGAR



BLOOD SUGAR
Fasting 90 mg.%.
½ hour 122 mgs.%.
1 hour 186 mgs.%.
1½ hour 130 mgs.%.
2 hour 126 mgs.%.
2½ hour
3 hour

G. M. POTGIETER
Signature

Age 47 years. C/O Discharge and " Hot flushes" 2 months. Menstruation 4/28 day type I.".P. 5/7/59. Uterus and adnexae normal- cervical erosion.

35.

Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

Serial No. 24127 - 8. Date 17.7.59.

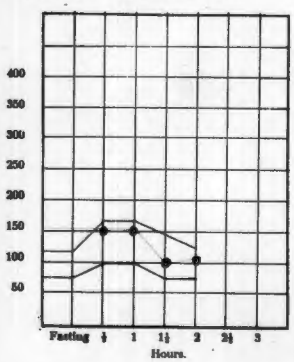
Patient's Identification GERDINA GILLISSEN (136722).

Ward A10. Physician or Surgeon Dr. Benjamin.

GLUCOSE TOLERANCE TEST.

URINE SUGAR
1.
Negative.

BLOOD SUGAR



BLOOD SUGAR
Insufficient
Fasting 152 mgs.%.
½ hour 152 mgs.%.
1 hour 200 mgs.%.
1½ hour 215 mgs.%.
2 hour
2½ hour
3 hour

G. M. POTGIETER
Signature

Age 45 years. Menorrhagia and " stress incontinence" 2 months. 28/7/59 Vaginal hysterectomy and repair. Cervicitis and secretory endometrium, more advanced than expected on the 18th. day of cycle.

36.

Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

Serial No. 24837-38 Date 23.7.59.

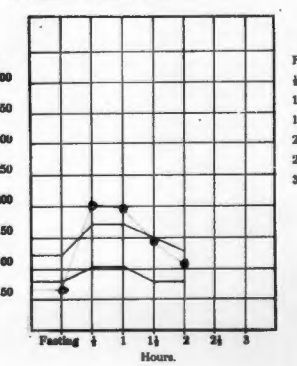
Patient's Identification NORA VOS. (34257)

Ward G2B. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR
1. Negative.
2. Negative.
3. Negative.

BLOOD SUGAR



BLOOD SUGAR
Fasting 43 mgs.%.
½ hour 202 mgs.%.
1 hour 299 mgs.%.
1½ hour 241 mgs.%.
2 hour 204 mgs.%.
2½ hour
3 hour

G. M. POTGIETER
Signature

Age 45 years. Profuse bleeding for 3 weeks. Histology: 1st. day cycle - late premenstrual phase - secretory endometrium.

37.

Report from Pathological Department.

UNIVERSITY OF CAPE TOWN.

Serial No. 15315 - 6. Date 17.5.59

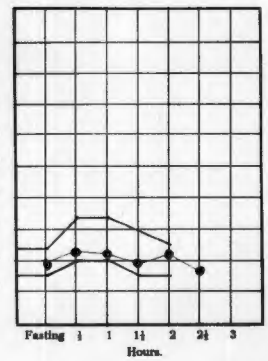
Patient's Identification MRS. M. FOURIE

Ward C30. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR
1. Negative.
2. Negative.
3. Negative.

BLOOD SUGAR



BLOOD SUGAR
Fasting 95mgs.%.(0.5ml
½ hour 112mgs.%.(" ")
1 hour 108mgs.%.("")
1½ hour 99 mgs.%.
2 hour 108mgs.%.(0.5
2½ hour 86mgs.%.(0.5ml
3 hour

J. J. v. D. WALT

Signature

Age 45 years. Menorrhagia: 3 years. Obese +++ . Fibroids size 16 weeks. D&C and Hysterectomy: Atrophic endometrium.

38.

Report from Pathological Department.

UNIVERSITY OF CAPE TOWN.

Serial No. 28216 - 18 Date 4.9.56

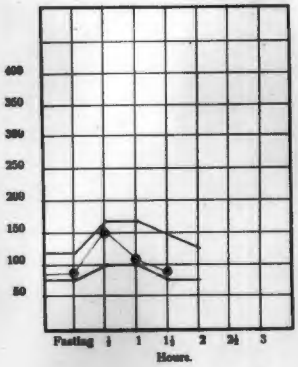
Patient's Identification CAROLINE GUERREAO (56/22923)

Ward A9 Physician or Surgeon Prof. Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR

BLOOD SUGAR



BLOOD SUGAR
Fasting 86 mgs.%.
½ hour 151 mgs.%.
1 hour 105 mgs.%.
1½ hour 85 mgs.%.
2 hourCoagulated.
2½ hour
3 hour

Signature

Age 56 years, still menstruating. Erosion. D&C and Hysterectomy: Proliferative endometrium - no hyperplasia or malignancy. (L.N.P. 1 week previously).

34.

Report from Pathological Department.

UNIVERSITY OF CAPE TOWN.

Serial No. Date 27.ch.59

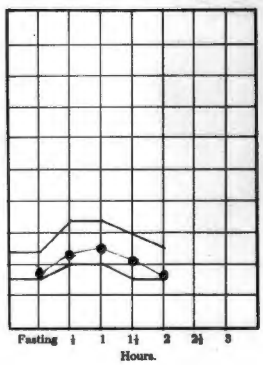
Patient's Identification SARAH ESSEN

Ward A9 Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR
1. +
2. Negative.

BLOOD SUGAR



BLOOD SUGAR
Fasting 85 mgs.%.
½ hour 115 mgs.%.
1 hour 119 mgs.%.
1½ hour 105 mgs.%.
2 hour 82 mgs.%. (clotted)
2½ hour
3 hour

Signature

Age 52 years. C/O Polymenorrhoea and intermenstrual bleeding : few months. D&C 29/7/59: Proliferative endometrium - also a benign endometrial poly. (L.N.P. 29/7/59.

40.

Report from Pathology Department.

(Chemical Pathology).

UNIVERSITY OF CAPE TOWN.

Serial No. 26887 - 8 Date 3.8.59.

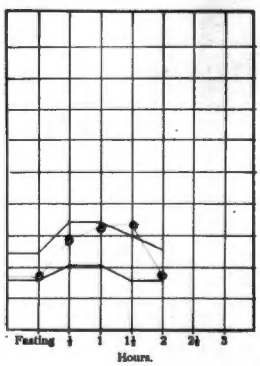
Patient's Identification ELIZA [illegible] (59/2888)

Ward A9. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR
1. -
2. -
3. -

BLOOD SUGAR



Received 4.8.59 at 11 a.m.

BLOOD SUGAR
Fasting 79 mgs.%.
½ hour 148 mgs.%.
1 hour 148 mgs.%.
1½ hour 170 mgs.%.
2 hour 86 mgs.%.
2½ hour
3 hour

G. M. POTGIETER

Signature

Age 48 years. Intermenstrual bleeding. Biopsy and D&C : Carcinoma of cervix, secretory endometrium.

57.

Report from Pathological Department.

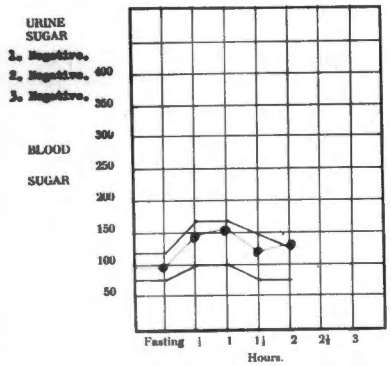
UNIVERSITY OF CAPE TOWN.

Serial No. 15473 - 4. Date 19.5.59

Patient's Identification C. DIRKES (167341)

Ward 37. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting	99 mgs.%.
½ hour	145 mgs.%.
1 hour	152 mgs.%.
1½ hour	122 mgs. %
2 hour	132 mgs.%.
2½ hour	
3 hour	

Signature D. J. Louw.

Age 47 years. Menorrhagia. D&C and cautery: Cervical erosion, normal secretory endometrium.

58.

Report from Pathology Department.

(Chemical Pathology).

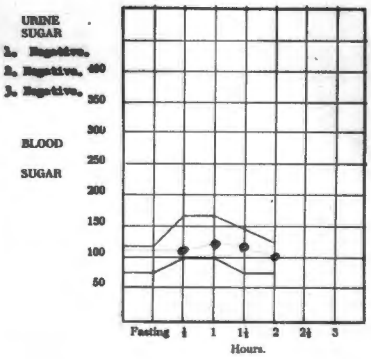
UNIVERSITY OF CAPE TOWN.

Serial No. 2939 - 60 Date 23.6.59.

Patient's Identification MAGDALENA BOOM (84302)

Ward 40 Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting	Insufficient.
½ hour	116 mgs.%.
1 hour	128 mgs.%.
1½ hour	121 mgs.%.
2 hour	104 mgs.%.
2½ hour	
3 hour	

Signature L. ANSTEY

Age 50 years. C/O Menorrhagia and discharge. Cervical erosion. D&C and cautery: Normal endometrium, erosions.

59.

Report from Pathology Department.

(Chemical Pathology).

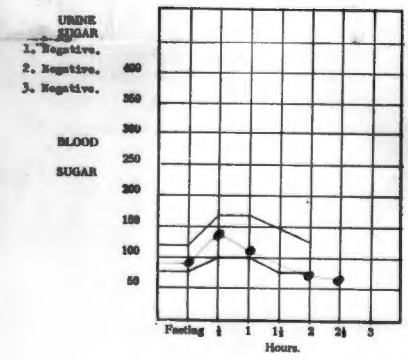
UNIVERSITY OF CAPE TOWN.

Serial No. 24271 - 72. Date 21.7.59.

Patient's Identification LUCY WALLIS (175070)

Ward A30. Physician or Surgeon Dr. Benjamin.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting	88 mgs.%.
½ hour	138 mgs.%.
1 hour	112 mgs.%.
1½ hour	
2 hour	76 mgs.%.
2½ hour	70 mgs.%
3 hour	

Signature G. M. POTGIETER

Age 45 years. D&C and cautery: Normal secretory endometrium.

60.

Report from Pathology Department.

(Chemical Pathology).

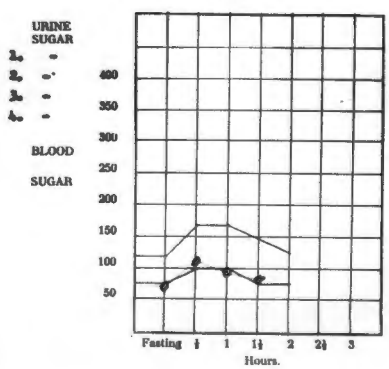
UNIVERSITY OF CAPE TOWN.

Serial No. 24833 - 34. Date 22.7.59.

Patient's Identification MARGARET KISTNASAMY (98/12757)

Ward 37. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting	76 mgs.%.
½ hour	112 mgs.%
1 hour	98 mgs.%.
1½ hour	80 mgs.%.
2 hour	
2½ hour	
3 hour	

Signature G. M. POTGIETER

Age 53 years. C/O offensive vaginal discharge. Menorrhagia ... L.M.P. 15/5/59. D&C 23/6/59: Late secretory endometrium. (Uterus 8 – 10 weeks size fibroids).

41.

Report from Pathology Department.

(Chemical Pathology).

UNIVERSITY OF CAPE TOWN.

Serial No. [illegible] – 60 Date [illegible]

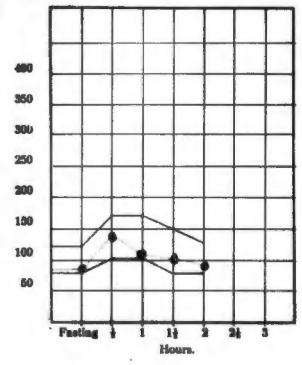
Patient's Identification [illegible] (29/1059)

Ward [illegible] Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR
1. –
2. –
3. –

BLOOD SUGAR



BLOOD SUGAR
Fasting [illegible] mgs.%.
½ hour 143 mgs.%.
1 hour 111 mgs.%.
1½ hour 104 mgs.%.
2 hour 95 mgs.%.
2½ hour
3 hour

G. M. POTGIETER

Signature

Age 51 years. C/O Profuse menorrhagia (Hb. 6 G.) Blood pressure 220/105 – congestive cardiac failure – hence later on X-ray menopause. 22/1/58 D&C : Proliferative endometrium ... plasia.

42.

Report from Pathology Department.

(Chemical Pathology).

UNIVERSITY OF CAPE TOWN.

Serial No. [illegible] – 10. Date 2.8.59.

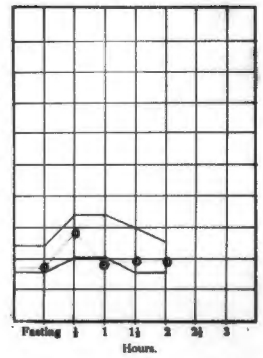
Patient's Identification [illegible] (27/59)

Ward [illegible] Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR
1. –
2. –
3. –

BLOOD SUGAR



Received 4.8.59 at 11 a.m.

BLOOD SUGAR
Fasting 88 mgs.%.
½ hour 140 mgs.%.
1 hour 98 mgs.%.
1½ hour 94 mgs.%.
2 hour 97 mgs.%.
2½ hour
3 hour

G. M. POTGIETER

Signature

Age 49 years. C/O Menorrhagia and irregular menstruation and "stress incontinence". D&C 17/6/59: Normal proliferative phase endometrium. Vaginal Hysterectomy ... Cervicitis.

43.

Report from Pathology Department.

(Chemical Pathology).

UNIVERSITY OF CAPE TOWN.

Serial No. [illegible] – 40. Date [illegible]

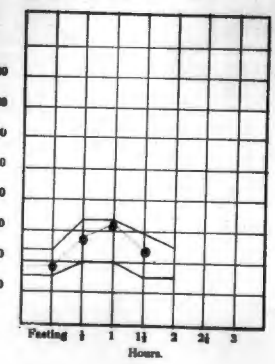
Patient's Identification [illegible]

Ward [illegible] Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR
1. ++
2. +
3. Negative.

BLOOD SUGAR



BLOOD SUGAR
Fasting 95 mgs.%.
½ hour 143 mgs.%.
1 hour 165 mgs.%.
1½ hour 127 mgs.%.
2 hour Insufficient.
2½ hour
3 hour

G. M. POTGIETER

Signature

Age 45 years. C/O Menorrhagia and dysmenorrhoea. D&C 31/3/58 Secretory endometrium. Sub-mucous fibroids in cavity of uterus.

44.

Report from Pathological Department.

UNIVERSITY OF CAPE TOWN.

Serial No. [illegible] Date 27.3.58.

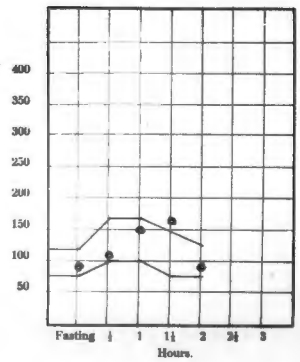
Patient's Identification [illegible]

Ward [illegible] Physician or Surgeon Prof. J.P. Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR
I. Negative.
II "
III "

BLOOD SUGAR



BLOOD SUGAR
Fasting 97 mgs.%.
½ hour 110 mgs.%.
1 hour 150 mgs.%.
1½ hour 140 mgs.%.
2 hour 93 mgs.%.
2½ hour
3 hour

Signature G. M. POTGIETER

Age 54 years. C/O menorrhagia: few months; prolapse with " stress incontinence". D&C 15/12/57 Secretory endometrium. Had vaginal hysterectomy and repair later.

45.

Report from Pathological Department.

UNIVERSITY OF CAPE TOWN.

Serial No. [illegible] Date [illegible]

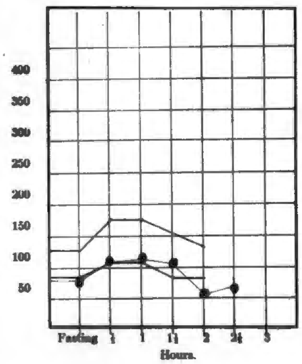
Patient's Identification [illegible] ([illegible])

Ward [illegible] Physician or Surgeon [illegible]

GLUCOSE TOLERANCE TEST.

URINE SUGAR

- o/s
- o/s
- o/s



BLOOD SUGAR

Fasting	79 mgs.%
½ hour	105 mgs.%
1 hour	116 mgs.%
1½ hour	105 mgs.%
2 hour	59 mgs.%
2½ hour	63 mgs.%
3 hour	

Signature

Age 45 years. C/O Menorrhagia: 3 years. Fibroids size 14 weeks. D&C 27/7/59 (L.M.P. 13/7/59) Normal mid-phase endometrium.

46.

Report from Pathology Department.

(Chemical Pathology).

UNIVERSITY OF CAPE TOWN.

Serial No. [illegible] Date [illegible]

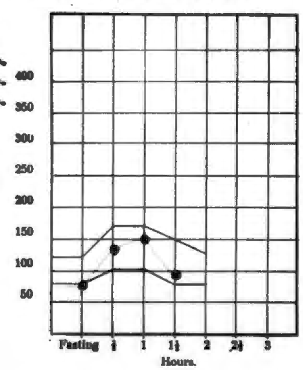
Patient's Identification [illegible] ([illegible]).

Ward [illegible] Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR

1. Negative.
2. Negative.
3. Negative.



BLOOD SUGAR

Fasting	79 mgs.%.
½ hour	139 mgs.%.
1 hour	152 mgs.%.
1½ hour	90 mgs.%.
2 hour	
2½ hour	
3 hour	

Signature G. M. POTGIETER

Age 46 years. C/O severe menorrhagia: 4 years. Fibroids size 12 weeks. D&C 9/10/59 Normal proliferative endometrium, corresponding to phase of cycle.

47.

Report from Pathology Department.

(Chemical Pathology).

UNIVERSITY OF CAPE TOWN.

Serial No. 34228 - 29 Date 2.10.59.

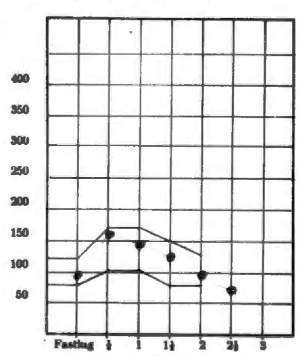
Patient's Identification LILLIAN WATTS (59/09848)

Ward C10. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR

- -
- -
- -



BLOOD SUGAR

Fasting	90 mgs.%.
½ hour	163 mgs.%.
1 hour	145 mgs.%.
1½ hour	125 mgs.%.
2 hour	95 mgs.%.
2½ hour	72 mgs.%.
3 hour	

Signature G. M. POTGIETER

Age 47 years. C/O Menorrhagia. [illegible]/59 Hysterectomy - fibroids, normal endometrium.

Groote Schuur Hospital. 48.

Report from Pathology Department.

(Chemical Pathology).

UNIVERSITY OF CAPE TOWN.

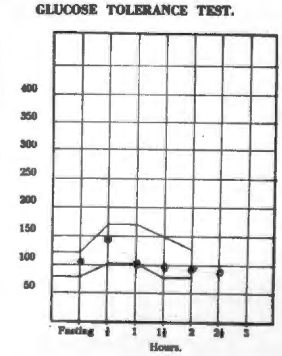
Serial No. [illegible] Date [illegible]

Patient's Identification [illegible] ([illegible])

Ward [illegible] Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

	[illegible]	URINE SUGAR
1.	0	0
2.	0	0
3.	0	0



BLOOD SUGAR

Fasting	102 mgs.%.
½ hour	144 mgs.%.
1 hour	100 mgs.%.
1½ hour	96 mgs.%.
2 hour	93 mgs.%.
2½ hour	84 mgs.%.
3 hour	

Signature G. M. POTGIETER

Age 46 years. C/O Haemorrhagia : 3 years. Fibroids. 2/10/39
D&C : Normal interval phase endometrium.

Groote Schuur Hospital

49.

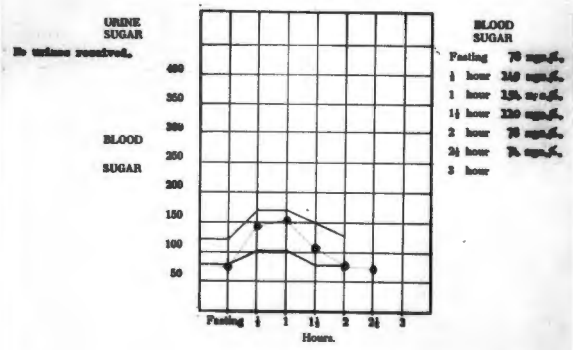
Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

Serial No. [illegible] Date [illegible]

Patient's Identification [illegible]

Ward [illegible] Physician or Surgeon [illegible]

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting	70 mgs.%
½ hour	140 mgs.%
1 hour	154 mgs.%
1½ hour	120 mgs.%
2 hour	78 mgs.%
2½ hour	74 mgs.%
3 hour	

[illegible] G. M. POTGIETER

DIRECT T.S.I. ADMINISTRATION.
Age 50 years. C/O Route of haemorrhage followed by bouts of bleeding 9 months. [illegible] 12/11/39 D&C: secretory endometrium.

50(a)

Report from Pathology Department.
(Chemical Pathology).
7777/39
UNIVERSITY OF CAPE TOWN.

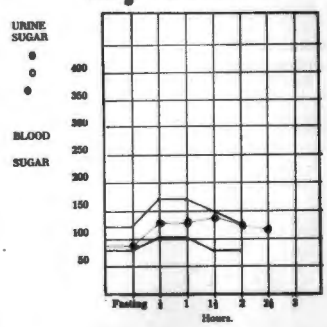
Serial No. [illegible] Date [illegible]

Patient's Identification [illegible]

Ward [illegible] Physician or Surgeon [illegible]

GLUCOSE TOLERANCE TEST.

Reactions	URINE SUGAR
1. 0	0
2. 0	0
3. 0	0



BLOOD SUGAR

Fasting	89 mgs.%
½ hour	138 mgs.%
1 hour	138 mgs.%
1½ hour	145 mgs.%
2 hour	129 mgs.%
2½ hour	128 mgs.%
3 hour	

[illegible] G. M. POTGIETER

Age 45 years. No haemorrhagia. Cycle 3 - 4/25 day type. D&C: Normal secretory endometrium.

[illegible] Schuur Hospital.

51.

Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

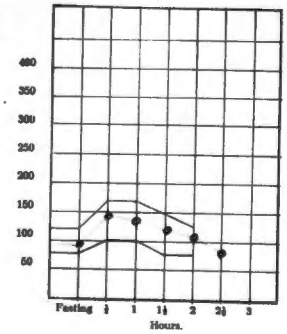
Serial No. [illegible] Date [illegible]

Patient's Identification [illegible]

Ward [illegible] Physician or Surgeon [illegible]

GLUCOSE TOLERANCE TEST.

Reactions	URINE SUGAR
1. 0	0.
2. 0	0
3. 0	0



BLOOD SUGAR

Fasting	94 mgs.%
½ hour	149 mgs.%
1 hour	139 mgs.%
1½ hour	124 mgs.%
2 hour	111 mgs.%
2½ hour	87 mgs.%
3 hour	

G. M POTGIETER

Age 60 years. Menopause at 53 years. Post-menopausal bleeding 4 months. D&C and biopsy : Carcinoma of cervix, atrophic post-menopausal endometrium.

52.

Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

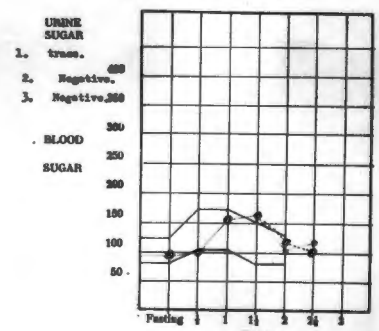
Serial No. [illegible] Date [illegible]

Patient's Identification [illegible]

Ward [illegible] Physician or Surgeon [illegible]

GLUCOSE TOLERANCE TEST.

	URINE SUGAR
1.	trace.
2.	Negative.
3.	Negative.



BLOOD SUGAR

Fasting	97 mgs.%
½ hour	100 mgs.%
1 hour	161 mgs.%
1½ hour	162 mgs.%
2 hour	104 mgs.%
2½ hour	121 mgs.%
3 hour	

Both the "2 h" and "2½ h" times were labelled "2 hours".

[illegible] L. ANSTEY.

Age 46 years. Menorrhagia 3 months. D&C 19.11.59: Normal proliferative endometrium.

53.

Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

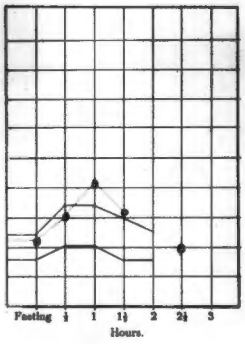
Serial No. 4406 - 407 Date 21.11.59.

Patient's Identification ANNA VAN NIE. (28896)

Ward 4th. Physician or Surgeon Dr. Reynolds.

GLUCOSE TOLERANCE TEST.

Specimen	URINE SUGAR
1.	0
2.	0
3.	0



BLOOD SUGAR	
Fasting	110 mgs.%.
½ hour	151 mgs.%.
1 hour	200 mgs.%.
1½ hour	155 mgs.%.
2 hour	Clotted.
2½ hour	91 mgs.%.
3 hour	

Signature G. M POTGIETER

Age 47 years. C/O Oligo-menorrhoea. D&C 14.11.59 : Normal secretory endometrium.

54.

Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

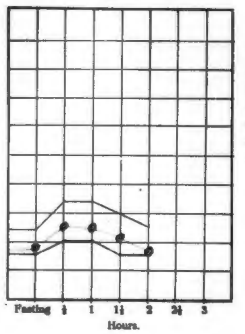
Serial No. 3906 - 07 Date 17.9.59.

Patient's Identification SHEILA BRAAM (28432)

Ward 4D Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR	
1.	trace.
2.	Negative
3.	Negative.



BLOOD SUGAR	
Fasting	90 mgs.%.
½ hour	158 mgs.%.
1 hour	182 mgs.%.
1½ hour	117 mgs.%.
2 hour	84 mgs.%.
2½ hour	
3 hour	

ceived 17.9.59 at 4 p.m.

Signature L. ANSTEY.

Age 47 years. C/E Fibroids size 16 weeks. 8/10/59D&C : Normal secretory endometrium. Hysterectomy not done - cardiac disease.

55.

Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

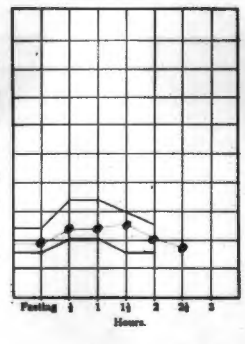
Serial No. 32730. Date 22.9.59.

Patient's Identification ALICE BE BRUYN (59/09621)

Ward A2. Physician or Surgeon Dr. Brown.

GLUCOSE TOLERANCE TEST.

URINE SUGAR
No urine received.



BLOOD SUGAR	
Fasting	93 mgs.%.
½ hour	121 mgs.%.
1 hour	121 mgs.%.
1½ hour	126 mgs.%.
2 hour	102 mgs.%.
2½ hour	89 mgs.%.
3 hour	

Signature G. M. POTGIETER

Age 46 years. Menorrhagia and discharge. D&C and biopsy: Cervicitis, normal secretory endometrium.

56.

Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

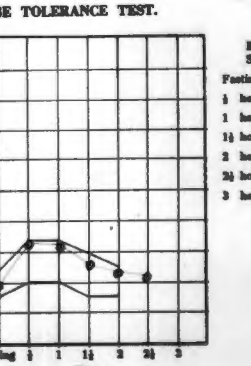
Serial No. 3956 - 67. Date 21.9.59.

Patient's Identification [illegible] (28025)

Ward 4th. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR	
1.	Negative.
2.	Negative.
3.	Negative.



BLOOD SUGAR	
Fasting	94 mgs.%.
½ hour	172 mgs.%.
1 hour	189 mgs.%.
1½ hour	138 mgs.%.
2 hour	139 mgs.%.
2½ hour	112 mgs.%.
3 hour	

Signature L. ANSTEY.

Age 51 years. C/O prolapse and discharge. Cervical erosion. D&C and cautery. Normal endometrium.

61.

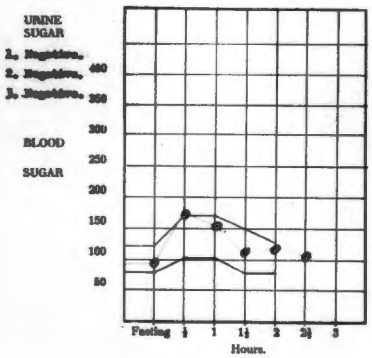
Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

Serial No. 3867 - 40 Date 15.9.39.

Patient's Identification DAPHNE KINNAN (39/2028)

Ward 220. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR
Fasting 97 mgs.%.
½ hour 175 mgs.%.
1 hour 155 mgs.%.
1½ hour 134 mgs.%.
2 hour 125 mgs.%.
2½ hour 109 mgs.%.
3 hour

Signature G. M. POTGIETER

Age 46 years. C/O Menorrhagia. D&C : Normal secretory endometrium, cervical erosion.

62.

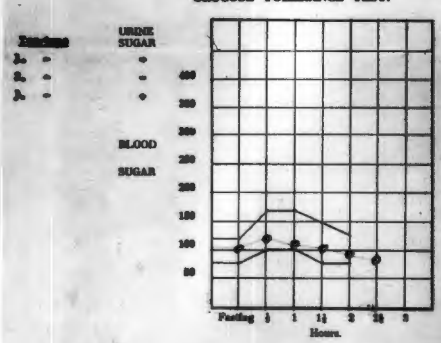
Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

Serial No. [illegible] Date [illegible]

Patient's Identification [illegible]

[illegible] Physician or Surgeon [illegible]

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR
Fasting 145 mgs.%.
½ hour 188 mgs.%.
1 hour 134 mgs.%.
1½ hour 105 mgs.%.
2 hour 93 mgs.%.
2½ hour 80 mgs.%.
3 hour

Signature G. M. POTGIETER

Age 52 years. Menorrhagia. Fibroids. Hysterectomy: Normal proliferative endometrium.

63.

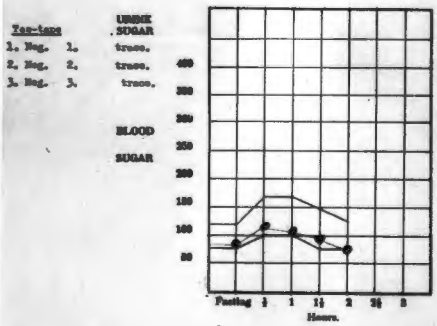
Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

Serial No. 673 Date 30.9.39.

Patient's Identification HELEN CORNELIUS. (56/2451)

Ward 37. Physician or Surgeon Dr. Heany.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR
Fasting 80 mgs.%.
½ hour 139 mgs.%.
1 hour 107 mgs.%.
1½ hour 94 mgs.%.
2 hour 76 mgs.%.
2½ hour
3 hour

Signature G. M. POTGIETER

Age 45 years. Prolapse. Vaginal Hysterectomy and repair: Normal secretory endometrium.

64.

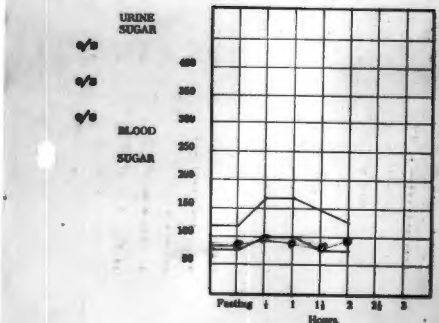
Report from Pathological Department.
UNIVERSITY OF CAPE TOWN.

Serial No. [illegible] Date 27.6.37

Patient's Identification IRENE ISAACS. (A704/6)

Ward A 2[illegible] Physician or Surgeon Prof. Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR
Fasting 85 mgs.%
½ hour 93 mgs.%
1 hour 90 mgs.%
1½ hour 81 mgs.%
2 hour 89 mgs.%
2½ hour
3 hour

Signature

Age 49 years. Menorrhagia and intermenstrual bleeding. D&C and Hysterectomy : Cervical polyp, atrophic endometrium.

65.

Report from Pathological Department.

UNIVERSITY OF CAPE TOWN.

Serial No. 30315-16/57 Date 11.9.57

Patient's Identification ANNA VAN ZYL (56/13453).

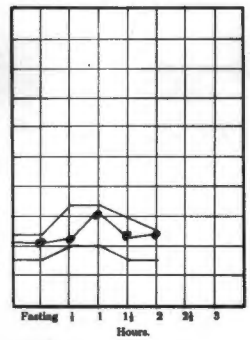
Ward G 10. Physician or Surgeon Prof. Louw

GLUCOSE TOLERANCE TEST.

URINE SUGAR

8 am. Negative.
6.30 negative.
9 am. Negative.

BLOOD SUGAR



BLOOD SUGAR

Fasting 101 mgs.%
½ hour 118 mgs.%
1 hour 152 mgs.%
1½ hour 120 mgs.%
2 hour 122 mgs.%
2½ hour
3 hour

G. LINDER

Signature

Age 48 years. C/O Menorrhagia. D&C : normal secretory endometrium.

66.

Report from Pathological Department.

UNIVERSITY OF CAPE TOWN.

Serial No. 17561-62/57 Date 22.5.57

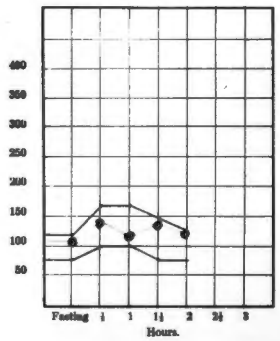
Patient's Identification LENA HLATI (57/16925).

Ward A 9. Physician or Surgeon Prof. Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR

BLOOD SUGAR



BLOOD SUGAR

Fasting 105 mgs.%
½ hour 114 mgs.%
1 hour 124 mgs.%
1½ hour 131 mgs.%
2 hour 129 mgs.%
2½ hour
3 hour

G. LINDER

Signature

Age 54 years. Prolapse. D&C and repair: No curettings.

67.

Report from Pathology Department.

(Chemical Pathology).

UNIVERSITY OF CAPE TOWN.

Serial No. 3832 - A33. Date 18.9.59.

Patient's Identification ROSIE NEMAN (59/07264)

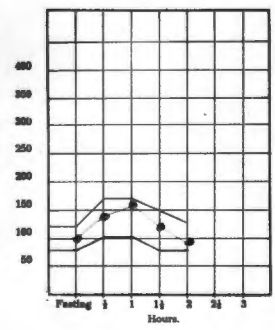
Ward A30. Physician or Surgeon Prof. Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR

1. Negative.
2. trace.
3. +

BLOOD SUGAR



BLOOD SUGAR

Fasting 100 mgs%.
½ hour 148 mgs%.
1 hour 156 mgs%.
1½ hour 138 mgs%.
2 hour 93 mgs%.
2½ hour
3 hour

received 18.9.59 at 3 p.m.

L. ANSTEY

Signature

Age 51 years. C/O Discharge and prolapse. D&C : Atrophic endometrium.

68.

Report from Pathological Department.

UNIVERSITY OF CAPE TOWN.

Serial No. 18830-31/57 Date 2.6.57

Patient's Identification PAULINA MAPHUMA (6296)

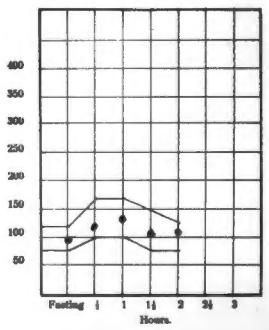
Ward G 10. Physician or Surgeon Prof. Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR

o/o
o/o
o/o

BLOOD SUGAR



BLOOD SUGAR

Fasting mgs.%
½ hour 137 mgs.%
1 hour 136 mgs.%
1½ hour 108 mgs.%
2 hour 105 mgs.%
2½ hour
3 hour

G. LINDER

Signature

Age 49 years. C/O Post-menopausal bleeding: 2½ months. Menopause at 48 years- 1 year ago. 8/2/59 D&C : cervicitis - atrophic endometrium.

69.

Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

Serial No. 23800 - 801. Date 13.7.59.

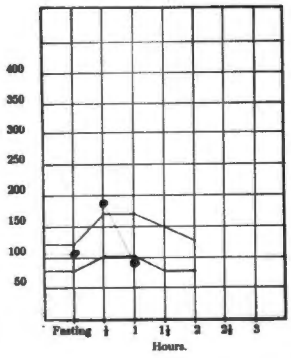
Patient's Identification MARY LUBANDE (58/30130)

Ward 49. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR
1. -
2. -
3. -

BLOOD SUGAR



BLOOD SUGAR
Fasting 101 mgs.%.
½ hour 167 mgs.%.
1 hour 96 mgs.%.
1½ hour Insufficient.
2 hour Insufficient.
2½ hour
3 hour

Signature G. M. POTGIETER

Age 47 years. Carcinoma of cervix (Radium and sertheim) Endometrium atrophic.

70.

Report from Pathological Department.
UNIVERSITY OF CAPE TOWN.

Serial No. 26912-13 Date 11.8.58.

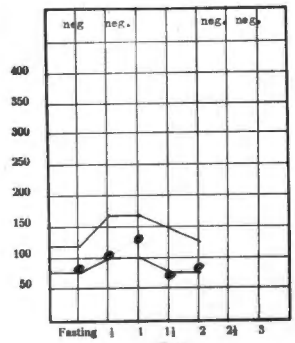
Patient's Identification ADAMS WILLIAM. (57/33735).

Ward B7 Physician or Surgeon Prof. Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR
neg neg. neg. neg.

BLOOD SUGAR



BLOOD SUGAR
Fasting 65 mgs.%.
½ hour 98 mgs.%.
1 hour 125 mgs.%.
1½ hour 71. mgs.%.
2 hour 86 mgs.%.
2½ hour
3 hour

Signature

Age 51 years. Post-menopausal bleeding - senile endometritis. Menopause at 46 years. Total hysterectomy.

71.

Report from Pathological Department.
UNIVERSITY OF CAPE TOWN.

Serial No. 31983-84/57 Date 24.9.57

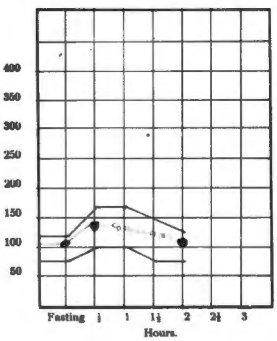
Patient's Identification LENA STAMMERS. (336513)

Ward A9 Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR
Urine specimens NEGATIVE.

BLOOD SUGAR



BLOOD SUGAR
Fasting 112 mgs.%.
½ hour 135 mgs.%.
1 hour Partially Coagulated.
1½ hour Coagulated.
2 hour 114(?
2½ hour
3 hour

Signature

Age 54 years. Post-menopausal bleeding. Atrophic endometrium. Menopause at 50 years.

72.

Report from Pathological Department.
UNIVERSITY OF CAPE TOWN.

Serial No. 34075/57 Date 11.10.57.

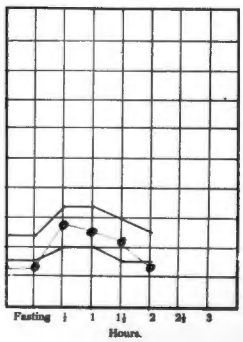
Patient's Identification KATHLEEN BEYERS. (149028).

Ward C 10 Physician or Surgeon Dr Buss.

GLUCOSE TOLERANCE TEST.

URINE SUGAR
Neg. I.
Neg II.
Neg. III.

BLOOD SUGAR



BLOOD SUGAR
9.00 am Fasting 72 mgs.%.
9.00 am 134 mgs.%.
9.30 am 126 mgs.%.
1 hour
10.00 am 118mgs.%.
1½ hour
10.30 am 68 mgs.%.
2 hour
2½ hour
3 hour

Signature

Age 50 years. Post-menopausal bleeding: 3 days. Menopause 5 months before. D&C Proliferative endometrium.

73.

Report from Pathological Department.

UNIVERSITY OF CAPE TOWN.

Serial No. 27240/57. Date 15.8.57.

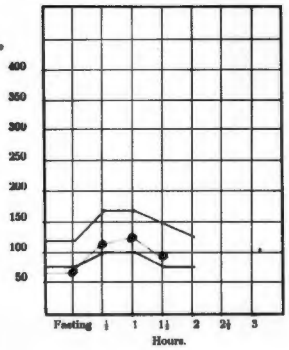
Patient's Identification EMILY NKOMBELO. (57/19759)

Ward B7 Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR

No sugar in any of the urine specimens.



BLOOD SUGAR

Fasting 66 mgs.%.
¼ hour 103 mgs.%.
1 hour 118 mgs.%.
1½ hour 97 mgs.%.
2 hour
2½ hour
3 hour

Signature

Age 52 years. C/O Post-menopausal bleeding : 3 months. Menopause at 45 years. Blood pressure 200/110. 12/6/59 D&C: Senile vaginitis, atrophic endometrium; one small intrauterine polyp in which the glands are atrophic and inactive.

74.

Report from Pathology Department.

(Chemical Pathology).

UNIVERSITY OF CAPE TOWN.

Serial No. 19193 - 94. Date 14.6.59.

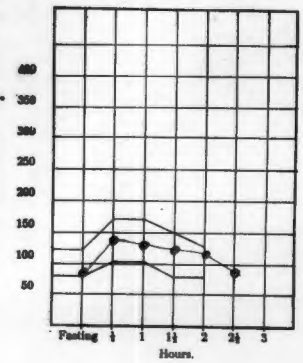
Patient's Identification MRS. A. HARLEMAN. (141811).

Ward Gyn. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR

1. -
2. -
3. No spec.



BLOOD SUGAR

Fasting 85 mgs.%.
¼ hour 141 mgs.%.
1 hour 134 mgs.%.
1½ hour 132 mgs.%.
2 hour 120 mgs.%.
2½ hour 88 mgs.%.
3 hour

J. J. V. D. WALT
Signature

Age 43 years. C/O Amenorrhoea: 3 months - due to early menopause "(flushes") D&C : Normal endometrium.

75.

Report from Pathological Department.

UNIVERSITY OF CAPE TOWN.

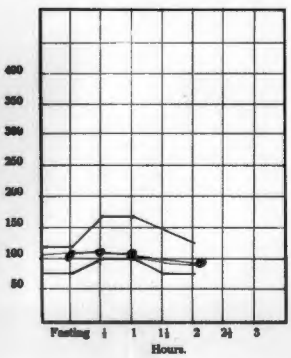
Serial No. 29027-28 Date 4.9.58.

Patient's Identification LANY HENDRICKS. (57/07635)

Ward B7 Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR



BLOOD SUGAR

Fasting 108 mgs%.
¼ hour 108 mgs.%.
1 hour 103 mgs.%.
1½ hour
2 ½ hour 96 mgs.%.
2½ hour
3 hour

Signature

Age 74 years. C/O Post-menopausal bleeding: 6 months. O/E Stage II Carcinoma cervix. D&C : no curettings.

76.

Report from Pathological Department.

UNIVERSITY OF CAPE TOWN.

Serial No. 580-581/58 Date 7.2.58.

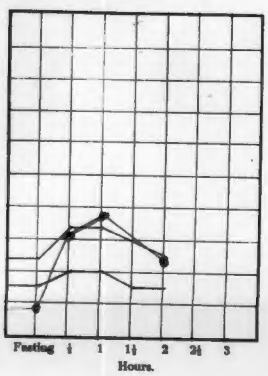
Patient's Identification ADA LOUW. (58/00097.)

Ward G 10 Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR

o/s Trace.



BLOOD SUGAR

Fasting 46 mgs%.
¼ hour 153 mgs.%.
1 hour 185 mgs.%.
1½ hour
2 hour 124 mgs.%.
2½ hour
3 hour

Signature

Age 56 years. C/O Post-menopausal bleeding : 3 weeks, prolapse. 8/5/59 D&C: Scanty atrophic endometrial glands - normal cervix - no malignancy. 18/6/59 Vaginal hysterectomy. Atrophic endometrium and myometrium.

77.

Report from Pathological Department.

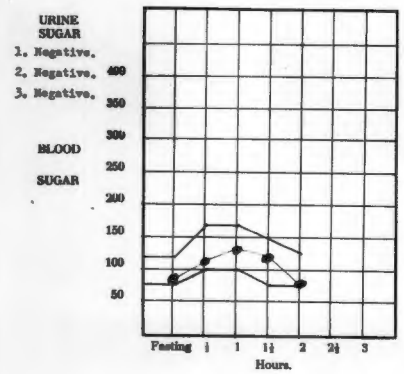
UNIVERSITY OF CAPE TOWN.

Serial No. 14524 - 525 Date 10.5.59

Patient's Identification MRS. MARIA ROUX. (59/05754)

Ward C10. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting 85 mgs.%.
½ hour 110 mgs.%.
1 hour 132 mgs.%.
1½ hour 120 mgs.%.
2 hour 83 mgs.%.
2½ hour
3 hour

Signature L. ANSTEY.

Age 57 years. Prolapse and slight menopausal

Age 57 years. Prolapse; slight post-menopausal bleeding. Atrophic endometrium. (Menopause at 52 years).

78.

Report from Pathological Department.

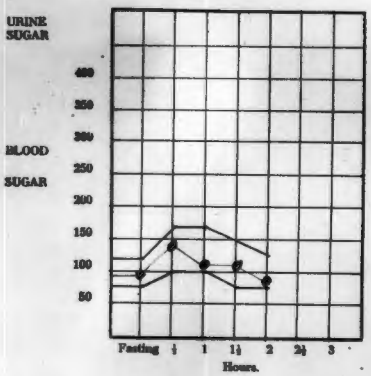
UNIVERSITY OF CAPE TOWN.

Serial No. 26598-99/57 Date 9.8.57.

Patient's Identification OLIVE ROBERTS (57/07632).

Ward C10. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting 94 mgs.%.
½ hour 126 mgs.%.
1 hour 107 mgs.%.
1½ hour 107 mgs.%.
2 hour 87 mgs.%.
2½ hour
3 hour

Signature

Age 60 years. Post-menopausal bleeding. Atrophic endometri (D&C and hysterectomy) (Menopause at 40 years.).

79.

Report from Pathological Department.

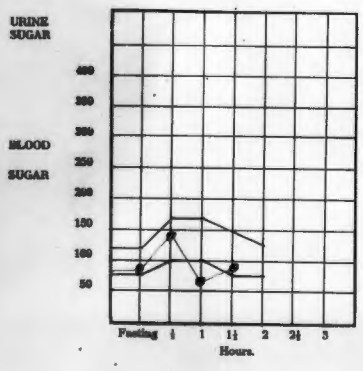
UNIVERSITY OF CAPE TOWN.

Serial No. 33519-20/57 Date 8.10.57.

Patient's Identification SARAH BARRY. (197836).

Ward A9 Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting 77 mgs.%.
½ hour 137 mgs.%.
1 hour 66 mgs.%
1½ hour 79 mgs.%.
2 hour
2½ hour
3 hour

Signature

Age 59 years. Post-menopausal bleeding - Senile vaginitis - normal atrophic endometrium.

80.

Report from Pathological Department.

UNIVERSITY OF CAPE TOWN.

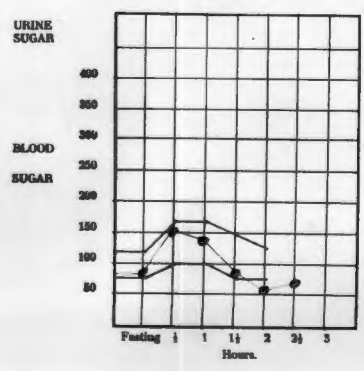
Serial No. 43446-7/57 Date 23.12.57.

Patient's Identification ALICE HAUSE. (57/12439).

Ward C 10. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

o/s
o/s
o/s.



BLOOD SUGAR

Fasting 82 mgs.%.
½ hour 161 mgs.%.
1 hour 139 mgs.%.
1½ hour 84 mgs.%.
2 hour 54 mgs.%.
2½ hour 73 mgs.%.
3 hour

Signature

Age 61 years. Menopause at 51 years. C/O scanty post-menopausal bleeding: 3 months. O/E Obese; blood pressure 205/130. 25/5/59 D&C: Scanty curettings: atrophic endometrium ; senile endometritis and senile vaginitis.

81.

Report from Pathological Department.

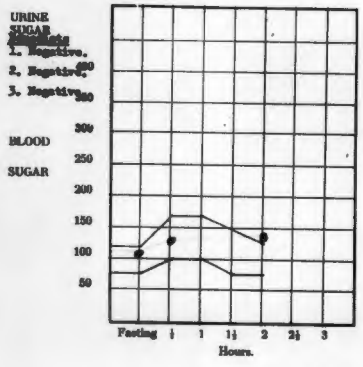
UNIVERSITY OF CAPE TOWN.

Serial No. 17384 - S. Date 29.5.59

Patient's Identification GAELJA SALIE (190962)

Ward E7. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting 107 mgs.%.
½ hour 128 mgs.%.
1 hour Insufficient
1½ hour Insufficient
2 hour 133 mgs.%.
2½ hour
3 hour

J. J. V. D. WALT

Signature

Age 60 years. Menopause at 54 years. C/O Post-menopausal bleeding: 4 months. 6/8/56 D&C and biopsy: Carcinoma cervix; corporal endometrium completely atrophic. Had Wertheim.

82.

Report from Pathology Department.

(Chemical Pathology).

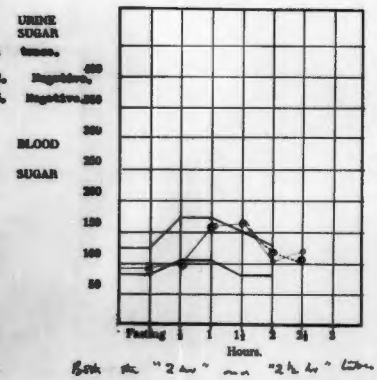
UNIVERSITY OF CAPE TOWN.

Serial No. [illegible] - 46 Date 28.8.59.

Patient's Identification [illegible]

Ward A2b. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting 97 mgs.%.
½ hour 200 mgs.%.
1 hour 162 mgs.%.
1½ hour 162 mgs.%.
2 hour 104 mgs.%.
2½ hour 121 mgs.%.
3 hour

L. ANSTEY.

Signature

Age 50 years. Menopause at 46 years. C/O Post-menopausal bleeding: 5 days. 1/7/59 D&C " Non-secretory and somewhat atrophic endometrium. Fibroids. (Serum Cholestrol 322).

83.

Report from Pathology Department.

(Chemical Pathology).

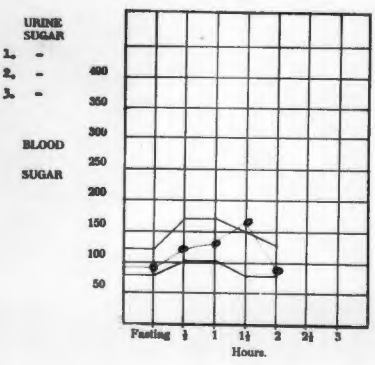
UNIVERSITY OF CAPE TOWN.

Serial No. 21364 - S. Date 26.6.59.

Patient's Identification ADA [illegible] (59/06243)

Ward G2b. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting 93 mgs.%.
½ hour 127 mgs.%.
1 hour 136 mgs.%.
1½ hour 165 mgs.%.
2 hour 90 mgs.%.
2½ hour
3 hour

L. ANSTEY

Signature

Age 62 years. Menopause at 45 years. C/O Prolapse . Blood pressure 195/110. Vaginal hysterectomy and repair 24/8/59: Fibroids; atrophic endometrium and myometrium.

84.

Report from Pathology Department.

(Chemical Pathology).

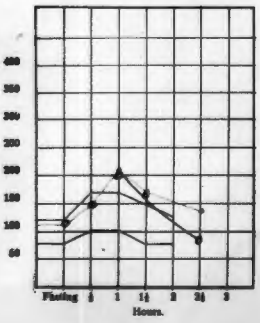
UNIVERSITY OF CAPE TOWN.

Serial No. [illegible] - 60. Date 2.9.59.

Patient's Identification [illegible]

Ward G2b. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting 116 mgs.%.
½ hour 150 mgs.%.
1 hour [illegible] mgs.%.
1½ hour 179 mgs.%.
2 hour [illegible].
2½ hour 82 mgs.%.
3 hour

G. M. POTGIETER

Signature

Age 54 years. Menopause at 53 years. C/O Post-menopausal bleeding. Fibroids. 27/8/59 D&C and biopsy: scanty curettings; inactive glands in an inactive stroma. Blood pressure 210/100.

85.

Report from Pathology Department.

(Chemical Pathology).

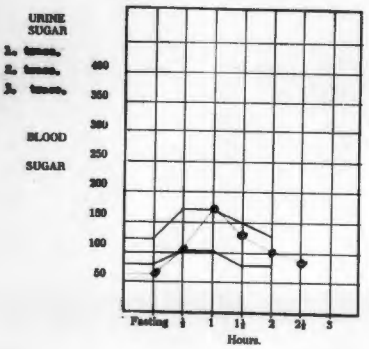
UNIVERSITY OF CAPE TOWN.

Serial No. 2941 - 42. Date 28.8.59.

Patient's Identification MARGOT KINNEAR (59/04825)

Ward 620. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR
Fasting 70 mgs.%.
½ hour 204 mgs.%.
1 hour 180 mgs.%.
1½ hour 134 mgs.%.
2 hour 104 mgs.%.
2½ hour 82 mgs.%.
3 hour

L. ANSTEY.

Age 57 years. C/O Postmenopausal bleeding: 4 months. Menopause at 56 years. D&C 15/6/59 - Secretory endometrium. Hysterectomy 24/6/59 - polyp in cavity; thin endometrium.

86.

Report from Pathology Department.

(Chemical Pathology).

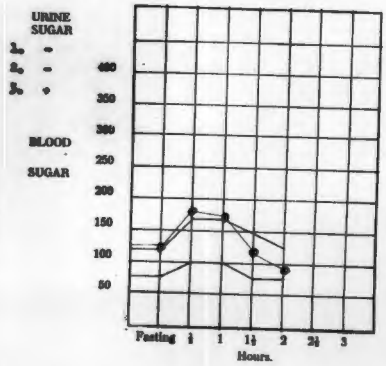
UNIVERSITY OF CAPE TOWN.

Serial No. 19202 - 203. Date 15.4.59.

Patient's Identification [illegible] (59063)

Ward 2E. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR
Fasting 138 mgs.%.
½ hour 188 mgs.%.
1 hour 179 mgs.%.
1½ hour 113 mgs.%.
2 hour 97 mgs.%.
2½ hour
3 hour

J. J. v. D. WALT

Age 54 years. C/O Post-menopausal bleeding. O/E Carcinoma cervix. Had 3 radium insertions and then Wertheim - no curettings.

87.

Report from Pathological Department.

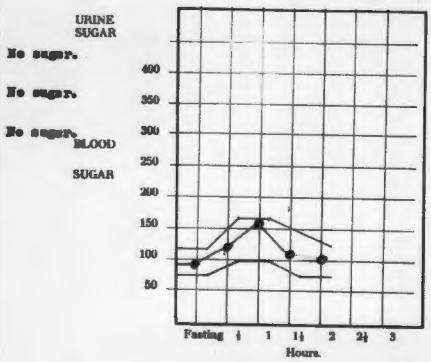
UNIVERSITY OF CAPE TOWN.

Serial No. 15510-11/57 Date 7.5.57

Patient's Identification MRS. JUDITH STEENKAMP. (57/03338).

Ward C 10. Physician or Surgeon Prof. Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR
Fasting 92 mgs.%
½ hour 129 mgs.%
1 hour 163 mgs.%
1½ hour 126 mgs.%
2 hour 105 mgs.%
2½ hour
3 hour

G. L[illegible]

Age 57 years. C/O Post-menopausal bleeding: 1 year. Menopause at 49 years. O/E Obese ++; Blood pressure 220/140. 19/5/57 D&C : Both glands and stroma show atrophy.

88.

Report from Pathological Department.

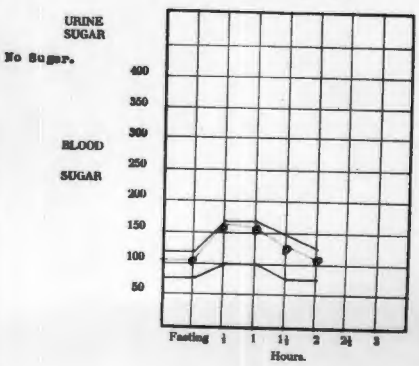
UNIVERSITY OF CAPE TOWN.

Serial No. 27[illegible]-5/57 Date 16.8.57

Patient's Identification GESINA CALLAGHAN. (57/07818).

Ward C 10. Physician or Surgeon Prof. Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR
Fasting 103 mgs.%
½ hour 170 mgs.%
1 hour 160 mgs.%
1½ hour 128 mgs.%
2 hour 107 mgs.%
2½ hour
3 hour

G. LINDER

Age 61 years. Menopause at 50 years. C/O Post-menopausal bleeding 2 months. 12/1099 D&C : Senile endometritis.

89.

Report from Pathological Department.

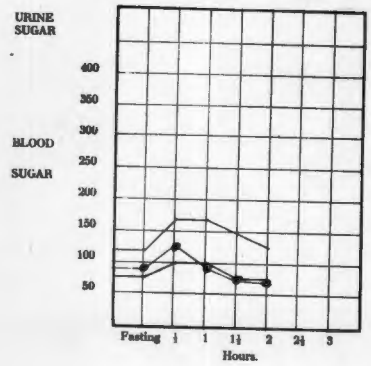
UNIVERSITY OF CAPE TOWN.

Serial No. 3bbdb-63/57 Date 18.10.57

Patient's Identification RACHEL SEPTEMBER. (57/23098).

Ward A 9. Physician or Surgeon Prof. Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR	
Fasting	87 mgs.%
½ hour	127 mgs.%
1 hour	87 mgs.%
1½ hour	71 mgs.%
2 hour	69 mgs.%
2½ hour	
3 hour	

Signature

Age 50 years. Menopause at 47 years. C/O Prolapse and stress incontinence. 2 episodes post-menopausal bleeding. D&C - no curettings. 13/9/57 Vaginal hysterectomy: post-menopausal endometrium.

90.

Report from Pathological Department.

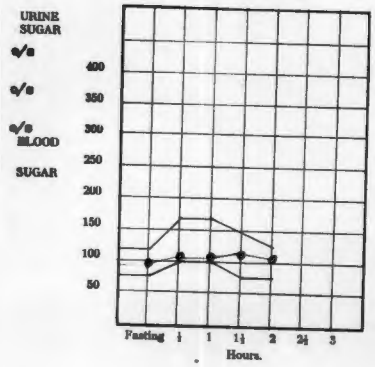
UNIVERSITY OF CAPE TOWN.

Serial No. 3-510-11/57 Date 13.9.57

Patient's Identification JOHANNA MURRAY. (56/00948).

Ward C 10. Physician or Surgeon Prof. Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR	
Fasting	91 mgs.%
½ hour	103 mgs.%
1 hour	103 mgs.%
1½ hour	119 mgs.%
2 hour	112 mgs.%
2½ hour	
3 hour	

G. LINDER

Signature

Age 57 years. C/O post-menopausal bleeding: 6 months. O/E Senile vaginitis . 4/9/57 D&C Scanty curettings : fragments of endometrial stroma only.

91.

Report from Pathological Department.

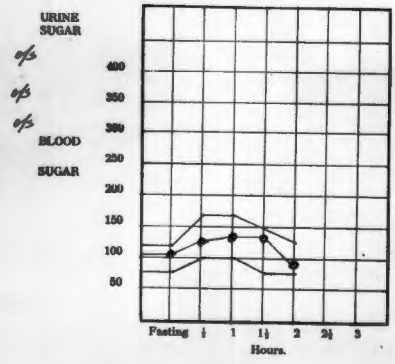
UNIVERSITY OF CAPE TOWN.

Serial No. 17277-78/57 Date 21.5.57

Patient's Identification MELVINA ALEXANDER. (55/00917).

Ward C 10. Physician or Surgeon Prof. Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR	
Fasting	101 mgs.%
½ hour	126 mgs.%
1 hour	129 mgs.%
1½ hour	129 mgs.%
2 hour	89 mgs.%
2½ hour	
3 hour	

G. LINDER

Signature

Age 68 years. C/O Post-menopausal bleeding: 4 months, O/E Fibroids and sarcomatous change . (D&C - no curettings.).

92.

Report from Pathological Department.

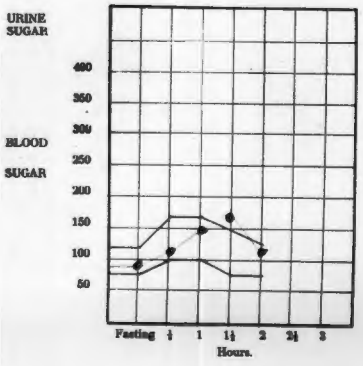
UNIVERSITY OF CAPE TOWN.

Serial No. 17277-78/57 Date 21.5.57

Patient's Identification CATHERINA BERGER (57/05108).

Ward C 10. Physician or Surgeon Prof. Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR	
Fasting	80 mgs.%
½ hour	125 mgs.%
1 hour	149 mgs.%
1½ hour	175 mgs.%
2 hour	125 mgs.%
2½ hour	
3 hour	

G. LINDER

Signature

Age 58 years. C/O Pruritis - Leukoplakia vulvae. Menopause at 45 years. D&C atrophic post-menopausal endometrium.

93.

Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

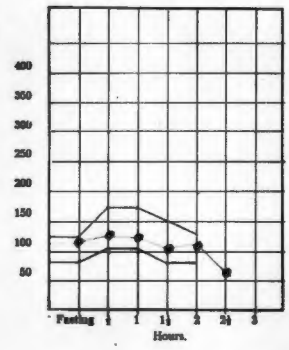
Serial No. 3823E - 33. Date 23.10.59.

Patient's Identification HILDA MEHLIN (29/6824)

Ward [illegible] Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

Urine	URINE SUGAR	BLOOD SUGAR	
2. 0	0	Fasting	139 mgms.
2. 0	0	½ hour	165 mgms.
3. 0	0	1 hour	125 mgms.
		1½ hour	104 mgms.
		2 hour	107 mgms.
		2½ hour	82 mgms.
		3 hour	



[illegible] G. M. POTGIETER
Signature

Age 52 years. C/O infrequent and not excessive menstruation (40 day type) L.M.P. 25/3/59. D&C 10/6/59 : Proliferative endometrium,not benign glandular hyperplasia . B.N.R. -10. Serum Chol 272.

94.

Report from Pathological Department.
UNIVERSITY OF CAPE TOWN.

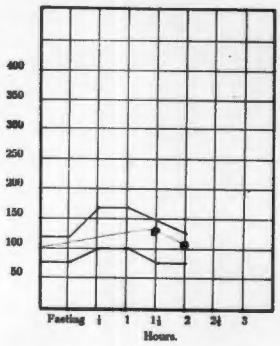
Serial No. [illegible] Date 17/10/59.

Patient's Identification Mrs. Ev. Long.

Ward P10. Physician or Surgeon Prof. J.T. Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR
Fasting : nil.
1-hour : nil.



BLOOD SUGAR
Fasting
½ hour
1 hour
1½ hour 133 mg. %.
2 hour 107 .
2½ hour
3 hour

[illegible] Signature Gm Potgieter.

Age 54 years. Menopause at 50 years. C/O prolapse. Vaginal hysterectomy done 29/1/59.

95.

Endosmic Research Lab.

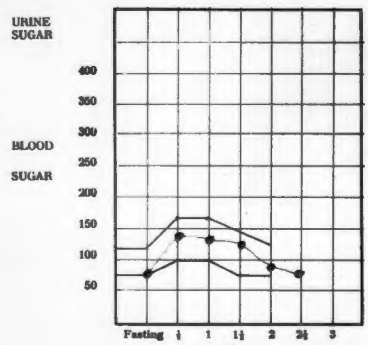
Report from Pathological Department.
UNIVERSITY OF CAPE TOWN.

Serial No. [illegible] Date 29/1/59.

Patient's Identification Mrs I. RIEGER.

Ward [illegible] Physician or Surgeon [illegible]

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR
Fasting 76 mgms %
½ hour 141
1 hour 153
1½ hour 178
2 hour 84
2½ hour 76.
3 hour

[illegible] Signature [illegible]

Age 59 years. C/O Prolapse. D&C: Scanty curettings - atrophic glands.

96.

Endosmic Research Lab.

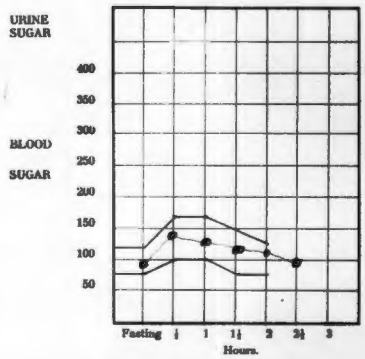
Report from Pathological Department.
UNIVERSITY OF CAPE TOWN.

Serial No. [illegible] Date 22/7/59.

Patient's Identification Mrs. VIOLET WOODHAM.

Ward P10. Physician or Surgeon [illegible]

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR
Fasting 94 mgms %
½ hour 104
1 hour 132
1½ hour 117
2 hour 116
2½ hour 99.
3 hour

[illegible] Signature [illegible]

TABLE 4. GLUCOSE TOLERANCE CURVES ON 100 CONTROL WOMEN WITH NORMAL ENDOMETRIA (i.e. NOT SUFFERING FROM BENIGN GLANDULAR HYPERPLASIA OR CARCINOMA OF THE ENDOMETRIUM) 45 YEARS AND OVER.

Patient			Blood Sugar Levels (Mgs.%)				
No	Initials	Age	Fasting	½ Hour	1 Hour	1½ Hours	2 Hours
A. Group with Diabetes.							
1.	O.T.	55		215	251	190	304
2.	G.J.	54	275	310		373	
3.	M.B.	48	251	304		431	438
4.	M.B.	79	137	158	213	201	176
5.	A.C.	54	233	266	326	337	294
6.	M.A.	57	189	271	374	368	319
7.	M.B.	71	137	186	238	300	219
8.	S.J.	71	243	333	399	344	333
9.	F.J.	70	206	240	236	362	369
10.	A.A.P.	78	94	295	254	234	234
11.	E.W.	89	109	177	235	246	298
12.	M.B.	79	157	193	254	301	286
13.	A.E.	53	186	186	212	202	227
B. Group with Mildly Impaired Glucose Tolerance.							
14.	W.N.	47	107		182	153	137
15.	R.A.	45	110	174	181	286	87
16.	F.S.	49		84	99	125	143
17.							
18.	Z.L.	47	96	144	157	171	162
19.	S.R.	47	97	151	128	180	153
20.	D.M.	53	102	165	221	200	142
21.	A.M.	68	110	147	172	190	160
22.	M.C.	50	89	171	215	219	128

Patient			Blood Sugar Levels (Mgs.%)				
No	Initials	Age	Fasting	½ Hour	1 Hour	1½ Hours	2 Hours
C. Group with Normal Glucose Tolerance Curves.							
23.	B.H.	51	99	121	142	133	76
24.	J.C.	50	79	128	167	107	88
25.	L.C.	52	67	105	143	124	106
26.	A.K.	53	77	119	146	121	110
27.	C.N.	48	87	115	158	94	80
28.	A.B.	49	79	114	116	107	
29.	H.B.	48	108	134	186	150	125
30.	M.O.	50	115	151	197	142	101
31.	A.D.	49	62	134	132	117	127
32.	A.M.	48	87	141	174	147	111
33.	C.M.	48	119	162	180	156	133
34.	Z.T.	46	90	122	126	130	126
35.	G.G.	47		151	151	100	115
36.	N.V.	45	63	202	199	141	104
37.	M.F.	45	98	112	108	99	108
38.	C.O.	45	86	151	105	85	
39.	S.L.	56	85	115	119	105	82
40.	A.G.	52	79	148	165	170	86
41.	V.W.	53	84	143	111	104	95
42.	E.W.	51	88	148	92	94	97
43.	S.C.	49	95	143	165	127	
44.	F.C.	45	97	110	150	169	93
45.	S.B.	54	79	104	116	105	59
46.	J.F.	45	79	139	152	90	
47.	L.W.	46	90	163	145	125	95
48.	J.H.	47	102	144	100	96	93
49.	A.C.	46	78	149	154	110	78
50.a	S.J.	50	88	132	132	145	129
51.	C.C.	45	94	149	138	124	111
52.	J.F.	60	97	100	161	162	104

Patient			Blood Sugar Levels (Mgs.%)				
No	Initials	Age	Fasting	½ Hour	1 Hour	1½ Hours	2 Hours
53.	A.V.	46	119	151	208	155	
54.	S.I.	47	90	128	121	117	84
55.	A.D.	47	93	121	121	126	102
56.	D.S.	46	94	172	165	138	129
57.	G.B.	48	99	145	152	122	132
58.	M.B.	47		116	128	121	104
59.	L.W.	50	88	138	112		76
60.	M.K.	45	76	112	98	80	
61.	D.K.	51	97	175	155	114	123
62.	P.B.	46	103	122	114	105	93
63.	H.O.	52	80	119	107	94	76
64.	I.I.	45	85	95	90	81	99
65.	A.V.	49	101	118	152	120	122
66.	L.H.	48	105	144	124	131	129
67.	S.B.	54	100	142	156	124	93
68.	P.H.	51	81	117	136	101	105
69.	M.L.	49	101	187	96		
70.	A.W.	47	65	98	125	71	86
71.	L.S.	51	112	135			114
72.	K.B.	54	72	134	126	118	68
73.	E.M.	50	66	103	118	97	
74.	A.H.	52	85	141	134	131	120
75.	M.H.	43	108	108	103		96
76.	A.L.	74	46	153	185		124
77.	M.R.	56	85	110	132	120	83
78.	O.F.	57	94	126	107	107	87
79.	S.B.	60	77	137	66	79	
80.	A.N.	59	82	161	139	84	54
81.	D.S.	61	107	128			133
82.	J.F.	60	97	100	161	162	104
83.	A.S.	50	93	127	136	165	90
84.	I.P.	62	116	150	214	179	82

Patient			Blood Sugar Levels (Mgs.%)				
No	Initials	Age	Fasting	½ Hour	1 Hour	1½ Hours	2 Hours
85.	M.Z.	54	70	104	180	134	104
86.	M.F.	57	124	188	179	113	97
87.	J.S.	54	91	129	163	126	105
88.	G.C.	57	103	170	160	128	107
89.	R.S.	61	87	127	87	71	69
90.	J.M.	50	91	103	103	119	112
91.	M.A.	57	101	126	129	129	89
92.	C.B.	68	80	125	149	175	125
93.	H.M.	58	119	125	123	104	107
94.	M.L.	52				133	107
95.	I.R.	54	75	141	132	128	84
96.	V.W.	59	94	134	132	117	115
97.	G.T.	47	60	142	160	132	109
98.	A.M.	46	80	107	115	117	105
99.	D.A.	45	88	92	205	108	105
100.	E.S.	62	119	187	180	129	120

The results can be summarised as follows:

TABLE 5. Glucose Tolerance Tests on 100 Control Women with normal Endometria (i.e. not suffering from Benign Glandular Hyperplasia or Carcinoma of the Endometrium), 45 years and over.

Normal Curves			
	Diabetes	Mildly Impaired Glucose Tolerance	Total
68%	13%	9%	22%

The percentage of diabetics (13%) and of women with mildly impaired glucose tolerance (9%), is therefore remarkably higher than that found in any of the aforementioned control series reported

in the literature, for women of the same age grouos; Spiegelman and Marks (1946), 1.02% diabetées; Wilkerson and Krall (1947), 5%; Joslin (1952), 2%; Walker (1959). 1.4%. However, the discrepancy is understandable. In none of these series was the population tested by routine glucose tolerance tests, the diabetics having been discovered by far less exacting methods. In each of my 100 control subjects, a full glucose tolerance test was done. This clearly demonstrates that many patients with diabetes remain undetected if full glucose tolerance tests are not done routinely.

(b) Control Subjects between 35 and 45 years of age:

Fifty consecutive women with normal endometria, between 35 and 45 years of age, were also examined and were subjected to the standard glucose tolerance test. The endometrium was examined histologically and the subject was only included if the endometrium was normal.

A photograph of each original glucose tolerance cure is seen in the following pages - on each graph is a summary of the clinical history and endometrial histology of the subject. Following the photographs is Table 6, on which are detailed the blood sugar results. A summary of the results (Table 7) follows the photographs and this Table of details.

Age 39 years. Menorrhagia. Normal secretory endometrium. 5 children; 3 abortions.

1.

Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

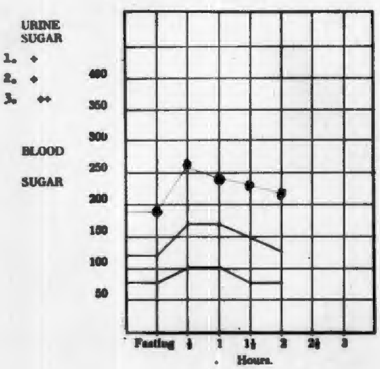
Serial No. 31515 - 516 Date 24.8.59

Patient's Identification FATIMA ISMAEL (3480)

Ward OPD Physician or Surgeon Prof Louw (pink)

GLUCOSE TOLERANCE TEST.

URINE SUGAR
1. +
2. +
3. ++



BLOOD SUGAR

Fasting	189	mgs.%.
½ hour	262	mgs.%.
1 hour	246	mgs.%.
1½ hour	237	mgs.%.
2 hour	227	mgs.%.
2½ hour		
3 hour		

Signature L. ANSTEY.

Age 39 years. C/O Menorrhagia : 9 months , prolapse. D&C 3/9/59
Interval phase endometrium with commencing secretory changes.

2.

Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

Serial No. 30637 - 38 Date 4.9.59.

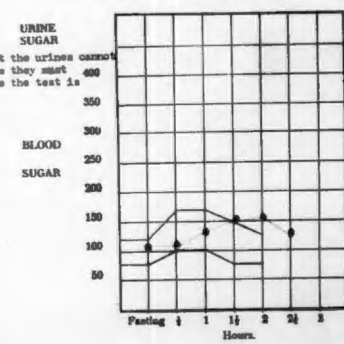
Patient's Identification CHRISTINE CROSS (59/09251)

Ward C10. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR

We regret that the urines cannot be accepted as they must be taken while the test is being done.



BLOOD SUGAR

Fasting	112	mgs.%.
½ hour	112	mgs.%.
1 hour	137	mgs.%.
1½ hour	150	mgs.%.
2 hour	155	mgs.%.
2½ hour	134	mgs.%.
3 hour		

Signature L. ANSTEY.

Age 38 years. C/O Pain and bleeding. Old ectopic, organised. Normal endometrium.

3.

Report from Pathological Department.
UNIVERSITY OF CAPE TOWN.

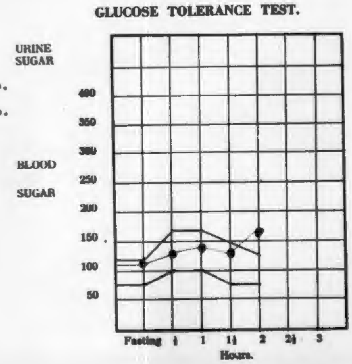
Serial No. 9263-64. Date 23.3.58.

Patient's Identification GIRLIE KHANTINI. (58/04959)

Ward 87. Physician or Surgeon Prof. Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR
I. trace.
II. +
III. Trace.



BLOOD SUGAR

Fasting	116	mgs.%.
½ hour	125.	mgs.%.
1 hour	145	mgs.%.
1½ hour	143	mgs.%.
2 hour	163	mgs.%.
2½ hour		
3 hour		

Signature

Age 36 years. Normal control. Normal menstruation and fertility.

4.

Report from ~~Pathological Department~~
ENDOCRINE RESEARCH LABORATORY.
UNIVERSITY OF CAPE TOWN.

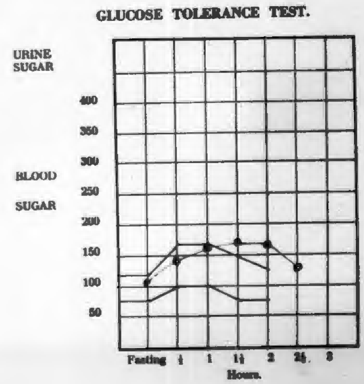
Serial No. Date 1959

Patient's Identification M. HANSEN.

Ward A 10. Physician or Surgeon Dr. F. Benjamin.

GLUCOSE TOLERANCE TEST.

URINE SUGAR



BLOOD SUGAR

Fasting	103	mgs.%.
½ hour	149	mgs.%.
1 hour	158	mgs.%.
1½ hour	163	mgs.%.
2 hour	161	mgs.%.
2½ hour	120	mgs.%.
3 hour		

Signature

Age 38 years. C/O Menorrhagia : 10 months. Fibroids size 12 weeks. Blood pressure 200/110, Hg 8.5 G. 1/9/59 hysterectomy - fibroids one of which had undergone sarcomatous change - endometrium thin and non-secretory (L.M.P. 22/8/59.

5.

Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

Serial No. 30165 - 06. Date [illegible]

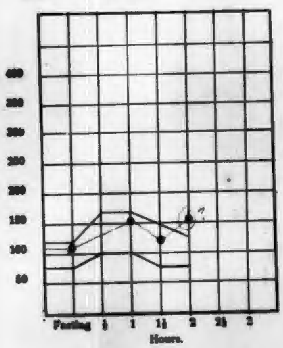
Patient's Identification [illegible] ([illegible])

Ward [illegible] Physician or Surgeon Prof [illegible]

GLUCOSE TOLERANCE TEST.

URINE SUGAR
1. Negative.
2. Negative.
3. Negative.

BLOOD SUGAR
Received [illegible].59 at 3 p.m.



BLOOD SUGAR
Fasting [illegible] mgm.%.
½ hour Insufficient.
1 hour [illegible] mgm.%.
1½ hour [illegible] mgm.%.
2 hour [illegible] mgm.%.
2½ hour
3 hour

Signature L. ANSTEY.

Age 43 years. C/O Menorrhagia : 6 months. Hysterectomy Normal uterus, normal endometrium.

6.

normal endometrium

Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

Serial No. 29963 - 6. Date 26.8.59.

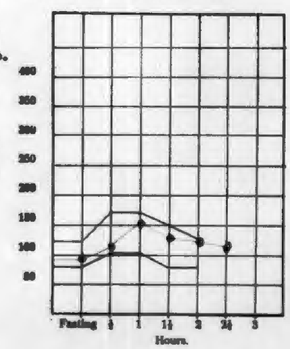
Patient's Identification [illegible] ([illegible])

Ward C10. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR
1. Negative.
2. trace.
3. trace.

BLOOD SUGAR



BLOOD SUGAR
Fasting 91 mgm.%.
½ hour 129 mgm.%.
1 hour 157 mgm.%.
1½ hour 137 mgm.%.
2 hour 135 mgm.%.
2½ hour 121 mgm.%.
3 hour

Signature L. ANSTEY.

Age 42 years. C/O Menorrhagia : 1 year. Obese 227 lbs. Hg. 12 G. Blood pressure 155/95. Fibroids of uterus. D&C 2/9/59 Proliferative endometrium, no metropathica, Hysterectomy 16/9/59 late secretory endometrium

7.

Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

Serial No. [illegible] Date [illegible]

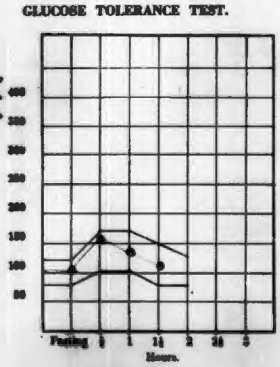
Patient's Identification [illegible] ([illegible])

Ward [illegible] Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR
1. Negative.
2. Negative.
3. Negative.

BLOOD SUGAR



BLOOD SUGAR
Fasting [illegible] mgm.%.
½ hour [illegible] mgm.%.
1 hour [illegible] mgm.%.
1½ hour [illegible] mgm.%.
2 hour Clotted.
2½ hour Clotted.
3 hour

G. M. POTGIETER
Signature

Age 35 years. C/O Menorrhagia : 6 months. D&C 18/9/59. 25th. day of cycle: secretory changes, but distended (had stilboestrol a few days prior to D&C).

8.

Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

Serial No. 31503 - 04. Date 14.9.59.

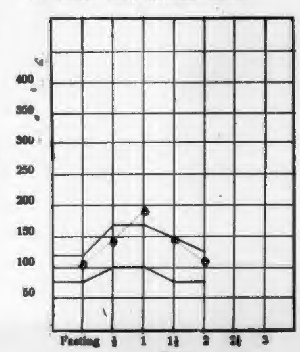
Patient's Identification C. [illegible] (59/23173)

Ward B7. Physician or Surgeon Dr. Massey.

GLUCOSE TOLERANCE TEST.

URINE SUGAR
1. -
2. -
3. -

BLOOD SUGAR



BLOOD SUGAR
Fasting 104 mgm.%.
½ hour 141 mgm.%.
1 hour 188 mgm.%.
1½ hour 141 mgm.%.
2 hour 116 mgm.%.
2½ hour
3 hour

Signature L. Anstey.

Age 40 years. C/O Menorrhagia and dysmenorrhoea: 7 months. D&C 25/5/57 : Secretory endometrium.

9.

Report from Pathological Department.

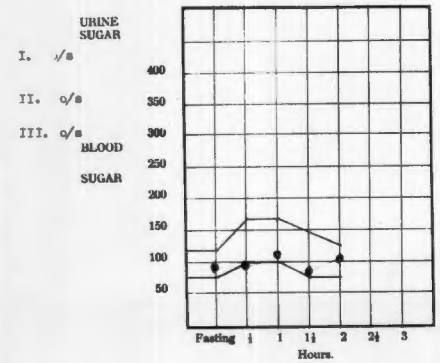
UNIVERSITY OF CAPE TOWN.

Serial No. 16998-99/57. Date 20.5.57

Patient's Identification MARY DE LILLIE. (57/10741)

Ward B 7 Physician or Surgeon Prof. Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR
Fasting 89 mgs.%
½ hour 96 mgs.%
1 hour 107 mgs.%
1½ hour 82 mgs.%
2 hour 105 mgs.%
2½ hour
3 hour

Signature

Age 42 years. C/O Menorrhagia: few months. D&C 20/3/58: Normal proliferative endometrium, chronic cervicitis.

10.

Report from Pathological Department.

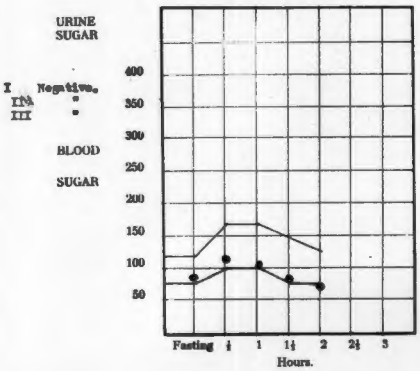
UNIVERSITY OF CAPE TOWN.

Serial No. 988-748 Date 28.3.58.

Patient's Identification CHRISTINA BARNES. (278851.)

Ward 4B Physician or Surgeon Prof. Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR
Fasting 89 mgs.%
½ hour 117 mgs.%
1 hour 102 mgs.%
1½ hour 79 mgs.%
2 hour 75 mgs.%
2½ hour
3 hour

Signature

Age 43 years. C/O Large abdomen. Tumour. O/E Fibroids size 16 weeks. Myomectomy 14/8/59.

11.

Report from Pathology Department.

(Chemical Pathology).

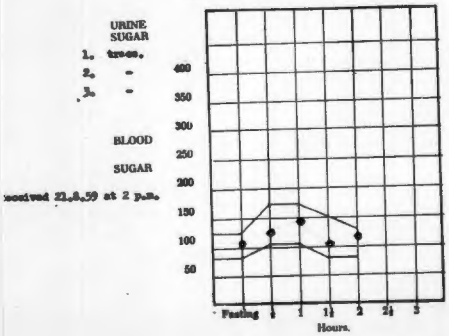
UNIVERSITY OF CAPE TOWN.

Serial No. 29017 - 18. Date 21.8.59.

Patient's Identification MRS. S.S. LAAUBSCH (59/09918)

Ward CHO. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR
Fasting 108 mgs.%
½ hour 127 mgs.%
1 hour 141 mgs.%
1½ hour 103 mgs.%
2 hour 124 mgs.%
2½ hour Clotted.
3 hour

Received 21.8.59 at 2 p.m.

G. M. POTGIETER
Signature

Age 40 years. C/O Menorrhagia: 2 years. Prolapse. 27/6/59 D&C : Normal proliferative endometrium. 2/10/59 Vaginal Hysterectomy Normal endometrium.

12.

Report from Pathology Department.

(Chemical Pathology).

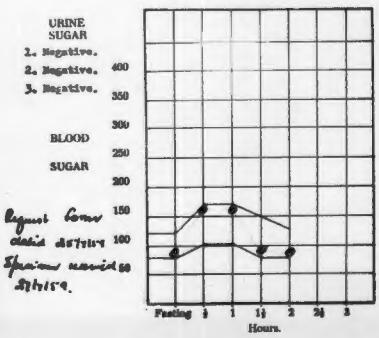
UNIVERSITY OF CAPE TOWN.

Serial No. 24995 - 6. Date 23.7.59.

Patient's Identification ALIDA CLAASSEN (59/07284)

Ward C.10. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.



Urgent Gynae clinic district Specimen received 23/7/59.

BLOOD SUGAR
Fasting 84 mgs.%
½ hour 164 mgs.%
1 hour 161 mgs.%
1½ hour 90 mgs.%
2 hour 88 mgs.%
2½ hour
3 hour

G. M. POTGIETER
Signature

Age 36 years. C/O Menorrhagia. Menstruation 5/38 day type, strong flow. 20/7/59 D&C: (L.M.P. 13/7/59) Normal proliferative endometrium.

13.

Report from Pathology Department.

(Chemical Pathology).

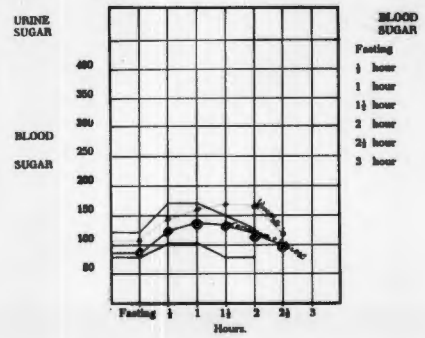
UNIVERSITY OF CAPE TOWN.

Serial No. _____ Date 7.8.59.

Patient's Identification Mrs. M. [illegible]

Ward _____ Physician or Surgeon Dr. [illegible]

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR
Fasting
½ hour
1 hour
1½ hour
2 hour
2½ hour
3 hour

Signature [illegible] KERSON

Age 40 years. C/O Polymenorrhoea and menorrhagia : 1 year. (5/0 - 7/21 day type) Total hysterectomy and bilateral salpingo-oophorectomy : Endometrium normal, follicular cysts of ovaries, true ulceration of cervix.

14.

Report from Pathological Department.

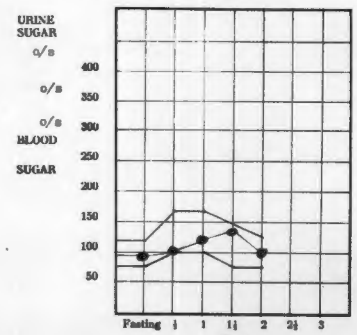
UNIVERSITY OF CAPE TOWN.

Serial No. 38740-41/57 Date 14.11.57

Patient's Identification J. SA. I.A STALDER. (57/10863.)

Ward C 10. Physician or Surgeon Prof. Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR
Fasting 96 mgs.%
½ hour 100 mgs.%
1 hour 124 mgs.%
1½ hour 136 mgs.%
2 hour 100 mgs.%
2½ hour
3 hour

Signature _____

Age 38 years. Habitual abortion: 7 abortions at 6-14 weeks. D&C: 3 days premenstrually - normal secretory endometrium (B.M.R.+ 10).

15.

Report from Pathological Department.

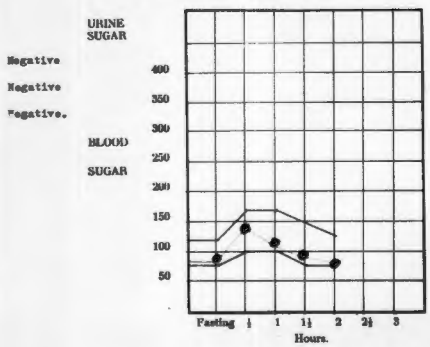
UNIVERSITY OF CAPE TOWN.

Serial No. 8389-99/-59 Date 19.3.59.

Patient's Identification HERNIA CANCRINUS. (56/13160).

Ward C 10. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR
Fasting 80 mgs.%
½ hour 137 mgs.%
1 hour 112 mgs.%
1½ hour 95 mgs.%
2 hour 75 mgs.%
2½ hour
3 hour

Signature _____

Age 39 years. C/O Menorrhagia. D&C - Normal secretory endometrium- Fibroids size 14 weeks - Hysterectomy done.

16.

Report from Pathological Department.

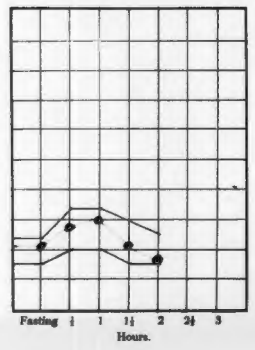
UNIVERSITY OF CAPE TOWN.

Serial No. 8553-54 Date 19.3.58.

Patient's Identification LENA FORTUIN. (56/06066).

Ward A9. Physician or Surgeon Prof. Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR
Fasting 101 mgs.%
½ hour 135 mgs. %
1 hour 151 mgs.%
1½ hour 103 mgs.%
2 hour 82 mgs.%
2½ hour
3 hour

Signature _____

Age 38 years. C/O Menorrhagia. D&C and Hysterectomy - Fibroids, Normal secretory endometrium.

17.

Report from Pathological Department.

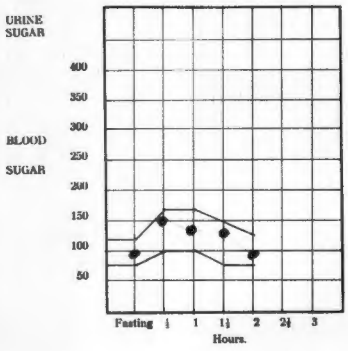
UNIVERSITY OF CAPE TOWN.

Serial No. 1132-33/58 Date 13.1.58.

Patient's Identification ROSIE TROUT (20894).

Ward A9. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting 8.15: 98 mgs.%.
8.45. 151 mgs.%.
9.15. 131 mgs.%.
9.45. 131 mgs.%.
10.15. 96 mgs.%.
3 hour

Signature

Age 42 years. C/O Menorrhagia - Fibroids - normal secretory endometrium.

18.

Report from Pathological Department.

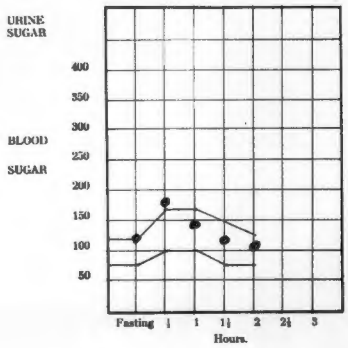
UNIVERSITY OF CAPE TOWN.

Serial No. 24934-35 Date 4.8.57.

Patient's Identification GEORGINA VAN DER SCHYFF. (249033)

Ward A 9. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting 122 mgs.%.
½ hour 170 mgs.%.
1 hour 144 mgs.%.
1½ hour 122 mgs.%.
2 hour 115 mgs.%.
2½ hour
3 hour

Signature

Age 43 years. C/O Menorrhagia: Obese : Normal secretory endometrium.

19.

Report from Pathological Department.

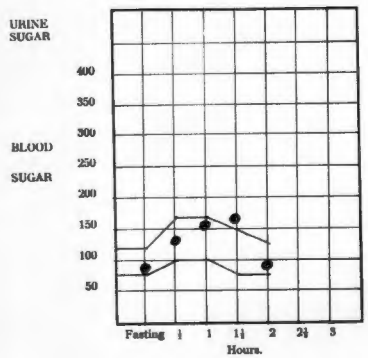
UNIVERSITY OF CAPE TOWN.

Serial No. 28213 - 15. Date 4.9.56.

Patient's Identification GAYA OSMAN (56/00244)

Ward A9 Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting 87 mgs.%.
½ hour 131 mgs.%.
1 hour 162 mgs.%.
1½ hour 171 mgs.%.
2 hour 86 mgs.%.
2½ hour
3 hour

Signature

Age 42 years. Menorrhagia ++: 4 months. D&C 23rd. day of cycle. Normal secretory endometrium.

20.

Report from Pathological Department.

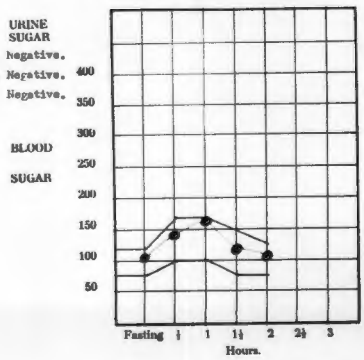
UNIVERSITY OF CAPE TOWN.

Serial No. 7569 - 70 Date 17.5.59

Patient's Identification ZOLA JOBS (57/10205)

Ward A9 Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting 101 mgs.%.
½ hour 147 mgs.%.
1 hour 167 mgs.%.
1½ hour 125 mgs.%.
2 hour 111 mgs.%.
2½ hour
3 hour

Signature D. Louw.

Age 40 years. C/O irregular menstruation 3-10/30 - 60 days. 17/6/59 D&C: Secretory endometrium indicating that ovulation has occurred. Hysterectomy 22/7/59: Normal endometrium.

21.

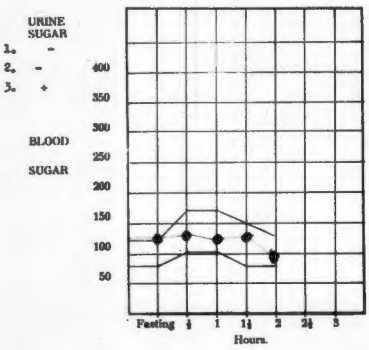
Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

Serial No. 21360 - 1. Date 25.6.59.

Patient's Identification HELEN LINDLEY (95408)

Ward A10. Physician or Surgeon Dr. Benjamin.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting	129 mgs.%.
½ hour	132 mgs.%.
1 hour	125 mgs.%.
1½ hour	128 mgs.%.
2 hour	91 mgs.%.
2½ hour	
3 hour	

Signature L. ANSTEY.

Age 40 years. C/O Polymenorrhoea and menorrhagia : 4 years. 7/2/58 Hysterectomy: Cervicitis; normal proliferative endometrium on the 11th. day of cycle (normal uterus).

22.

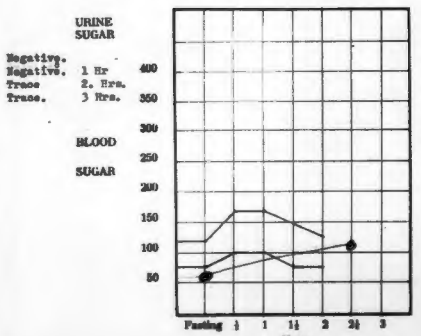
Report from Pathological Department.
UNIVERSITY OF CAPE TOWN.

Serial No. 3720-29 Date 6.2.59

Patient's Identification MRS. P. KENNEDY. (58/01469)

Ward C 10 Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting	77 mgs.%.
½ hour	
1 hour	
1½ hour	
2 hour	
2½ hour	123 mgs.%.
3 hour	

Signature

Age 38 years. C/O Menorrhagia for 2 years - had sterilisation 15/5/59 D&C: 1 month after L.V.P. - " Proliferative endometrium with no secretion; glands however are not hyperplastic". 29/8/59 Hysterectomy: normal premenstrual endometrium.

23.

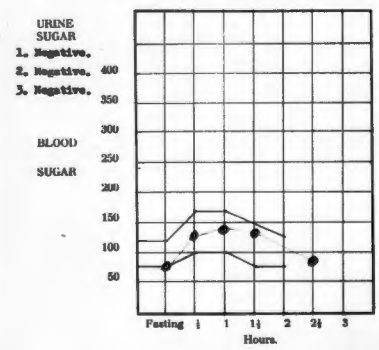
Report from Pathological Department.
UNIVERSITY OF CAPE TOWN.

Serial No. 1754 - 5. Date 28.5.59

Patient's Identification MRS. T. RAMSEW. (59/01852)

Ward C10. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting	71 mgs.%.
½ hour	134 mgs.%.
1 hour	144mgs.%.
1½ hour	142 mgs.%.
2 hour	Insufficient
2½ hour	78 mgs.%.
3 hour	

Signature J. J. V. D. WALT

Age 43 years. C/O Polymenorrhoea and menorrhagia : 3 years. 1/28 - 8/20 day type. 27/6/59 D&C: Normal secretory endometrium.

24.

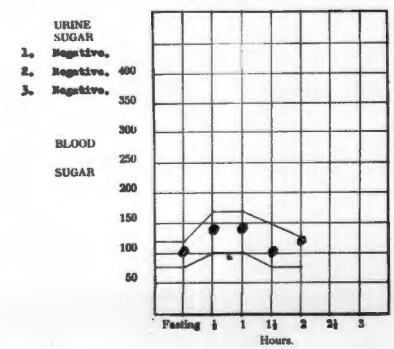
Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

Serial No. 24878 Date 29.7.59.

Patient's Identification JOSEPHINE WEIR NS (304783)

Ward C.10. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting	104 mgs.%.
½ hour	146 mgs.%.
1 hour	146 mgs.%.
1½ hour	102 mgs.%.
2 hour	132 mgs.%.
2½ hour	
3 hour	

Signature G. M. POTGIETER

Age 38 years. C/O Progressive menorrhagia: 2 years. Secretory endometrium.

25.

Report from Pathological Department.

UNIVERSITY OF CAPE TOWN.

Serial No. 5529-30 Date 20.2.58

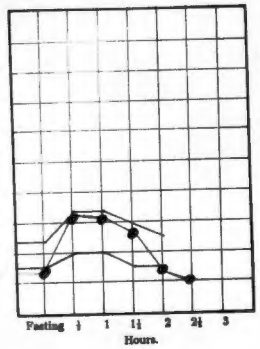
Patient's Identification JULIA UORLI (58/03063)

Ward C 10 Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR
1. No Sugar.
2. " "
3. " "

BLOOD SUGAR



BLOOD SUGAR	
Fasting	68 mgs.%.
½ hour	153 mgs.%.
1 hour	152 mgs.%.
1½ hour	136 mgs.%.
2 hour	79 mgs.%.
2½ hour	50 mgs.%.
3 hour	

Signature

Age 35 years. Menorrhagia. DAC 24/6/59: Normal endometrium.

26.

Report from Pathology Department.

(Chemical Pathology).

UNIVERSITY OF CAPE TOWN.

Serial No. 20631 - 2. Date 22.6.59.

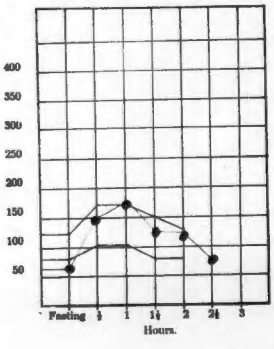
Patient's Identification V. KIRSCHNER.

Ward Physician or Surgeon Dr. Benjamin.

GLUCOSE TOLERANCE TEST.

URINE SUGAR
1. -
2. -
3. -

BLOOD SUGAR



BLOOD SUGAR	
Fasting	99 mgs.%.
½ hour	150 mgs.%.
1 hour	179 mgs.%.
1½ hour	135 mgs.%.
2 hour	12[illegible] mgs.%.
2½ hour	77 mgs.%.
3 hour	

J. J. V. D. WALT

Signature

Age 43 years. C/O Menorrhagia for 8 months. O/E Nervous; blood pressure 170/110; Hb 10.5 G. Uterus and adnexae normal, but erosion and polyp. D&C and removal of polyp 20/10/59.: Adenomatous polyp; normal curettings.

27.

Report from Pathology Department.

(Chemical Pathology).

UNIVERSITY OF CAPE TOWN.

Serial No. 20866 - 867 Date 30.11.59.

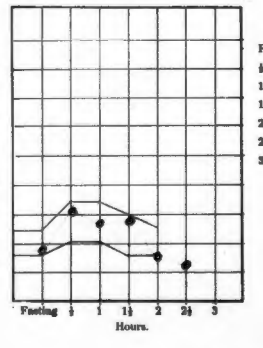
Patient's Identification WINIFRED PETERSEN (26/12468)

Ward A30. Physician or Surgeon Prof. Louw.

GLUCOSE TOLERANCE TEST.

Test-tube	URINE SUGAR
1.	0 trace.
2.	0 0
3.	trace +

BLOOD SUGAR



BLOOD SUGAR	
Fasting	80 mgs.%.
½ hour	138 mgs.%.
1 hour	138 mgs.%.
1½ hour	130 mgs.%.
2 hour	75 mgs.%.
2½ hour	63 mgs.%.
3 hour	

G. M. POTGIETER

Signature

Age 36 years. Normal menstruation. L.M.P. 11/7/59 . Glucose Tolerance test on 17th. day of cycle - day after marked basal temperature rise.

28.

Report from Pathology Department.

(Chemical Pathology).

UNIVERSITY OF CAPE TOWN.

Serial No. 21994 - 5. Date 27.7.59.

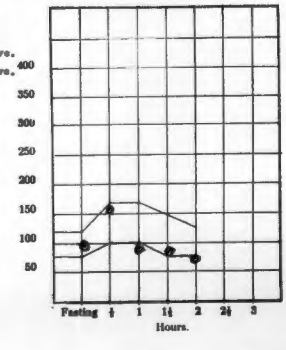
Patient's Identification V. D. VILLIERS (59/691801)

Ward A.10. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR
1. Negative.
2. Negative.

BLOOD SUGAR



BLOOD SUGAR	
Fasting	100 mgs.%.
½ hour	161 mgs.%.
1 hour	90 mgs.%.
1½ hour	81 mgs.%.
2 hour	72 mgs.%.
2½ hour	
3 hour	

G. M. POTGIETER

Signature

Age 36 years. Normal menstruation. L.N.P. 11/7/59. Glucose Tolerance test on 10th. day of cycle - pre-ovulation temperature level.

29.

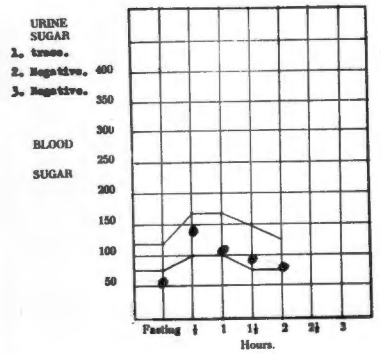
Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

Serial No. 2401 - 02 Date 22.7.59.

Patient's Identification V. DE VILLIERS. (59/1801)

Ward A11. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR
Fasting 63 mgs.%.
½ hour 247 mgs.%.
1 hour 137 mgs.%.
1½ hour Insufficient.
2 hour 82 mgs.%.
2½ hour
3 hour

Signature [illegible]

Age 35 years. Habitual abortion. D&C 20/1/59. : Premenstrual endometrium.

30.

Report from Pathology Department. 369/59.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

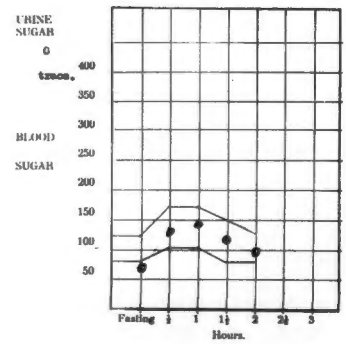
Serial No. 3868 - 869 Date 28.11.59.

Patient's Identification [illegible] (59/0945)

Ward A10. Physician or Surgeon Dr. Benjamin

GLUCOSE TOLERANCE TEST.

Test-tube	URINE SUGAR
1.	0
2.	+



BLOOD SUGAR
Fasting 70 mgs.%.
½ hour 134 mgs.%.
1 hour 148 mgs.%.
1½ hour 115 mgs.%.
2 hour 99 mgs.%.
2½ hour Insufficient.
3 hour

Signature G. M. POTGIETER

Age 35 years. Normal control. Normal menstruation and fertility.

31.

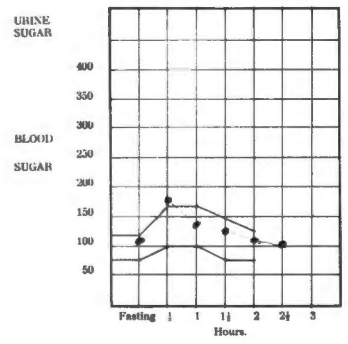
Report from [illegible]
ENDOCRINE RESEARCH LABORATORY.
UNIVERSITY OF CAPE TOWN.

Serial No. [illegible] Date 1959 1959.

Patient's Identification Mrs. S. [illegible]

Ward A 10. Physician or Surgeon Dr. F. Benjamin

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR
Fasting 96 mgs.%.
½ hour 167 mgs.%.
1 hour 138 mgs.%.
1½ hour 131 mgs.%.
2 hour 114 mgs.%.
2½ hour 105 mgs.%.
3 hour

Signature [illegible]

Age 35 years. Normal control. Normal menstruation and fertility.

32.

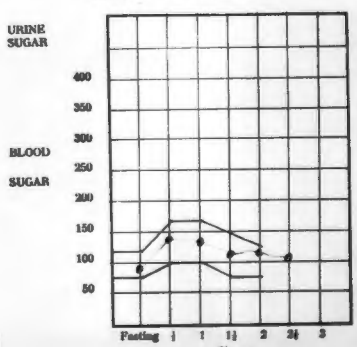
Report from [illegible]
ENDOCRINE RESEARCH LABORATORY.
UNIVERSITY OF CAPE TOWN.

Serial No. [illegible] Date 1959.

Patient's Identification J. [illegible]

Ward A 10. Physician or Surgeon Dr. F. Benjamin

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR
Fasting 92 mgs.%.
½ hour 144 mgs.%.
1 hour 136 mgs.%.
1½ hour 139 mgs.%.
2 hour 122 mgs.%.
2½ hour 101 mgs.%.
3 hour

Signature [illegible]

Age 39 years. Normal control. Normal menstruation and fertility.

33.

Report from Pathological Department.

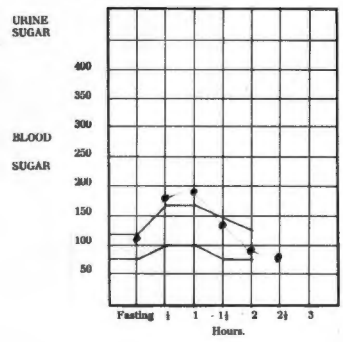
UNIVERSITY OF CAPE TOWN. ENDOCRINE RESEARCH LABORATORY

Serial No. ..... Date 1959.

Patient's Identification E. COHN.

Ward A 10. Physician or Surgeon Dr. F. Benjamin.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting 96 mgs.%.
½ hour 127 mgs.%.
1 hour 178 mgs.%.
1½ hour 144 mgs.%.
2 hour 94 mgs.%.
2½ hour 74 mgs.%.
3 hour

Signature. .....

Age 38 years. Normal control. Normal menstruation and fertility.

34.

Report from Pathological Department.

ENDOCRINE RESEARCH LABORATORY.

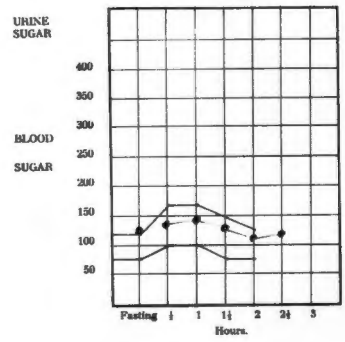
UNIVERSITY OF CAPE TOWN.

Serial No. ..... Date 1959.

Patient's Identification A. ISAAC.

Ward A 10. Physician or Surgeon Dr. F. Benjamin.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting 101 mgs.%.
½ hour 136 mgs.%.
1 hour 140 mgs.%.
1½ hour 109 mgs.%.
2 hour 74 mgs.%.
2½ hour 83 mgs.%.
3 hour

Signature. .....

Age 36 years. Normal control. Normal menstruation and fertility.

35.

Report from Pathological Department.

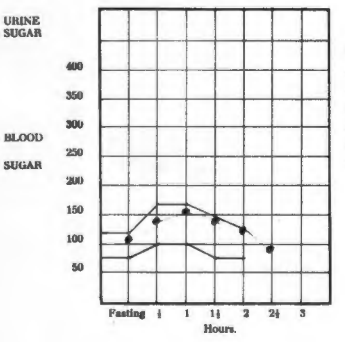
UNIVERSITY OF CAPE TOWN.

Serial No. ..... Date 1959.

Patient's Identification J. FAHN.

Ward A 10. Physician or Surgeon Dr. F. Benjamin.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting 91 mgs.%.
½ hour 144 mgs.%.
1 hour 151 mgs.%.
1½ hour 144 mgs.%.
2 hour 131 mgs.%.
2½ hour 96 mgs.%.
3 hour

Signature. .....

Age 40 years. Normal control. Normal menstruation and fertility.

36.

Report from Pathological Department.

UNIVERSITY OF CAPE TOWN.

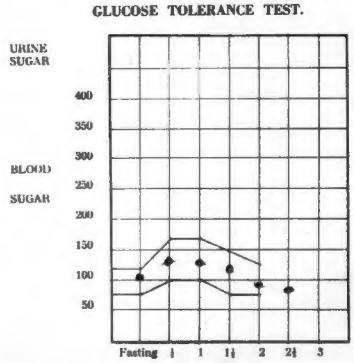
ENDOCRINE RESEARCH LABORATORY.

Serial No. ..... Date 1959.

Patient's Identification A. SITES.

Ward A 10. Physician or Surgeon Dr. F. Benjamin.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting 83. mgs.%.
½ hour 135
1 hour 133
1½ hour 126
2 hour 94
2½ hour 65.
3 hour

Signature. .....

Age 35 years. Normal control. Normal menstruation and fertility.

37.

Report from Pathological Department.

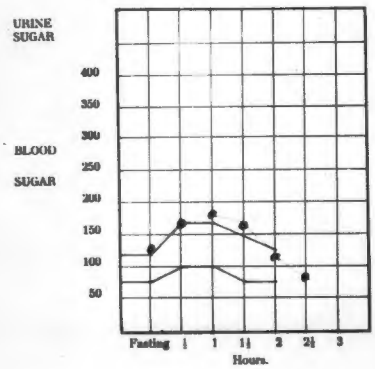
UNIVERSITY OF CAPE TOWN.

Serial No. ____ Date 1939

Patient's Identification A. ISAACS.

Ward A 20 Physician or Surgeon Dr F. Benjamin.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting 106 mgs.%.
½ hour 169
1 hour 176
1½ hour 160
2 hour 86
2½ hour 71.
3 hour

Signature ____

Age 38 years. Control : Normal secretory endometrium.

38.

Report from Pathology Department.

(Chemical Pathology).

UNIVERSITY OF CAPE TOWN.

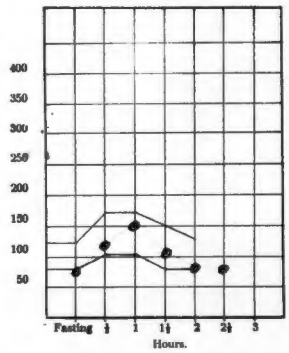
Serial No. 31500 - 501. Date 14.8.39.

Patient's Identification GLORIA YAWA (806/39)

Ward ____ Physician or Surgeon Dr. Bunn (MRC)

GLUCOSE TOLERANCE TEST.

URINE SUGAR
1. -
2. -
3. trace.



BLOOD SUGAR

Fasting 77 mgs.%.
½ hour 121 mgs.%.
1 hour 154 mgs.%.
1½ hour 112 mgs.%.
2 hour 79 mgs.%.
2½ hour 75 mgs.%.
3 hour

Signature L. ANSTEY

Age 36 years. Control : Normal secretory endometrium.

39.

Report from Pathology Department.

(Chemical Pathology).

UNIVERSITY OF CAPE TOWN.

Serial No. 41090 - 91. Date 24.11.39.

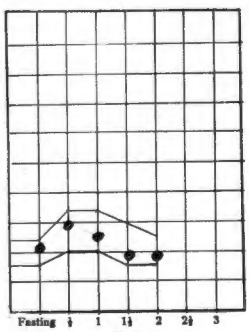
Patient's Identification MRS. [illegible] (973/39)

Ward ____ Physician or Surgeon Dr. Bunn (MRC)

GLUCOSE TOLERANCE TEST.

Test-tube
1. Neg.
2. Neg.
3. Neg.

URINE SUGAR
+
+
+



BLOOD SUGAR

Fasting 120 mgs.%.
½ hour 150 mgs.%.
1 hour 127 mgs.%.
1½ hour 93 mgs.%.
2 hour 95 mgs.%.
2½ hour
3 hour

Signature G. M. POTGIETER

Age 37 years. Control: Normal secretory endometrium.

40.

Report from Pathology Department.

(Chemical Pathology).

UNIVERSITY OF CAPE TOWN.

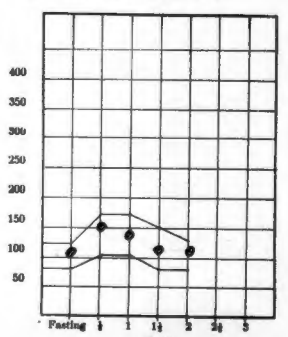
Serial No. 31953 - 54. Date 25.9.39.

Patient's Identification MRS. FLORENCE GROOP. (786)

Ward ____ Physician or Surgeon Dr. Bunn (MRC)

GLUCOSE TOLERANCE TEST.

URINE SUGAR
1. +
2. -
3. -



BLOOD SUGAR

Fasting 111 mgs.%.
½ hour 158 mgs.%.
1 hour 137 mgs.%.
1½ hour 112 mgs.%.
2 hour 116 mgs.%.
2½ hour
3 hour

Signature G. M. POTGIETER

Age 38 years. Control; obese: Normal secretory endometrium.

41.

Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

Serial No. 3585 - 86 Date

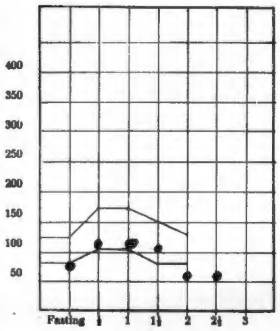
Patient's Identification MRS. [redacted] (953/39)

Ward Physician or Surgeon Dr. Bunt (MED)

GLUCOSE TOLERANCE TEST.

Ten-hour	URINE SUGAR
-	trace.
-	trace.
-	trace.

BLOOD SUGAR



BLOOD SUGAR	
Fasting	78 mgm.%.
½ hour	139 mgm.%.
1 hour	115 mgm.%.
1½ hour	109 mgm.%.
2 hour	60 mgm.%.
2½ hour	60 mgm.%.
3 hour	

Signature G. M. POTGIETER

Age 39 years. Control: Normal secretory endometrium.

42.

Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

Serial No. 3798 - 799 Date 17.11.39

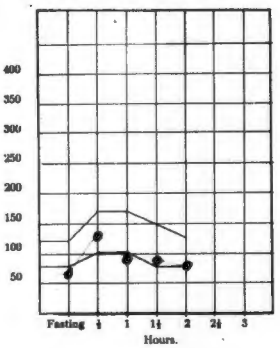
Patient's Identification MRS. C. [redacted] (963)

Ward Physician or Surgeon Dr. Bunt (MED)

GLUCOSE TOLERANCE TEST.

Ten-hour	URINE SUGAR
1. 0	0
2. 0	0
3. 0	0

BLOOD SUGAR



BLOOD SUGAR	
Fasting	67 mgm.%.
½ hour	138 mgm.%.
1 hour	92 mgm.%.
1½ hour	92 mgm.%.
2 hour	79 mgm.%.
2½ hour	
3 hour	

Signature

Age 35 years. Control; weight 234 lbs. : Normal secretory endometrium.

43.

Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

Serial No. 4033 - 34. Date

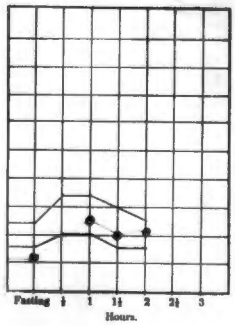
Patient's Identification MRS. [redacted] (MED)

Ward Physician or Surgeon Dr. Bunt (MED)

GLUCOSE TOLERANCE TEST.

Ten-hour	URINE SUGAR
1. 0	0
2. 0	0
3. 0	0

BLOOD SUGAR



BLOOD SUGAR	
Fasting	96 mgm.%.
½ hour	[illegible]
1 hour	215 mgm.%.
1½ hour	208 mgm.%.
2 hour	[illegible]
2½ hour	[illegible]
3 hour	

Signature G. M. POTGIETER

Age 36 years. Control; menstruation 5/28 day type. [illegible] Normal secretory endometrium.

44.

Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

Serial No. 36099 - 100. Date

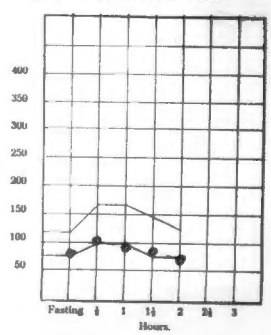
Patient's Identification [redacted] (MED).

Ward OPD Physician or Surgeon [illegible]

GLUCOSE TOLERANCE TEST.

Ten-hour	URINE SUGAR
1. 0	+
2. 0	+
3. 0	+

BLOOD SUGAR



BLOOD SUGAR	
Fasting	75 mgm.%.
½ hour	201 mgm.%.
1 hour	93 mgm.%.
1½ hour	86 mgm.%.
2 hour	73 mgm.%.
2½ hour	
3 hour	

Signature L. ANSTEY

Age 39 years. Control Menstruation ... regular ... endometrium.

45.

Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

Serial No. 4680 - 39. Date ...

Patient's Identification ...

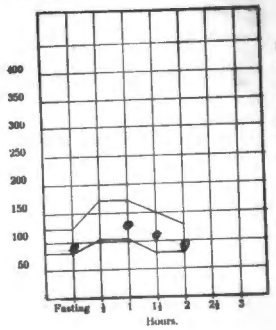
Ward ... Physician or Surgeon ...

GLUCOSE TOLERANCE TEST.

Test-hour	URINE SUGAR
1. 0	+
2. 0	0
3. 0	+

BLOOD SUGAR

Fasting	85 mgs%.
½ hour	Clotted.
1 hour	139 mgs%.
1½ hour	111 mgs%.
2 hour	89 mgs%.
2½ hour	
3 hour	



Signature G. M. POTGIETER

Age 40 years. Menstruation regular 5 days. Normal ... endometrium.

46.

Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

Serial No. 4695 - 39 Date ...

Patient's Identification ...

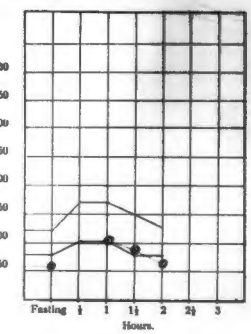
Ward OPD Physician or Surgeon ...

GLUCOSE TOLERANCE TEST.

Test-hour	URINE SUGAR
1.	Negative.
2.	Negative.
3.	Negative

BLOOD SUGAR

Fasting	81 mgs%.
½ hour	Partially clotted.
1 hour	155 mgs%.
1½ hour	88 mgs%.
2 hour	66 mgs%.
2½ hour	
3 hour	



Signature G. M. POTGIETER

Age 37 years. Control Menstruation regular 3-4 days. ... secretory endometrium.

47.

Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

Serial No. 4682 - 39. Date ...

Patient's Identification ...

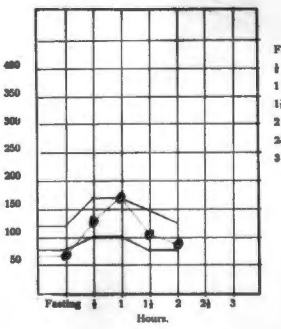
Ward OPD Physician or Surgeon ...

GLUCOSE TOLERANCE TEST.

Test-hour	URINE SUGAR
1.	+
2.	++
3.	++

BLOOD SUGAR

Fasting	63 mgs%.
½ hour	127 mgs%.
1 hour	170 mgs%.
1½ hour	126 mgs%.
2 hour	83 mgs%.
2½ hour	
3 hour	



Signature G. M POTGIETER

Age 36 years. Menstruation regular 5 days. Normal ... endometrium.

48.

Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

Serial No. 4696 - 39 Date ...

Patient's Identification ...

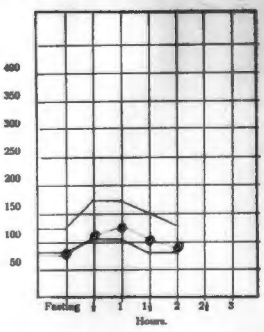
Ward OPD Physician or Surgeon ...

GLUCOSE TOLERANCE TEST.

Test-hour	URINE SUGAR
1.	+
2.	++
3.	+++

BLOOD SUGAR

Fasting	77 mgs%.
½ hour	135 mgs%.
1 hour	187 mgs%.
1½ hour	160 mgs%.
2 hour	83 mgs%.
2½ hour	
3 hour	



Signature G. M POTGIETER

Age 36 years. Menstruation regular. Normal secretory endometrium.
Control. [illegible] HOSPITAL.

44.

Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

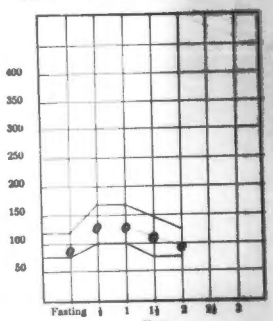
Serial No. 3,386 - 7 Date 5.10.59.

Patient's Identification GLADYS [illegible] (COL)

Ward OPD Physician or Surgeon [illegible]

GLUCOSE TOLERANCE TEST.

Specimens	URINE SUGAR
1.	trace.
2.	trace.
3.	trace.



BLOOD SUGAR	
Fasting	80 mgs%.
½ hour	139 mgs%.
1 hour	185 mgs%.
1½ hour	134 mgs%.
2 hour	97 mgs%.
2½ hour	
3 hour	

G. M. POTGIETER
Signature

Age 39 years. Menstruation normal 28/5 regular. Normal secretory endometrium. CARCINOMA.

50.

Report: Chronic [illegible]

Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

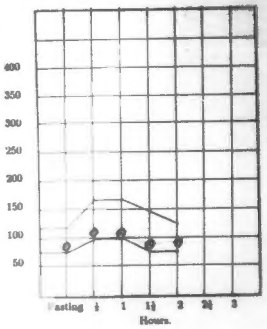
Serial No. 3,384 - 5. Date 5.10.59.

Patient's Identification MRS. JOSINA [illegible] (COL)

Ward OPD Physician or Surgeon Prof Louw (PAT)

GLUCOSE TOLERANCE TEST.

Specimens	URINE SUGAR
1.	trace.
2.	-
3.	trace.



BLOOD SUGAR	
Fasting	86 mgs%.
½ hour	226 mgs%.
1 hour	224 mgs%.
1½ hour	86 mgs%.
2 hour	90 mgs%.
2½ hour	
3 hour	

G. M. POTGIETER
Signature

TABLE 6. GLUCOSE TOLERANCE IN 50 CONSECUTIVE WOMEN BETWEEN 35 and 45 YEARS OF AGE WITH NORMAL ENDOMETRIA.

Patient No	Initials	Age	Fasting	Hour	Hour	Hours	Hours
A. Group with Diabetes.							
1.	F.I.	39	189	262	246	237	227
B. Group with Mildly Impaired Glucose Tolerance.							
2.	C.C.	39	112	112	137	150	155
3.	G.X.	38	116	125	145	143	163
4.	M.H.	36	103	149	158	163	161
5.	F.D.	38	116		152	134	152
C. Group with normal Glucose Tolerance Curves.							
6.	L.L.	43	91	119	157	137	135
7.	E.J.	42	102	164	130	107	
8.	G.K.	45	104	141	188	141	116
9.	M.D.	40	89	96	107	82	105
10.	C.B.	42	89	117	102	79	75
11.	S.Z.	43	108	127	141	103	124
12.	A.C.	40	84	164	161	90	88
13.	M.H.	36	70	120	142	140	112
14.	S.S.	40	96	100	124	136	100
15.	H.C.	38	80	137	112	95	65
16.	L.F.	39	101	135	151	103	82
17.	R.T.	38	98	151	131	131	96
18.	G.V.	42	122	170	144	122	115
19.	G.O.	43	87	131	162	171	86
20.	R.J.	42	101	147	167	125	111

Patient			Blood Sugar Levels (Mgs.%)				
No.	Initials	Age	Fasting	Hour	Hour	Hours	Hours
21.	H.L.	40	129	132	125	128	91
22.	P.K.	40	77				123
23.	T.H.	38	71	124	144	142	
24.	J.W.	43	104	146	146	102	122
25.	J.M.	38	68	153	152	136	79
26.	V.K.	35	59	150	179	135	114
27.	W.F.	43	88	155	132	139	75
28.	V.D.	36	100	161	90	81	72
29.	V.V.	36	63	147	117		81
30.	H.L.	35	70	134	145	115	99
31.	Z.J.	35	96	167	138	131	114
32.	J.K.	35	92	144	136	119	122
33.	E.K.	39	96	167	171	144	94
34.	A.I.	38	101	136	140	109	74
35.	J.K.	36	91	144	151	144	131
36.	A.S.	40	83	135	133	126	94
37.	A.I.	35	106	169	176	160	86
38.	G.Y.	37	77	121	150	112	79
39.	M.B.	36	110	150	127	93	95
40.	F.G.	37	111	152	137	112	116
41.	M.M.	38	78	119	115	109	60
42.	C.B.	39	67	132	93	92	79
43.	S.R.	35	56		129	102	108
44.	K.R.	36	75	104	93	86	73
45.	M.H.	39	85		129	111	85
46.	F.E.	40	61		104	88	66
47.	M.V.	37	63	127	170	106	83
48.	L.E.	38	77	115	127	108	83
49.	G.S.	36	80	130	126	114	97
50.	J.K.	39	86	116	114	86	90

The results are summarised as follows:

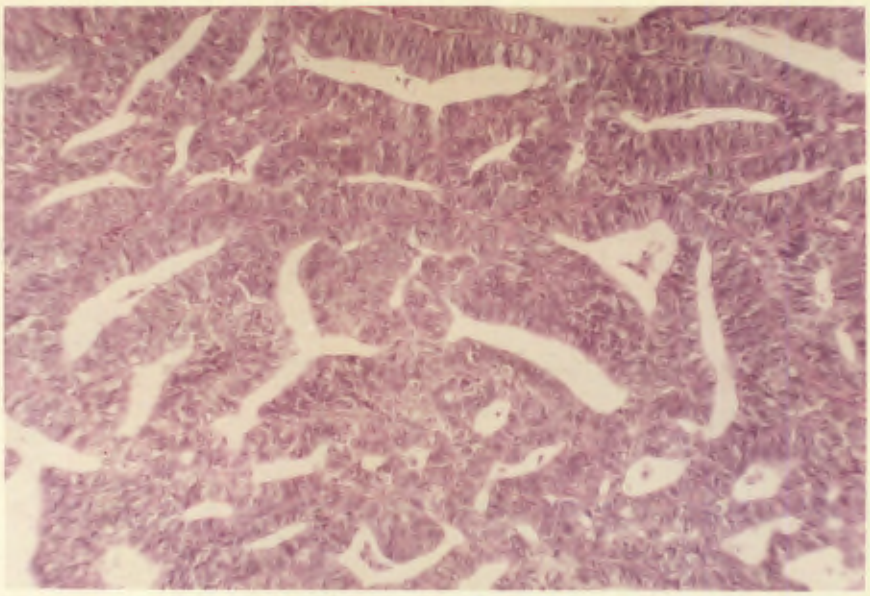
TABLE 7. Glucose Tolerance in 50 consecutive Women, between 35 and 45 years of age, with normal endometria.

Normal Curves	Abnormal Curves		
	Diabetes	Mildly Impaired Glucose Tolerance	Total
90%	2%	8%	10%

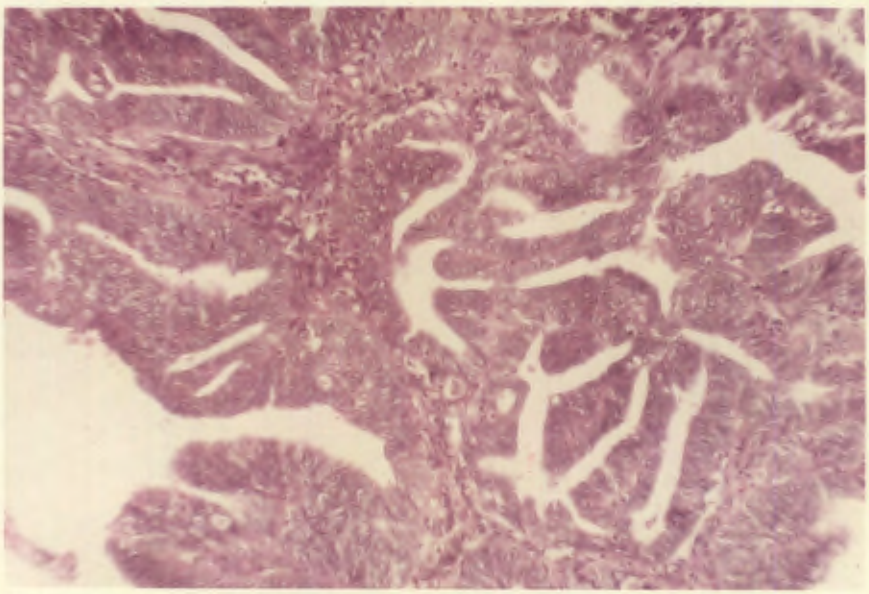
2. CANCER OF THE ENDOMETRIUM.

Under the same conditions, 50 consecutive cases of cancer of the endometrium were investigated by glucose tolerance tests. In order to compare this group with the 100 controls, only those who were 45 years and over were included.

On the following pages are photographs of all the original glucose tolerance curves. Attached to each curve is a summary of the clinical history of the patient. Here then follows Table 8, which presents the details of the blood sugar levels. And finally is shown a summary of the results obtained. (Table 9).



CA 1.



CA 2.

Photomicrograph of curettings in Casel. of the Adenocarcinoma of the Endometrium group.

Age 42 years. D&C 4/11/59: Adenocarcinoma endometrium.

1.

Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

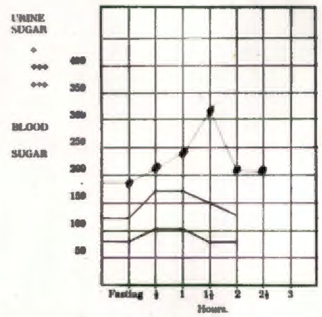
Serial No. 37973 - 7b. Date 3.11.59.

Patient's Identification ACHMAT [illegible] (16.84)

Ward 430. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

Too-tone	URINE SUGAR
1. +	+
2. ++	+++
3. +	+++



BLOOD SUGAR	
Fasting	183 mgm.%.
½ hour	209 mgm.%.
1 hour	238 mgm.%.
1½ hour	300 mgm.%.
2 hour	210 mgm %.
2½ hour	206 mgm.%.
3 hour	

(47.)

Signature G. M. POTGIETER

07740/50/5/50. S.P.

Age 59 years. Post-menopausal bleeding: 1 months. Menopause at 43 years. D&C (fractional): Endometrial carcinoma.

2.

Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

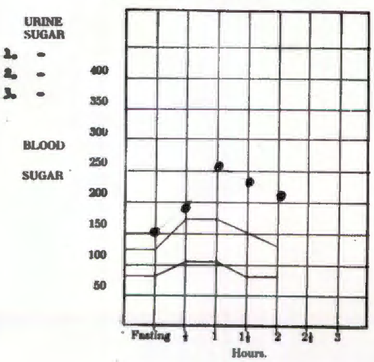
Serial No. 82963 - 6b. Date 29.6.59

Patient's Identification MRS. ELIZABETH LINDENFELDER (59/06540)

Ward C10. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR	
1.	-
2.	-
3.	-



BLOOD SUGAR	
Fasting	130 mgm.%.
½ hour	192 mgm.%.
1 hour	254 mgm.%.
1½ hour	242 mgm.%.
2 hour	220 mgm.%.
2½ hour	
3 hour	

L. ANSTEY.

Signature

07740/50/5/50. S.P.

Age 77 years. C/o Post-menopausal bleeding: 2 months. Menopause at 47 years. 8/6/59 D&C Adenocarcinoma of body of uterus.

3.

Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

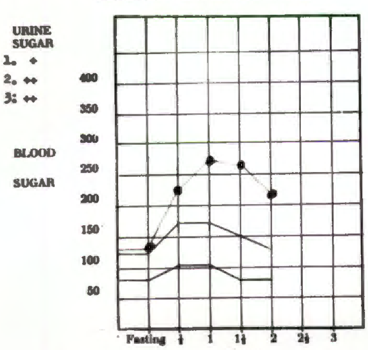
Serial No. 18548 - 9. Date 7.6.59

Patient's Identification MRS. A. VAN ZYL (59/05985)

Ward G10. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR	
1.	+
2.	++
3.	++



BLOOD SUGAR	
Fasting	131 mgm.%.
½ hour	232 mgm.%.
1 hour	273 mgm.%.
1½ hour	265 mgm.%.
2 hour	221 mgm.%.
2½ hour	Clotted.
3 hour	

Signature J. J. V. D. WALT

07740/50/5/50. S.P.

Age 73 years. Menopause at ± 45 years. Adenocarcinoma of body of uterus.

4.

Report from Pathological Department.
UNIVERSITY OF CAPE TOWN.

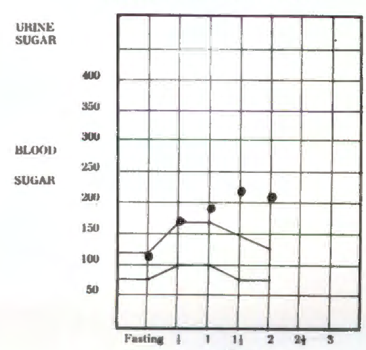
Serial No. 15006/55. Date 27.7.55.

Patient's Identification MARIA BEUKES. (299583.)

Ward B 7. Physician or Surgeon Prof. Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR



BLOOD SUGAR	
Fasting	119.%
½ hour	176 %
1 hour	198 %
1½ hour	228 %
2 hour	221 %
~~2½ hour~~	
~~3 hour~~	

G. LINDER

Signature

2741 Im 5.45. S.P.

Age 63 years. Post-menopausal bleeding: 8 months. Menopause at 50 years. Adenocarcinoma of endometrium. D&C, Radium, Wertheim.

5.

Report from Pathological Department.

UNIVERSITY OF CAPE TOWN.

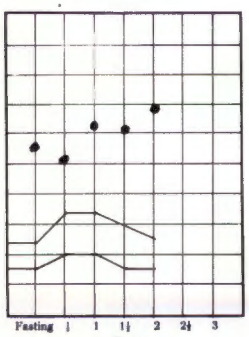
Serial No. 14910-11 Date 18.5.56.

Patient's Identification ALETTA BOTHA (04385)

Ward C 10. Physician or Surgeon Prof. Louw.

GLUCOSE TOLERANCE TEST.

	URINE SUGAR
Fasting	+
60 min.	++
120 min.	+++



	BLOOD SUGAR
Fasting	278 mgs.%
½ hour	258 mgs.%
1 hour	320 mgs.%
1½ hour	308 mgs.%
2 hour	315 mgs.%
2½ hour	346 mgs.%
3 hour	COMA.....

Signature

Age 64 years. Adenocarcinoma of endometrium. Polyp - cervical no malignancy.

6.

Report from Pathological Department.

UNIVERSITY OF CAPE TOWN.

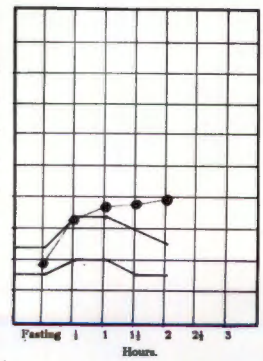
Serial No. 14526 - 527 Date 30.5.59

Patient's Identification MRS. ISOBEL PASTANA . (59/04154)

Ward C10. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR
1. Negative.
2. Trace.
3. +



	BLOOD SUGAR
Fasting	97 mgs.%
½ hour	141 mgs.%
1 hour	163 mgs.%
1½ hour	188 mgs.%
2 hour	197 mgs.%
2½ hour	
3 hour	

Signature L. ANSTEY.

Age 63 years. Post-menopausal bleeding: 9 months. Menopause at 56 years. D&C - adenocarcinoma of endometrium. Treated with Radium.

7.

Report from Pathological Department.

UNIVERSITY OF CAPE TOWN.

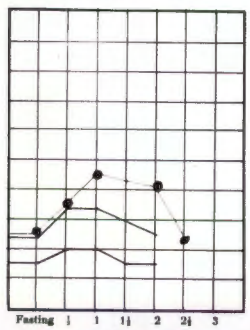
Serial No. 17690 - 91. Date 1.6.59

Patient's Identification MRS. L.E. SMART. (59/05043)

Ward C10. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

	URINE SUGAR Re-test
Immediate	-
1. Negative	-
2. Negative	-
3. Negative	-



	BLOOD SUGAR
Fasting	127 mgs.%.
½ hour	183 mgs.%.
1 hour	289 mgs.%.
1½ hour	234 mgs.%.
2 hour	205 mgs.%.
2½ hour	117 mgs.%.
3 hour	

Signature J. J. V. D. WALT

Age 59 years. C/O post-menopausal bleeding: 4 months. Menopause at 43 years. 1/5/59 D&C Well differentiated adenocarcinoma endometrium.

8.

Report from Pathological Department.

UNIVERSITY OF CAPE TOWN.

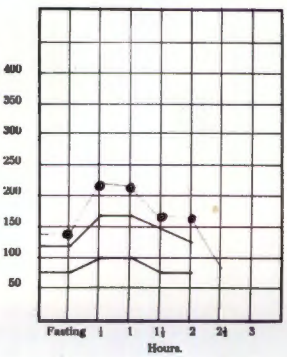
Serial No. 17649 - 50. Date 1.6.59

Patient's Identification MRS. S. GROENEWALD (163572)

Ward C.10. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR
1. Negative.
2. Negative.
3. Negative.



	BLOOD SUGAR
Fasting	139 mgs.%. (Confirmed) 225 mgs.%.
½ hour	
1 hour	217 mgs.%.
1½ hour	163 mgs.%.
2 hour	163 mgs.%.
2½ hour	87 mgs.%. (Confirmed)
3 hour	

Signature J. J. V. D. WALT

Age 61 years. Menopause at 51 years. D&C 12/7/58: Adenocarcinoma of endometrium.

9.

Report from Pathological Department.

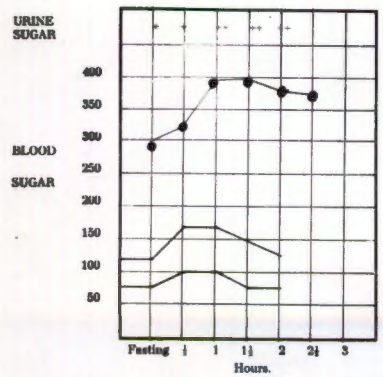
UNIVERSITY OF CAPE TOWN.

Serial No. 24527-28 Date 29.7.58.

Patient's Identification CLAUDINA VAN WYK. (58/0888)

Ward C10. Physician or Surgeon Prof. Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting 295 mgs. %
½ hour 316 mgs. %
1 hour 397 mgs. %
1½ hour 398 mgs. %
2 hour 381 mgs. %
2½ hour 380 mgs. %
3 hour

Signature J. J. V. D. WALT

Age 60 years. Menopause at 55 years. Adenocarcinoma of endometrium.

10.

Report from Pathological Department.

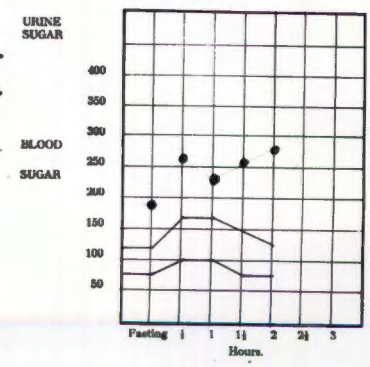
UNIVERSITY OF CAPE TOWN.

Serial No. 23255-6/57 Date 10.7.57

Patient's Identification CHRISTINA VRAAGOM. (57/16783.)

Ward B 7. Physician or Surgeon Prof. Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting 183 mgs. %
½ hour 268 mgs. %
1 hour 230 mgs. %
1½ hour 254 mgs. %
2 hour 276 mgs. %
2½ hour
3 hour

Signature

Age 64 years. Menopause at 45 years. D&C 21/12/56 Adenocarcinoma of endometrium.

11.

Report from Pathological Department.

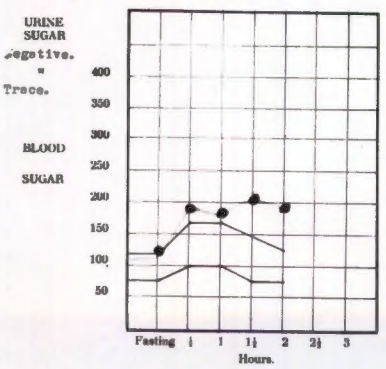
UNIVERSITY OF CAPE TOWN.

Serial No. 15129-30/57 Date 3.5.57

Patient's Identification ELIZABETH KINNEY. (56/32495)

Ward A 9. Physician or Surgeon Prof. Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting 122 mgs. %
½ hour 190 mgs. %
1 hour 187 mgs. %
1½ hour 201 mgs. %
2 hour 199 mgs. %
2½ hour
3 hour

Signature

Age 56 years. Menopause at 49 years. D&C 13/10/56 Adenocarcinoma of endometrium.

12.

Report from Pathological Department.

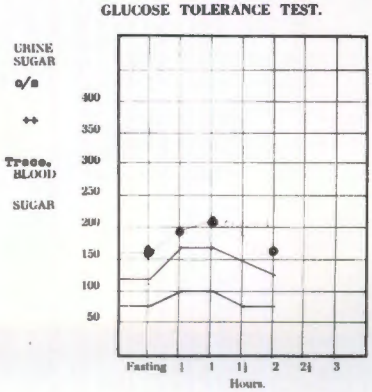
UNIVERSITY OF CAPE TOWN.

Serial No. 30508-9/57 Date 13.9.57

Patient's Identification GERTRUDE WESSELS. (55/11644).

Ward C 10. Physician or Surgeon Prof. Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting 160 mgs. %
½ hour 191 mgs. %
1 hour 213 mgs. %
1½ hour Coagulated.
2 hour 156 mgs%
2½ hour
3 hour

Signature

Age 63 years. Menopause at 50 years. D&C 16/4/57 Carcinoma of endometrium.

13.

Report from Pathological Department.

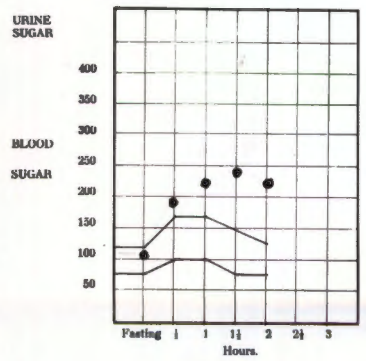
UNIVERSITY OF CAPE TOWN.

Serial No. 13989/57 Date 25.4.57

Patient's Identification CATHERINA WOLFAARD. (57/03707)

Ward C 10. Physician or Surgeon Prof. Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting 116 mgs.%
½ hour 199 mgs.%
1 hour 225 mgs.%
1½ hour 247 mgs.%
2 hour 222 mgs.%
2½ hour
3 hour

Signature

Age 61 years. C/O Postmenopausal bleeding. D&C: Adenocarcinoma of endometrium.

14.

Report from Pathology Department.

(Chemical Pathology).

UNIVERSITY OF CAPE TOWN.

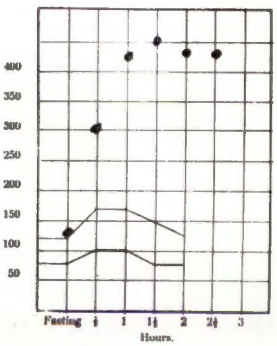
Serial No. 40610 - 611. Date 20.11.59.

Patient's Identification KOOLSUM JACO. S (59/2486)

Ward A10. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

Tes-tape	URINE SUGAR
1.	++++
2.	++++
3.	++++



BLOOD SUGAR

Fasting 126 mgs.%.
½ hour 301 mgs.%.
1 hour 430 mgs.%.
1½ hour 451 mgs.%.
2 hour 436 mgs.%.
2½ hour 436 mgs.%.
3 hour

Signature G M POTGIETER

Age 50 years. Menopause at 48 years. C/O Blood stained post-menopausal discharge: 6 months. D&C 15/10/59 - Wynberg Hospital - De-differintiated carcinoma of the endometrium. Total Hysterectomy 5/11/59.

15.

Report from Pathology Department.

(Chemical Pathology).

UNIVERSITY OF CAPE TOWN.

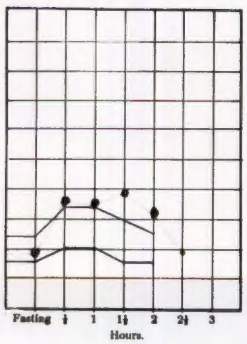
Serial No. 39075 - 6 Date 3.11.59.

Patient's Identification MAY SMITH (59/20948)

Ward A10. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

Tes-tape	URINE SUGAR
1. 0	0
2. 0	trace.



BLOOD SUGAR

Fasting 94 mgs.%.
½ hour 176 mgs.%.
1 hour 172 mgs.%.
1½ hour 192 mgs.%.
2 hour 159 mgs.%.
2½ hour 98 mgs.%.
3 hour

Signature G. M. POTGIETER

Age 64 years. Postmenopausal discharge and bleeding 6 months. Menopause at 49 years. Endometrial carcinoma. Serum Cholesterol 290.

16.

Report from Pathological Department.

UNIVERSITY OF CAPE TOWN.

Serial No. C 23990- 91 Date 14..8.56.

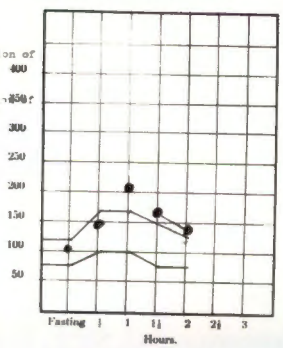
Patient's Identification FATIE GEORGE.

Ward A 9 Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR

1. Very slight reduction of [illegible]
2. o/s
3. o/s
4. o/s
5. o/s



BLOOD SUGAR

Fasting 101 mgs.%.
½ hour 147 mgs.%.
1 hour 208 mgs.%.
1½ hour 163 mgs.%.
2 hour 124 mgs.%.
2½ hour
3 hour

Signature

Age 70 years. Menopause at 47 years. D&C 3/4/58: Adenocarcinoma of endometrium.

17.

Report from Pathological Department.

UNIVERSITY OF CAPE TOWN.

Serial No. 10,397-98 Date 2.4.58.

Patient's Identification TERESSA IAKSY. (58/08237)

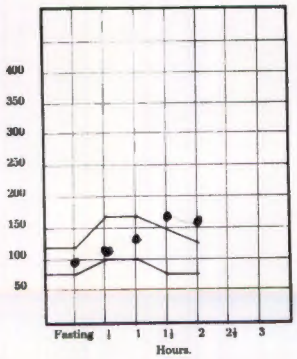
Ward B7 Physician or Surgeon Prof. Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR

I N Sugar
II " "
III " "

BLOOD SUGAR



BLOOD SUGAR	
Fasting	92 mgs.%
¼ hour	119 mgs.%
1 hour	135 mgs.%
1½ hour	169 mgs.%
2 hour	153 mgs.%
2½ hour	
3 hour	

Signature

Age 80 years. Menopause at 50 years. D&C 3/8/57: Adenocarcinoma of endometrium.

18.

Report from Pathological Department.

UNIVERSITY OF CAPE TOWN.

Serial No. 272h203/57 Date 15.8.57

Patient's Identification ANNA DE VILLIERS. (57/07279).

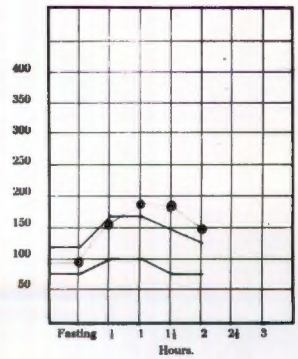
Ward C 10. Physician or Surgeon Dr. Massey.

GLUCOSE TOLERANCE TEST.

URINE SUGAR

Trace.
++
+

BLOOD SUGAR



BLOOD SUGAR	
Fasting	91 mgs.%
9.30 a.m.	157 mgs.%
10 a.m.	189 mgs.%
10.30 a.m.	182 mgs.%
11 a.m.	150 mgs.%
2 hour	
2½ hour	
3 hour	

Signature

Age 72 years. Menopause at 52 years. D&C 30/8/57: Adenocarcinoma of endometrium.

19.

Report from Pathological Department.

UNIVERSITY OF CAPE TOWN.

Serial No. 29133-34/57 Date

Patient's Identification ELIZABETH HORTJE. (57/08273).

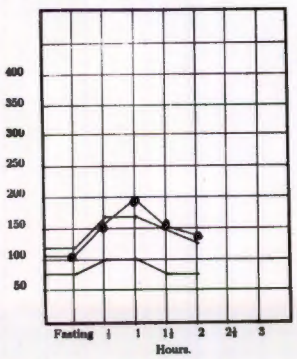
Ward C 10. Physician or Surgeon Prof. Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR

o/s
o/s
o/s

BLOOD SUGAR



BLOOD SUGAR	
Fasting	107 mgs.%
¼ hour	154 mgs.%
1 hour	199 mgs.%
1½ hour	153 mgs.%
2 hour	138 mgs.%
2½ hour	
3 hour	

Signature

Age 75 years. Post-menopausal bleeding: 5 months. Menopause at 47 years. Adenocarcinoma of endometrium. D&C and Radium.

20.

Report from Pathological Department.

UNIVERSITY OF CAPE TOWN.

Serial No. [illegible] 28001-02 Date 31.8.56

Patient's Identification Philidah WILDE.

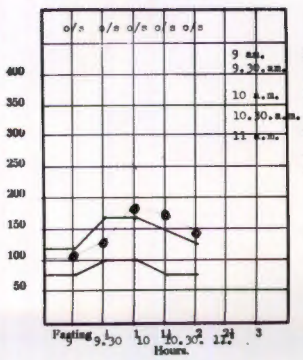
Ward A9 Physician or Surgeon Prof. Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR

o/s o/s o/s o/s o/s

BLOOD SUGAR



BLOOD SUGAR	
Fasting	104 mgs.%
¼ hour	126 mgs.%
1 hour	174 mgs.%
1½ hour	172 mgs.%
2 hour	145 mgs.%
2½ hour	
3 hour	

Signature

Age 75 years. Menopause at 50 years. D&C 15/11/56: Adenocarcinoma of endometrium.

21.

Report from Pathological Department.

UNIVERSITY OF CAPE TOWN.

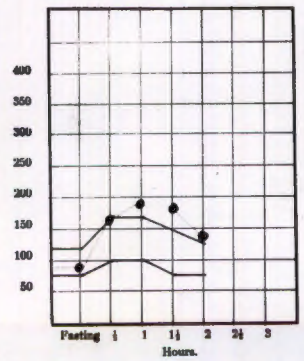
Serial No. C 35422 - 23 Date 21/11/56

Patient's Identification SANNIE TITOS (248078)

Ward A9 Physician or Surgeon Dr Buss.

GLUCOSE TOLERANCE TEST.

URINE SUGAR
1. Negative.
2. Negative.
3. Negative.



BLOOD SUGAR
Fasting 93 mgs.%
½ hour 167 mgs.%
1 hour 192 mgs.%
1½ hour 192 mgs.%
2 hour 131 mgs.%
2½ hour
3 hour

Signature G. LINDER

Age 71 years. Menopause at 53 years. D&C 16/3/56: Adenocarcinoma of endometrium.

22.

Report from Pathological Department.

UNIVERSITY OF CAPE TOWN.

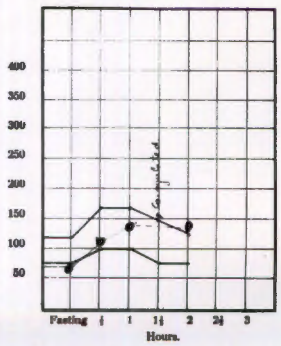
Serial No. C 8568 Date 26/3/56

Patient's Identification LOUISA BASSON

Ward B 7 Physician or Surgeon Prof. Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR
1. : o/s
2. : o/s
3. : o/s



BLOOD SUGAR
Fasting 68 mgs.%
½ hour 107 mgs.%
1 hour 136 mgs.%
1½ hour Coagulated.
2 hour 132 mgs.%
2½ hour
3 hour

Signature

Age 45 years. Post-menopausal bleeding, Menopause 7 months previously. Adenocarcinoma of endometrium.

23.

Report from Pathological Department.

UNIVERSITY OF CAPE TOWN.

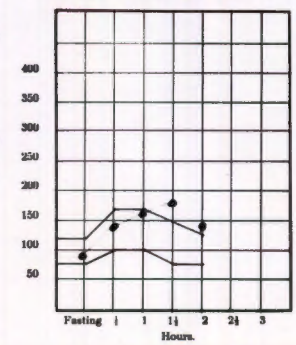
Serial No. C 33848 - 49 Date 8.11.56

Patient's Identification LIEBERT LOUREIRO.

Ward A9 Physician or Surgeon Prof. Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR
1. Negative.
2. Negative.
3. Negative.



BLOOD SUGAR
Fasting 94 mgs.%
½ hour 140 mgs.%
1 hour 165 mgs.%
1½ hour 179 mgs.%
2 hour 140 mgs.%
2½ hour
3 hour

Signature

Age 65 years. Menopause at 50 years. D&C 6/8/56: Adenocarcinoma of endometrium.

24.

Report from Pathological Department.

UNIVERSITY OF CAPE TOWN.

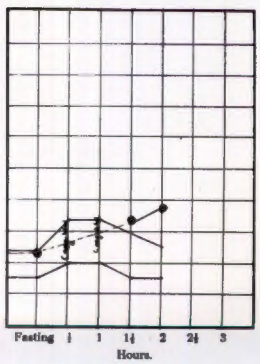
Serial No. 28155-56/57 Date 26.8.57

Patient's Identification SABINA ENGELBRECHT. (57/13225).

Ward A 9. Physician or Surgeon Dr. Buss.

GLUCOSE TOLERANCE TEST.

URINE SUGAR
o/s
+
+



BLOOD SUGAR
Fasting 122 mgs.%
½ hour Coagulated.
1 hour Coagulated.
1½ hour 165 mgs.%
2 hour 188 mgs.%
2½ hour
3 hour

Signature

Age 58 years. Menopause at 54 years. DEC 20/2/57 : Adenocarcinoma of endometrium. Radium and then Wertheim.

25 (a)

Report from Pathological Department.

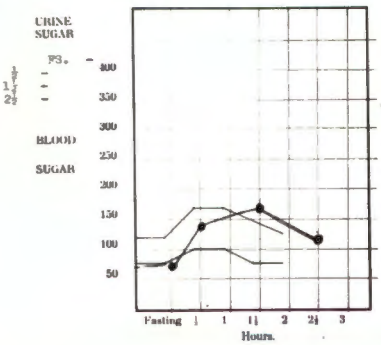
UNIVERSITY OF CAPE TOWN.

Serial No. 5805-6 Date 20.2.57

Patient's Identification CHRISTINA MILLIARD. (96260)

Ward A 3. Physician or Surgeon Dr. Buss.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting	81 mgs.%
½ hour	141 mgs.%
1½ hour	166 mgs.%
2½ hour	128 mgs.%
2 hour	
2½ hour	
3 hour	

Signature

Age 51 years. Intermenstrual bleeding; 2 years. Weight: 175 lbs. Height 5' 10''. D&C 13/10/59: Adenocarcinoma of Endometrium.

26.

Report from Pathology Department.

(Chemical Pathology).

UNIVERSITY OF CAPE TOWN.

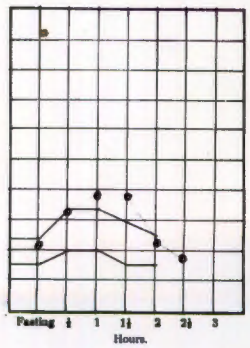
Serial No. 3578 - 79. Date 6.10.59.

Patient's Identification PEGGY BISCHOFF (56/01402)

Ward G10. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

Test-tape	URINE SUGAR
1.	– trace.
2.	– trace.
3.	+ trace.



BLOOD SUGAR

Fasting	103 mgs.%.
½ hour	163 mgs.%.
1 hour	189 mgs.%.
1½ hour	181 mgs.%.
2 hour	124 mgs.%.
2½ hour	75 mgs.%.
3 hour	

Signature G. M. POTGIETER

Age 53 years. Menorrhagia. Adenocarcinoma of endometrium (secondary in round ligament).

27.

Report from Pathological Department.

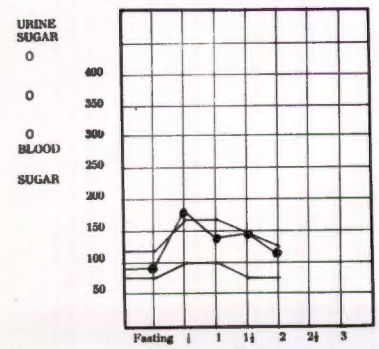
UNIVERSITY OF CAPE TOWN.

Serial No. 27733-34/57 Date 20.8.57

Patient's Identification ELIZABETH MYBURGH. (57/07862).

Ward C 10. Physician or Surgeon Prof. Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting	90 mgs.%
½ hour	180 mgs.%
1 hour	136 mgs.%
1½ hour	145 mgs.%
2 hour	120 mgs.%
2½ hour	
3 hour	

Signature

Age 58 years. Menopause at 52 years. Post-menopausal bleeding: 1 year. D&C Adenocarcinoma of endometrium. Total Hysterectomy.

28.

Report from Pathological Department.

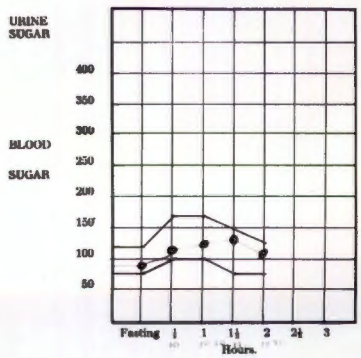
UNIVERSITY OF CAPE TOWN.

Serial No. G. 2306. Date 2.2.55.

Patient's Identification Mrs. Helen Payne.

Ward C 10. Physician or Surgeon Prof Louw

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting	90 mgs.%
½ hour 10.0	128 mgs.%
1 hour 10.30.	138 mgs.%
1½ hour 11.0.	144 mgs.%
2 hour 11.30.	111 mgs.%
2½ hour	
3 hour	

Signature

Age 49 years. Oligo-amenorrhoea and menorrhagia. D&C Adenocarcinoma of endometrium. Obesity. Radium and Wertheim p no residual carcinoma.

29.

Report from Pathological Department.

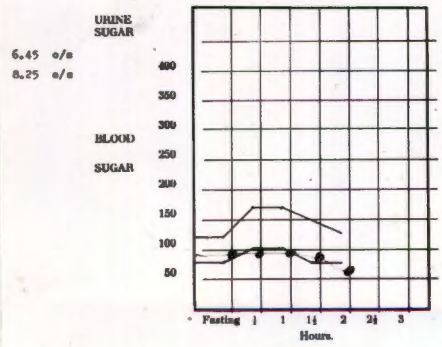
UNIVERSITY OF CAPE TOWN.

Serial No. 6907 Date 12.3.56.

Patient's Identification SARIEKA SOLKER

Ward A9 Physician or Surgeon Prof. Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting 93 mgs.%.
½ hour 93 mgs.%.
1 hour 99 mgs.%.
1½ hour 93 mgs.%.
2 hour 79 mgs.%.
2½ hour
3 hour

Signature

Age 59 years. Menopause at 45 years. D&C 21/10/57: Adenocarcinoma of endometrium.

30.

Report from Pathological Department.

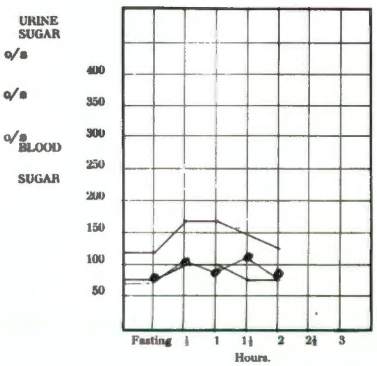
UNIVERSITY OF CAPE TOWN.

Serial No. 3443-74/57 Date 15.10.57

Patient's Identification MARTHA BOTHA. (170491.)

Ward C 10. Physician or Surgeon Prof. Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting 72 mgs.%
½ hour 105 mgs.%
1 hour 85 mgs.%
1½ hour 112 mgs.%
2 hour 76 mgs.%
2½ hour
3 hour

Signature

Age 80 years (thin woman). Menopause at 54 years. C/O post-menopausal bleeding: 2 months. D&C 30/6/59. Papillary Adeno-carcinoma of endometrium.

31.

Report from Pathology Department.

(Chemical Pathology).

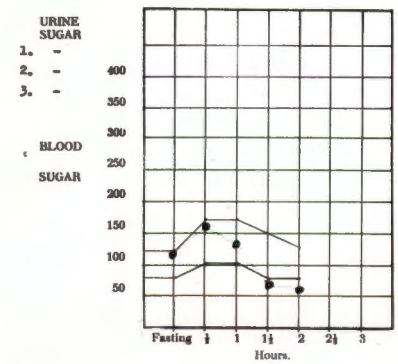
UNIVERSITY OF CAPE TOWN.

Serial No. 2362 - 3. Date 26.6.59.

Patient's Identification ADELAIDE BERNHARDT (59/06527)

Ward C10. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting 118 mgs.%.
½ hour 157 mgs.%.
1 hour 138 mgs.%.
1½ hour 65 mgs.%.
2 hour 61 mgs.%.
2½ hour
3 hour

L. ANSTEY.

Signature

Age 79 years. Post-menopausal bleeding. Adenocarcinoma of endometrium.

32.

Report from Pathological Department.

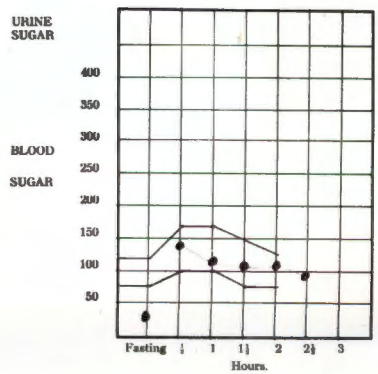
UNIVERSITY OF CAPE TOWN.

Serial No. 388-89/58 Date 7.1.58.

Patient's Identification ANGELIK COETZEE. (56/14272).

Ward C10. Physician or Surgeon Prof.Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting 42 mgs.%
½ hour 142 mgs.%
1 hour 121 mgs.%
1½ hour 117 mgs.%
2 hour 117 mgs.%
2½ hour 93 mgs.%
3 hour

Signature

Age 76 years. Menopause at 63 years. D&C 11/9/57: Papillary Adenocarcinoma of endometrium.

33.

Report from Pathological Department.

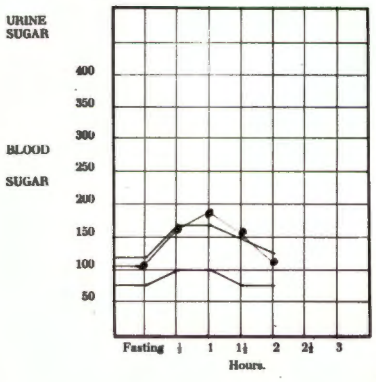
UNIVERSITY OF CAPE TOWN.

Serial No. 29989-90/57 Date 10.9.57

Patient's Identification FREDERIKA STEPHAN. (131803).

Ward 1. C/P Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting 110 mgs.%
½ hour 162 mgs.%
1 hour 183 mgs.%
1½ hour 156 mgs.%
2 hour 117 mgs.%
2½ hour
3 hour

Signature

Age 53 years. (Menstruating until 53 years) Adenocarcinoma of endometrium.

34

Report from Pathological Department.

UNIVERSITY OF CAPE TOWN.

Serial No. 32081-2/57 Date 24.9.57.

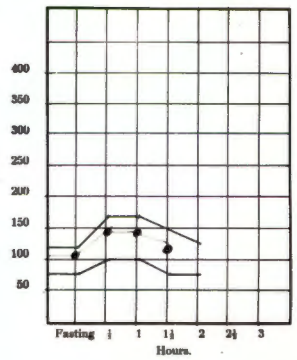
Patient's Identification JANAP ELY (5702205)

Ward A9. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR

Urines Negative.



BLOOD SUGAR

Fasting 107 mgs.%
½ hour 147 mgs.%
1 hour 146 mgs.%
1½ hour 119 mgs.%
2 hour
2½ hour
3 hour

Signature

Age 57 years. Menopause at 54 years. Adenocarcinoma of endometrium.

35.

Report from Pathological Department.

UNIVERSITY OF CAPE TOWN.

Serial No. 33521-22/57 Date 7.10.57.

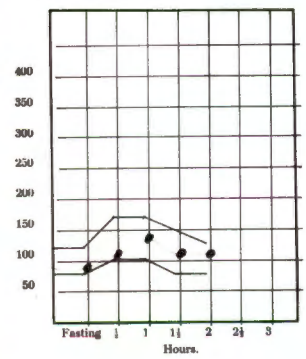
Patient's Identification ANNIE HARVE. (57/09487)

Ward C 10. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR

Urine 1. Negative.
" 2. Negative.



BLOOD SUGAR

Fasting 82 mgs.%
½ hour 107 mgs.%
1 hour 139 mgs.%
1½ hour 119 mgs.%
2 hour 119 mgs.%
2½ hour
3 hour

Signature

Age 51 years. Irregular menstruation and intermenstrual bleeding, sometimes profuse, sometimes scanty. D&C: Adenocarcinoma of endometrium. Radium and Wertheim.

36

Report from Pathological Department.

UNIVERSITY OF CAPE TOWN.

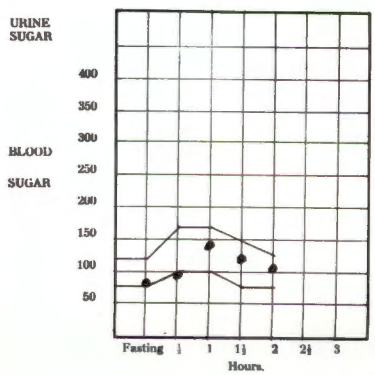
Serial No. C 13236 - 37 Date 8.5.56.

Patient's Identification MARTHA DE WET. (56/02061)

Ward C 10. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR



BLOOD SUGAR

Fasting 86 mgs.%
½ hour 96 mgs.%
1 hour 144 mgs.%
1½ hour 130 mgs.%
2 hour 107 mgs.%
2½ hour
3 hour

Signature

Age 52 years. Menopause at 47 years. C/O post-menopausal bleeding. D&C 8/6/99: Adenocarcinoma of endometrium.

37.

Body of uterus

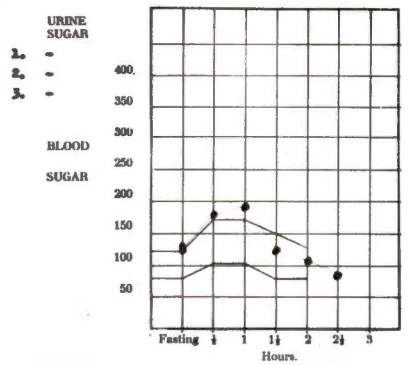
Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

Serial No. 21548 - 49. Date 28.6.99.

Patient's Identification MRS. MARGARIETA M. UITENDOGAARDT (59/05452).

Ward C10. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR	
Fasting	117 mgs.%.
½ hour	183 mgs.%.
1 hour	197 mgs.%.
1½ hour	138 mgs.%.
2 hour	103 mgs.%.
2½ hour	94 mgs.%.
3 hour	

L. ANSTEY.

Signature

Age 52 years. Menopause at 42 years. D&C 25/8/55: Adenocarcinoma of endometrium.

38.

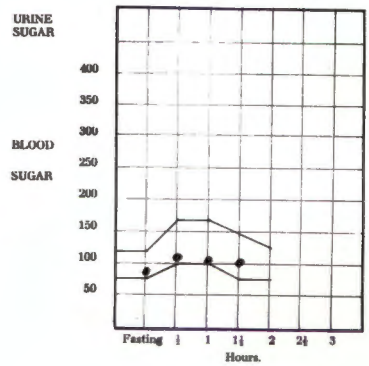
Report from Pathological Department.
UNIVERSITY OF CAPE TOWN.

Serial No. 19765. Date 29. 9. 55.

Patient's Identification MRS. LEMMET. 55/09283.

Ward C10. Physician or Surgeon Prof. James Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR	
Fasting	86 %
½ hour	108.
1 hour	108
1½ hour	105.
2 hour	100.
2½ hour	
3 hour	

Signature

Age 50 years. C/O Menorrhagia and bleeding: 1 year. D&C 19/4/55. Adenocarcinoma fo endometrium.

39

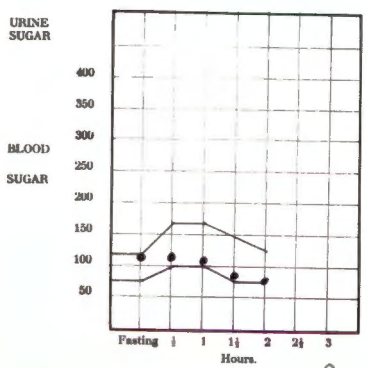
Report from Pathological Department.
UNIVERSITY OF CAPE TOWN.

Serial No. 14781/55. Date 20.7.55.

Patient's Identification MRS. MULLER. (34311)

Ward C 10. Physician or Surgeon Prof. Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR	
Fasting	Coagulated.
½ hour	122
1 hour	113
1½ hour	85
2 hour	83
2½ hour	
3 hour	

Signature

Age 52 years. Menopause at 48 years. D&C 30/8/55: Adenocarcinoma of endometrium.

40.

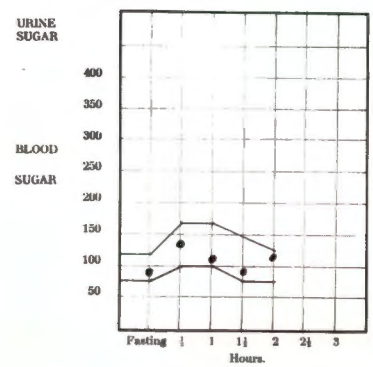
Report from Pathological Department.
UNIVERSITY OF CAPE TOWN.

Serial No. 23256. Date 9. 11. 55.

Patient's Identification RICHARD, JOSSIA. (116985)

Ward C.10. Physician or Surgeon ????

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR	
Fasting	93.%
½ hour	139
1 hour	117
1½ hour	93
2 hour	121
2½ hour	
3 hour	

Signature

Age 53 years. C/O Menorrhagia 5 years. D&C 8/8/57: Adenocarcinoma of endometrium.

41.

Report from Pathological Department.

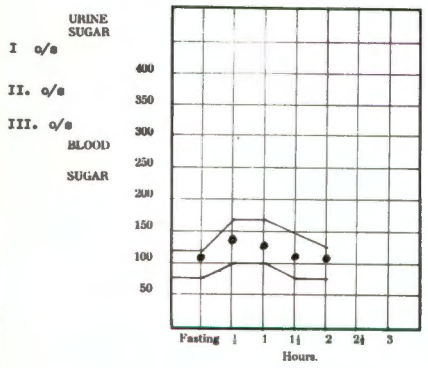
UNIVERSITY OF CAPE TOWN.

Serial No. 25446-7/57 Date 30.7.57

Patient's Identification JEANETTE HERMAN. (57/17629).

Ward B 7. Physician or Surgeon Prof. Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR
Fasting 112 mgs.%
½ hour 131 mgs.%
1 hour 126 mgs.%
1½ hour 115 mgs.%
2 hour 105 mgs.%
2½ hour
3 hour

Signature

Age 46 years. C/O Menorrhagia: 3 months. D&C 9/5/57: Adenocarcinoma of endometrium.

42.

Report from Pathological Department.

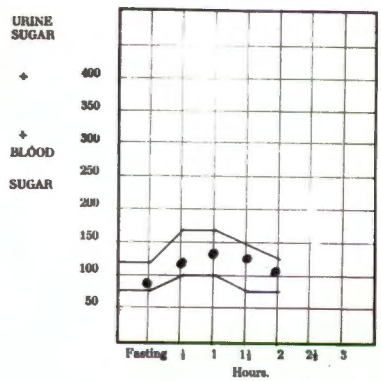
UNIVERSITY OF CAPE TOWN.

Serial No. 17955-6/57 Date 24.5.57

Patient's Identification MINNIE ENGEL. (20504).

Ward A 9. Physician or Surgeon Dr. Buss.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR
Fasting 82 mgs.%
½ hour 128 mgs.%
1 hour 135 mgs.%
1½ hour 130 mgs.%
2 hour 104 mgs.%
2½ hour
3 hour

G. LINDER
Signature

Age 52 years. Menopause at 50 years. D&C 29/10/57: Adenocarcinoma of the endometrium.

43.

Report from Pathological Department.

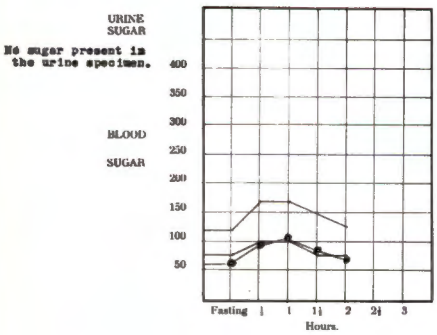
UNIVERSITY OF CAPE TOWN.

Serial No. 32808-09/57 Date 1.10.57

Patient's Identification KATRINA WAGNER (HOSP.NO. 57/09461.)

Ward C 10. Physician or Surgeon Prof. Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR
Fasting 64 mgs.%
½ hour 90 mgs.%
1 hour 104 mgs.%
1½ hour 82 mgs.%
2 hour 72 mgs.%
2½ hour
3 hour

Signature

Age 56 years. Menopause at 46 years. D&C 17/8/56: Adenocarcinoma of the endometrium.

44.

Report from Pathological Department.

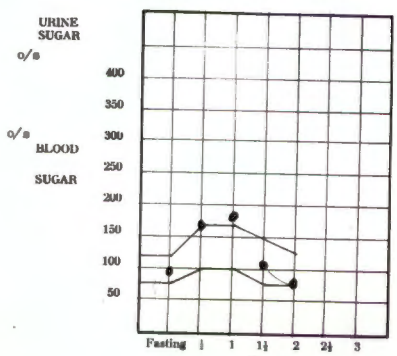
UNIVERSITY OF CAPE TOWN.

Serial No. 18832-33/57 Date 2.6.57

Patient's Identification MAY VISAGIE. (56/07493).

Ward C 10. Physician or Surgeon Prof. Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR
Fasting 92 mgs.%
½ hour 161 mgs.%
1 hour 176 mgs.%
1½ hour 101 mgs.%
2 hour 74 mgs.%
2½ hour
3 hour

G. LINDER
Signature

Age 60 years. Menopause at 50 years. D&C 6/10/58: Adenocarcinoma of endometrium.

45.

Report from Pathological Department.

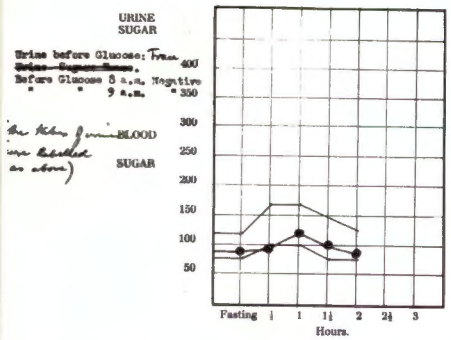
UNIVERSITY OF CAPE TOWN.

Serial No. 33352 Date 6.10.58.

Patient's Identification ALETTA WILLIAMS. (58/24384)

Ward A9. Physician or Surgeon Prof. Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR
Fasting 85 mgs.%
½ hour 9[illegible] mgs.%
1 hour 121 mgs.%
1½ hour 100 mgs.%
2 hour 87 mgs.%
2½ hour
3 hour

Signature H. GOLBY

Age 46 years. C/O Menorrhagia: 2 months. D&C 1/7/57: Adenocarcinoma of endometrium.

46.

Report from Pathological Department.

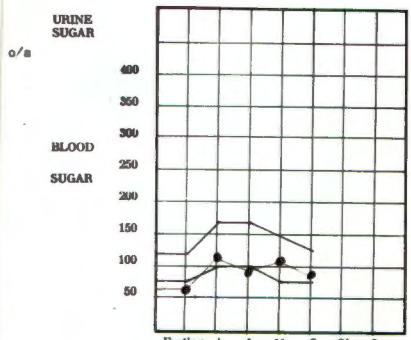
UNIVERSITY OF CAPE TOWN.

Serial No. 20458-9/57 Date 18.6.57

Patient's Identification FATIMA MAJAL. (57/11168).

Ward B 7. Physician or Surgeon Dr. Meaney.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR
Fasting 63 mgs.%
½ hour 124 mgs.%
1 hour 95 mgs.%
1½ hour 115 mgs.%
2 hour 83 mgs.%
2½ hour
3 hour

G. LINDER

Signature

Age 50 years. C/O Menorrhagia: 1 year. Hysterectomy 22/8/55: Adenocarcinoma of endometrium, and fibroids.

47.

Report from Pathological Department.

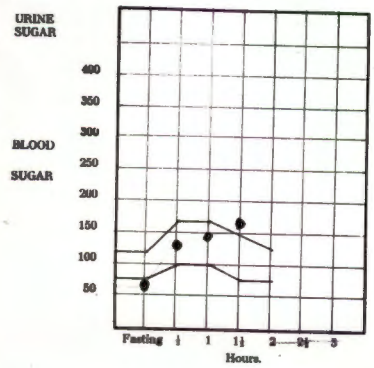
UNIVERSITY OF CAPE TOWN.

Serial No. 17899/55. Date 2.9.55.

Patient's Identification MISS KEILLY. (185882.)

Ward C 10. Physician or Surgeon Prof. James Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR
Fasting 71.
½ hour 131
1 hour 149
1½ hour 160
2 hour Coagulated.
2½ hour
3 hour

G. LINDER

Signature

Age 62 years. C/O Irregular vaginal bleeding: 14 months. D&C and hysterectomy 6/1/59: Adenocarcinoma of endometrium.

48.

Report from Pathological Department.

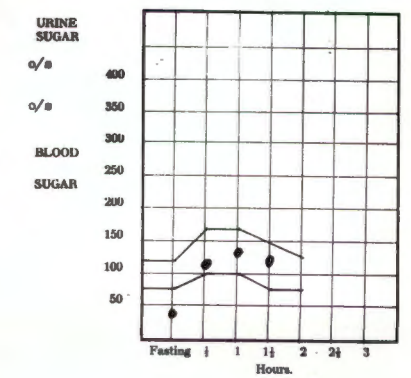
UNIVERSITY OF CAPE TOWN.

Serial No. 18277-8/57 Date 28.5.57

Patient's Identification MRS. A.L. RICHARDS. (88576).

Ward C 10. Physician or Surgeon Prof. Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR
Fasting 44 mgs.%
½ hour 113 mgs.%
1 hour 135 mgs.%
1½ hour 126 mgs.%
2 hour
2½ hour
3 hour

G. LINDER

Signature

Age 49 years. Menorrhagia. D&C 25/11/59: Well differentiated adenocarcinoma of the endometrium.

49.

Report from Pathology Department.

(Chemical Pathology).

UNIVERSITY OF CAPE TOWN.

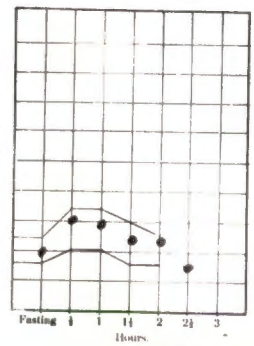
Serial No. 40604 - 605. Date 23.11.59.

Patient's Identification JOHANNA CURTIS (59/2311.5)

Ward A9 Physician or Surgeon Dr. Benjamin.

GLUCOSE TOLERANCE TEST.

Test-tape	URINE SUGAR
1. Negative	Negative.
2. Negative	Negative.
3. Negative	Negative.



BLOOD SUGAR	
Fasting	93 mgs.%.
½ hour	150 mgs.%.
1 hour	145 mgs.%.
1½ hour	139 mgs.%.
2 hour	117 mgs.%.
2½ hour	68 mgs.%.
3 hour	

Signature

Age 64 years. Post-menopausal bleeding. D&C : Adenocarcinoma endometrium.

50.

Report from Pathology Department.

(Chemical Pathology).

UNIVERSITY OF CAPE TOWN.

Serial No. A2983 - 84. Date 4.12.59.

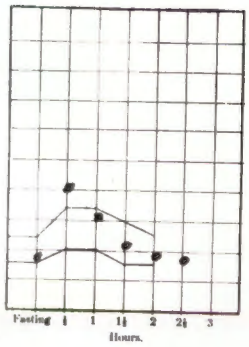
Patient's Identification HEL A JAY (119849)

Ward A1C. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR

Urines negative with tes tape and Benedicts



BLOOD SUGAR	
Fasting	84 mgs.%.
½ hour	204 mgs.%.
1 hour	196 m s.%.
1½ hour	188 mgs.%.
2 hour	94 mgs.%.
2½ hour	87 mgs.%.
3 hour	

Signature

TABLE 8. GLUCOSE TOLERANCE CURVES IN CASES WITH CARCINOMA OF THE ENDOMETRIUM.

Patient			Blood Sugar Levels (Mgs.%)				
No	Initials	Age	Fasting	½ Hour	1 Hour	1½ Hours	2 Hours
A. Group with Diabetes.							
1.	A.K.	46	183	219	238	320	210
2.	E.L.	59	150	192	254	242	210
3.	A.V.Z.	77	131	232	273	265	221
4.	M.B.	73	119	176	198	228	221
5.	A.B.	63	278	258	320	308	315
6.	I.P.	64	97	161	183	288	197
7.	L.E.S.	63	127	183	229	214	203
8.	S.G.	59	139	225	217	163	163
9.	C.V.W.	61	295	316	397	398	381
10.	C.V.	60	183	268	230	254	276
11.	E.K.	64	122	190	187	201	199
12.	G.W.	56	160	191	213		156
13.	C.W.	63	116	199	225	247	222
14.	K.J.	61	126	301	430	451	436
B. Group with Mildly Impaired Glucose Tolerance.							
15.	M.S.	50	94	176	172	192	159
16.	K.G.	64	101	147	208	163	124
17.	T.L.	70	92	119	135	169	153
18.	A.D.V.	80	91	157	189	182	150
19.	E.M.	72	107	154	199	153	138
20.	P.M.	75	104	126	174	172	145
21.	S.T.	75	93	167	192	182	131
22.	L.B.	71	68	107	136		132
23.	L.L.	45	94	140	165	179	140
24.	S.E.	65	122			165	188
25.	C.M.	58	81	141		166	128
26.	P.S.	51	103	163	189	181	124

Patient			Blood Sugar Levels (Mgs. %)				
No	Initials	Age	Fasting	½ Hour	1 Hour	1½ Hours	2 Hours
27.	E.M.	53	90	180	136	145	120
28.	H.P.	58	90	128	138	144	111
29.	S.S.	49	93	93	99	93	79
30.	M.B.	59	72	105	85	112	76
31.	A.B.	80	118	157	138	65	61
32.	A.C.	79	42	142	121	117	117
33.	F.S.	76	110	162	183	156	117
34.	J.E.	53	107	147	146	119	
35.	A.H.	57	82	107	139	119	119
36.	M.D.W.	51	86	96	144	130	107
37.	M.M.V.	52	117	183	197	131	103
38.	M.L.	52	86	108	108	105	100
39.	M.M.	50		122	113	85	83
40.	J.E.	52	93	139	117	93	121
41.	J.H.	53	112	131	126	115	105
42.	M.E.	46	82	128	135	130	104
43.	K.M	52	64	90	104	82	72
44.	M.V.	56	92	161	176	101	74
45.	A.W.	60	85	94	121	100	87
46.	F.M.	47	63	124	95	115	83
47.	M.K.	50	71	131	149	160	
48.	A.L.R.	62	44	113	135	126	
49.	J.C.	49	93	150	145	119	117
50.	H.J.	64	84	204	198	198	94

The results can be summarised as follows:

TABLE 9.

Normal Curves	Abnormal Curves		
	Diabetes	Mildly Impaired Glucose Tolerance	Total
48%	28%	24%	52%

This high incidence of diabetes and less severely impaired glucose tolerance in cases of cancer of the endrometrium, corresponds with the findings of Moss (1947), Gernet (1958), Way (1954), Louw (1958), all of whom also carried out glucose tolerance tests in their patients. A much lower incidence of diabetes was found by Smith (1941), Vander (1958), Hertig et al., (1940), Scheffey et al., (1943), and Palmer et al, (1949). The explanation for this discrepancy has been discussed (see page 18) and is found in the method used in

diagnosing the diabetes.

3. PATIENTS WITH BENIGN GLANDULAR HYPERPLASIA:

Fifty consecutive patients who were proved by histological examination of the endometrium to be suffering from benign glandular hyperplasia were investigated along the same lines for glucose tolerance.

It is necessary first to consider what is meant by the term Benign Glandular Hyperplasis, and to discuss the pathology thereof:

The Pathology of Huperplasia of the Endometrium:

In recent years our ideas concerning hyperplasia of the endometrium have undergone considerable change. This applies particularly to its histology and significance. Hyperplasia of the endometrium is the commonest form of the so-called "dysfunctional uterine bleedings". The term dysfunctional uterine bleeding is applied to abnormal uterine bleeding where there is no gross pathology

detectable clinically in the female ganital tract. Such dysfunctional bleeding may be due to general diseases such as thrombocytopoenic purpura, or to local pathology not detectable clinically, like endometrial tuberculosis. But in this discussion we are only concerned with those abnormal haemorrhages that are due to disturbances in the hormones produced by the ovary. Some of these endocrine disturbances are ovular, but the majority are non-ovular, and it is the latter that cause the various types of endometrial hyperplasia. Primarily, therefore, they are due to persistence of unruptured Graafian follicles. Consequently the endometrium is subjected to an abnormally prolonged or excessive action of oestrogenic hormones. Since, under these conditions the corpus lutum is not formed, the endometrium shows no evidence of the secretory changes which, as far as we know, can be produced only by progesterone.

The degree of abnormal growth response of the ovary depends apparently, not only on the amount and duration of the oestrogenic stimulus, but also on the degree of sensitivity of the particular

endometrium. It is also known that an endometrium which shows comparatively little hyperplastic change may at times be associated with profuse bleeding, while in other cases a marked degree of hyperplasia may exhibit very much less bleeding. The most constant feature of this type of dysfunctional bleeding is the absence of secretory changes, as would be expected from the absence of corpora lutea and therefore of progesterone. It must be added, however, that there are other types of dysfunctional bleeding than the one under discussion, and that bleeding can occur from practically any type of endometrium.

The Definition of Hyperplasia:

Confusion has arisen with regard to the use of the term endometrial hyperplasia. Some limit it to the fully-developed hyperplastic and cystic picture which is often seen; others extend its use to include the lesser degrees of hyperplasia; and others, again, would even include non-ovular "menstruation", which occurs from an endometrium indistinguishable from that seen in the first half

of a normal ovulatory cycle. These varieties are probably merely different degrees of the same basic pathology. For the purpose of this investigation the cases have been divided into two groups, namely 1) genuine hyperplasia of the endometrium, and 2) bleeding from a non-ovulatory endometrium, where definite hyperplastic changes are not seen. It is, howeverm likely that a diagnosis of hyperplasia would be more frequently made if the curettage were always done before the bleeding had persisted for too long; and not too soom after the cessation of a bleeding episode. Continued bleeding may lead to desquamation of most of the surface epithelium, so that later curettage may show only a proliferative, non hyperplastic pattern.

Macroscopic appearance of Genuine Hyperplasia:

In the most pronounced cases the endometrium is greatly thickened and polypoidal, so that curettage in these cases yield great quantities of polypoidal tissue. Superficially this may resemble

endometrial carcinoma, but in the latter case the tissue is usually more friable and necrotic. In most cases, however, the lestion is much less gross, the curettings being moderately or slightly thickened, or even normal looking.

Miscroscopic Appearances:

The microscopic appearance in a well-developed case is very characteristic and the diagnosis can be made at a glance.

In hyperplasia there is an increase in the number of tissue elements, both epithelial and stromal. The surface epithelium and epithelium of the glands is taller than normal, with heavily stained nuclei which not infrequently show many mitoses. The stroma is compact-looking and abundant, also with a tendency to increased mitotic activity.

The gland pattern may also be most distinctive. In any phase of the normal menstrual cycle there is a rather striking uniformity in the size and shape of the glands, but in hyperplasia

the glands show marked disparity. Some are large and cystic, while others are very small. There is thus produced the so-called "Swiss-cheese pattern", like the large and small holes of Swiss cheese. (This term which has been widely adopted in the literature, was first suggested by Novak in 1924 - (Novak and Martzloff, 1924).

The degree of hyperplasia, however, varies, and may be mild and difficult to distinguish from the appearances seen in the proliferative phase of a normal ovulatory cycle.

The typical hyperplasia, however, is a frankly benign lesion, both histologically and clinically. Whether it predisposes to carcinoma is an unsolved question, but there is little evidence that it does. However, cases are encountered where there is such an unusual degree of epithelial proliferative activity that adenocarcinoma may be suspected. In such cases the glands may be closely placed, and there may be pseudostratification, or actual stratification, especially in the smaller glands. The epithelium may be markedly convoluted, with intra-luminal tufting and budding. There may

also be evidence of unusual mitotic activity. In the re-productive years only 1.3% of cases (Novak and Novak, 1958) of the common hyperplasia present pictures which may lead to the suspicion of adenocarcinoma.

Another interesting, though common, epithelial change is the so-called squamous metaplasia. It involves primarily the glands, often filling the lumen, although surface epidermidization is not infrequent. The squamous cells are well-differentiated; in no way do they indicate a malignant tendency. The prognosis depends entirely on the glandular component, for if this is benign, then so is the whole process, irrespective of the associated acanthosis (if the adenomatous element is malignant, then the lesion is called an adenoacanthoma, a variety of adenocarcinoma). The explanation of this change is probably a direct transition from columnar to squamous epithelium, but the point is a debatable one.

Post-menopausal Hyperplasia:

A rather surprising development of

recent years is the finding of what is apparently a perfectly typical and actively growing benign hyperplasia in women who are far beyond the menopause. The general view had always been that such hyperplasia was a disease of reproductive life, though also commonly occurring round about the menopause. However, it was though unlikely that it could persist for more than a year or two after the cessation of menstruation. However, Novak and Yui (1936) found a considerable number of cases in women from 1 - 40 years after the last menstrual period. In my series 19 were of this type. (The possible pre-malignant role of post-menopausal hyperplasia is discussed later).

In some of these cases, however, the hyperplasia is not active, but "retrogressive". In other words, the Swiss-cheese pattern has persisted for many years as a relic of the non-ovular menstrual cycles just prior to the menopause. It does not represent a response to further oestrogenic stimulation. In such retrogressive cases, even though the Swiss-cheese pattern persists, the stroma is likely to be more or less fibrotic, while the gland epithelium is low, and inactive, with none of the

mitosis so commonly seen in active hyperplasia.

All cases where histological examination of the endometrium showed the hyperplastic picture just described, were investigated by glucose tolerance tests. The same standard and conditions that applied to the control and other groups were used. Fifty such cases were encountered and studied. The clinical features in each case are described.

The detailed results are shown in Table 10. A photograph of each glucose tolerance test is attahhed. The histological picture of the endometrium of every patient is seen in the accompanying photo-microhraphs. A summary of the results (Table 11) is shown after all these photographs, photomicrographs and Tables have been presented.

Age 42 years. C/O Menorrhagia: 2 months. D&C 23/5/57: Benign glandular hyperplasia 27/5/57 Hysterectomy: Marked adenomyosis; also benign glandular hyperplasia.

1.

Report from Pathological Department.

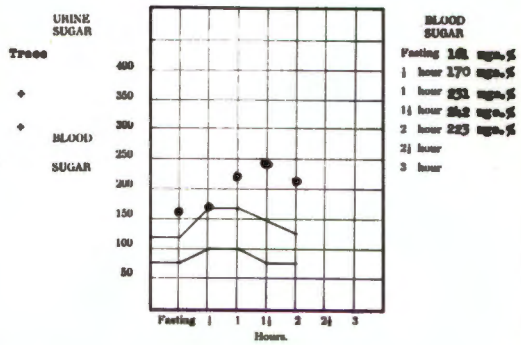
UNIVERSITY OF CAPE TOWN.

Serial No. 17953-4/57. Date 24. 5. 57

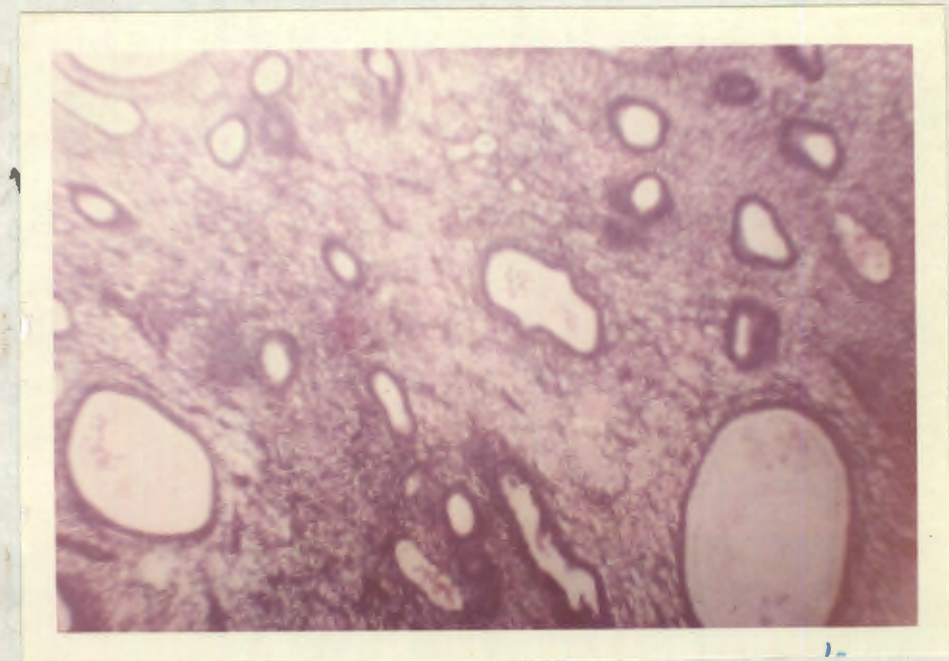
Patient's Identification [redacted] (57/1014).

Ward B 7. Physician or Surgeon Prof. Louw.

GLUCOSE TOLERANCE TEST.



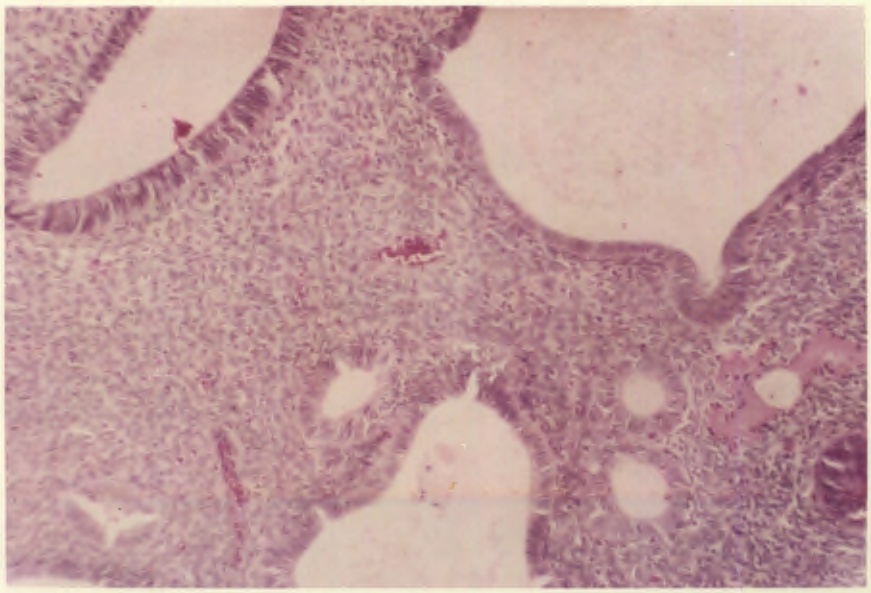
Signature [redacted]



CASE 1.

Age 42 years. Complained of menorrhagia: 2 months. D and C 23/5/57: Benign glandular hyperplasia. 27/5/57 Hysterectomy: Marked adenomyosis; also benign glandular hyperplasia.

CASE 1. (Photomicrograph b.).



1b

Age 54 years. Post-menopausal bleeding. D&C: Proliferative hyperplastic endometrium. (In 1949 had menorrhagia - D&C - Normal endometrium - no glycosuria then).

2. (

Report from Pathological Department.

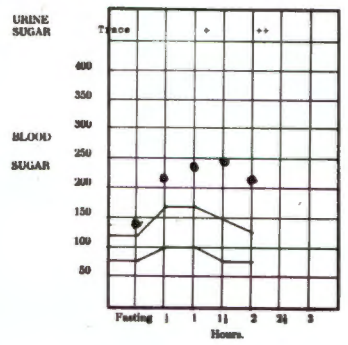
UNIVERSITY OF CAPE TOWN. 7/4458 437.

Serial No. 6660-61. Date 3.3.58.

Patient's Identification SUSAN SWANSON. (190816)

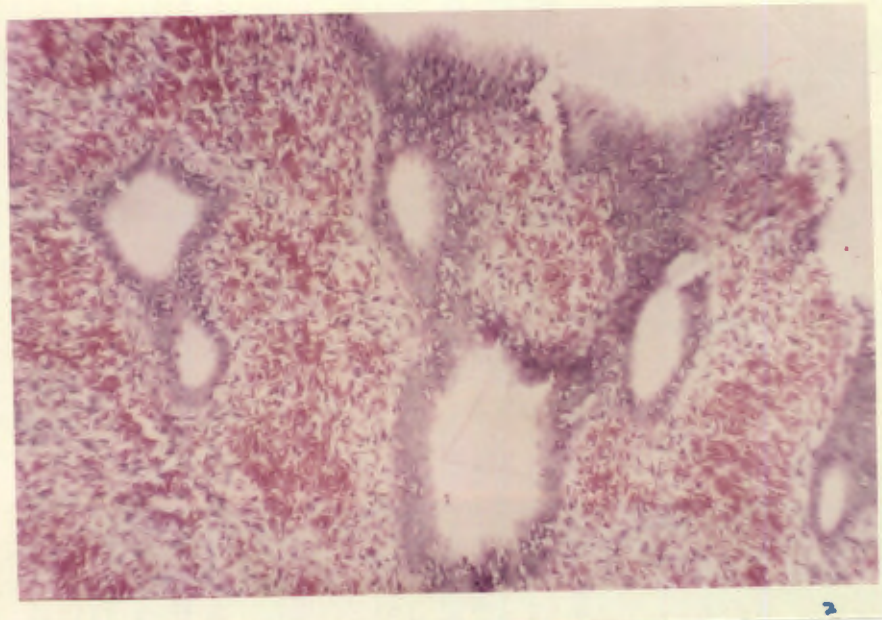
Ward 37 Physician or Surgeon Prof. Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR
Fasting 135 mgs.%.
½ hour 218 mgs.%.
1 hour 237 mgs.%.
1½ hour 240 mgs.%.
2 hour 212 mgs. %.
2½ hour
3 hour

Signature



2

CASE 2.

Age 54 years. Postmenopausal bleeding. D and C: Proliferative hyperplastic endometrium. (In 1949 had menorrhagia - D and C - Normal endometrium - no glycosuria then).

Age 42 years. Menorrhagia. D.&C., Hyperplastic cystic endometrium.

3.

Report from Pathological Department.

UNIVERSITY OF CAPE TOWN.

Serial No. 17582 - 3. Date 29.5.59

Patient's Identification RAYNA ABRAHAMS (59/07300)

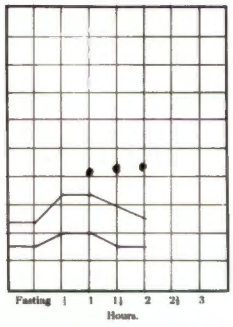
Ward 37. Physician or Surgeon Mr. Mackay.

GLUCOSE TOLERANCE TEST.

URINE SUGAR

Benedicts	Test-tape
1. +	++
2. ++	+++
3. +++	+++

BLOOD SUGAR

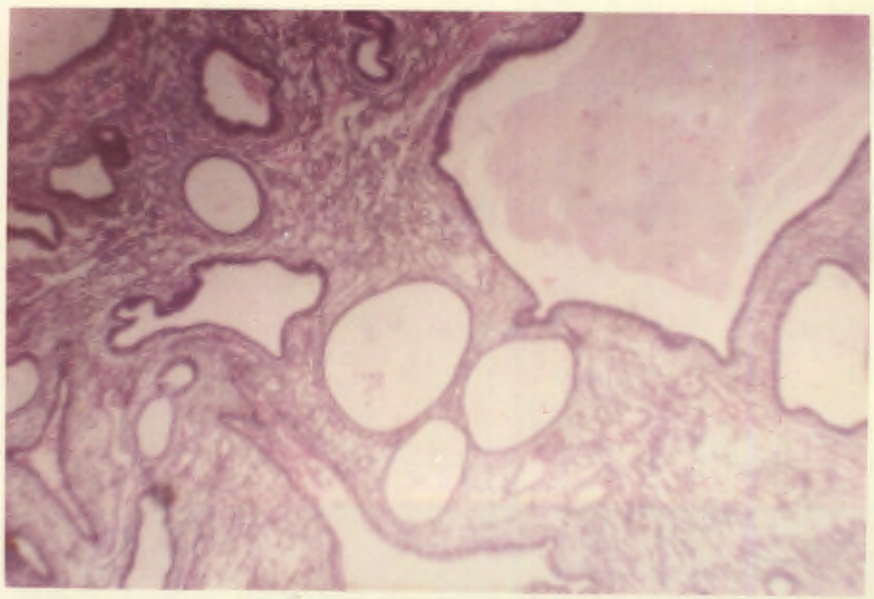


BLOOD SUGAR

	Insufficient
Fasting	
½ hour	Clotted
1 hour	304 mgs.%.
1½ hour	205 mgs.%.
2 hour	205 mgs.%.
2½ hour	
3 hour	

S. J. v d WALT

Signature



CASE 3.

Age 42 years. Menorrhagia. D and C: Hyperplastic cystic endometrium.

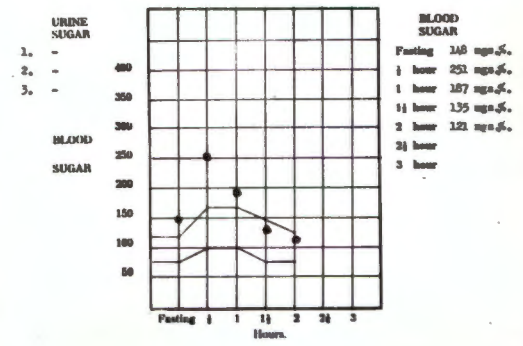
Age 55 years. C/O Bouts of amenorrhoea followed by bouts of prolonged bleeding - now bleeding for 2 months. D&C 3/7/59: Proliferative endometrium, cystic dilation of glands, and areas are hyperplastic.

4.

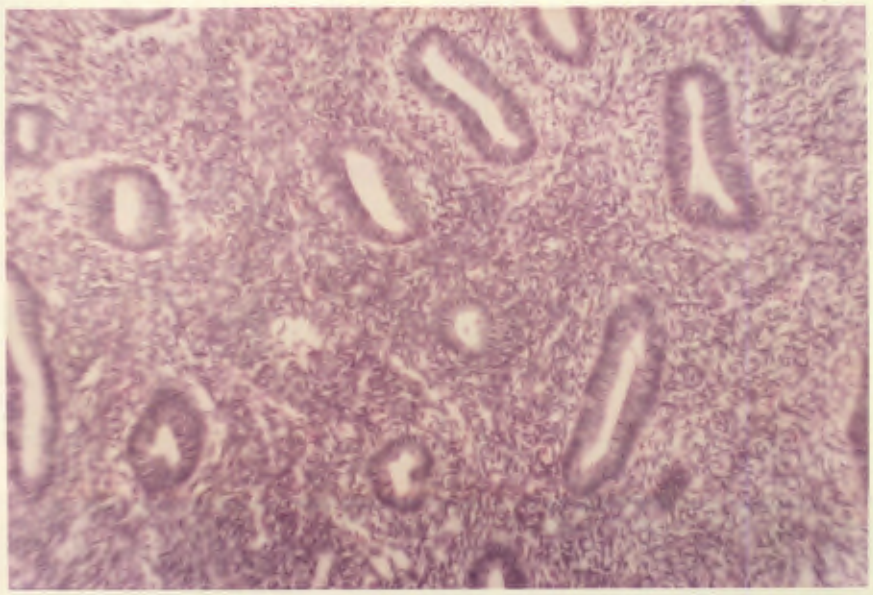
Report from Pathology Department.
(Chemical Pathology). 4472
UNIVERSITY OF CAPE TOWN.

Serial No. 22759 - 60. Date 3.7.59
Patient's Identification C. KRIEL (59/07.99)
Ward C10. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.



G. M. POTGIETER
Signature

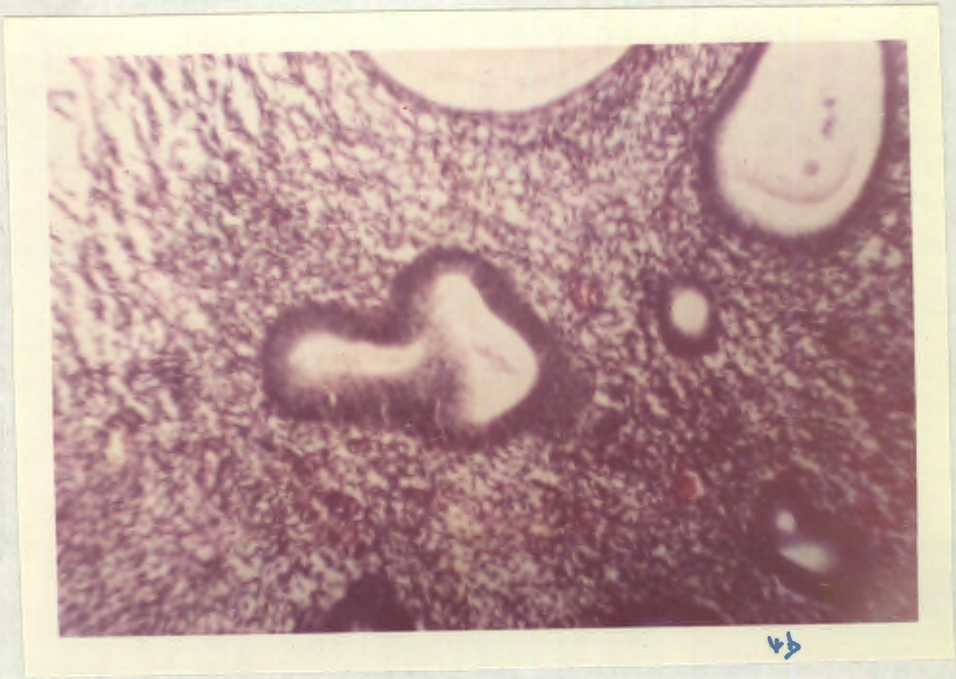


4a

CASE 4.

Age 55 years. Complained of bouts of amenorrhoea followed by prolonged bleeding - now bleeding for 2 months. D and C 3/7/59: Proliferative endometrium, cystic dilatation of glands, and areas are hyperplastic.

CASE 4 (Photomicrograph b.).



Age 47 years. Menopause at 33 years. C/O post-menopausal bleeding: 1 week. D&C 20/8/59: * Endometrium suggests active oestrogen secretion. The appearance is that of benign glandular hyperplasia with no secretory activity. No malignancy.

5.

Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN. 24/8/59 5659

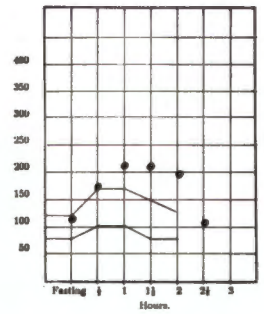
Serial No. 3.896 - 57. Date 9.10.59.

Patient's Identification CHELSTINA JACOBS (56/2545)

Ward A10. Physician or Surgeon Prof Louw.

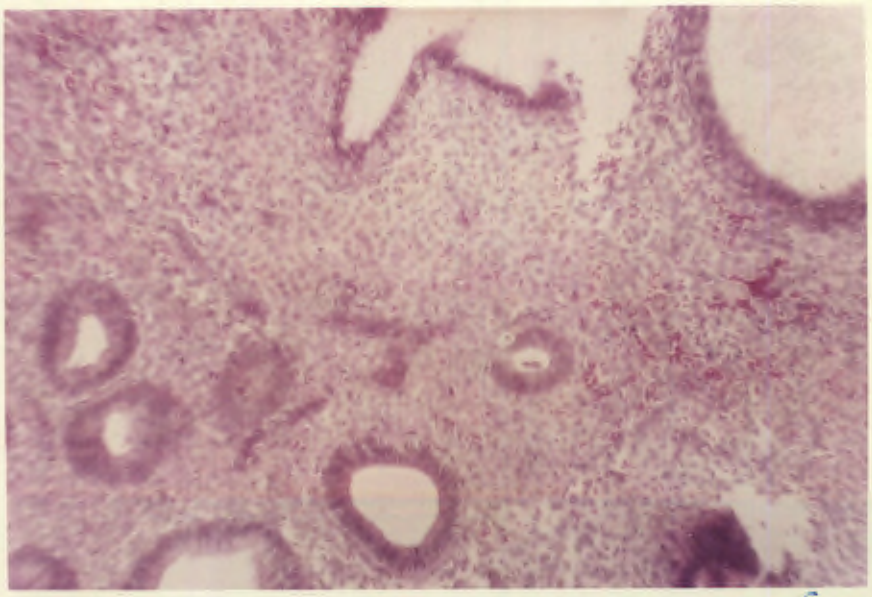
GLUCOSE TOLERANCE TEST.

Test-tape	URINE SUGAR
1. -	-
2. -	-
3. trace	+



BLOOD SUGAR	
Fasting	117 mgs.%.
½ hour	173 mgs.%.
1 hour	215 mgs.%.
1½ hour	213 mgs.%.
2 hour	199 mgs.%.
2½ hour	104 mgs.%.
3 hour	

Signature [illegible]

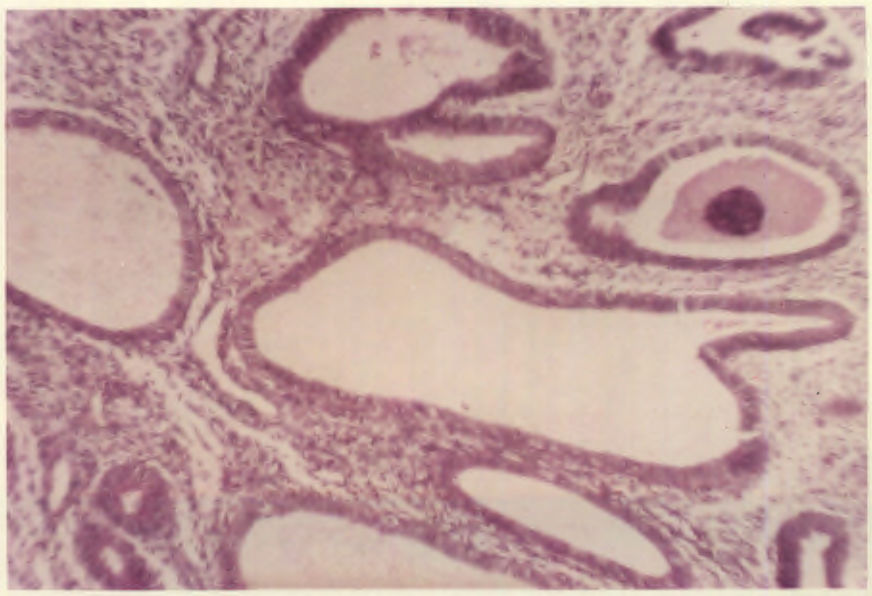


5a

CASE 5.

Age 47 years. Menopause at 33 years. Complained of post-menopausal bleeding: 1 week. D and C 20/8/59: Endometrium suggests active oestrogen secretion. The appearance is that of benign glandular hyperplasia with no secretory activity. No malignancy.

CASE 5. (Photomicrograph b.).



5b

Age 62 years. Marked hyperplastic endometrium.

6 (a)

Report from Pathological Department.

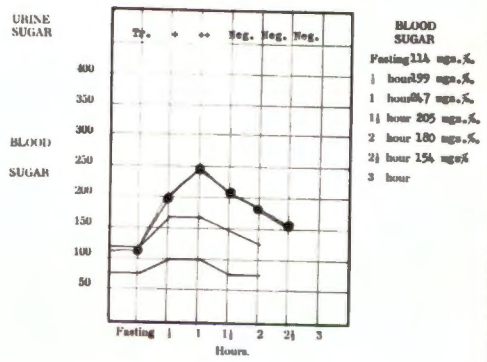
UNIVERSITY OF CAPE TOWN.

Serial No. 32684-85 Date 30.9.58.

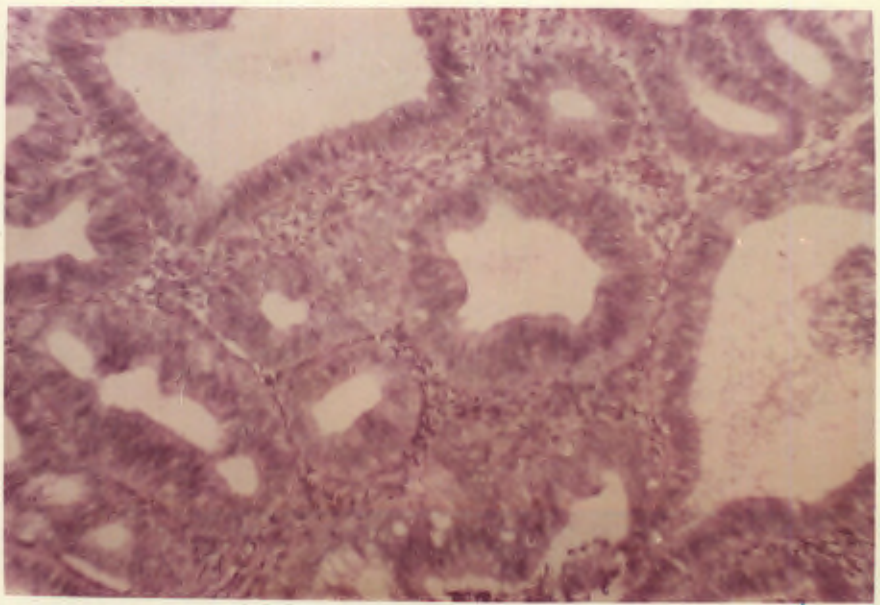
Patient's Identification MRS. TSUNWICH. (29911)

Ward C10. Physician or Surgeon Prof. Louw.

GLUCOSE TOLERANCE TEST.

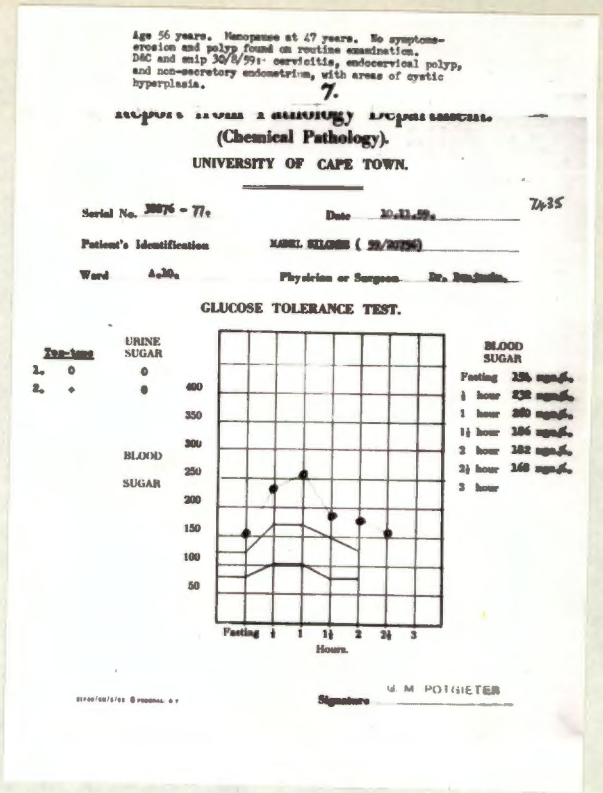


Signature



CASE 6.

Age 62 years. Markedly hyperplastic endometrium.



Age 56 years. Menopause at 47 years. No symptoms- erosion and polyp found on routine examination. D&C and snip 30/8/59:- cervicitis, endocervical polyp, and non-secretory endometrium, with areas of cystic hyperplasia.

7.

(Chemical Pathology).

UNIVERSITY OF CAPE TOWN.

Serial No. 30876 - 77. Date 30.11.59. 74.35

Patient's Identification MABEL GILCHRIST (59/30756)

Ward A.30. Physician or Surgeon Dr. Bamford

GLUCOSE TOLERANCE TEST.

Test-tube	URINE SUGAR
1. 0	0
2. +	0

BLOOD SUGAR

400 350 300 250 200 150 100 50

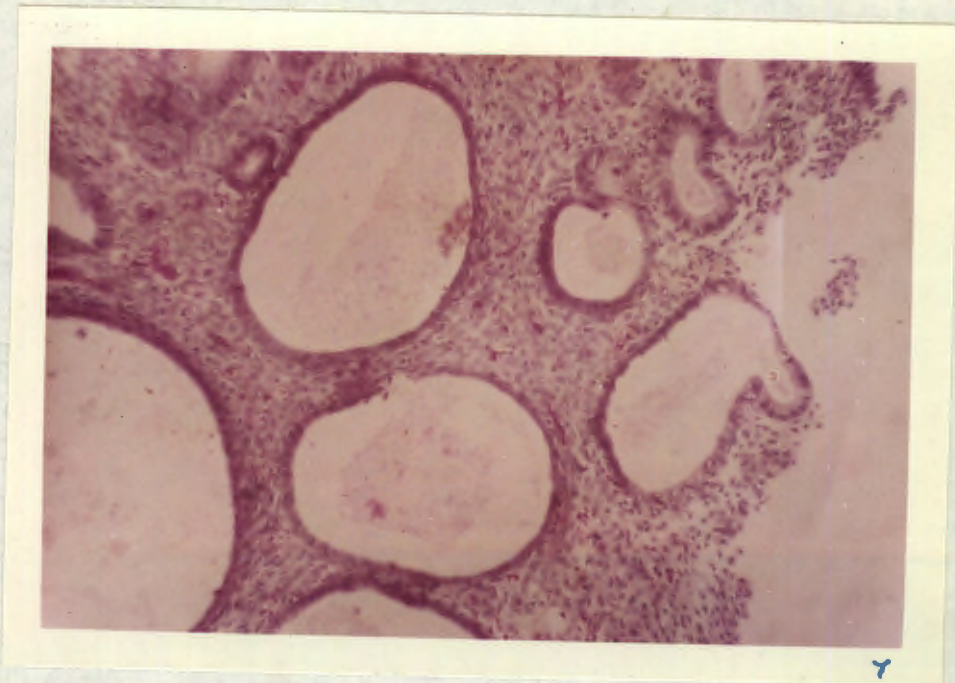
Fasting ½ 1 1½ 2 2½ 3

Hours.

BLOOD SUGAR

Fasting 158 mgms.
½ hour 232 mgms.
1 hour 280 mgms.
1½ hour 186 mgms.
2 hour 182 mgms.
2½ hour 160 mgms.
3 hour

Signature G. M. POTGIETER



CASE 7.

Age 56 years. Menopause at 47 years. No symptoms - erosion and polyp found on routine examination. D and C and snip 30/8/59: Cervicitis, endocervical polyp, and non-secretory endometrium, with areas of cystic hyperplasia.

Age 53 years. Postmenopausal bleeding 2 years. (Menopause at 50 years - then one year amenorrhoea - then continual bleeding, daily, 2 years). D&C : Marked hyperplasia, cystic glands, and crowding together of glands. Hysterectomy: no evidence of carcinoma.

8.

Report from Pathological Department.

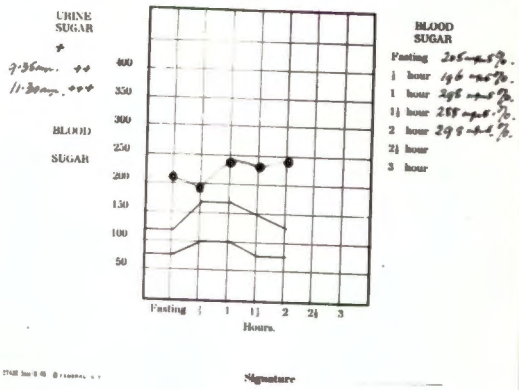
UNIVERSITY OF CAPE TOWN.

Serial No. 16972-3/57 Date 16/5/57.

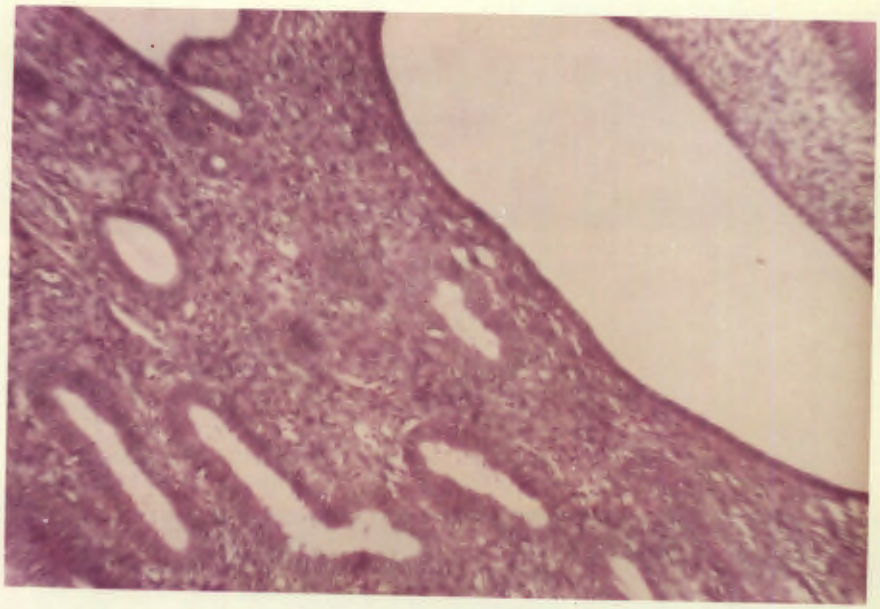
Patient's Identification [illegible] (57/05037)

Ward C10. Physician or Surgeon Prof. F. Lewis.

GLUCOSE TOLERANCE TEST.



Signature



8

CASE 8.

Age 53 years. Postmenopausal bleeding 2 years. (Menopause at 50 years - then one year amenorrhoea - then continual bleeding, daily, 2 years). D and C: Marked hyperplasia, cystic glands, and crowding together of glands. Hysterectomy: no evidence of carcinoma.

Age 48 years. C/O Menorrhagia: 5 years G/E Fairly obese, Blood pressure 168/88. D&C 27/4/59: Marked cystic dilatation of some of the glands.

9.

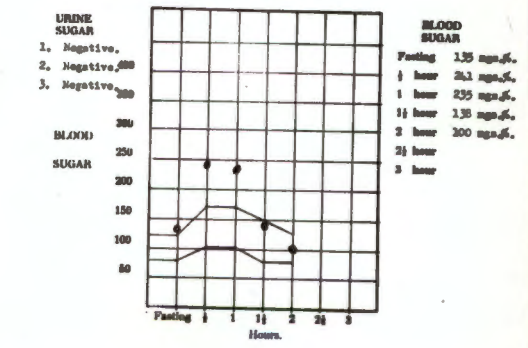
Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

Serial No. 28043 - 50. Date 3.7.59.

Patient's Identification SOPHIE MICHAU (56/2316)

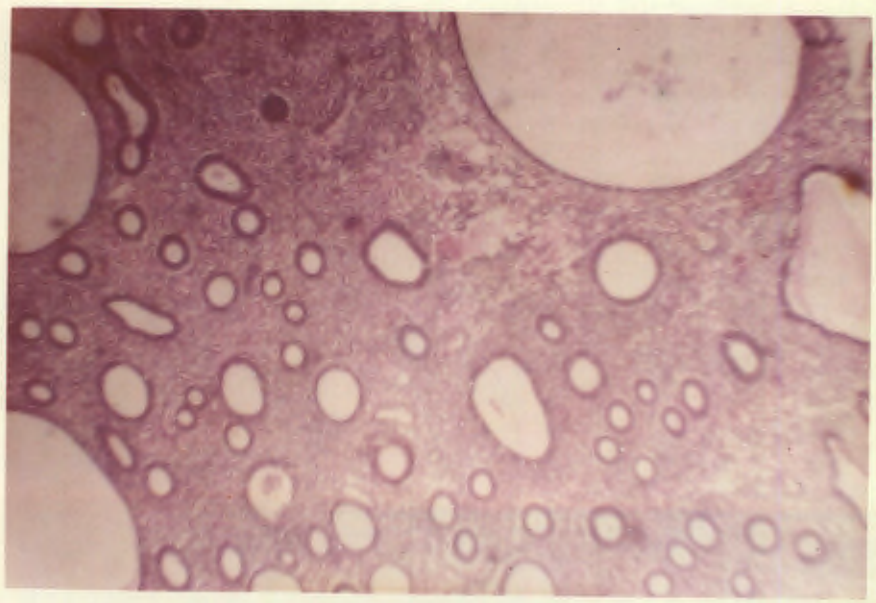
Ward 49. Physician or Surgeon Prof. Lewis.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR
Fasting 135 mgs.%.
½ hour 241 mgs.%.
1 hour 235 mgs.%.
1½ hour 138 mgs.%.
2 hour 100 mgs.%.
2½ hour
3 hour

Signature G. M. POTGIETER



9.

CASE 9.

Age 48 years. Complained of menorrhagia: 5 years. On examination fairly obese, blood pressure 168/88. D and C 27/4/59: Marked cystic dilatation of some of the glands.

BEFORE P.S.H. administration.

Age 50 years. C/O Bouts of amenorrhoea followed by bouts of menorrhagia. for 1 year. D&C 11/11/59: L.M.P. 23/9/59. Hyperplastic Proliferative endometrium - some cystic dilatation of glands.

10 (a)

Report from Pathology Department.

(Chemical Pathology).

UNIVERSITY OF CAPE TOWN.

7/12/59.

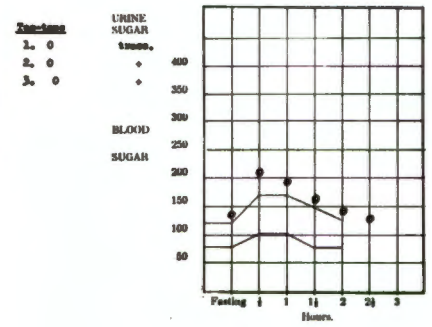
Serial No. 3904 - 905. Date 13.11.59.

Patient's Identification MAUD V.D. WESTHUIZEN (59/04.682)

Ward A30. Physician or Surgeon Dr. Benjamin.

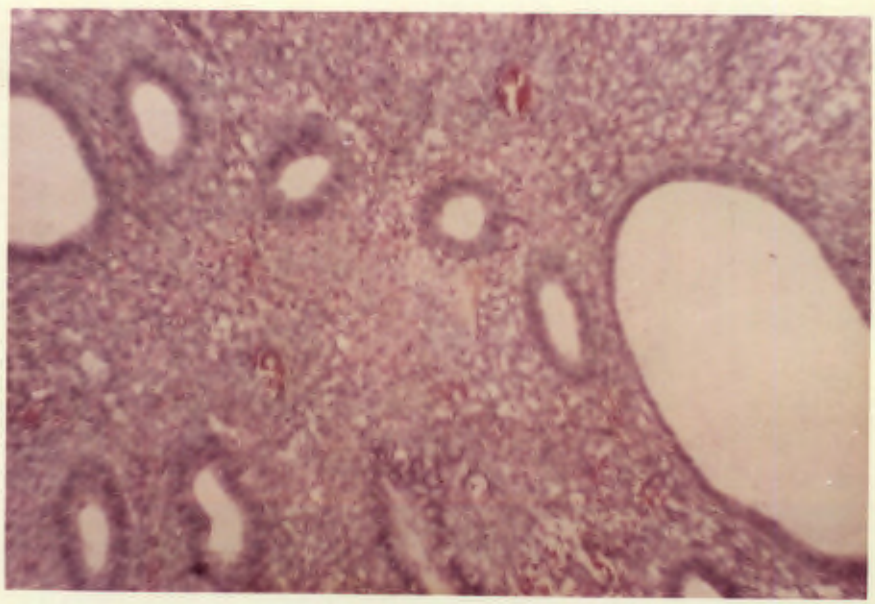
GLUCOSE TOLERANCE TEST.

Test-tube	URINE SUGAR
1. 0	trace.
2. 0	+
3. 0	+



BLOOD SUGAR	
Fasting	139 mgm.%.
½ hour	213 mgm.%.
1 hour	209 mgm.%.
1½ hour	164 mgm.%.
2 hour	148 mgm.%.
2½ hour	138 mgm.%.
3 hour	

Signature N. M. POTGIETER



10

CASE 10.

Age 50 years. Complained of bouts of amenorrhoea followed by bouts of menorrhagia: 1 year. D and C 11/11/59: L.M.P. 23/9/59. Hyperplastic proliferative endometrium - some cystic dilatation of glands.

Age 56 years. Menopause at 49 years. C/O post-menopausal bleeding. D&C and removal of polyp 31/8/59: Non-secretory endometrium with some hyperplasia atypical epithelium; must be reported with suspicion.

11(a)

Report from Pathology Department.

(Chemical Pathology).

UNIVERSITY OF CAPE TOWN.

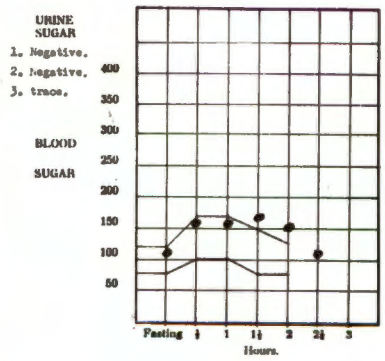
Staff

Serial No. 29967 - 68. Date 26.8.59.

Patient's Identification LYDIA CHEBE (59/0-853)

Ward C10. Physician or Surgeon Prof Louw.

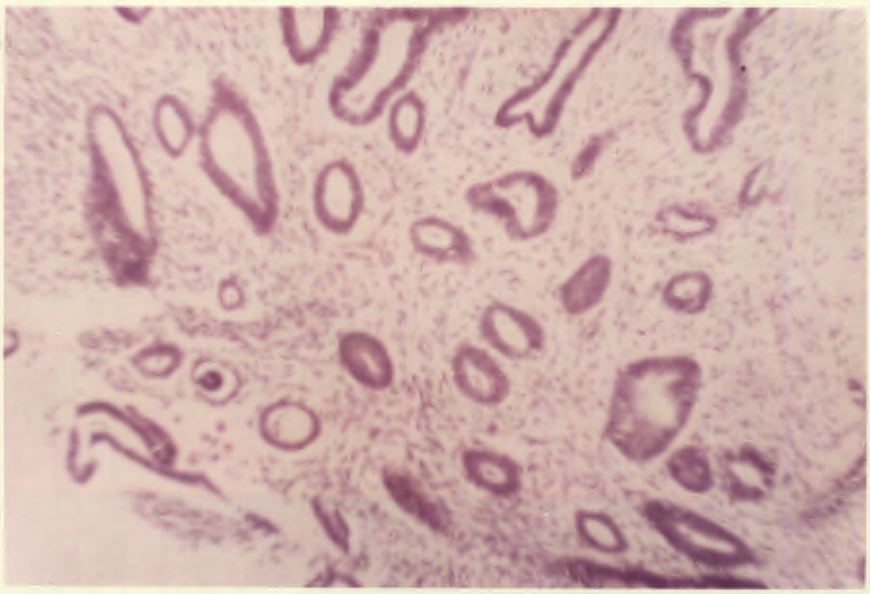
GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting	112 mgs.%.
½ hour	162 mgs.%.
1 hour	161 mgs.%.
1½ hour	171 mgs.%.
2 hour	152 mgs.%.
2½ hour	112 mgs.%.
3 hour	

Signature L. ANSTEY



11.

CASE 11.

Age 56 years. Menopause at 49 years. Complained of postmenopausal bleeding. D and C and removal of polyp 31/8/59: Non-secretory endometrium with some hyperplasia atypicality enpithlium; must be reported with suspicion.

Age 60 years. C/O Post-menopausal bleeding: 2 months. D&C 1/7/59: Histology: Active glandular hyperplasia with several cystically dilated glands. There is no marked crowding atypically of the glands and therefor nothing to suggest malignancy.

12.

Report from Pathology Department.

(Chemical Pathology).

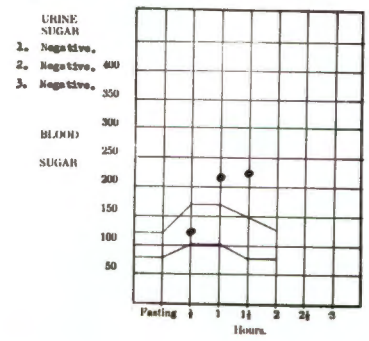
UNIVERSITY OF CAPE TOWN.

Serial No. 22996 - 7. Date 6.7.59.

Patient's Identification A. ABRAHAMS (55/18706)

Ward 37. Physician or Surgeon Prof Louw.

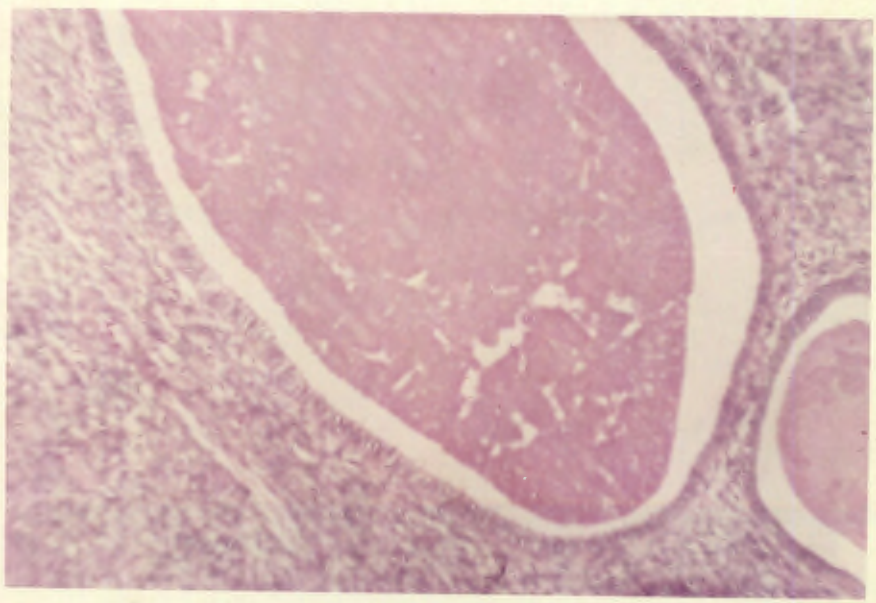
GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting [illegible] blood [illegible] for [illegible] hour 123 mg.%.
1 hour 219 mg.%.
1½ hour 223 mg.%.
2 hour No spec. received.
2½ hour
3 hour

Signature G. M. POTGIETER



12

CASE 12.

Age 60 years. Complained of postmenopausal bleeding: 2 months. D and C 1/7/59: Histology: active glandular hyperplasia with several cystically dilated glands. There is no marked crowding atypicality of the glands and therefore nothing to suggest malignancy.

Age 55 years. C/O vaginal bleeding, still menstruating: 3/28 day type: L.M.P. 8/6/59. D&C 1/7/59. Hysterectomy 3/8/59: Benign Glandular hyperplasia.

18.

Report from Pathology Department.

(Chemical Pathology).

UNIVERSITY OF CAPE TOWN.

Serial No. 23550 - 51. Date 29.6.59. 42[illegible]/59

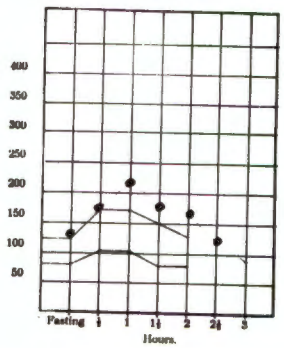
Patient's Identification MRS. IRIS M. MC GREGOR (59/0622A).

Ward C10. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

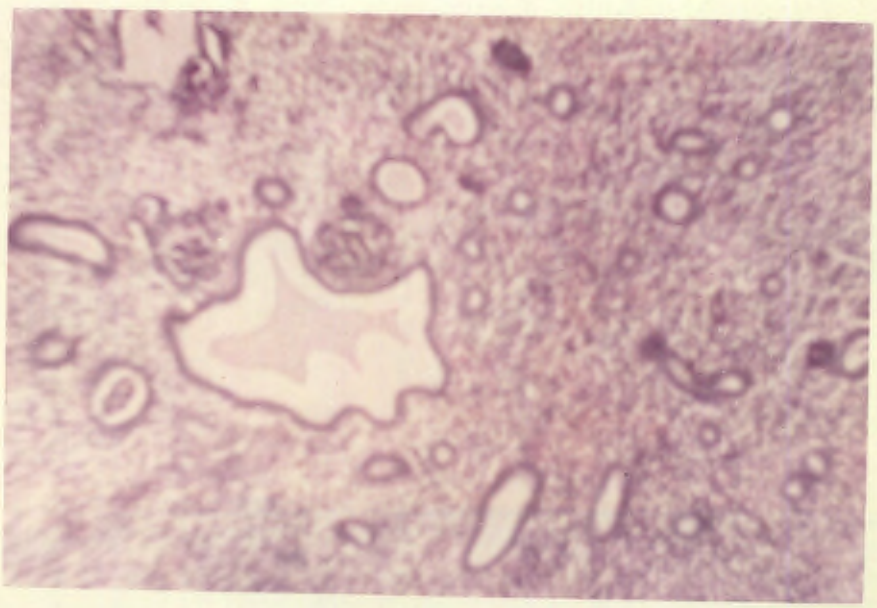
URINE SUGAR
1. trace.
2. trace.
3. trace.

BLOOD SUGAR



BLOOD SUGAR
Fasting 128 mgms%.
½ hour 178 mgms%.
1 hour 222 mgms%.
1½ hour 203 mgms%.
2 hour 167 mgms%.
2½ hour 122 mgms%.
3 hour

Signature L. ANSTEY.



13

CASE 13.

Age 55 years. Complained of vaginal bleeding intermenstrually; 3/28 day cycle: L.M.P. 8/6/59. D and C 1/7/59. Hysterectomy 3/8/59: Benign glandular hyperplasia.

Age 44 years. Menorrhagia and bouts of amenorrhoea. D&C 21/10/59: Benign glandular hyperplasia with cystic changes.

14.

Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

Serial No. 3496 - 77. Date 20.10.59.

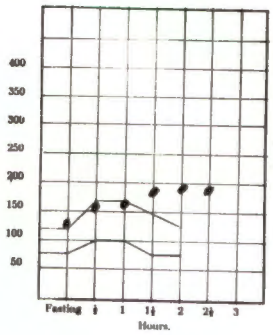
Patient's Identification CARA ATTWOOD (192343).

Ward A30. Physician or Surgeon Prof [illegible].

GLUCOSE TOLERANCE TEST.

Test-hour	URINE SUGAR
1. 0	0
2. 0	0
3. 0	0

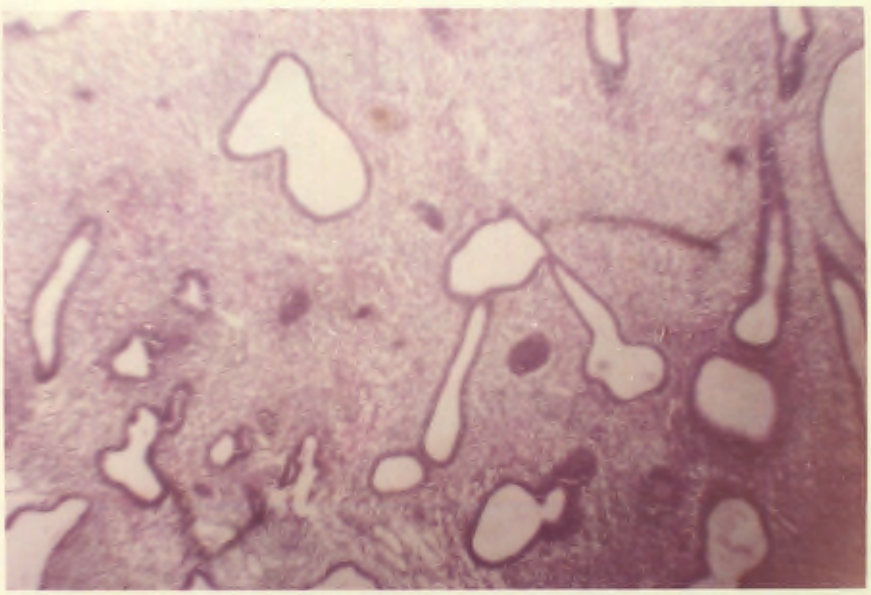
BLOOD SUGAR



BLOOD SUGAR

Fasting 186 mgms%.
½ hour 198 mgms%.
1 hour 162 mgms%.
1½ hour 180 mgms%.
2 hour 167 mgms%.
2½ hour 186 mgms%.
3 hour

Signature [illegible]



14

CASE 14.

Age 44 years. Menorrhagia and bouts of amenorrhoea. D and C 21/1059: benign glandular hyperplasia with cystic changes.

Age 30 years. C/O vaginal bleeding 2 months. D&C 8/11/58: benign glandular hyperplasia with several cystic and dilated glands.

15.

Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

7/189

Serial No. 21366 -7. Date 26.6.59.

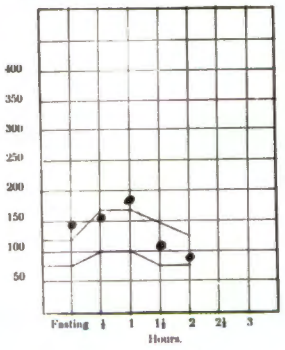
Patient's Identification GAILJA LAWRENCE (55/2,845).

Ward A10. Physician or Surgeon Dr. Benjamin.

GLUCOSE TOLERANCE TEST.

URINE SUGAR
1. -
2. -
3. -

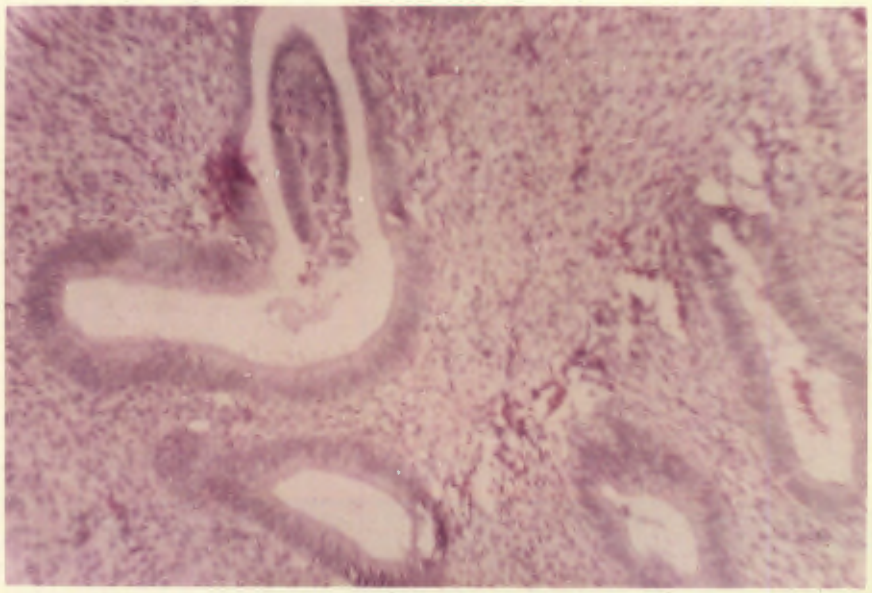
BLOOD SUGAR



BLOOD SUGAR
Fasting 145 mgs.%.
½ hour 152 mgs.%.
1 hour 183 mgs.%.
1½ hour 105 mgs.%.
2 hour 88 mgs.%.
2½ hour
3 hour

[illegible]NSTEY.

Signature



15

CASE 15.

Age 30 years. Complained of vaginal bleeding 2 months. D and C 8/11/58: benign glandular hyperplasia with several cystic and dilated glands.

Age 55 years. Post-menopausal bleeding. D&C : Metropathica.

16.

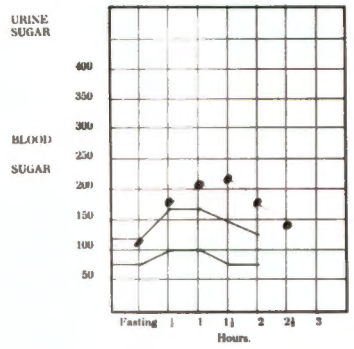
Report from Pathological Department.
ENDOCRINE RESEARCH LABORATORY.
UNIVERSITY OF CAPE TOWN.

Serial No. _____ Date 1950.

Patient's Identification V.F. ALLERS.

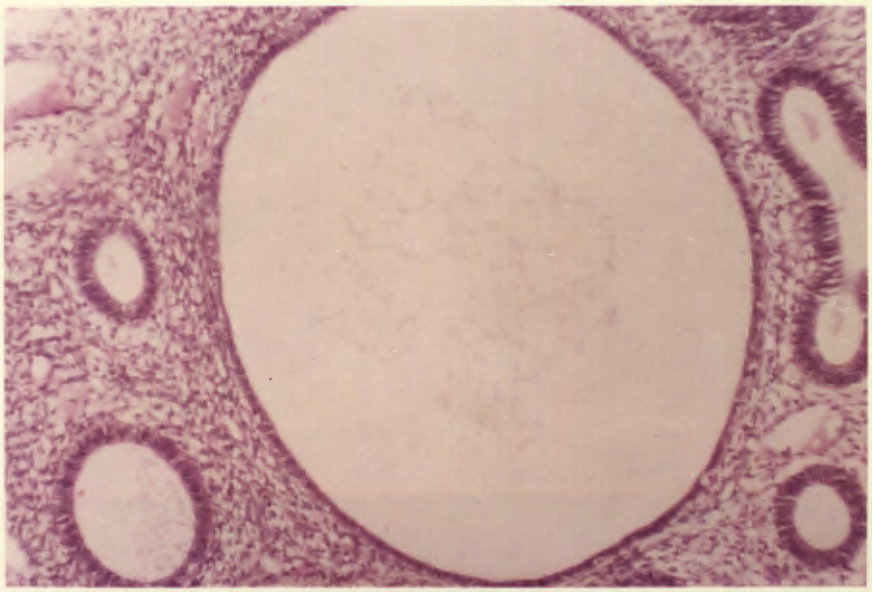
Ward A 10. Physician or Surgeon Dr F. Benjamin.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR	
Fasting	117 mgms.%.
½ hour	161
1 hour	207
1½ hour	218
2 hour	170
2½ hour	140.
3 hour	

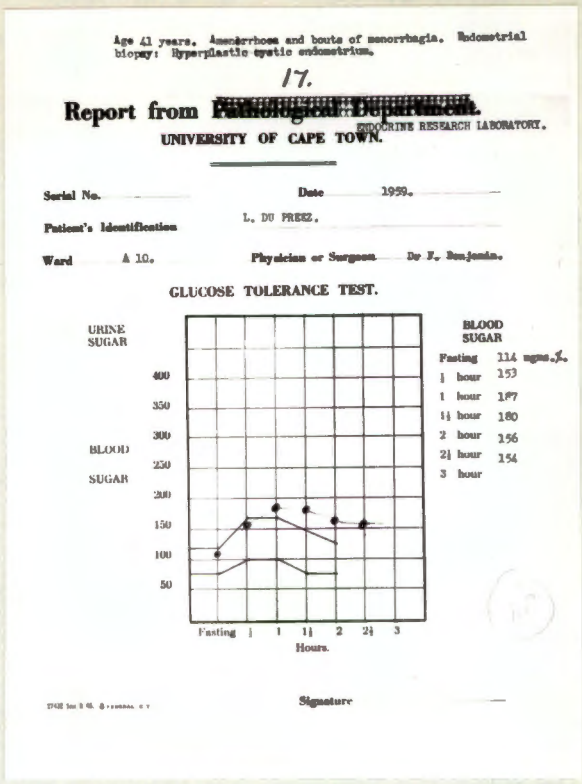
Signature _____



16

CASE 16.

Age 55 years. Postmenopausal bleeding. D and C: Metropathic endometrial picture.



Age 41 years. Amenorrhoea and bouts of menorrhagia. Endometrial biopsy: Hyperplastic cystic endometrium.

17.

Report from [redacted] Department.

ENDOCRINE RESEARCH LABORATORY.

UNIVERSITY OF CAPE TOWN.

Serial No. _____ Date 1959.

Patient's Identification L. DU PREEZ.

Ward A 10. Physician or Surgeon Dr F. Benjamin.

GLUCOSE TOLERANCE TEST.

URINE SUGAR

BLOOD SUGAR

400
350
300
250
200
150
100
50

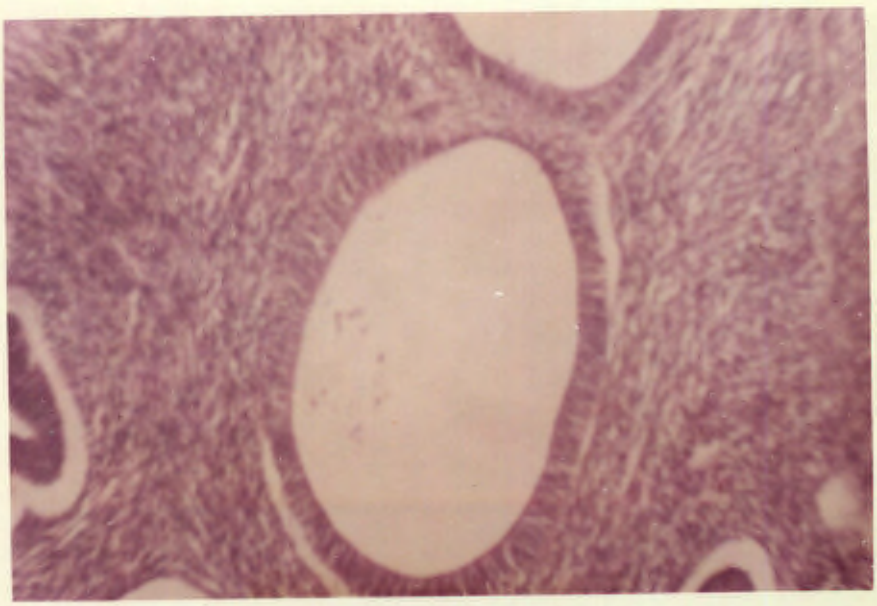
Fasting ½ 1 1½ 2 2½ 3

Hours.

BLOOD SUGAR

Fasting	114 mgms.%
½ hour	153
1 hour	187
1½ hour	180
2 hour	156
2½ hour	154
3 hour	

Signature _____



17

CASE 17.

Age 41 years. Amenorrhoea and bouts of menorrhagia. Endometrial biopsy: hyperplastic cystic endometrium.

Age 39 years. Cycle 4-7/28 - 35 day type with menorrhagia. D&C: Hyperplastic cystic endometrium.

18.

Report from Pathology Department.

(Chemical Pathology).

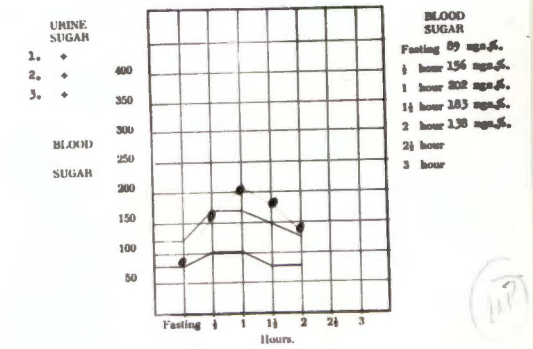
UNIVERSITY OF CAPE TOWN.

Serial No. 1854 - 5. Date 7.6.39

Patient's Identification [illegible] (297529)

Ward A9 Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

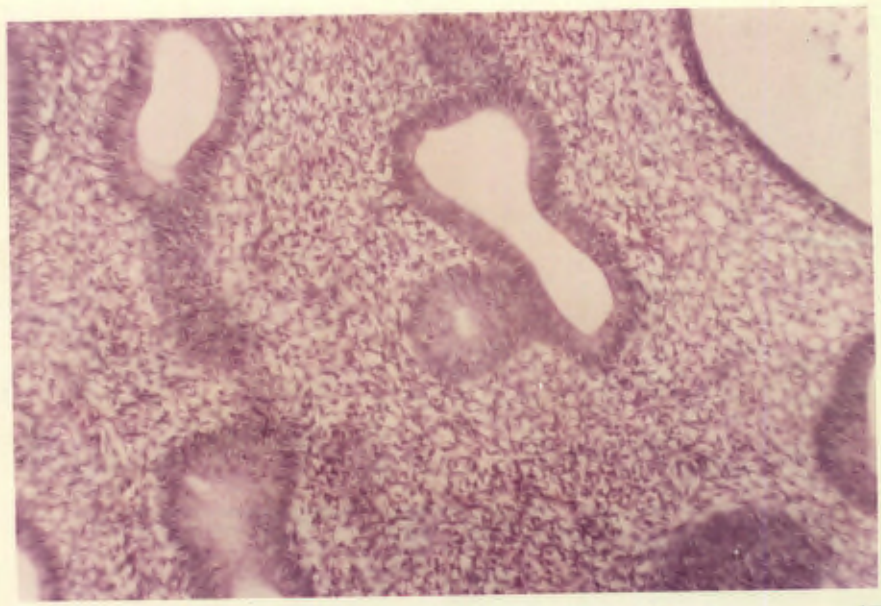


BLOOD SUGAR

Fasting 89 mgs.%.
½ hour 156 mgs.%.
1 hour 202 mgs.%.
1½ hour 183 mgs.%.
2 hour 138 mgs.%.
2½ hour
3 hour

J. J. V D WALT

Signature



18.

CASE 18.

Age 39 years. Cycle 4-7/28-35 day type with menorrhagia. D and C: hyperplastic cystic endometrium.

Age 45 years. C/O Menorrhagia: 2 years. Intermenstrual bleeding: 2 years. o/e Obese, Blood pressure 120/95, Hb 11.5. , 12-14 weeks fibroids. D&C 25/9/59: Large and proliferative glands - non-secretory endometrium.

19.

Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

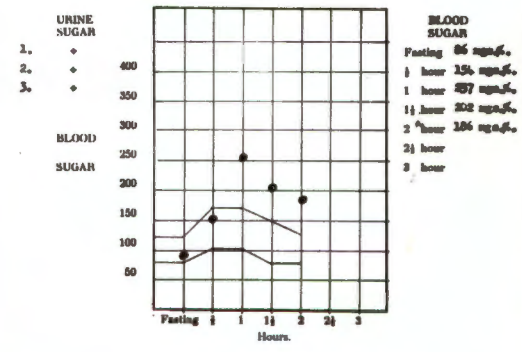
6562

Serial No. 33319 - 320. Date 25.9.59.

Patient's Identification HELEN GERBER (58/00492)

Ward G3C. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

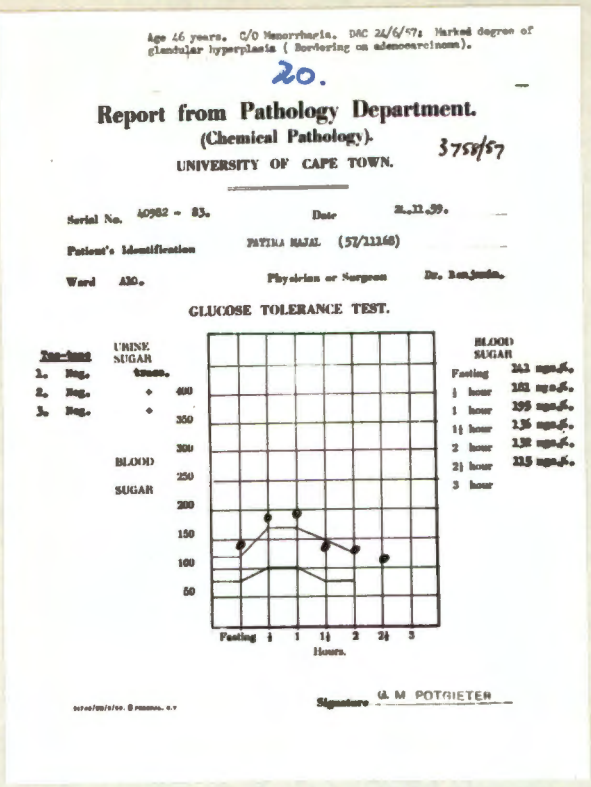


Signature G. M. POTGIETER

19.

CASE 19.

Age 45 years. Menorrhagia: 2 years. Intermenstrual bleeding: 2 years. Obese, blood pressure 120/95, Hb. 11.5.G., 12-14 weeks size fibroids. D and C 25/9/59: Large and proliferative glands - non-secretory endometrium.



Age 46 years. C/O Menorrhagia. D&C 24/6/57: Marked degree of glandular hyperplasia (Bordering on adenocarcinoma).

20.

Report from Pathology Department.

(Chemical Pathology). 375/57

UNIVERSITY OF CAPE TOWN.

Serial No. 40982 - 83. Date 24.12.59.

Patient's Identification PATIMA HAJAL (57/11168)

Ward A30. Physician or Surgeon Dr. Benjamin.

GLUCOSE TOLERANCE TEST.

Test-time	URINE SUGAR
1.	Neg.
2.	Neg.
3.	Neg.

	BLOOD SUGAR
Fasting	243 mgs.%
½ hour	283 mgs.%
1 hour	295 mgs.%
1½ hour	135 mgs.%
2 hour	132 mgs.%
2½ hour	215 mgs.%
3 hour	

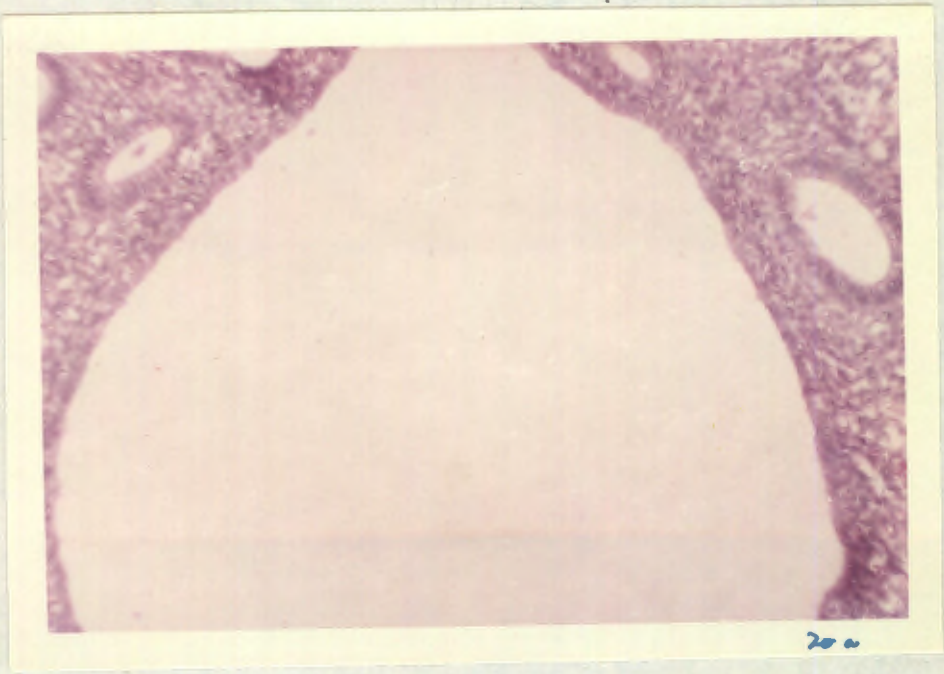
BLOOD SUGAR

400 350 300 250 200 150 100 50

Fasting ½ 1 1½ 2 2½ 3

Hours.

Signature G. M. POTGIETER

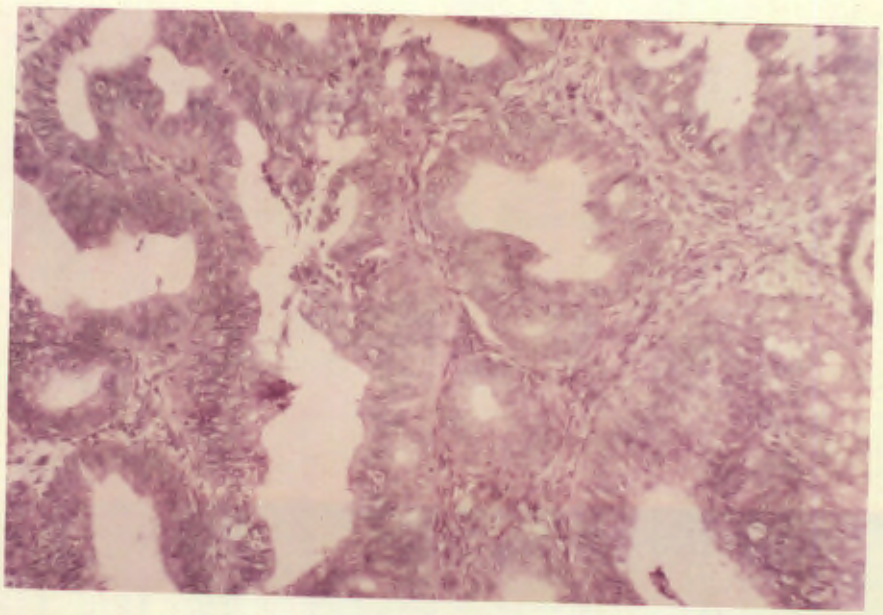


20 a

CASE 20.

Age 46 years. Complained of menorrhagia. D and C 24/6/57: Marked degree of glandular hyperplasia (Bordering on adenocarcinoma).

CASE 20. (Photomicrograph b.).



20b

Age 49 years. Menopause 8 months before. C/O Post-menopausal bleeding. D&C 19/8/59: Proliferative endometrium - some hyperplasia.

21.

Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

5613

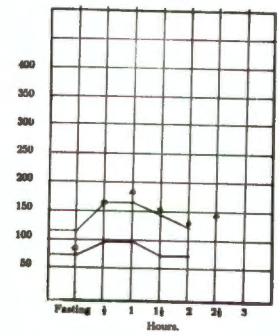
Serial No. 3673A - 35. Date 23.10.59.

Patient's Identification ELIZABETH FERREIRA (330899)

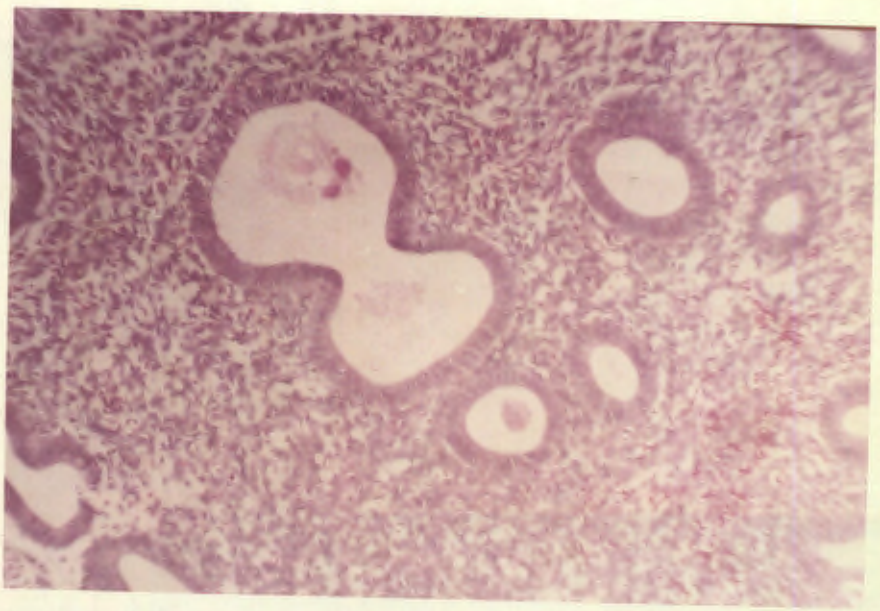
Ward A10. Physician or Surgeon Dr. Benjamin

GLUCOSE TOLERANCE TEST.

Fasting	URINE SUGAR	BLOOD SUGAR
1. 0	0	
2. 0	0	Fasting 109 mgs.%
3. 0	0	½ hour 180 mgs.%
		1 hour 159 mgs.%
		1½ hour 133 mgs.%
		2 hour 148 mgs.%
		2½ hour
		3 hour



Signature G. M. POTGIETER



21

CASE 21.

Age 49 years. Menopause 8 months before. Complained of post-menopausal bleeding. D and C 19/8/59: Proliferative endometrium - some hyperplasia.

Age 55 years. Menopause at 53½ year. C/O post-menopausal bleeding. D&C 27/7/59: Polyp showing benign glandular hyperplasia; (rest of endometrium also takes part in this process).

22.

Report from Pathology Department.

(Chemical Pathology). 5041 6262

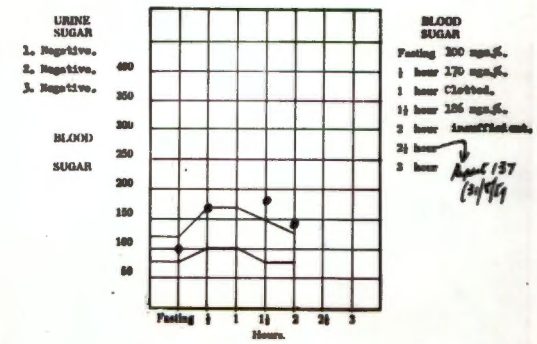
UNIVERSITY OF CAPE TOWN.

Serial No. 25001 - 2. Date 25.7.59.

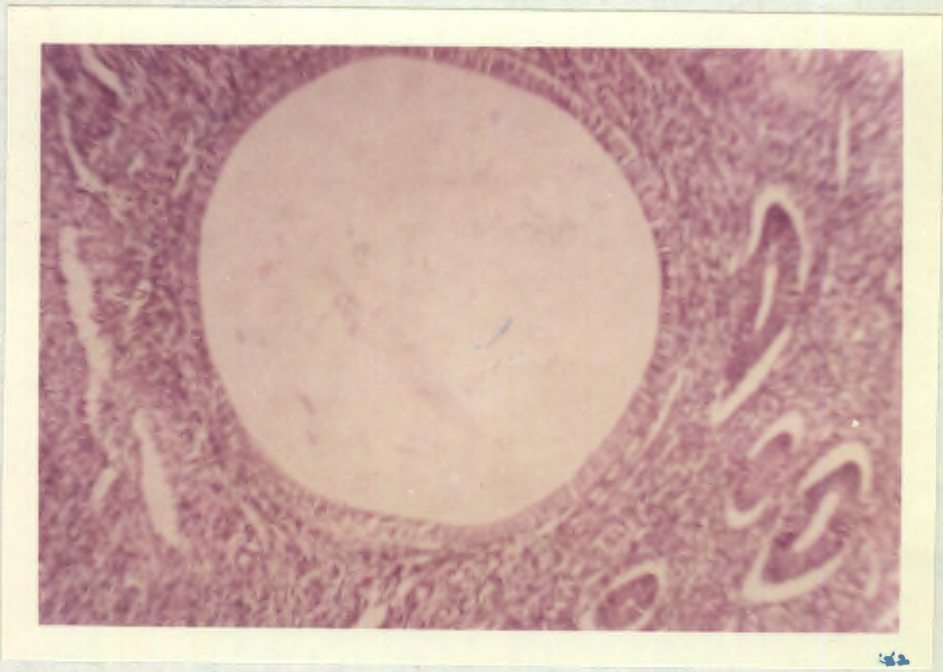
Patient's Identification MIRIL KATZ. (29/07ZR).

Ward GIO. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.



Signature A. M. POTGIETER



CASE 22.

Age 55 years. Menopause at 53 years. Complained of postmenopausal bleeding. D and C 27/7/59: polyp showing benign glandular hyperplasia; (rest of endometrium also takes part in this process).

Age 51 years. Amenorrhoea followed by continual bleeding for 3 months. D&C 2/2/59 : Benign Glandular Hyperplasia.

23.

Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

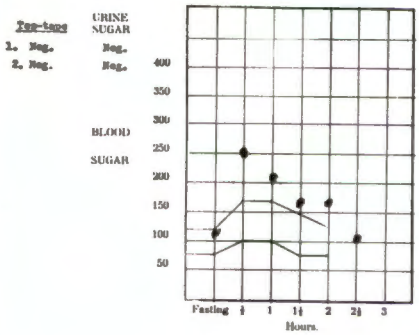
Serial No. 42713 - 14. Date 3.12.59.

Patient's Identification HENA KHOZE (59/08783)

Ward Alo. Physician or Surgeon Dr. Benjamin.

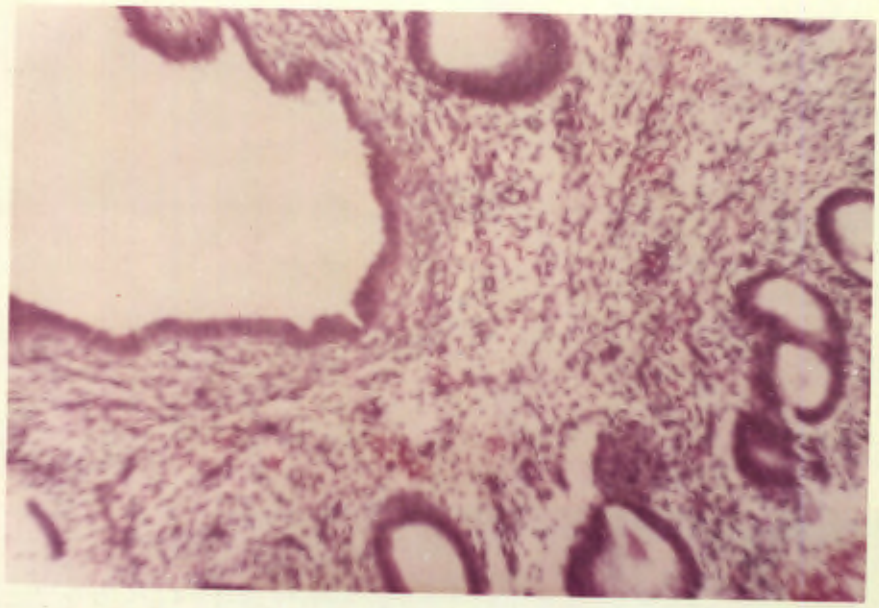
GLUCOSE TOLERANCE TEST.

Test-taps	URINE SUGAR
1. Neg.	Neg.
2. Neg.	Neg.



BLOOD SUGAR	
Fasting	117 mgs.%.
½ hour	253 mgs.%.
1 hour	221 mgs.%.
1½ hour	163 mgs.%.
2 hour	166 mgs.%.
2½ hour	102 mgs.%.
3 hour	

Signature G M [illegible]ETER



23

CASE 23.

Age 51 years. Amenorrhoea followed by continual bleeding for 3 months. D and C 2/2/59: benign glandular hyperplasia.

Age 25 years. Bouts of amenorrhoea followed by bouts of profuse bleeding (also hirsutism and infertility). D&C (i) 30th. day of cycle - proliferative endometrium. D&C (ii) Cystic endometrial hyperplasia.

24.

Report from Pathological Department.

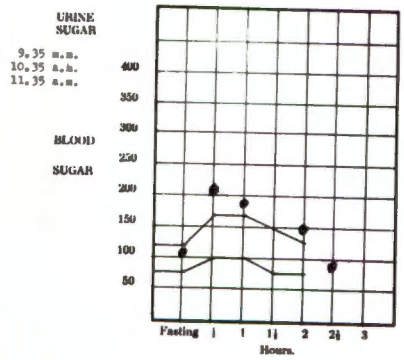
UNIVERSITY OF CAPE TOWN.

Serial No. 1226 Date 17.1.56

Patient's Identification DORO..Y ST ..IDT.

Ward C10' Physician or Surgeon Prof Louw.

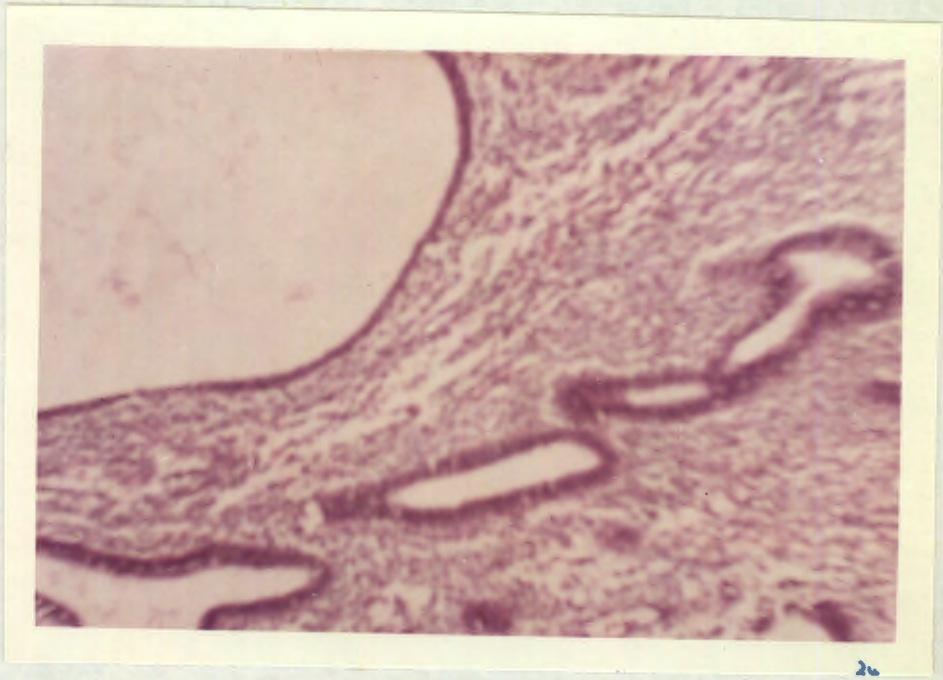
GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting	111 mgs.%.
½ hour	212 mgs.%.
1 hour	180 mgs.%.
1½ hour	No specimen
2 hour	No specimen.
2½ hour	86 mgs.%.
3 hour	

Signature



24

CASE 24.

Age 25 years. Bouts of amenorrhoea followed by bouts of profuse bleeding (also hirsutism and infertility). D and C (i) 30th. day of cycle - proliferative endometrium. D and C (ii) Cystic endometrial hyperplasia.

Age 45 years. Continual vaginal bleeding 2 months. D&C 5/12/59: Cystic endometrial hyperplasia.

25.

Report from Pathology Department.

(Chemical Pathology).

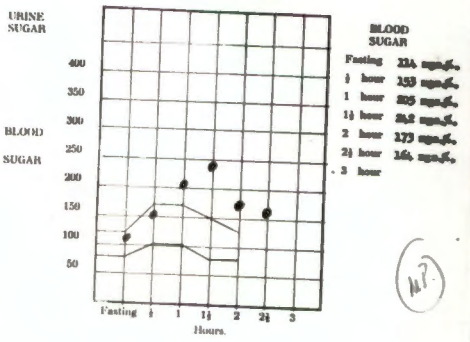
UNIVERSITY OF CAPE TOWN.

Serial No. A2288 - 59 Date 4.12.59.

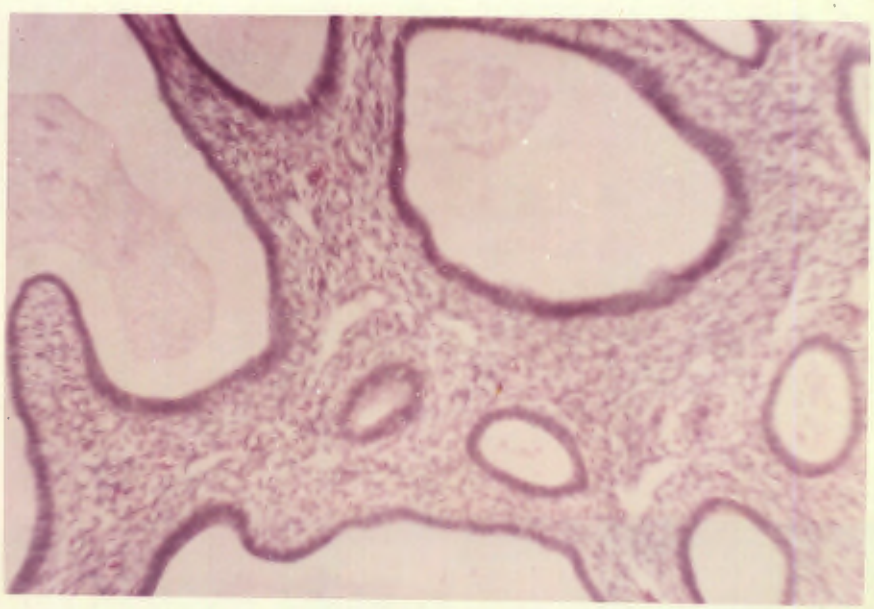
Patient's Identification MACHLETHA BARNARD (38/2830)

Ward A30. Physician or Surgeon Dr. Benjamin

GLUCOSE TOLERANCE TEST.



Signature



25

CASE 25.

Age 45 years. Continual vaginal bleeding 2 months. D and C 5/12/59: cystic endometrial hyperplasia.

Age 50 years. C/O Bouts of amenorrhoea (up to 7 months) followed by bouts of menorrhagia. D&C 17/6/59: benign glandular hyperplasia - glands show active proliferation and no evidence of secretory activity; marked irregularity in size and disposition of the glands.

26.

Report from Pathology Department.

(Chemical Pathology).

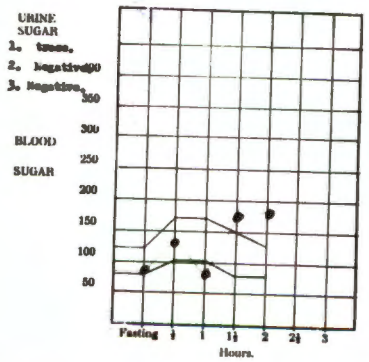
UNIVERSITY OF CAPE TOWN.

Serial No. 19197 - 28. Date 24.6.59.

Patient's Identification MR IRINA FLORIS (243490).

Ward A9 Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

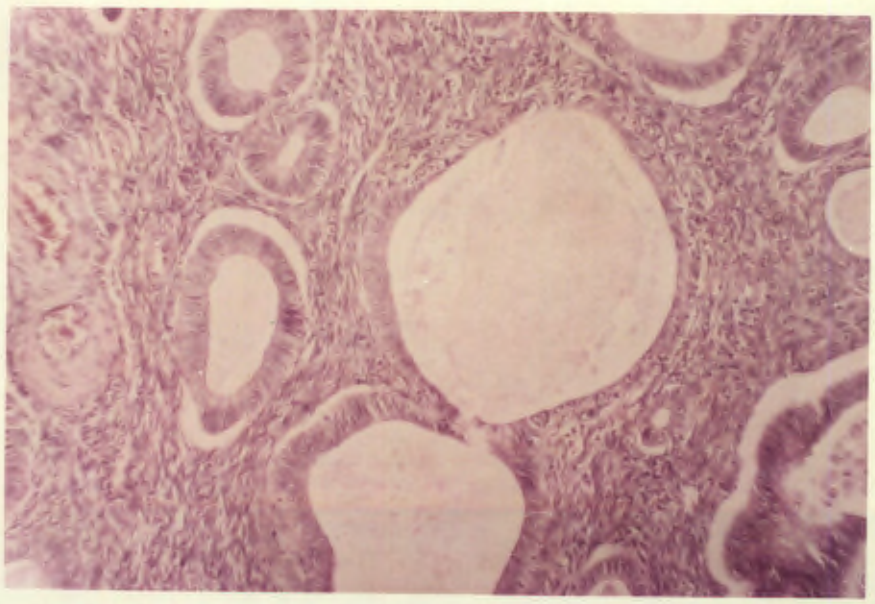


BLOOD SUGAR

Fasting	88 mgm.%.
½ hour	141 mgm.%.
1 hour	82 mgm.%.
1½ hour	135 mgm.%.
2 hour	105 mgm.%.
2½ hour	
3 hour	

S. J. V. D. WALT

Signature



26.

CASE 26.

Age 50 years. Complained of bouts of amenorrhoea (up to 7 months) followed by bouts of menorrhagia. D and C 17/6/59: benign glandular hyperplasia - glands show active proliferation and no evidence of secretory activity; marked irregularity in size and disposition of the glands.

Age 51 years. Menorrhagia few years, worse 1 year (hb 5.5G)
Fibroids size 14 weeks. D&C 15/6/59 - 21st day of 26 day cycle :
Hyperplastic proliferative endometrium - moderately dilated glands.

27 (a)

Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

19/6/59
3 9 77.

Serial No. 1987a - 275. Date 11.6.59.

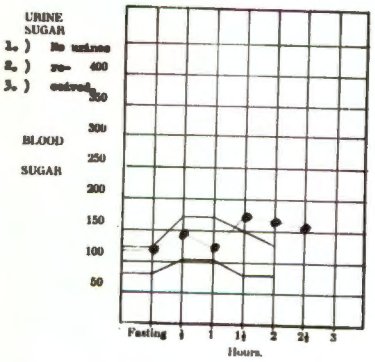
Patient's Identification MRS. C. KAPLAN (59/06249)

Ward C10. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

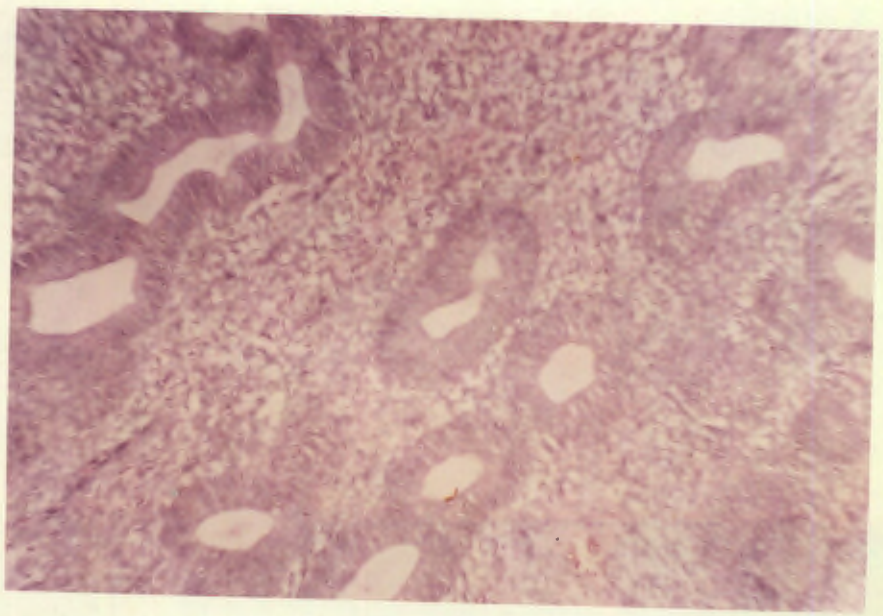
URINE SUGAR
1.) No sugar
2.) Pre- 400
3.) centred.

BLOOD SUGAR



BLOOD SUGAR
Fasting 122 mgms%.
½ hour 149 mgms%.
1 hour 122 mgms%.
1½ hour 178 mgms%.
2 hour 169 mgms%.
2½ hour 156 mgms%.
3 hour

J. J. V. D. WALT
Signature



27.

CASE 27.

Age 51 years. Menorrhagia few years, worse 1 year (hb. 5.5g.) Fibroids size 14 weeks. D and C 15/6/59:- 21st. day of 26 day cycle: hyperplastic proliferative endometrium - moderately dilated glands.

Age 63 years. C/O Post-menopausal bleeding: 2 months. (Menopause at 51 years.). 15/6/59 D&C: Actively hyperplastic - benign glandular hyperplasia - some atypically ? non-benign. Hysterectomy 29/6/59 Same histological features, but no definite malignancy.

28.

Report from Pathology Department.

(Chemical Pathology).

UNIVERSITY OF CAPE TOWN. 3475

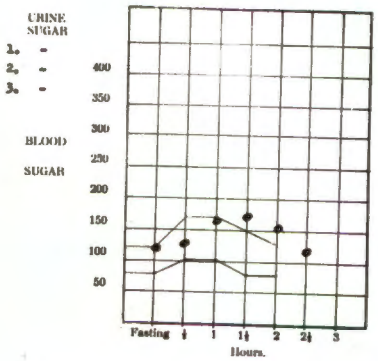
Serial No. 2152 - 53. Date 29.6.59.

Patient's Identification MRS. WILHELMINA C. BURGER (54088)

Ward CMO. Physician or Surgeon Prof Louw.

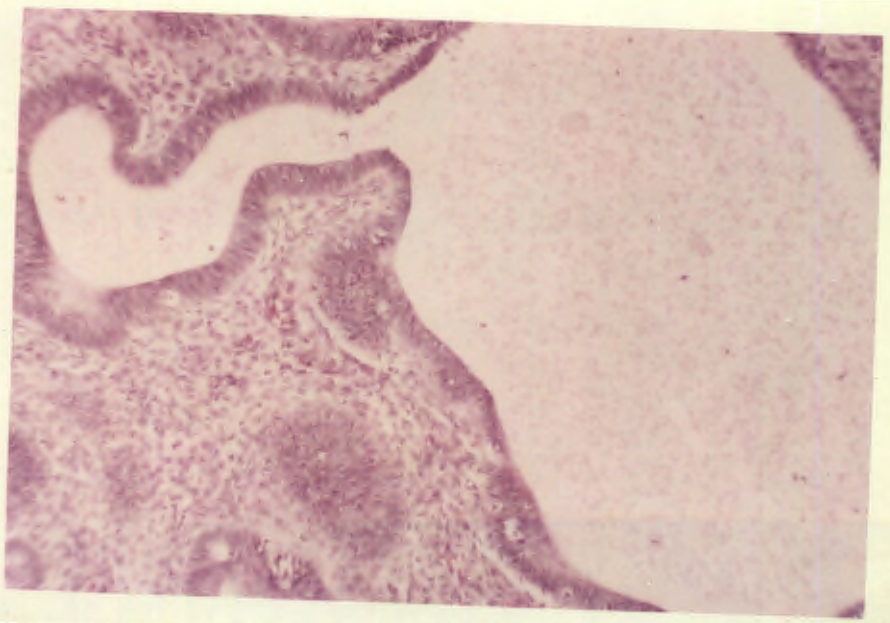
GLUCOSE TOLERANCE TEST.

URINE SUGAR
1. -
2. -
3. -



BLOOD SUGAR
Fasting 134 mgms%.
½ hour 134 mgms%.
1 hour 163 mgms%.
1½ hour 176 mgms%.
2 hour 158 mgms%.
2½ hour 122 mgms%.
3 hour

Signature L. ANSTEY.



28

CASE 28.

Age 63 years. Complained of postmenopausal bleeding: 2 months. (Menopause at 51 years). 15/6/59 D and C: Actively hyperplastic-benign glandular hyperplasia - some atypicality. Hysterectomy 29/6/59: same histological features, but no definiate malignancy.

Age 51 years. C/O post-menopausal bleeding. O/E Uterus 14 weeks - fibroid. 30/7/59 D&C: Endometrium shows glands in which there are mitotic figures, the general picture resembles that seen in hyperplastic proliferative endometrium. Tendency towards slight dilatation of glands. No malignancy.

29.

[illegible] Pathology Department.

(Chemical Pathology).

UNIVERSITY OF CAPE TOWN.

5/58 5/59

Serial No. 26289 - 210. Date 3.8.59.

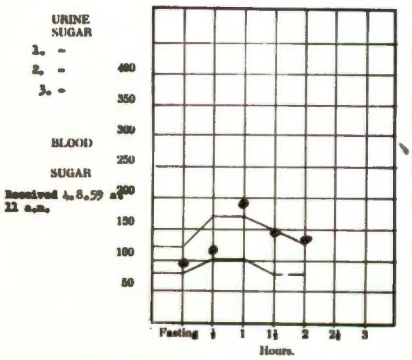
Patient's Identification SUSAN PEP BS (19273)

Ward B7. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

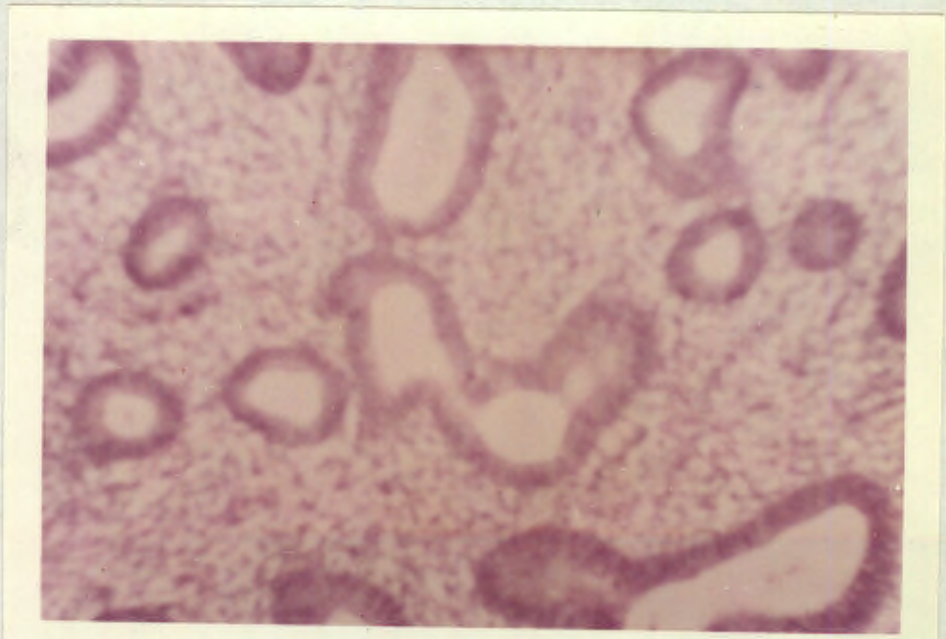
URINE SUGAR
1. -
2. -
3. -

BLOOD SUGAR
Received 4.8.59 at 11 a.m.



BLOOD SUGAR
Fasting 95 mgs.%.
½ hour 122 mgs.%.
1 hour 193 mgs.%.
1½ hour 148 mgs.%.
2 hour 139 mgs.%.
2½ hour
3 hour

Signature G. M. POTGIETER



20.

CASE 29.

Age 51 years. Postmenopausal bleeding. Uterus 14 weeks size fibroid. 30/7/59 D and C: Mitotic figures; picture resembles that seen in hyperplastic proliferative endometrium. Tendency towards slight dilatation of glands. No malignancy.

Age 52 years. C/O Menorrhagia: 1 year. Biopsy: Endocervical carcinoma. Wertheim Hysterectomy 26/8/59 : Endocervical carcinoma. Endometrium hyperplastic and cystic.

30.

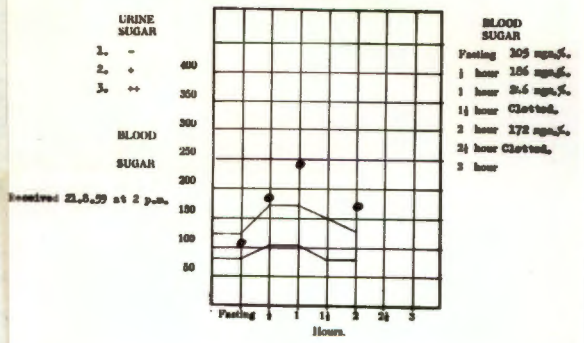
Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

Serial No. 29085 - 6. Date 21.8.59.

Patient's Identification Mrs. C.M. ABRT. (58/6855A.)

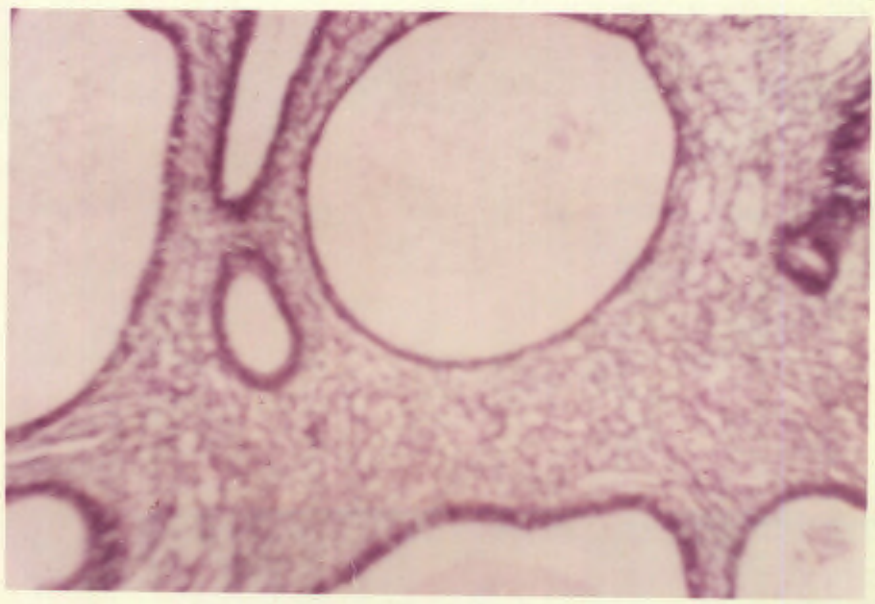
Ward C10. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.



Received 21.8.59 at 2 p.m.

Signature G. M. POTGIETER

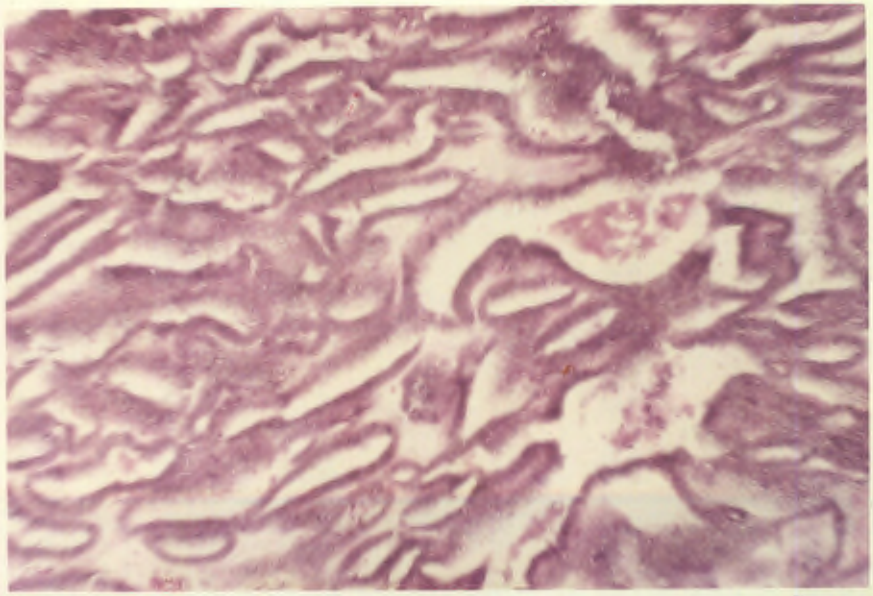


306

CASE 30.

Age 52 years. Complained of menorrhagia: 1 year. Biopsy: Endocervical carcinoma. Wertheim hysterectomy 26/8/59: Endocervical carcinoma. Endometrium hyperplastic and cystic.

CAS E 30. (Photomicrograph b.).
(Endocervical Carcinoma).



30a

Age 57 years. C/O Post-menopausal bleeding. Hyperplastic cystic endometrium.

31.

Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

Serial No. 29083 - 4. Date 22.8.39.

Patient's Identification D. ELST. (17730)

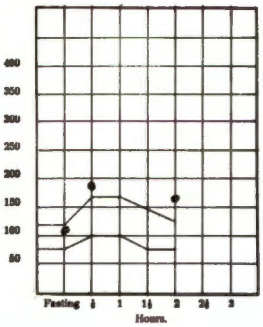
Ward GYO. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR
1. +
2. -
3. -

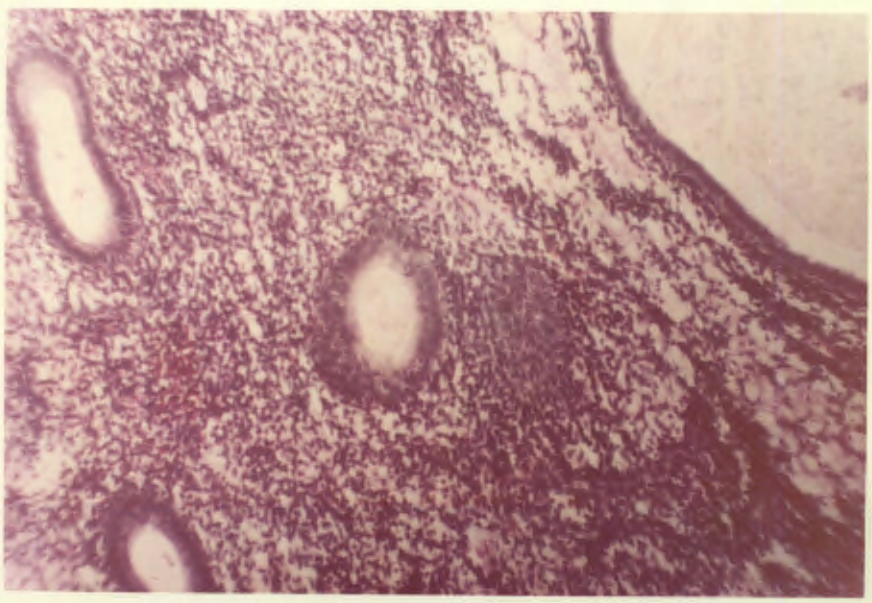
BLOOD SUGAR

Received 22.8.39 at 2 p.m.



BLOOD SUGAR
Fasting 115 mgm.%.
½ hour 195 mgm.%.
1 hour Clotted.
1½ hour Clotted.
2 hour 170 mgm.%.
2½ hour Clotted.
3 hour

G. M. POTGIETER
Signature



31.

CASE 31.

Age 57 years. Complained of postmenopausal bleeding. Hyperplastic cystic endometrium.

Age 49 years. Menorrhagia. Fibroids size 14 weeks. Endometrial hyperplasia. On the 26th. day of cycle D&C;- then Hysterectomy.

32.

Report from Pathological Department. 2254

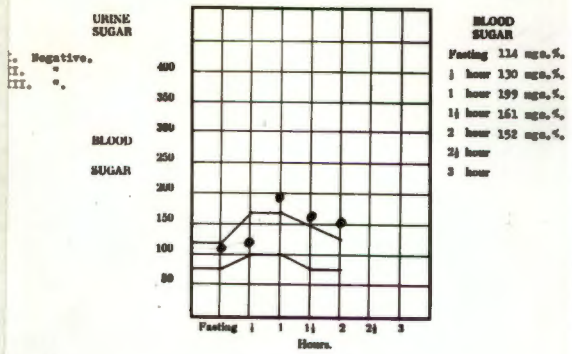
UNIVERSITY OF CAPE TOWN.

Serial No. 9261-62. Date 23.3.58.

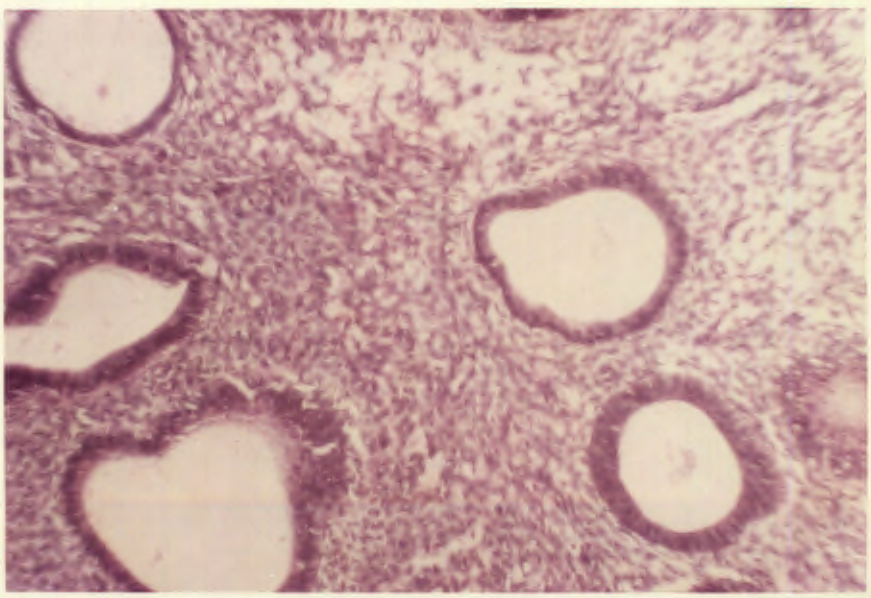
Patient's Identification EILEEN JONES. (58/06059)

Ward E7. Physician or Surgeon Prof. Louw.

GLUCOSE TOLERANCE TEST.



Signature.



32.

CASE 32.

Age 49 years. Menorrhagia. Fibroids size 14 weeks. Endometrial hyperplasia. On the 26th. day of cycle D and C:- then hysterectomy.

Age 53 years. Menorrhagia: 2 months, followed by 8 weeks amenorrhoea. Hyperplastic endometrium of benign glandular hyperplasia. (D&C and Hysterectomy) (Also small fibroids.).

33.

Report from Pathological Department.

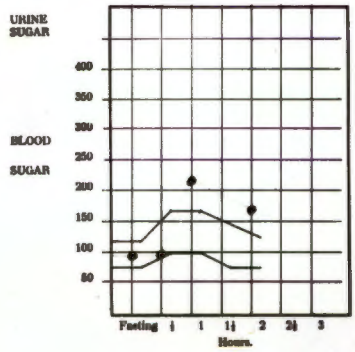
UNIVERSITY OF CAPE TOWN.

Serial No. 33521-24/57 Date 8.10.57.

Patient's Identification BEATRICE SALMEGRAVE. (57/21868).

Ward A9 Physician or Surgeon Prof Louw.

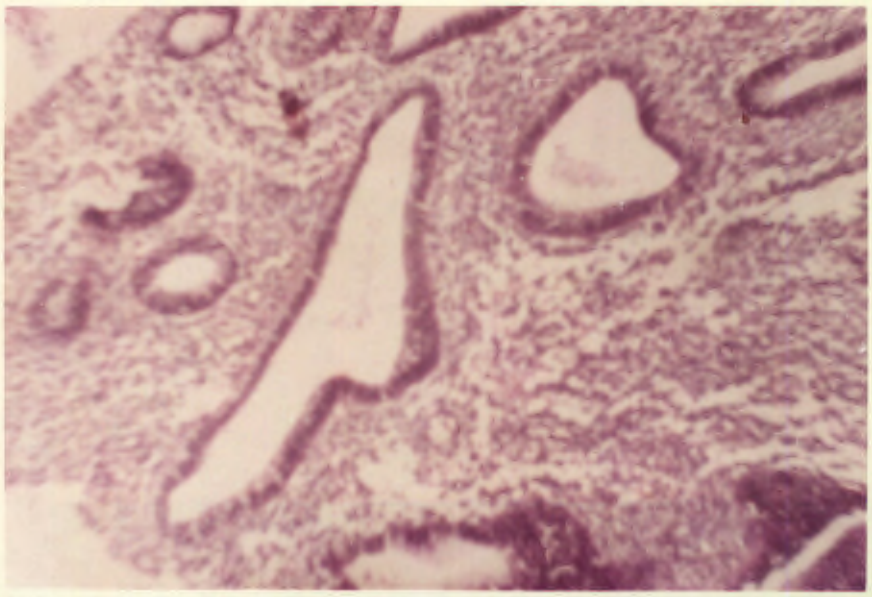
GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting 97 mgs.%.
½ hour 97 mgs.%.
1 hour 235 mgs.%.
1½ hour
2 hour 173 mgs.%.
2½ hour
3 hour

Signature



33.

CASE 33.

Age 53 years. Menorrhagia: 2 months, followed by 8 weeks amenorrhoea. D and C and hysterectomy: hyperplastic endometrium of benign glandular hyperplasia, and small fibroids.

Age 52 years. Menorrhagia: 3 months. D&C and then Hysterectomy : Benign glandular hyperplasia, and small fibroid.

34.

Report from Pathological Department.

UNIVERSITY OF CAPE TOWN.

Serial No. C 14663- 64. Date 22.5.56.

Patient's Identification JEANNIE RAE (56/ 06187)

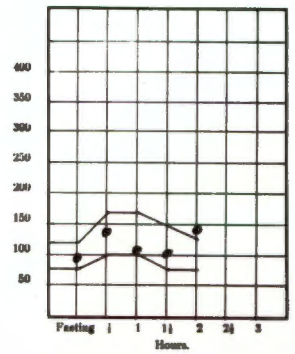
Ward O 10. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR

1. +
2. Trace.
3. Trace .

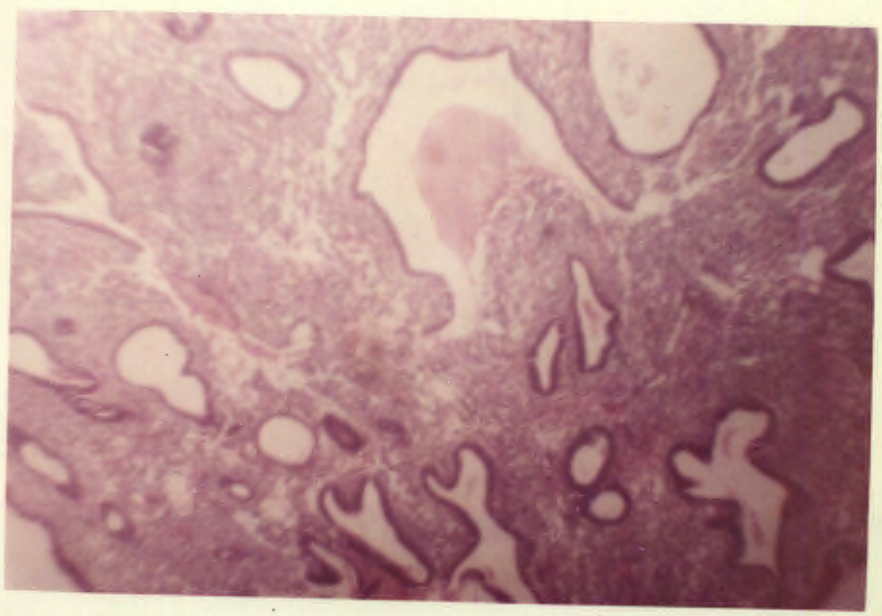
BLOOD SUGAR



BLOOD SUGAR

Fasting	98 mgs.%
½ hour	134 mgs.%
1 hour	110 mgs.%
1½ hour	107 mgs.%
2 hour	141 mgs.%
2½ hour	
3 hour	

Signature.



34.

CASE 34.

Age 52 years. Menorrhagia: 3 months. D and C and then hysterectomy: benign glandular hyperplasia, and small fibroid.

Age 52 years. Menorrhagia and irregular menstruation: 8 months. D&C and hysterectomy: Marked endometrial hyperplasia - also a fibroid. [illegible] Hospital. 35.

Report from Pathological Department.

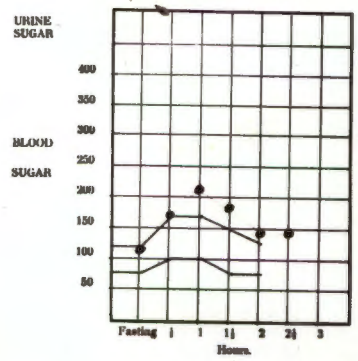
UNIVERSITY OF CAPE TOWN.

Serial No. C 20624 - 25 Date 16.7.56.

Patient's Identification MRS METTA HEPP. (56/07723)

Ward C 10 Physician or Surgeon Prof. Louw.

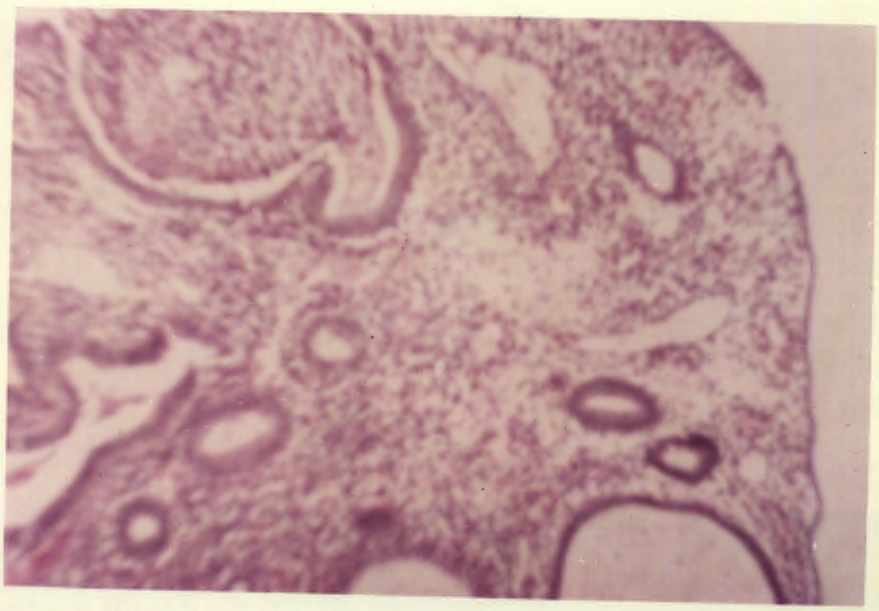
GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting 111 mgs.%
1 hour 168 mgs.%
1 hour 206 mgs.%
1½ hour 182 mgs.%
2 hour 143 mgs.%
2½ hour 146 mgs.%
3 hour

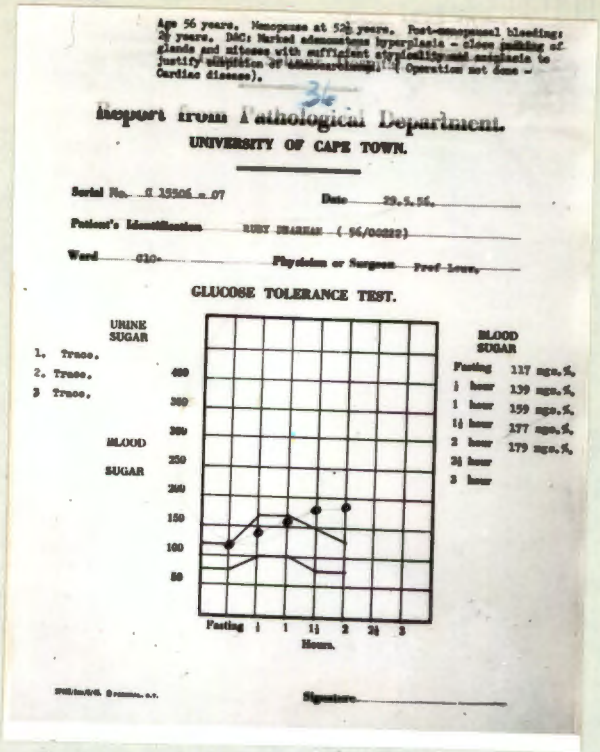
[illegible] Signature



35.

CASE 35.

Age 52 years. Menorrhagia and irregular menstruation: 8 months. D and C and hysterectomy: Marked endometrial hyperplasia - also a fibroid.



Age 56 years. Menopause at 52½ years. Post-menopausal bleeding: 2½ years. D&C: Marked adenomatous hyperplasia - close packing of glands and mitoses with sufficient atypicality and anaplasia to justify suspicion of adenocarcinoma. (Operation not done - Cardiac disease).

36

Report from Pathological Department.

UNIVERSITY OF CAPE TOWN.

Serial No. S 19506 - 07 Date 29.5.56.

Patient's Identification RUBY SHARRAH (56/00222)

Ward G10. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR

1. Trace.
2. Trace.
3 Trace.

BLOOD SUGAR

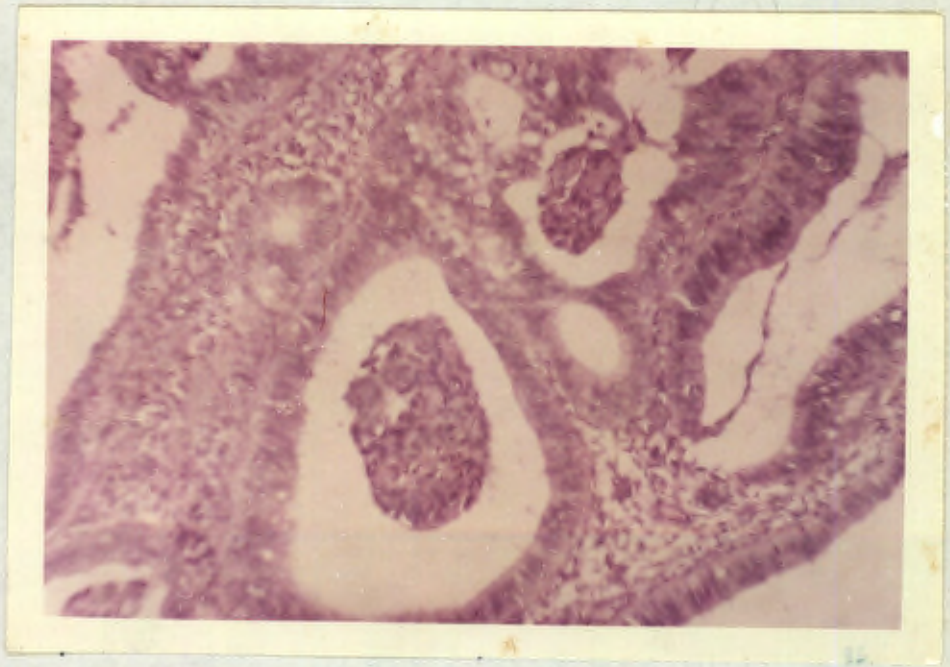
400
300
200
250
200
150
100
50

Fasting 1 1 1½ 2 2½ 3
Hours.

BLOOD SUGAR

Fasting 117 mgs.%
½ hour 139 mgs.%
1 hour 159 mgs.%
1½ hour 177 mgs.%
2 hour 179 mgs.%
2½ hour
3 hour

Signature



CASE 36.

Age 56 years. Menopause at 52 years. Postmenopausal bleeding: 2½ years. D and C: Marked adenomatous hyperplasia - close packing of glands and mitoses with sufficient atypicality for suspicion of adenocarcinoma. (Operation not done - cardiac disease).

Age 57 years. Post-menopausal bleeding: 1 year. D&C and Hysterectomy: Fibroid, and non-secretory hyperplastic cystic endometrium with compact stroma.

37.

Report from Pathological Department.

UNIVERSITY OF CAPE TOWN.

Serial No. C 29497 - 98 Date 2.10.56

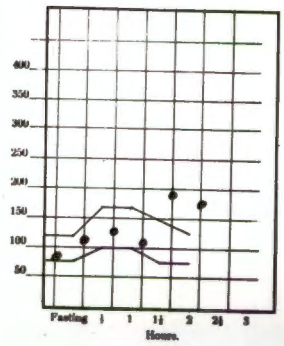
Patient's Identification MRS. ALETTA ZEHNER (56/11273).

Ward C 10 Physician or Surgeon Prof Louw

GLUCOSE TOLERANCE TEST.

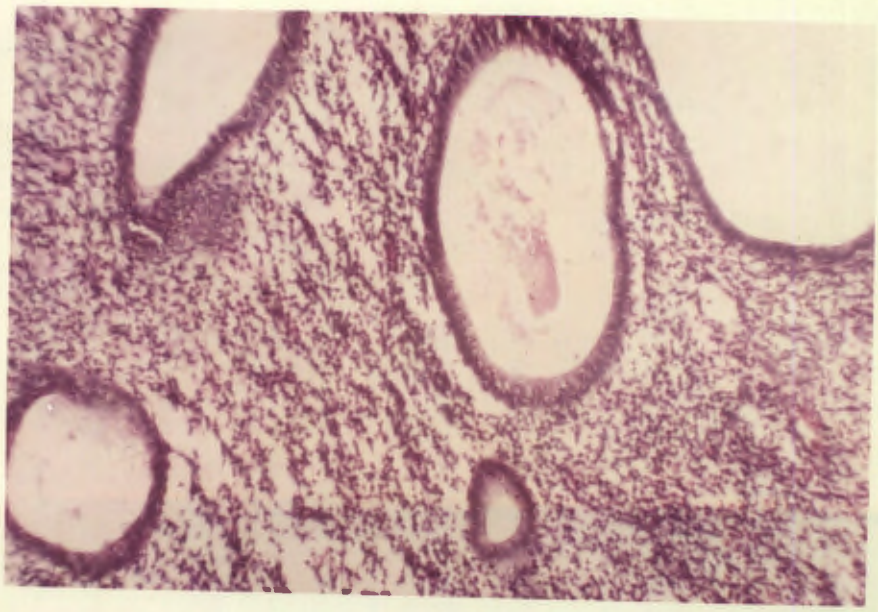
URINE SUGAR
1. Negative
2. Negative.
3. Negative

BLOOD SUGAR



BLOOD SUGAR
Fasting 76 mgs.%
½ hour 111 mgs.%
1 hour 133 mgs.%
1½ hour 101 mgs.%
2 hour 190 mgs.%
2½ hour 183 mgs.%
3 hour

Signature



37.

CASE 37.

Age 57 years. Postmenopausal bleeding: 1 year. D and C and hysterectomy: fibroid, and non-secretory hyperplastic cystic endometrium with compact stroma.

Age 54 years. C/O Post-menopausal bleeding: 2 months. 7/6/57 Total Hysterectomy: Fibroids and endometrium; benign endometrial polyp. Endometrium has a dense stroma but the glands are hyperplastic and cystic.

38.

Report from Pathological Department.

UNIVERSITY OF CAPE TOWN.

Serial No. 18836-37/57 Date 4.6.57

Patient's Identification KATHERINA LOUW. (57/05448).

Ward C 10. Physician or Surgeon Prof. Lewis

GLUCOSE TOLERANCE TEST.

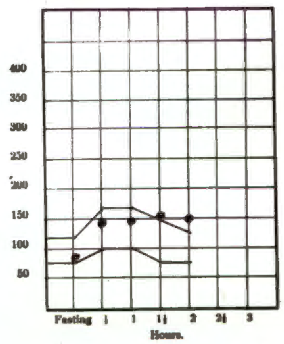
URINE SUGAR

o/o

o/o

o/o

BLOOD SUGAR

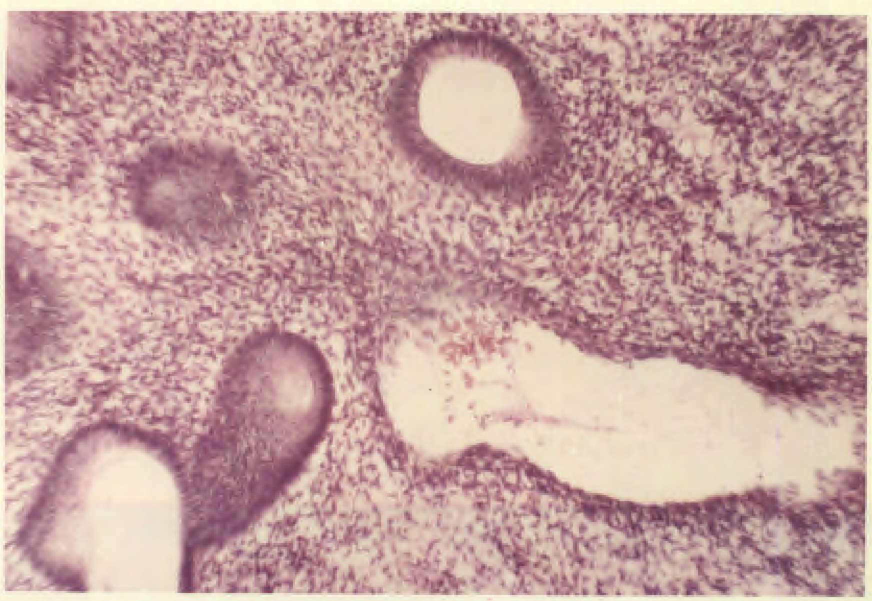


BLOOD SUGAR

Fasting 78 mgs.%
½ hour 147 mgs.%
1 hour 149 mgs.%
1½ hour 154 mgs.%
2 hour 149 mgs.%
2½ hour
3 hour

G. LINDER

Signature



38.

CASE 38.

Age 54 years. Postmenopausal bleeding: 2 months. 7/6/57 Total hysterectomy: fibroids and benign endometrial polyp. Endometrium has a dense stroma and the glands are hyperplastic.

Age 35 years. C/O Menorrhagia, polymenorrhoea and dysmenorrhoea: O/E Prolapse. Vaginal hysterectomy and repair: Benign Glandular hyperplasia.

39.

Report from Pathology Department.
(Chemical Pathology). 7/58
UNIVERSITY OF CAPE TOWN.

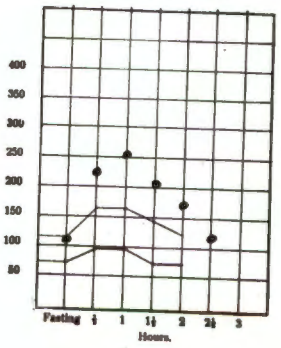
Serial No. 3638 - 53. Date 20.10.59.

Patient's Identification ELLA NICHOLSON (25/07975)

Ward G20. Physician or Surgeon Prof Louw.

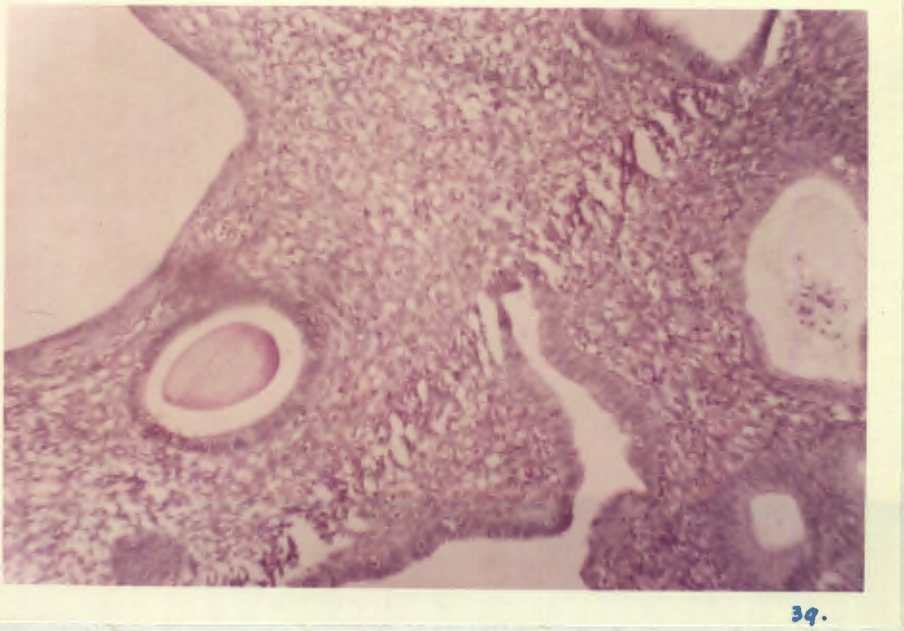
GLUCOSE TOLERANCE TEST.

Time-hours	URINE SUGAR
½	trace
1	++
2	++



BLOOD SUGAR
Fasting 224 mgms%.
½ hour 265 mgms%.
1 hour 296 mgms%.
1½ hour 283 mgms%.
2 hour 234 mgms%.
2½ hour 218 mgms%.
3 hour

Signature G. M. POTGIETER



39.

CASE 39.

Age 35 years. Complained of menorrhagia, polymenorrhoea and dysmenorrhoea: On examination prolapse. Vaginal hysterectomy and repair: benign glandular hyperplasia.

Age 34 years. Menorrhagia: D&C and hysterectomy. Benign glandular hyperplasia.

40.

Report from Pathological Department.

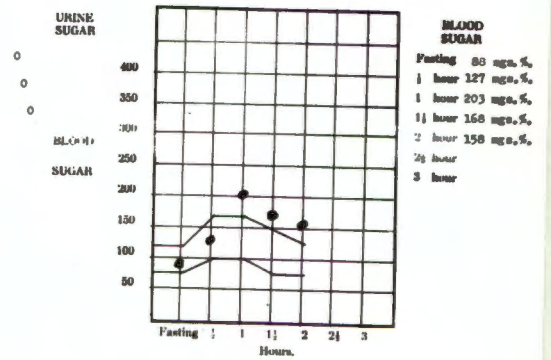
UNIVERSITY OF CAPE TOWN.

Serial No. 27740-41/57 Date 29.8.57.

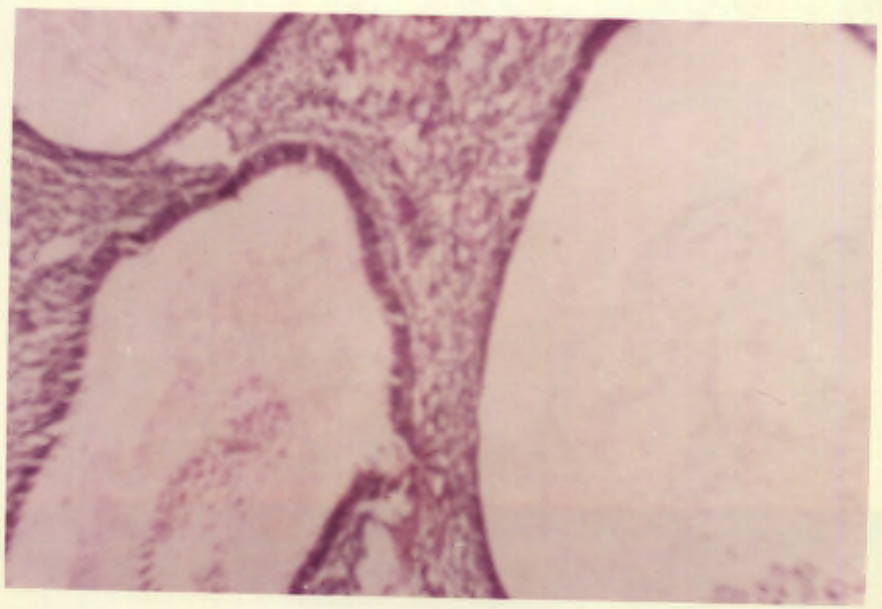
Patient's Identification MAGDALENE HENDRICKS. (57/19701).

Ward 27 Physician or Surgeon Prof. Louw.

GLUCOSE TOLERANCE TEST.



Signature



40.

CASE 40.

Age 34 years. Menorrhagia. D and C and hysterectomy: benign glandular hyperplasia.

Age 30 years. Intractable metropathia (continual menorrhagia since the age of 17 years - Menarche at 14 years) - 4 D&C's, all metropathia. Euthyroid, Obese.

41.

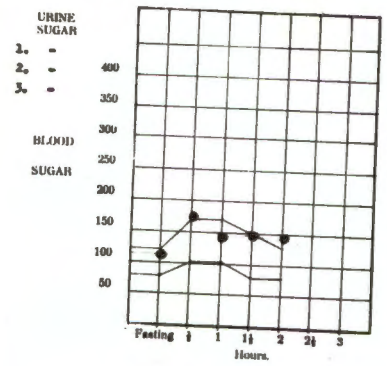
Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

Serial No. 21967 - 8. Date 1.7.59.

Patient's Identification RITA JACOBS.

Ward 230. Physician or Surgeon Dr. Benjamin.

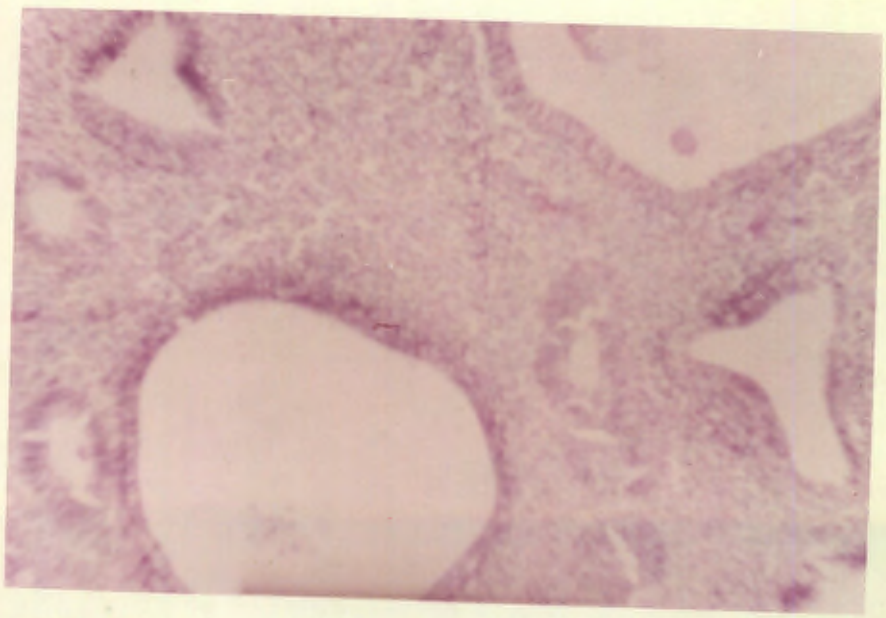
GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting	230 mgs.%.
½ hour	273 mgs.%.
1 hour	247 mgs.%.
1½ hour	249 mgs.%.
2 hour	246 mgs.%.
2½ hour	
3 hour	

Signature G. M. POTGIETER



41.

CASE 41.

Age 30 years. Intractable menorrhagia since the age of 17 years (Menarche at 14 years) - 4 D and C's, all metropathia. Euthyroid. obese.

BEFORE F.S.H. ADMINISTRATION.

Age 32 years. C/O Menorrhagia for 4 years following sterilization 4/11/59 Hysterectomy (13th. day of cycle) (Endometrium not reported on(Had D&C in George : Metropathia).

42 (a)

Report from Pathology Department.
(Chemical Pathology). M&A 7582.

UNIVERSITY OF CAPE TOWN.

Serial No. 3947 - 48 Date 13.11.59.

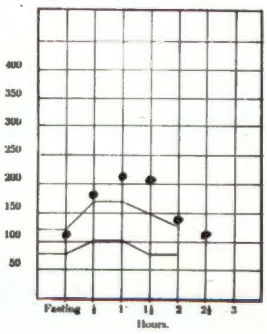
Patient's Identification MARIA VAN RIJSBURG (59/13885)

Ward Alt. Physician or Surgeon Dr. Benjamin.

GLUCOSE TOLERANCE TEST.

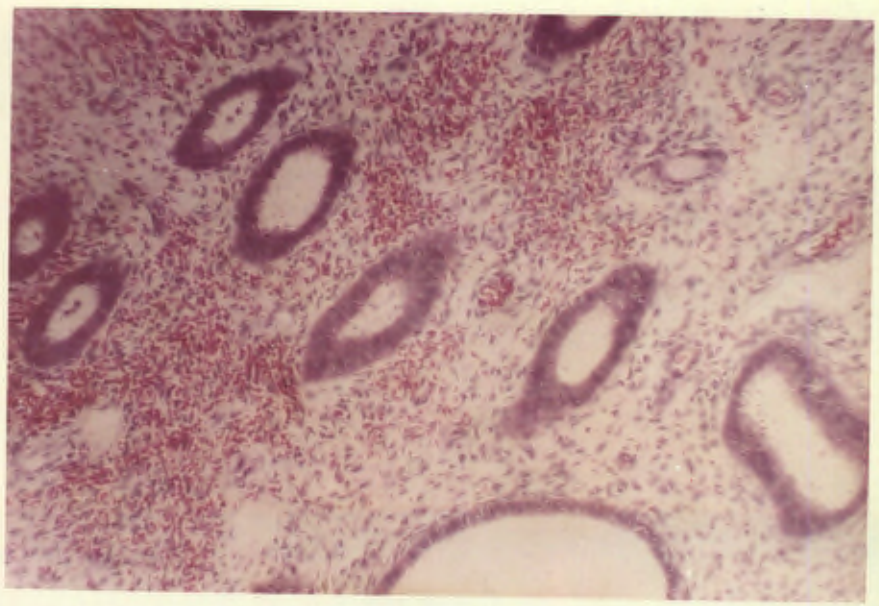
Time-taken	URINE SUGAR
2. 0	0
Specimen marked 2½ hours sugar	
72½ hours urine.	

BLOOD SUGAR



BLOOD SUGAR	
Fasting	135 mgm.%.
½ hour	183 m.g.%.
1 hour	229 mgm.%.
1½ hour	213 mgm.%.
2 hour	146 m.g.%.
2½ hour	117 m.g.%.
3 hour	

Signature G. M. POTGIETER



42.

CASE 42.

Age 32 years. Complained of menorrhagia for 4 years following sterilization. 4/11/59 hysterectomy: Metropathia.

Age 61 years. Menopause at 51 years. C/O Post-menopausal bleeding : few months. 24/7/56 D&C Endometrial tissue showing " retrogressive benign glandular hyperplasia. Glands show cystic dilatation but no evidence of epithelial hyperplasia. Stroma is inactive". 27/5/57 Hysterectomy: " Follicular cysts with hyperactive glandular cells. Endometrium shows benign glandular hyperplasia".

43.

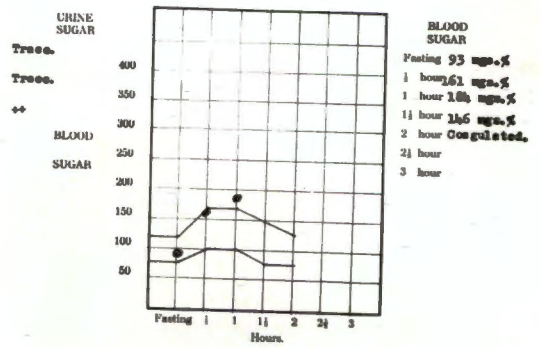
UNIVERSITY OF CAPE TOWN.

Serial No. 17951-52/57 Date 21. 5. 57

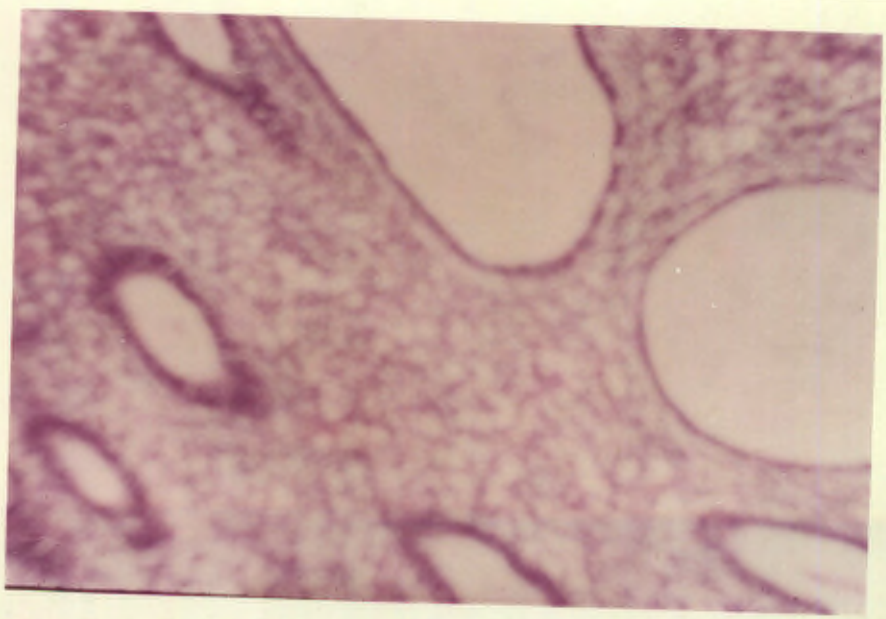
Patient's Identification GINA COROMA. (56/08686).

Ward G 10. Physician or Surgeon Prof. Louw.

GLUCOSE TOLERANCE TEST.



Signature



43.

CASE 43.

Age 61 years. Menopause at 51 years. Postmenopausal bleeding: few months. 24/7/56 D and C "Retrogressive benign glandular hyperplasia. Glands show cystic dilatation but no evidence of epithelial hyperplasia. Stroma is inactive". 27/5/57 hysterectomy.

Age 53 years. Menorrhagia: 9 months.
D&C and hysterectomy: - Marked benign glandular hyperplasia.
(Menopause not reached).

Report from Pathological Department.

UNIVERSITY OF CAPE TOWN.

Serial No. 3208?-8/57 Date 24.9.57

Patient's Identification JOHANNA GLOETE. (51243).

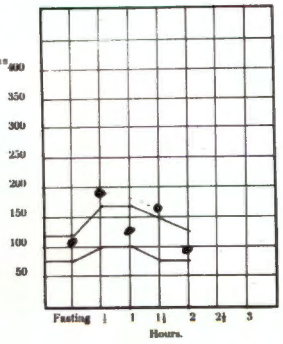
Ward 87 Physician or Surgeon Prof. Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR

Urine Specimens SUGAR %.

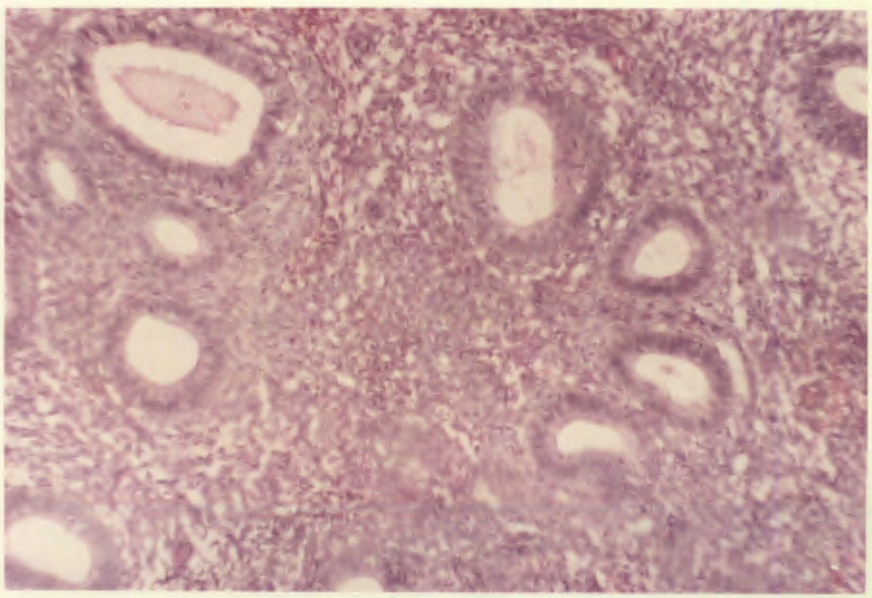
BLOOD SUGAR



BLOOD SUGAR

Fasting	107 mgs.%
½ hour	191 mgs.%
1 hour	122 mgs.%
1½ hour	169 mgs.%
2 hour	85 mgs.%
2½ hour	
3 hour	

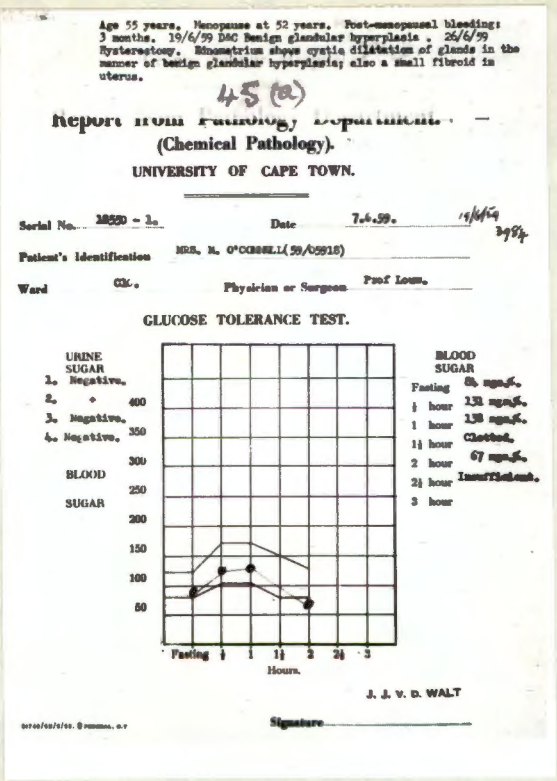
Signature



44

CASE 44.

Age 53 years. Menorrhagia: 9 months. D and C and hysterectomy: marked glandular hyperplasia (Menopause not reached.).



Age 55 years. Menopause at 52 years. Post-menopausal bleeding: 3 months. 19/6/59 D&C Benign glandular hyperplasia . 26/6/59 Hysterectomy. Endometrium shows cystic dilatation of glands in the manner of benign glandular hyperplasia; also a small fibroid in uterus.

45 (a)

Report from Pathology Department.
(Chemical Pathology).

UNIVERSITY OF CAPE TOWN.

Serial No. 2859 - 2. Date 7.6.59. 4/6/59 3484

Patient's Identification MRS. M. O'CONNELL (59/05918)

Ward CM. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR
1. Negative.
2. +
3. Negative.
4. Negative.

BLOOD SUGAR

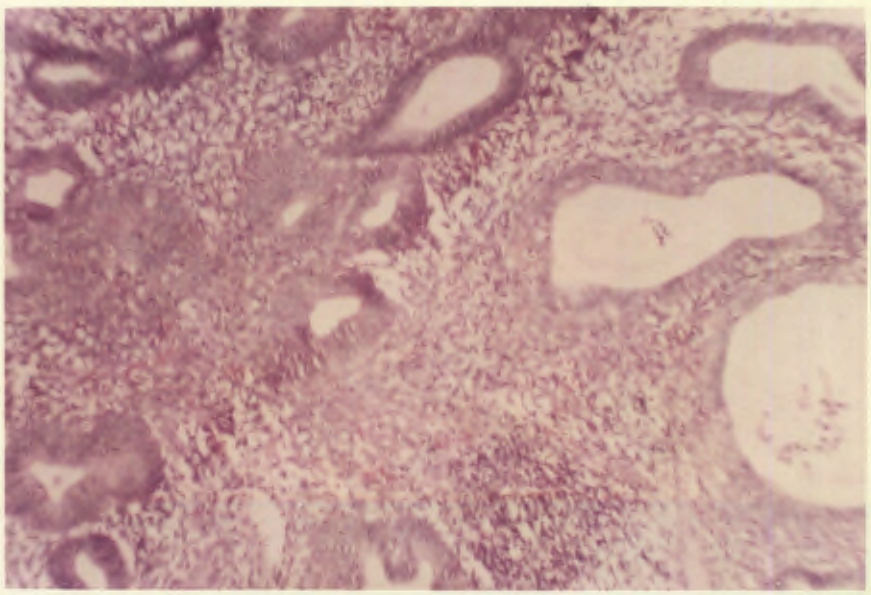
400
350
300
250
200
150
100
50

Fasting ½ 1 1½ 2 2½ 3
Hours.

BLOOD SUGAR
Fasting 81 mgms%.
½ hour 131 mgms%.
1 hour 138 mgms%.
1½ hour Clotted.
2 hour 67 mgms%.
2½ hour Insufficient.
3 hour

J. J. V. D. WALT

Signature

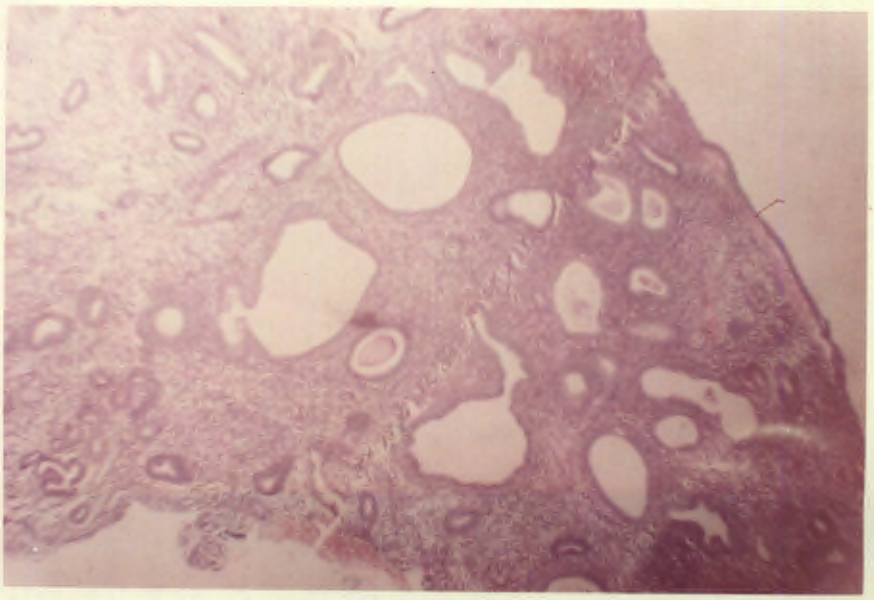


45 a

CASE 45.

Age 55 years. Menopause at 52 years. Postmenopausal bleeding: 3 months. 19/6/59 D and C: Benign glandular hyperplasia. 26/6/59 hysterectomy: endometrium shows cystic dilatation of glands in the manner of benign glandular hyperplasia; also a small fibroid in uterus.

CASE 45. (Photomicrograph b.).



Age 61 years. Post-menopausal bleeding. Hyperplastic endometrium.

— 46. —

7589.

Report from Pathological Department.

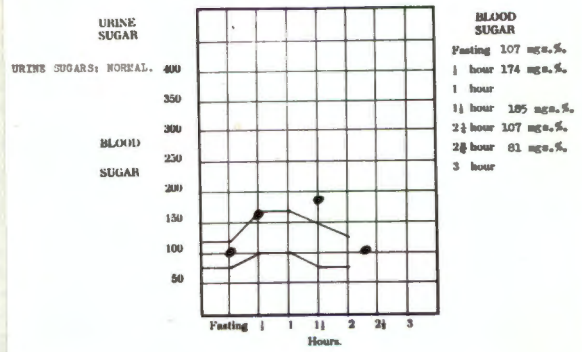
UNIVERSITY OF CAPE TOWN.

Serial No. 38782-83 Date 11.11.58.

Patient's Identification S. GEORGE. (10741)

Ward A9 Physician or Surgeon Prof Louw.

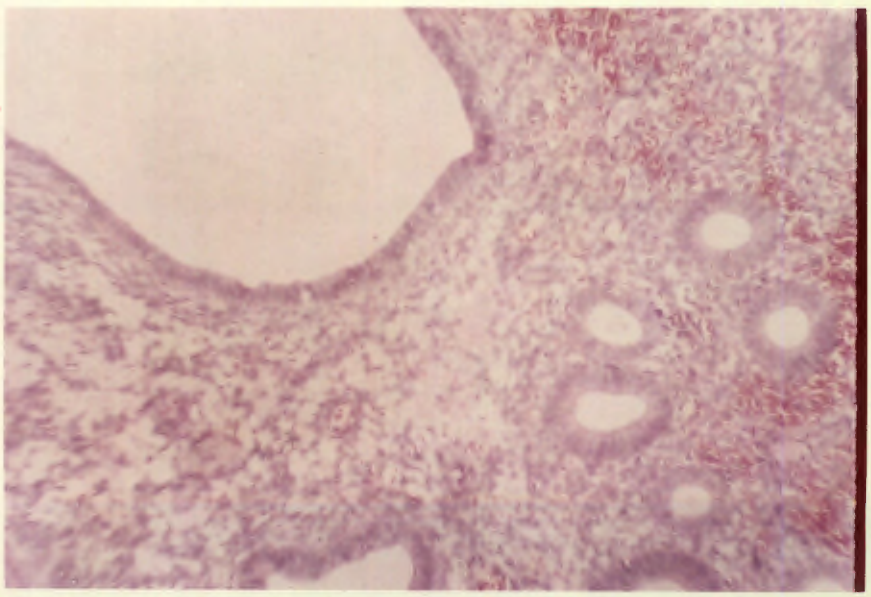
GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting 107 mgs.%.
¼ hour 174 mgs.%.
1 hour
1½ hour 185 mgs.%.
2½ hour 107 mgs.%.
2¾ hour 81 mgs.%.
3 hour

Signature



46.

CASE 46.

Age 61 years. Postmenopausal bleeding. Hyperplastic endometrium.

Abe 57 years. C/O Menorrhagia: 1 year. 17/3/59 D&C, 7/10/59
Hysterectomy: Small fibroids and benign glandular hyperplasia.

47.

Glandular Hyperplasia.

Report from Pathology Department. 6917

(Chemical Pathology).

UNIVERSITY OF CAPE TOWN.

Serial No. 26384 - 25 Date 20.10.59.

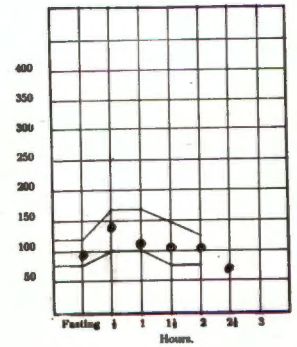
Patient's Identification IDA MEYER (122926)

Ward A30. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

Testing	URINE SUGAR
1. 0	0
2. 0	0
3. 0	0

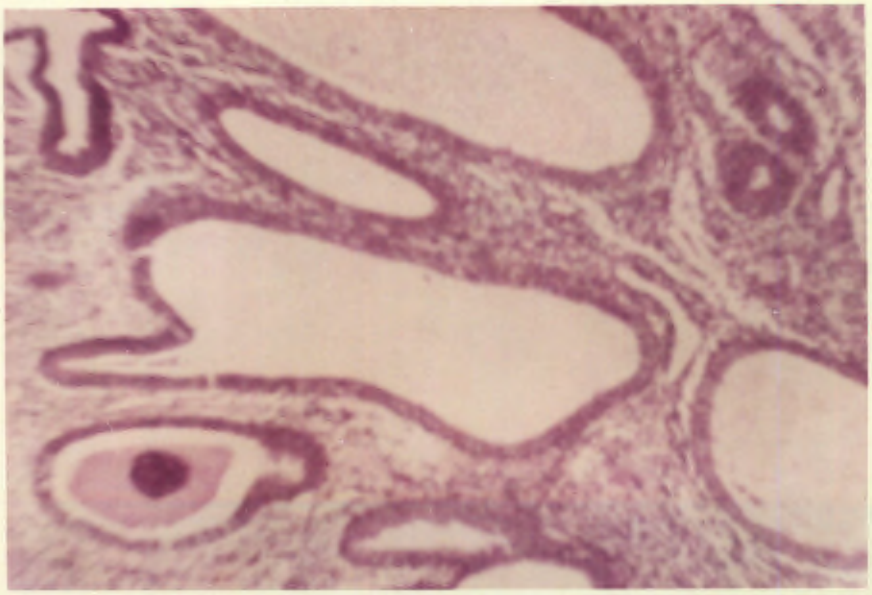
BLOOD SUGAR



BLOOD SUGAR

Fasting	89 mgms%.
½ hour	240 mgms%.
1 hour	132 mgms%.
1½ hour	130 mgms%.
2 hour	107 mgms%.
2½ hour	67 mgms%.
3 hour	

Signature G. M POTGIETER



47.

CASE 47.

Age 57 years. Complained of menorrhagia: 1 year. 17/3/59 Dand C, 7/10/59 hysterectomy: small fibroids and benign glandular hyperplasia.

Age 47 years. C/O Menorrhagia. 2/1059 Hysterectomy: Benign glandular hyperplasia.

Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

Serial No. 30167 - 68 Date 3.8.59.

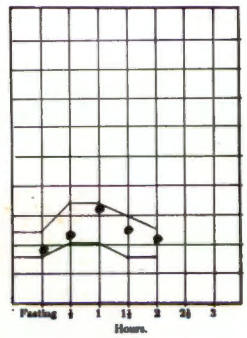
Patient's Identification MARGARET MILLER. (29236)

Ward 49 Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

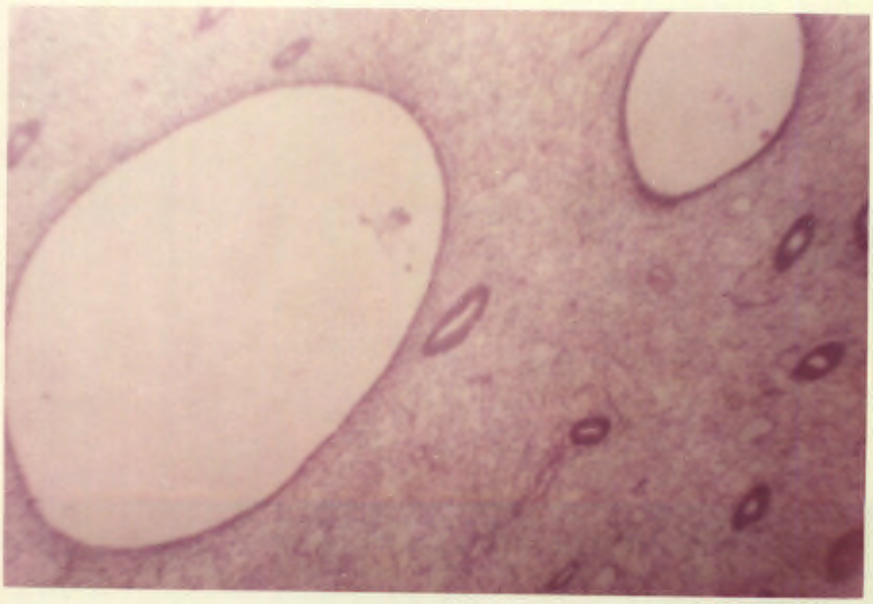
URINE SUGAR
1. Negative.
2. Negative.
3. Negative.

BLOOD SUGAR
Received 1.9.59 at 20 a.m.



BLOOD SUGAR
Fasting 86 mgs.%.
½ hour 125 mgs.%.
1 hour 161 mgs.%.
1½ hour 134 mgs.%.
2 hour 108 mgs.%.
2½ hour
3 hour

Signature L. ANSTEY.

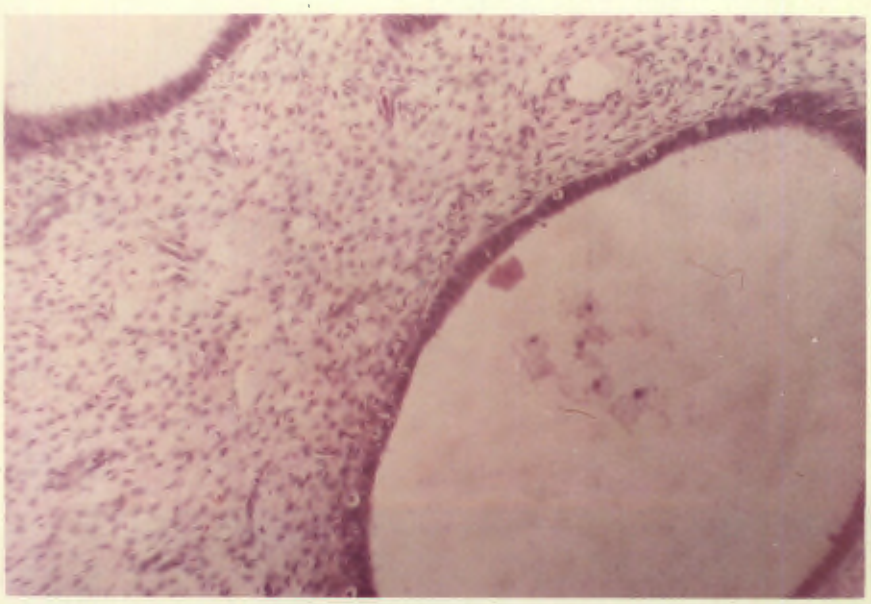


48

CASE 48.

Age 47 years. Complained of menorrhagia. 2/10/59 hysterectomy: benign glandular hyperplasia.

CASE 48. (Photomicrograph b.).



48 b.

Age 39 years. C/O Bouts of amenorrhoea followed by bouts of prolonged bleeding. D&C 20/12/59 : Hyperplastic Cystic endometrium.

49.

Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

Serial No. 43686 - 87. Date 27.11.59.

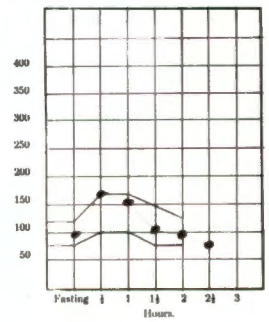
Patient's Identification MAKELIKA SALIE (57/06217)

Ward A10. Physician or Surgeon Dr. Benjamin

GLUCOSE TOLERANCE TEST.

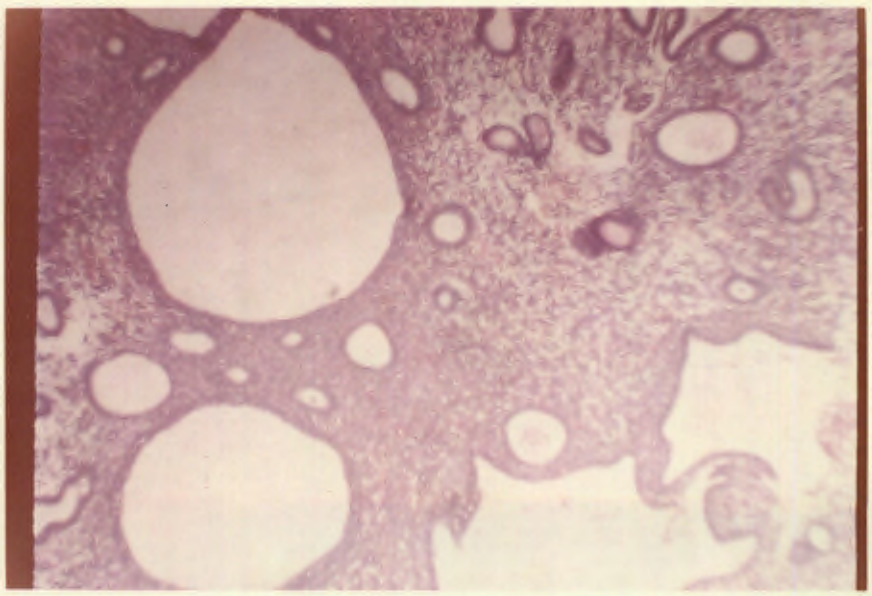
Tes-tape	URINE SUGAR
1. Neg.	Neg.
2. Neg.	Neg.
3. Neg.	Neg.

BLOOD SUGAR



BLOOD SUGAR	
Fasting	99 mgs.%.
½ hour	178 mgs.%.
1 hour	154 mgs.%.
1½ hour	103 mgs.%.
2 hour	99 mgs.%.
2½ hour	76 mgs.%.
3 hour	

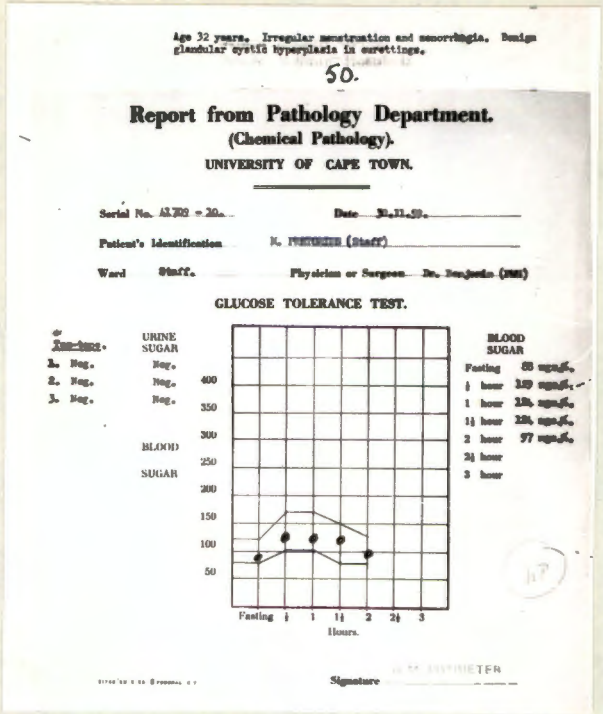
G. M POTGIETER
Signature



49.

CASE 49.

Age 39 years. Complained of bouts of amenorrhoea followed by bouts of prolonged bleeding. D and C 20/12/59: Hyperplastic cystic endometrium.



Age 32 years. Irregular menstruation and menorrhagia. Benign glandular cystic hyperplasia in curettings.

50.

Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

Serial No. A1705 = 20... Date 30.11.50.

Patient's Identification M. PIETERSE (Staff)

Ward Staff. Physician or Surgeon Dr. Benjamin (PMH)

GLUCOSE TOLERANCE TEST.

Specimen.	URINE SUGAR
1.	Neg.
2.	Neg.
3.	Neg.

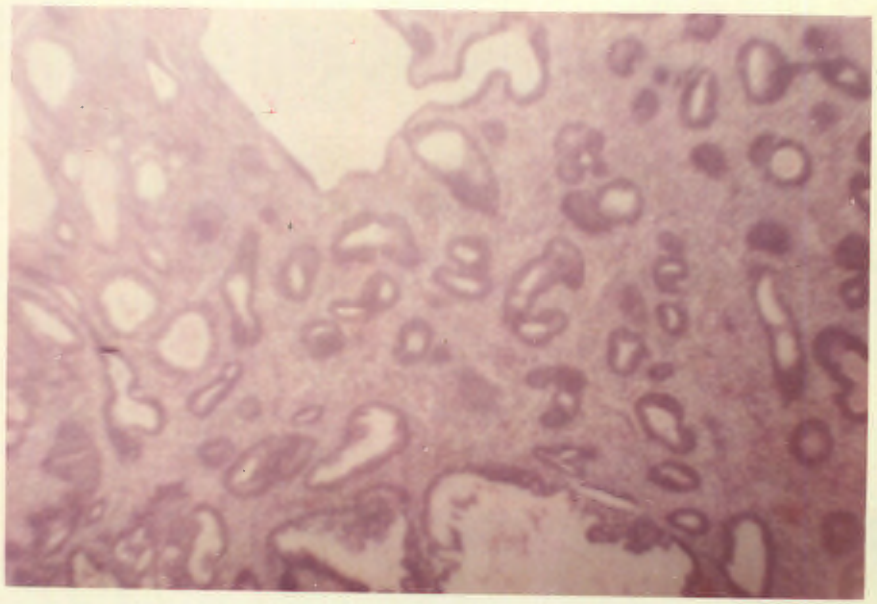
BLOOD SUGAR

400
350
300
250
200
150
100
50

Fasting ½ 1 1½ 2 2½ 3
Hours.

BLOOD SUGAR	
Fasting	88 mgm.%
½ hour	189 mgm.%
1 hour	238 mgm.%
1½ hour	238 mgm.%
2 hour	97 mgm.%
2½ hour	
3 hour	

Signature



50.

CASE 50.

Age 32 years. Irregular menstruation and menorrhagia. Benign glandular cystic hyperplasia in curettings.

TABLE 10. GLUCOSE TOLERANCE CURVES IN PATIENTS WITH BENIGN GLANDULAR HYPERPLASIA.

Patient			Blood Sugar Levels (Mgs.%)				
No.	Initials	Age	Fasting	½ Hour	1 Hour	1½ Hours	2 Hours
A. Group with Diabetes.							
1.	A.R.	42	161	170	231	242	223
2.	S.S.	54	135	218	237	240	212
3.	B.A.	42			204	205	205
4.	C.K.	55	148	251	187	135	121
5.	C.J.	47	117	173	215	213	199
6b.	M.T.	62	183	248	265	201	
7.	M.S.	56	154	232	260	286	182
8.	P.S.	53	205	196	298	288	298
9.	S.M.	48	135	241	235	138	100
10.a	M.V.W.	50	139	213	199	164	141
B. Group with Mildly Impaired Glucose Tolerance.							
11a.	L.G.	56	112	162	161	171	152
12.	A.A.	60		123	219	223	
13.	I.M.G.	55	124	174	221	183	167
14.	G.A.	44	126	158	162	180	187
15.	G.L.	30	145	152	183	105	88
16.	V.F.A.	55	117	161	207	218	170
17.	L.D.P.	41	114	153	187	180	156
18.	D.O.	39	89	156	202	183	138
19.	H.G.	45	86	154	267	202	186
20.	F.M.	46	141	181	195	136	132
21.	E.F.	49	84	169	180	159	148
22.	M.K.	55	100	170		186	137
23.	D.K.	51	117	253	211	163	166
24.	D.S.	25	111	212	180		
25.	M.B.	45	114	153	205	242	173
26.	H.F.	50	88	141	81	176	185

Patient			Blood Sugar Levels (Mgs.%)				
No	Initials	Age	Fasting	½ Hour	1 Hour	1½ Hours	2 Hours
27a.	G.K.	51	122	149	122	178	165
28.	W.C.B.	63	114	134	163	176	152
29.	S.P.	51	95	122	193	148	139
30.	C.M.S.	52	105	186	246		172
31.	D.Z.	57	115	195			178
32.	E.J.	49	114	130	199	161	152
33.	B.W.	53	97	97	235		173
34.	J.R.	52	98	134	110	107	141
35.	M.S.	52	111	168	206	182	143
36.	R.D.	56	117	139	159	177	179
37.	A.G.	57	76	111	133	101	190
38.	K.D.	54	78	147	149	154	149
39.	E.N.	35	114	226	256	211	174
40.	M.H.	34	88	127	203	168	158
41.	R.J.	30	110	173	147	149	146
42a.	M.V.R.	32	115	181	219	213	146
C. Group with normal Glucose Tolerance Curves.							
43.	G.C.	61	93	161	184	146	
44.	J.C.	53	107	191	122	169	85
45a.	M.O.C.	55	84	131	138		67
46.	S.G.	61	107	174		185	107
47.	I.M.	57	89	140	112	110	107
48.	M.M.	47	86	125	161	134	105
49.	M.S.	39	99	174	154	103	99
50.	M.P.	32	88	129	124	124	97

A summary of the results is as follows:

TABLE 11. Glucose Tolerance Curves in 50 Consecutive cases of Benign Glandular Hyperplasia.

Normal Curves	Abnormal Curves		
	Diabetes	Mildly Impaired Glucose Tolerance	Total
16%	20%	64%	84%

In patients in whom benign glandular hyperplasia is found, it is a striking feature that the incidence of impaired glucose tolerance is very high, being in the region of 84%. This figure is considerably higher than that found in normal control cases, and in cases of abnormal uterine bleeding where the endometrium is atrophic or normal. This incidence is also appreciably greater than that encountered

even in cases with carcinoma of the endometrium.

4. PATIENTS WITH NON-OVULAR CYCLES BUT IN WHOM THE ENDOMETRIUM WAS NOT HYPERPLASTIC.

Ten patients were encountered in whom histologial examination of the endometrium indicated that ovulation had not taken place, but the endometrium was not hyperplastic. In each case the endometrical biopsy was done at a stage of the cycle when secretory changes should have been present - such changes were not seen, the endometrium being proliferative in type. These patients were all investigated by glucose tolerance tests.

The results are shown in Table 12 and photographs of the actual glucose tolerance tests are attached, together with a summary of the clinical history and histological description of the endometrium.

The same high incidence of abnormal glucose tolerance was found as in cases of benign glandular hyperplasia.

Age 49 years. Menorrhagia 1 year. Obese. B.P. 230/130. D&C 30/10/59 i.e. 20th. day of cycle: proliferative endometrium with no secretory activity.

1.

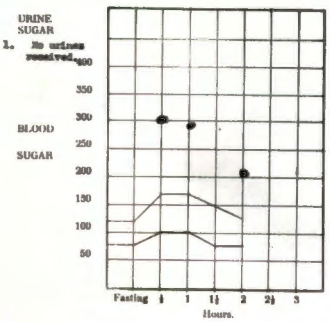
Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

Serial No. 28762 Date 30.10.59.

Patient's Identification [illegible] (59/04147)

Ward C30. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting	Clotted
½ hour	302 mgs.%.
1 hour	293 mgs.%.
1½ hour	Clotted
2 hour	223 mgs.%.
2½ hour	
3 hour	

Signature G. M. [illegible]

Age 47 years. Menorrhagia. Fibroids size of 14 weeks. D&C 25th. day of cycle: Proliferative endometrium.

2.

1728

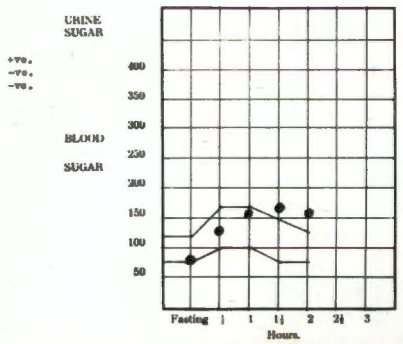
Report from Pathological Department.
UNIVERSITY OF CAPE TOWN.

Serial No. 8148-49 Date 13.3.58

Patient's Identification EMILY ABRAHAMS. (58/06726)

Ward 37 Physician or Surgeon Prof. Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting	73 mgs.%.
½ hour	139 mgs.%.
1 hour	162 mgs.%.
1½ hour	167 mgs.%.
2 hour	162 mgs.%.
2½ hour	
3 hour	

Signature

Age 48 years. Menorrhagia. Non-secreting endometrium on 24th. day of 28 day cycle.

3.

=

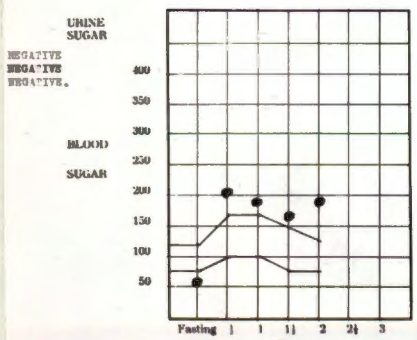
Report from Pathological Department.
UNIVERSITY OF CAPE TOWN.

Serial No. 2686-7/58 Date 29.1.58.

Patient's Identification SOPHIA JENKINS. (57/07921).

Ward A9 Physician or Surgeon Prof J.T. Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting	54 mgs.%.
½ hour	202 mgs.%.
1 hour	181 mgs.%.
1½ hour	172 mgs.%.
2 hour	189 mgs.%.
2½ hour	
3 hour	

Signature

Age 40 years. C/O Menorrhagia: 2 months. Fibroids size 14 - 16 weeks. 13/5/59 D&C: On the 40th. day of cycle - " ..." phase - thus decided leg".

4.

3776

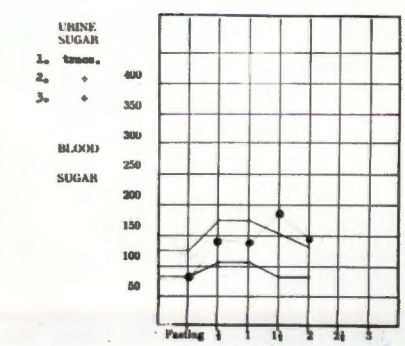
Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

Serial No. 19199 - 200. Date 14.6.59.

Patient's Identification MARY MOSALA (59/04415).

Ward A9. Physician or Surgeon Pmf Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting	74 mgs.%.
½ hour	147 mgs.%.
1 hour	140 mgs.%.
1½ hour	190 mgs.%.
2 hour	145 mgs.%.
2½ hour	
3 hour	

S. J. V. D. WALT
Signature

Age 45 years. C/O Severe dysmenorrhoea 3 months. Menstruation: 14/28-30 day type. L.M.P. 30/5/59. 26/6/59 Endometrial biopsy done as Outpatient on 26th. day of cycle - proliferative endometrium.

5.

Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

42%

Serial No. 19280 - 281. Date 12.6.59.

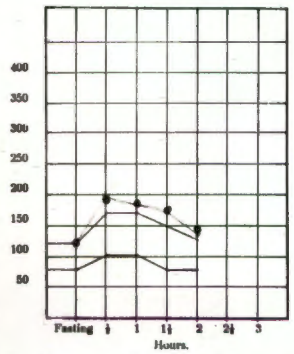
Patient's Identification RACHAEL DAVIDS (59/14544).

Ward A10 Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR
1. -
2. +
3. trace

BLOOD SUGAR



BLOOD SUGAR
Fasting 117 mgs.%.
½ hour 199 mgs.%. (Clotted)
1 hour 183 mgs.%.
1½ hour 176 mgs.%.
2 hour 140 mgs.%.
2½ hour
3 hour

A. J. V. D. WALT
Signature

Age 49 years. C/O Pruritis Vulvae. Menstruation 1-2/28 day type L.M.P. 5/6/59. In view of abnormal curve in control, endometrial biopsy (O.P.D.) done 26/6/59 on 21st. day of cycle. Non-secretory endometrium.

6

Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

Serial No. 19282 - 283. Date 12.6.59.

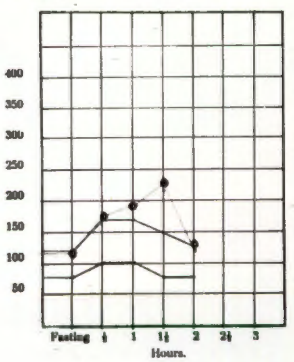
Patient's Identification JANIE ISRAELSON, (183019).

Ward A10. Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR
1. +
2. -
3. -

BLOOD SUGAR



BLOOD SUGAR
Fasting 113 mgs.%.
½ hour 178 mgs.%.
1 hour 194 mgs.%.
1½ hour 233 mgs.%.
2 hour 120 mgs.%.
2½ hour
3 hour

A. J. V. D. WALT
Signature

Age 47 years. Bouts of amenorrhoea and oligo-menorrhoea and infertility. Menstruation 1-3/2 months type. D&C on 23rd. day of cycle: Proliferative endometrium. Serum Cholertrol 273 mgs%.

7.

Report from Pathological Department.
UNIVERSITY OF CAPE TOWN.

Serial No. 27651 - 2. Date 1.6.59

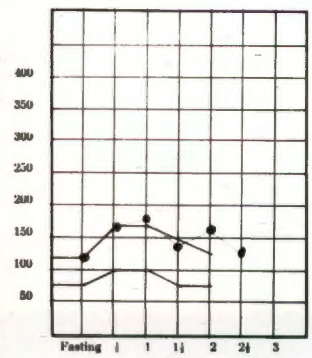
Patient's Identification FARIDA BADEROEN (146319)

Ward A.2 Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR
1. Negative.
2. Negative.
3. Negative.

BLOOD SUGAR



BLOOD SUGAR
Fasting 120 mgs.%.
½ hour 161 mgs.%.
1 hour 177 mgs.%.
1½ hour 141 mgs.%.
2 hour 163 mgs.%.
2½ hour 133 mgs.%.
3 hour

J. J. V. D. WALT
Signature

Age 44 years. C/O Menorrhagia: 1 year. Menstruation 7/32 day type. D&C on 30th. day of cycle: Proliferative endometrium, no secretory changes.

8.

Report from Pathological Department.
UNIVERSITY OF CAPE TOWN.

Serial No. 28242-3/57 Date 27.8.57

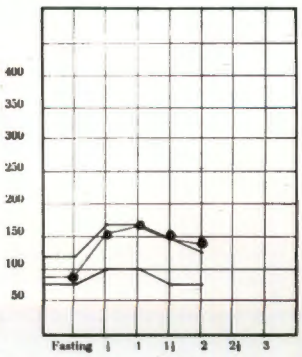
Patient's Identification CAROLINE WARREN. (42024).

Ward C10. Physician or Surgeon Prof. Louw.

GLUCOSE TOLERANCE TEST.

URINE SUGAR

BLOOD SUGAR



BLOOD SUGAR
Fasting 89 mgs.%
½ hour 152 mgs.%
1 hour 165 mgs.%
1½ hour 149 mgs.%
2 hour 138 mgs.%
2½ hour
3 hour

Signature

Age 43 years. C/O Menorrhagia and prolongation of cycle. Menstruation 6/30 -35 day type. DAC on 32 day of cycle: Proliferative endometrium, no secretory changes.

9.

Report from Pathological Department.

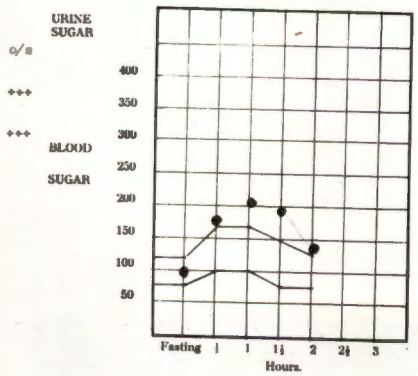
UNIVERSITY OF CAPE TOWN.

Serial No. 20574-5/57. Date 10.6.57

Patient's Identification Mrs. SIMPRITZ. (8418)

Ward A 9. Physician or Surgeon Prof. Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting 85 mgs.%
½ hour 176 mgs.%
1 hour 203 mgs.%
1½ hour 181 mgs.%
2 hour 128 mgs.%
2½ hour
3 hour

G. LINDER

Signature

Age 45 years. C/O Menorrhagia: 9 months. DAC 27/7/59 (L.M.P. 1/7/59). Normal proliferative endometrium, but should be secretory at 26th. day.

10.

Report from Pathology Department.

(Chemical Pathology).

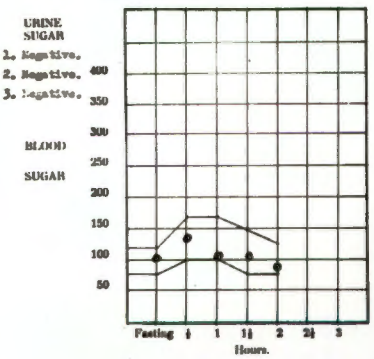
UNIVERSITY OF CAPE TOWN.

Serial No. 24,999 - 25000. Date 26.7.59.

Patient's Identification SARAH ISAACS (1...30)

Ward A9 Physician or Surgeon Prof Louw.

GLUCOSE TOLERANCE TEST.



BLOOD SUGAR

Fasting 104 mgs.%.
½ hour 136 mgs.%.
1 hour 104 mgs.%.
1½ hour 104 mgs.%.
2 hour 86 mgs.%.
2½ hour
3 hour

G. M. POTGIETER

Signature

TABLE 12. GLUCOSE TOLERANCE CURVES IN 10 CONSECUTIVE PATIENTS WITH ABNORMAL UTERINE BLEEDING IN WHOM THE ENDOMETRIUM WAS NOT HYPERPLASTIC, BUT WHERE SECRETORY CHANGES WERE ABSENT (NON-OVULAR CYCLES).

Patient			Blood Sugar Levels (Mgs.%)				
No	Initials	Age	Fasting	½ Hour	1 Hour	1½ Hours	2 Hours
A. Diabetic.							
1.	D.W.	49		302	291		213
B. Mildly Impaired Glucose Tolerance.							
2.	E.A.	47	73	139	162	167	162
3.	S.J.	48	54	202	181	172	189
4.	M.M.	40	74	147	140	190	145
5.	R.D.	45	117	199	183	176	140
6.	J.I.	49	113	178	194	233	120
7.	F.B.	47	120	161	177	141	163
8.	C.W.	44	89	152	165	149	138
9.	A.S.	43	85	176	203	181	128
C. Normal Glucose Tolerance.							
10.	S.I.	45	104	136	104	104	86

SUMMARY.

Five different groups were investigated for glucose tolerance, and a photograph of each original glucose tolerance curve is shown. In each case the endometrium was examined histologically, and the clinical findings are presented. In the control group (of 100 subjects) who were 45 years and over (with normal endometria) 78% had normal curves, 13% diabetic curves, and 9% mildly impaired curves. The 50 women between 35 and 45 years with normal endometria showed 90% normal curves, 2% diabetic curves and 8% mildly impaired curves.

The findings in 50 consecutive cases of cancer of the endometrium showed a statistically significant high incidence of abnormal curves - 52%. A most striking incidence of abnormal curves (84%) was demonstrated in benign glandular hyperplasia. Only one of 10 subjects with non-hyperplastic endometria) had a normal glucose tolerance curve.

Further investigations revealed that 1) glucose tolerance remained the same at different stages of the menstrual cycle 2) oestrogen administration did not affect normal glucose tolerance 3) follicle-

stimulating hormone actually improved glucose tolerance, and 4) there was no improvement in the impaired glucose tolerance in cases of benign glandular hyperplasia after total hysterectomy and bilateral salpingo-oophorectomy.

CHAPTER 5.

DISCUSSION OF THE FINDINGS.

CHAPTER 5.

DISCUSSION OF THE FINDINGS.

It is thus seen that 5 different groups of subjects were investigated by glucose tolerance tests. Several interesting findings came to light which warrant discussion:

1. Women who acted as control subjects - 45 years of age and over with normal endometria.

In this random control group, the surprising finding was the high incidence of impaired glucose tolerance in older women, when compared with previous diabetic surveys of entire populations. Thus in this group, as many as 22% had abnormal glucose tolerance curves (13% being frankly diabetic curves, and 9% showing lesser degrees of impairment of glucose tolerance). This incidence is remarkably high when compared with diabetic surveys of populations reported in the literature; for example,

in the same age groups Spiegelman and Marks (1946) reported the presence of 1.02% diabetics; Wilkerson and Krall, (1947) 5%, Joslin (1952) 2%, and Walker (1959) 1.4%.

There are 3 main explanations for the apparent discrepancy: (a) In the population surveys the methods of detecting the diabetics were far less exacting than those employed in this investigation. In this study each and every subject was submitted to a full glucose tolerance test, and the test was often repeated. In none of the population surveys were routine glucose tolerance tests done. Thus Spiegelman and Marks (1946) simply carried out single fasting blood sugar estimation on each subject. In the Oxford Survey (Wilkerson and Krall, 1947) blood samples were taken one hour after a meal, and specimens of urine were examined; if the results were thought suspicious, the tests were repeated, and only if still abnormal, was a glucose tolerance test performed. The British Diabetic Association Survey reported by Walker (1959) was carried out by testing specimens of urine passed after a high carbohydrate meal - when glycosuria was detected, a glucose tolerance test was carried out. The strictest criteria applied to the Oxford Survey, and the

incidence here was by far the highest. Yet this method was not nearly as exacting as the one used in my investigation. (b) The criteria for impaired glucose tolerance laid down in my investigation were carefully adhered to, were standard and were strict, and minor aberations in the curves were taken into consideration. (See chapter on "The Glucose Tolerance Test"). (c) It is possible that the presence of some pelvic pathology and vaginal bleeding may cause minor aberrations in the curves.

This investigation demonstrates inter alia; that a great many patients with diabetes and less severely impaired glucose tolerance are not detected unless full glucose tolerance tests are carried out. The remark made by Walker (1959) that "for every known case of diabetes, there is another as yet undetected and untreated", is probably an understatement. This present control investigation also indicates that the population diabetic surveys would have been hopelessly inaccurate as a control group for my other investigations, because every subject of mine, whether normal or abnormal, underwent a full glucose tolerance test.

2. Women who acted as control subjects - between 35 and 45 years of age, with normal endometria:

The bulk population surveys previously referred to, as well as all other investigations in the field, showed the increasing incidence of diabetes and lesser degrees of impaired glucose tolerance with age. Practically all such studies showed that the most abrupt rise in incidence was round about the age of 45 years. Having obtained control series over 45 years of age, I considered it necessary to investigate a group under 45 years of age, (a) to confirm the rising incidence with age, and (b) to determine whether the rise was abrupt after the age of 45 years. Fifty such consecutive women with normal endometria were studied by glucose tolerance tests. The much lower incidence of impaired glucose tolerance in this group (10%) confirmed both these points.

3. Patients with cancer of the Endometrium:

In the group with cancer of the endometrium it was found that 48% had normal glucose tolerance curves, while in 52% the curves were abnormal (28% being diabetic and 24% showing less severely impaired glucose tolerance). Comparing these figures with the control group of the same age 78% with normal

glucose tolerance and 22% with impaired glucose tolerance, these figures are statistically significant. This investigation confirms the finding of an increased incidence of impaired glucose tolerance in cases of endometrial cancer, by Moss (1947), Garnet (1958), Way (1954) and Loux (1958) - all these investigators also carried out glucose tolerance tests in every patient. Smith (1941), Vander (1958), Hertig et al., (1940), Scheffey et al. (1943), and Palmer et al. (1949) did not find a significantly increased incidence of diabetes in their cancer cases; however, none of them carried out glucose tolerance tests - the diagnosis of diabetes was made by much less refined methods, such as the level of the fasting blood sugar only, or the clinical presence of diabetes.

The increased incidence of abnormal glucose tolerance curves in cases of cancer of the endometrium compared with suitable and strict controls was therefore shown to be indisputable, in this investigation.

4. Patients with benign glandular hyperplasia:

The most striking findings were those seen in the cases of benign glandular hyperplasia. In

this group it was found that as many as 84% of the curves were abnormal. Most of the aberrations in the curves were not gross - 22% were diabetic type curves, and 62% showed less severely impaired glucose tolerance. This figure is considerably higher than that found in the control group (i.e. 84% compared with 22%), and is statistically most significant. The figure of 84% is also appreciably higher than that obtained even in cases of cancer of the endometrium (52%).

There is another reason why the findings in this group are most significant. The increased incidence of diabetes and mildly impaired glucose tolerance in older people has constantly been referred to in this thesis. Many of the patients with cancer of the endometrium were old women, and this fact had to be taken into account in that group. But in this series with benign glandular hyperplasia, the average age was low: 6 were under 35 years, 7 were between 35 and 44 years, 22 between 45 and 54 years, and 15 between 55 and 64 years (the oldest patient was 63 years of age).

5. Women with non-ovular cycles but in whom the endometrium was not hyperplastic:

Such cases are rare, and I was able to find only 10 of them during the period of this investigation. It is interesting that out of these 10, only 1 showed a normal glucose tolerance curve, 1 showing definite diabetes, and 8 showing less gross alterations in the curves. This group, of course, had this in common with benign glandular hyperplasia series, that both were non-ovular groups with an absence of progestational changes in the endometrium.

Two important questions arise as an outcome of these findings:

1. What is the cause of the remarkably high incidence of abnormal glucose tolerance curves in cases of benign glandular hyperplasia?

The whole question is discussed in the next chapter (Chapter 6).

2. Since a high incidence of abnormal glucose tolerance is a factor common to both benign glandular hyperplasia and cancer of the endometrium, are these two conditions related? And does this metabolic disturbance give a clue as to the cause of benign glandular hyperplasia, and the cause of endometrial cancer?

The finding of this common factor in both cancer of the endometrium and benign glandular hyperplasia, lends weight to the argument that these 2 conditions are related. Their association has often been reported (Novak and Yui, 1936; Way, 1954; Turnbull, 1956; Barr and Charteris, 1955; Hunter et al., 1954; Mazolla, 1938; Speert, 1949; Strachan, 1936; Orrahood and Wyatt, 1953; Purdie, 1945; Hertig and Somers 1949). Novak and Richardson (1941), and McBride (1955) reported the frequent association of "retrogressive hyperplasia or cystic atrophy" and carcinoma of the endometrium. Benign glandular hyperplasia (cystic and non-cystic) may be found in conjunction with frank cancer, and all gradations may occur, just as all degrees of typical hyperplasia may precede the development of endometrial carcinoma (Novak and Novak, 1958).

What is the cause of the endometrial hyperplasia, and what is the factor that determines the development of carcinoma?

Excessive or prolonged oestrogen activity has been suggested, for the following reasons:

1. An increasing number of cases is being reported with a history of prolonged oestrogen therapy and later development of uterine cancer (Novak and Novak, 1958).

2. There is evidence that there is a high incidence of endometrial carcinoma in feminising mesenchymal ovarian tumours (Novak and Novak, 1958).

3. Endometrial hyperplasia is due to abnormal oestrogen activity (Brown et al., 1959).

However, oestrogens alone do not explain the malignant development, for the following reasons:

1. McBride (1955) showed that in postmenopausal women with endometrial carcinoma there was no evidence of continuing abnormal oestrogen activity.

2. Jones and Brewer (1941) reported on a group of young menstruating women whose endometria showed cancer in conjunction with a progestational endometrium. There is no question that this occurs, but as the exception to the usual picture of postmenopausal adenocarcinoma.

3. Cirrhotics were thought to be more prone to develop endometrial cancer, because of hepatic inability to detoxify oestrogen. However Brewer and Foley (1953) seem to have disproved this fairly conclusively.

Thus while the role of abnormal oestrogen activity in the development of benign glandular hyperplasia is undoubted, the part it plays in the

development of carcinoma of the endometrium remains uncertain. However, my investigation leaves no doubt that there is a common factor in a high percentage of patients with both these diseases, namely an aberration of carbohydrate metabolism. This finding may pave the way towards determining the missing factor which results in the development of endometrial malignancy. Such disturbances in carbohydrate metabolism may render the patient more liable to the development of carcinoma, just as diabetes predisposes, for example, to certain infections.

STATISTICAL ANALYSES.

GLUCOSE TOLERANCE IN CONTROL SUBJECTS, IN CASES OF CANCER OF THE ENDOMETRIUM, AND IN PATIENTS WITH BENIGN GLANDULAR HYPERPLASIA.

Group Investigated	Normal Curves	Abnormal Curves		
		Diabetes	Mildly Impaired Glucose Tol.	Total
100 Control Subjects	78%	13%	9%	22%
50 Cases with Cancer of the Endometrium	48%	28%	24%	52%
50 Cases with Benign Glandular Hyperplasia.	16%	20%	64%	84%

1. CANCER OF THE ENDOMETRIUM GROUP COMPARED WITH CONTROL GROUP.

(X_1^2 Test).

	Normal Curves	Abnormal Curves	Total.
Control Group	78	22	100
Cancer of Endo-metrium Group	24	26	50
Total	102	48	150

$$\chi^2 = \frac{(78 \times 26 - 24 \times 22)^2 \, 150}{102 \times 48 \times 50 \times 100}$$

$$= \frac{(2028 - 528)^2 \, 150}{102 \times 48 \times 50 \times 100}$$

$$= \frac{1500 \times 1500 \times 150}{120 \times 48 \times 50 \times 100}$$

$$= 13.6$$

The difference between the control group and the cancer of the endometrium group is significant at less than .1% level, tested by the X_1^2 Test.

2. BENIGN GLANDULAR HYPERPLASIA GROUP COMPARED WITH CONTROL GROUP.

(X_1^2 Test).

	Normal Curves	Abnormal Curves	Total.
Control Group	78	22	100
Benign Glandular Hyperplasia Group	8	42	50
Total	86	64	150

$$X_1^2 = \frac{(78 \times 42 - 8 \times 22)^2 \, 150}{86 \times 64 \times 50 \times 100}$$

$$= \frac{(3276 - 176)^2 \, 150}{86 \times 64 \times 50 \times 100}$$

$$= 52.5$$

The difference between the control group and the Benign Glandular Hyperplasia group is significant at less than .1%, tested by the X_1^2 Test.

3. CANCER OF THE ENDOMETRIUM GROUP COMPARED WITH BENIGN GLANDULAR HYPERPLASIA GROUP.

(X_1^2 Test).

	Normal Curves	Abnormal Curves	Total.
Endometrial Cancer Group	24	26	50
Benign Glandular Hyperplasia Group	8	42	50
Total	32	68	100

$$X_1^2 = \frac{(24 \times 42 - 8 \times 26)^2 \, 100}{32 \times 68 \times 50 \times 50}$$

$$= \frac{(1008 - 208)^2 \, 100}{32 \times 68 \times 50 \times 50}$$

$$= \frac{800 \times 800 \times 100}{32 \times 68 \times 50 \times 50}$$

$$= \frac{200}{17}$$

$$= 11.7$$

The difference between the endometrial cancer group and the Benign Glandular Hyperplasia group is significant al less than .1% level, tested by the X_1^2 test.

CHAPTER 6.

HYPOTHESES CONCERNING THE IMPAIRED GLUCOSE TOLERANCE IN BENIGN GLANDULAR HYPERPLASIA.

CHAPTER 6.

HYPOTHESES CONCERNING THE IMPAIRED GLUCOSE TOLERANCE IN BENIGN GLANDULAR HYPERPLASIA.

"and now remains
That we find out the cause of this effect,
Or rather say, the cause of this defect,
For this effect defective comes by cause".
SHAKESPEARE, Hamlet, Act II, Scene II.

Having discovered that there was such a remarkably high incidence of impaired glucose tolerance in cases of benign glandular hyperplasia, an attempt was made to detect the reason for this association.

In conducting this enquiry, several factors had to be considered:

The hormones concerned in the development of benign glandular hyperplasia must come under

discussion i.e. oestrogens, progresterone, and follicle-stimulating hormone. The effect of these hormones on carbohydrate metabolism, and the effect of carbohydrate metabolism on the secretion of these hormones needs to be discussed. In addition, other hormones which are known to be concerned with carbohydrate metabolism, and which may affect the pituitary-ovarian control of the endometrium, must be considered. These include the growth hormone of the anterior pituitary, prolactin, the adrenal cortical hormones, and the thyroid hormone. And finally it is necessary to discuss the physiology of carbohydrate metabolism in the endometrium itself, the site where the main histo-pathology is evident.

1. OESTROGENS:

Oestrogen levels in Benign Glandular Hyperplasia and other conditions:

It has always been considered that the hyperplastic pattern of the endometrium in benign glandular hyperplasia is due to an exessive or abnormal oestrogen activity, or to an abnormal response of the endometrium

to such oestrogens. There is no reason to doubt this. Assays have shown that there is a direct correlation between the histological state of the endometrium and the level of urinary oestrogens (Brown et al., 1959).

Brown et al. (1959) investigated the urinary oestrogen excretion in the normal menstrual cycle, and in cases of amenorrhoea and dysfunctional bleeding. They found fluctuations during the normal menstrual cycle. During the first 2 or 3 days of menstrual bleeding in a 28 day cycle, the oestrogen excretion is low, the mean being about 12µg. per day. The oestrogen levels then rise to a well-defined peak about the 13th. day of the cycle, when the average excretion is 57 µg. per 24 hours. Following this temporary elevation, there is a short period of decrease, followed by another rise which averages about 33 µg. daily. During the last few days of the cycle the oestrogen excretion falls rapidly and by the first day of the next cycle it has again reached an average figure of 12 µg. daily. These mean excretion rates must be borne in mind when considering

abnormalities of the cycle.

In benign glandular hyperplasia these workers found that the urinary oestrogen levels are maintained for some time in the region of 30 µg. per 24 hours or more; in some cases the levels remain reasonably constant, in others there is a fluctuation; but in the latter the elevated levels were maintained for a longer time, and were usually greater than those associated with the ovulatory peak of the normal cycle.

In non-ovulatory cycles the oestrogen excretion level remains remarkably constant, in the region of 20 µg. per 24 hours. Unlike normal menstruation, this bleeding occurs without withdrawal of the oestrogenic stimulus.

Normal postmenopausal women, with normal atrophic endometria excreted an average of 5 µg. per 24 hours. In postmenopausal women whose endometria showed a patter of benign glandular hyperplasia the oestrogen excretion rate was about the same as in the pre-menopausal groups with this condition.

Applying this knowledge to my findings of abnormal glucose tolerance, it appears that when the

? continuously

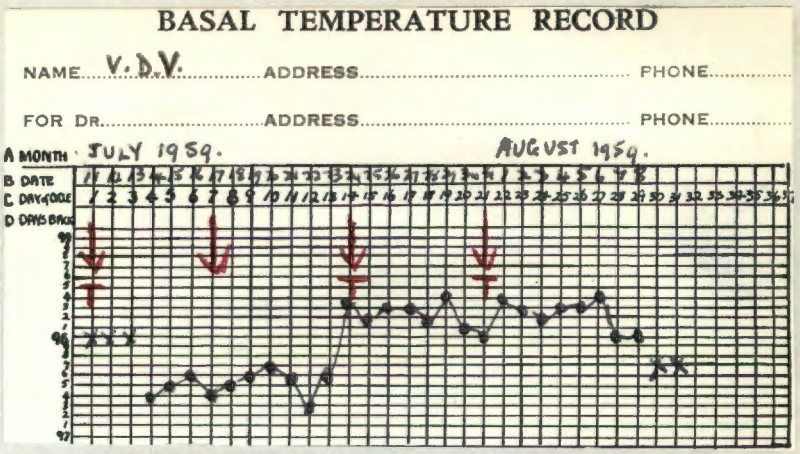
oestrogen level is constantly high (30 µg. or 20 µg.) as in benign glandular hyperplasia and non-ovulatory cycles, glucose tolerance is impaired. When the level is high, but does fluctuate regularly to lower rates, as in the normal menstrual cycle (12 ug. to 33 µg.), glucose tolerance is normal. And when the level is constantly low, as in postmenopausal women (5 µg.) glucose tolerance is also normal.

Investigation:

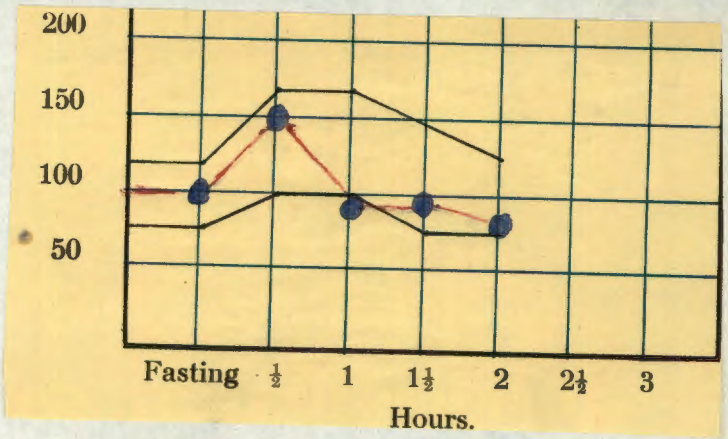
In order to create an artificial high and constant level of oestrogen activity (such as occurs in benign glandular hyperplasia) and observe the effect of this on glucose tolerance 5 normal postmenopausal patients were each given 2.5 mgm. Premarin b.d. for 6 weeks. Glucose tolerance tests were carried out before and during the 6 weeks' of course of the hormone. No effect on glucose tolerance was observed. As examples the curves of 2 of the subjects are attached. Likewise 5 normal subjects were submitted to glucose tolerance investigations immediately after menstruation and at ovulation time (covered by basal body temperature recordings). As an example, one of the subject's basal temperature recordings and

glucose tolerance curves is attached. There is no change in glucose tolerance in these normal subjects during the menstrual cycle.

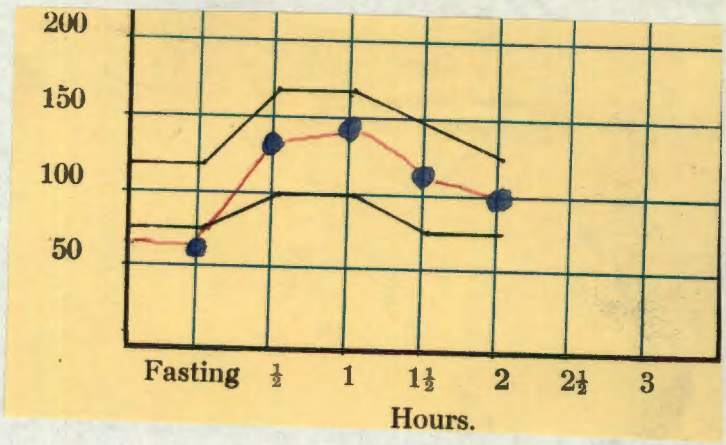
Basal body temperature recordings in the cycle investigated.



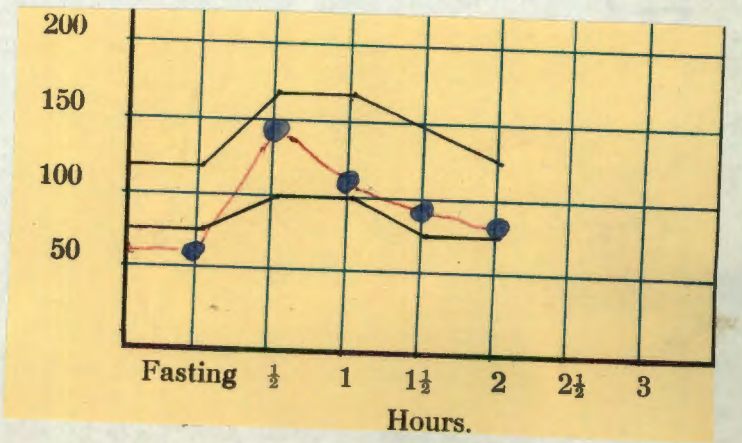
Glucose Tolerance Test on the 1st day of menstruation.



Glucose Tolerance Test at the time of ovulation.



Glucose Tolerance Test on the 21st day (luteal phase).



Influence of Oestrogens on Carbohydrate Metabolism.

There is evidence from animal and human studies that small amounts of oestrogens may aggravate diabetes, while large amounts may improve it.

Studies in animals have usually demonstrated an amelioration and occasionally an aggravation in the intensity of diabetes following oestrogen therapy. In 1933 Barnes et. al. reported that oestrogen therapy attenuated the diabetes which follows total pancreatectomy. Nelson and Overholser (1936) confirmed this effect and showed that estrone also reduced the hyperglycaemia and glycosuria which appeared in monkeys given crude pituitary extract. In both sets of studies it was suggested that the beneficial effects probably resulted from suppression of the pituitary. The later studies of Foglia et al., (1947), Lewis et al., (1950), Rodriguez (1950, 1951, 1954), Houssay (1951) and Houssay et al., (1954) have clearly demonstrated that oestrogens do ameliorate experimental diabetes. They have pointed out that in subtotally pancreatectomized rats, maintained with or without forced feeding, the diabetes is more frequent and appears earlier in the males than in the females, that

castration slightly protects the males and sensitises the females to experimental diabetes of this type, and that oestrogens have a protective action in both sexes (Foglia et al., 1954). However Rodriguez (1950) has noted that in some animals stilboestrol therapy produced an immediate transitory rise in blood sugar and in glycosuria with a later protective action. Young (1941) found that estrone, estriol, or diethyl-stilbestrol had no obvious anti-diabetic action in four pituitary diabetic dogs, and one depancreatized animal, and that as a matter of fact aggravation of the diabetic state may have occurred in some. Similarly, Ingle (1944), using adrenalectomized-hyophysectomized-partially de-pancreatized male rats as test animal, observed that diethylstilbestrol produced diabetes, or aggravated it if it were submaximal.

The clinical observation that in some women diabetes appears at the time of the menopause, or if pre-existent, the diabetic state may be aggravated after the menopause has lead to therapeutic trials of estrogens. However most of the early reports, based on the use of small amounts of relatively impure

extracts, indicated either no change or an actual aggravation of the diabetic state (Lewis et al., 1949; Ingle, 1941; Dolin et al., 1941; Griffiths et al., 1941; Lawrence et al., 1941; Collens et al., 1936). Other workers using larger amounts have noted amelioration. Thus Mazer and Israel (1937) reported three menopausal diabetic females in whom therapy with 2,000 to 10,000 R.U. every 4 days controlled diabetes without insulin. Gessler et al., (1939) treated five diabetic females with similar or larger dosages of estradiol benzoate, and noted that in two in whom the appearance of diabetes and menopause coincided, blood sugar levels were lower. This amount of estrogen suppressed the urinary output of follicle-stimulating hormone in the four patients in whom this measurement was made. Spiegelman (1940) indicated that 10,000 R.U. of estrogen twice weekly reduced insulin requirements in diabetic females, and that this effect persisted for three months after withdrawal of the medication. Gitlow and Kurschner (1943) describing 15 menopausal females with coincident development of diabetes suggested that 60,000 to 80,000 R.U. of estrogen per week ameliorated the diabetes and that this paralleled the remission in menopausal symptoms. Cantilo's (1941) findings,

again in menopausal or postmenopausal diabetics, were similar. Morton and McGavack's (1946) case is of particular interest since the diabetes was aggravated at each menstrual period and ameliorated by large amounts of estrogens. Others have since voiced the opinion that in their patients such beneficial responses have been produced. (Marcus et al., 1948; McCullach et al., 1952; McCullach et al., 1955). The most interesting of these series is McCullagh's (1955), consisting of six acromegalic diabetics. Estrogen therapy not only improved the diabetes clinically and, in five patients, in terms of the glucose tolerance response as well, but also controlled the acromegaly as reflected in a lowering of the elevated serum inorganic phosphorus levels, and a decrease in hand and foot size.

These isolated observations do suggest that there may be clinical support for a beneficial action in diabetes of large amounts of estrogens and the aggravating effect of small dosages. It is probably premature to consider this evidence conclusive because of the difficulties of clinical experimentation in a group of patients where many variables other than estrogen administration are undoubtedly operative.

One might cite among these the "placebo effect", and the natural tendency of newly discovered diabetes to ameliorate.

Summary:

There is some suggestive evidence that the abnormal oestrogen activity in benign glandular activity may be a factor in the production of the abnormal glucose tolerance that occurs in such a high percentage of these cases. 1) Such abnormal activity is present in benign glandular hyperplasia. 2) In my cases of non-ovulatory menstruation where oestrogen activity is also abnormal, this disturbance in glucose tolerance was also present. 3) There is the abovementioned evidence from animal and human studies that small amounts of oestrogens may aggravate diabetes. On the other hand there is evidence that oestrogenic activity may have nothing to do with the cause of the abnormal glucose tolerance in benign glandular hyperplasia, and for the following reasons:

1) The increasing incidence of diabetes mellitus after the menopause (when oestrogen levels are low

in all series studied.) 2) The fact that there was no alteration in glucose tolerance at different stages of the normal menstrual cycle in the subjects I studied. 3) There was no alteration in the glucose tolerance test when I had administered oestrogens to postmenopausal women. 4) Animal and human studies have shown that large amounts of oestrogens may be beneficial in cases of diabetes mellitus. 5) A follow up study was made of cases with benign glandular hyperplasia which had been treated and cured by total hysterectomy and bilateral salpingo - oophorectomy; in all these the abnormality in the glucose tolerance curve persisted or became worse.

The position is therefore far from clear. Although there is evidence that glucose tolerance may be altered by oestrogens, it seems that in benign glandular hyperplasia it is not the altered oestrogen activity that causes the aberrations in the glucose tolerance curve - it is much more likely that benign glandular hyperplasia is a manifestation of the disturbed metabolic state.

2. PROGESTERONE.

I have demonstrated the same high incidence of impaired glucose tolerance in both benign glandular hyperplasia, and in patients with non-ovular bleeding where the endometrium was not hyperplastic. In both groups progesterone activity of the ovary is absent. During the course of this investigation the association of impaired glucose tolerance and endometrial carcinoma has also been proven. This disease, too, nearly always arises from an endometrium which is not progestational (Jones and Brewer, 1941).

All these 3 groups of patients with disturbed glucose tolerance, then, have this in common, that the secretion of progesterone from the ovary is absent. The significance of this fact is unknown, and is worthy of investigation. It would be well worth while studying the effect of progesterone on carbohydrate metabolism. Little is known about this (Pasckis, Rakhoff, and Cantarow, 1958; Marrian, Prof. G.F. - Personal Communication by correspondence). Joslin et al. (1948), studied the menstrual cycles of 24 diabetic girls at a rest camp in 1939. With an almost unchanged diet and insulin dosage, the glycosuria appeared to be greatest in the premenstrual

and menstrual periods, falling to low levels between 7 and 21 days. It may be significant that glucose tolerance was at its worst at the time when pregosterone secretion was minimal or absent.

A careful search through the literature failed to disclose any studies on the effect of progesterone on carbohydrate metabolism. Since Professor Houssay of Buenos Aires, and Professor Marrian of Edinburgh had made many studies on the effect of steriods on diabetes, I wrote to them (1959). They, too, had not investigated the effect of progesterone on carbohydrate metabolism.

I have suggested to Dr. W.P.U. Jackson, who is in charge of the Diabetic Clinic in the Groote Schuur Hospital, Cape Town, that he administer progesterone to diabetic patients to observe whether there is any amelioration in the diabetes. Such research is now being contemplated.

3. FOLLICLE-STIMULATING HORMONE:

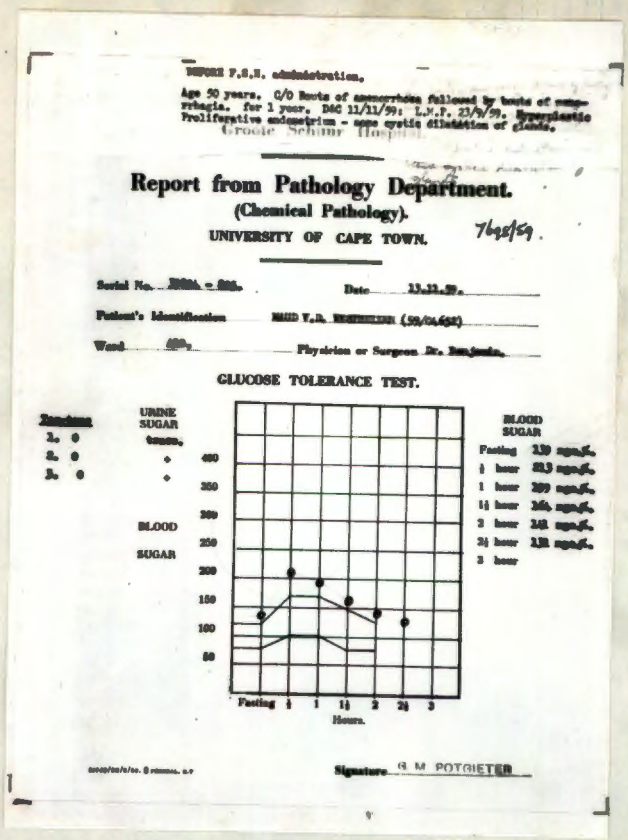
The cause of the ovarian dysfunction in benign glandular hyperplasia may, at times, be

primarily in the ovary, but faulty gonadotrophic function of the anterior pituitary is inevitably often blamed. This gland may secrete follicle-stimulating hormone continuously instead of intermittently, or may fail to produce the lutenising hormone. However, all this is theoretical, and is based on our knowledge of the physiology of the normal menstrual cycle. An exploration of the literature fails to reveal any definite studies of the F.S.H. levels in cases of benign glandular hyperplasia. Brown et al. (1959) state that they are at present working on such assays in the Clinical Endocrinology Research Unit (Medical Research Council) at the University of Edinburgh. Their results are awaited with interest.

Since, however, there may theoretically, be an excess of prolonged F.S.H. secretion, I felt that it was necessary to determine the effect of F.S.H. on glucose tolerance. Such an investigation has not been done before. Three patients with abnormal uterine bleeding were submitted to glucose tolerance tests; large doses of F.S.H. were administered, and the glucose tolerance tests were repeated. The details of this investigation are as follows:

Case 1.

Mrs. M. vd. W., age 50 years complained of bouts of amenorrhoea, followed by bouts of menorrhagia for a year. There were no abnormal physical signs in the pelvis. On 11/11/1959 a curettage was done (her previous episode of bleeding having been on 23/9/1959). The endometrial picture was one of benign glandular hyperplasia, a proliferative picture, with cystic dilatation of glands. On the 10th, 11th and 12th November an injection of 5,000 units of F.S.H. was given, and the glucose tolerance test was repeated again. The blood sugar levels before the F.S.H. were: fasting 139, ½ hour 213, 1 hour 199, 1. hours 164, 2 hours 141, and 2½ hours 131 mgs.%. After the F.S.H. the respective readings were, 99,155, 177, 155, 110, and 68 mgs.% respectively.



BEFORE F.S.H. administration.

Age 30 years. G/O Route of amenorrhoea followed by bouts of menorrhagia. for 1 year. D&C 11/11/59: L.M.P. 23/9/59. Hyperplastic Proliferative endometrium - some cystic dilatation of glands.

Groote Schuur Hospital

Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN. 7645/59.

Serial No. [illegible] Date 13.11.59.

Patient's Identification MAUD V.D. WESTHUIZEN (59/04652)

Ward [illegible] Physician or Surgeon Dr. Benjamin.

GLUCOSE TOLERANCE TEST.

Specimen	URINE SUGAR
1. 0	Ounce.
2. 0	+
3. 0	+

BLOOD SUGAR

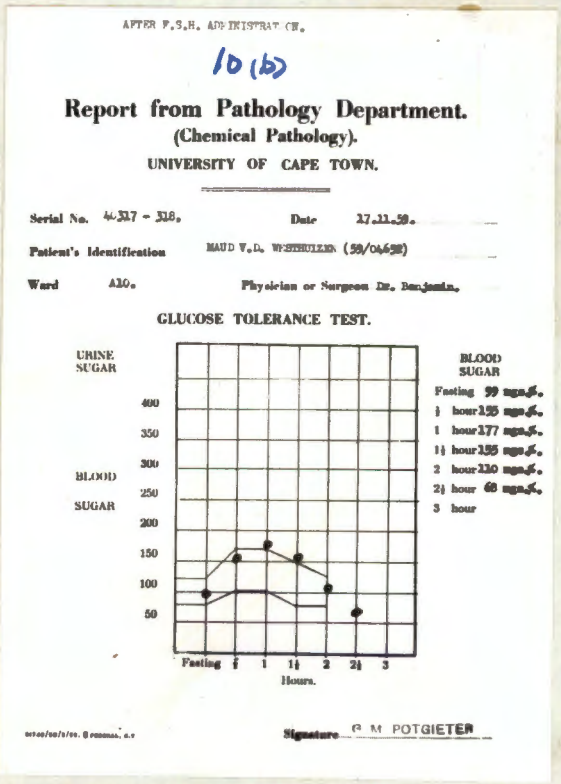
400 350 300 250 200 150 100 50

Fasting ½ 1 1½ 2 2½ 3
Hours.

BLOOD SUGAR	
Fasting	139 mgs.%
½ hour	183 mgs.%
1 hour	209 mgs.%
1½ hour	164 mgs.%
2 hour	142 mgs.%
2½ hour	138 mgs.%
3 hour	

Signature G. M. POTGIETER

Glucose Tolerance Test Before F.S.H. Administration.



AFTER F.S.H. ADMINISTRATION.

10 (b)

Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

Serial No. 4037 - 318. Date 17.11.59.

Patient's Identification MAUD V.D. WESTHUIZEN (59/04652)

Ward A10. Physician or Surgeon Dr. Benjamin.

GLUCOSE TOLERANCE TEST.

URINE SUGAR

BLOOD SUGAR

400 350 300 250 200 150 100 50

Fasting ½ 1 1½ 2 2½ 3
Hours.

BLOOD SUGAR	
Fasting	99 mgs.%
½ hour	155 mgs.%
1 hour	177 mgs.%
1½ hour	155 mgs.%
2 hour	130 mgs.%
2½ hour	68 mgs.%
3 hour	

Signature G. M. POTGIETER

Glucose Tolerance Test After F.S.H. Administration.

Case 2:

Mrs. M. v. R., aged 32 years complained of menorrhagia for 4 years, following a sterilization operation. A curettage yielded curettings, histological examination of which showed the picture of benign glandular hyperplasia. An identical course of F.S.H. as that given to Case 1. was administered. The blood sugar levels before the F.S.H. administration were: Fasting 115, ½ hour 181, 1 hour 219, 1½ hours 213, 2 hours 146 and 2½ hours 117 mgm.%. After the F.S.H., the readings were 84, 125, 134, 161, 148, and 104 mgs. respectively.

BEFORE F.S.H. ADMINISTRATION.

Age 32 years. C/O Menorrhagia for 4 years following sterilisation 4/11/59 Hysterectomy (13th. day of cycle) (Endometrium not reported on(Had D&C in George : Metropathia).

42 (a)

Report from Pathology Department.
(Chemical Pathology). MGA 7532.
UNIVERSITY OF CAPE TOWN.

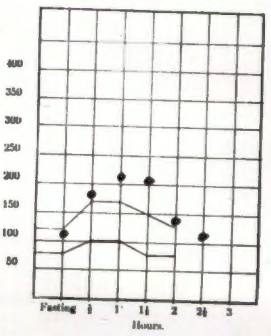
Serial No. 39447 - 448 Date 13.11.59.

Patient's Identification MARIA VAN RENSBURG (59/11885)

Ward A3. Physician or Surgeon Dr. Benjamin.

GLUCOSE TOLERANCE TEST.

Test-hour	URINE SUGAR
1. 0	0
Specimen mixed 2½ hours sugar	
72½ hours urine.	



BLOOD SUGAR	
Fasting	125 mgs.%.
½ hour	181 mgs.%.
1 hour	229 mgs.%.
1½ hour	223 mgs.%.
2 hour	146 mgs.%.
2½ hour	117 mgs.%.
3 hour	

Signature G M POTGIETER

Glucose Tolerance Test Before F.S.H. Administration.

AFTER F.S.H. ADMINISTRATION.
3 daily injections: 5,000 units each.

42 (b)

Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

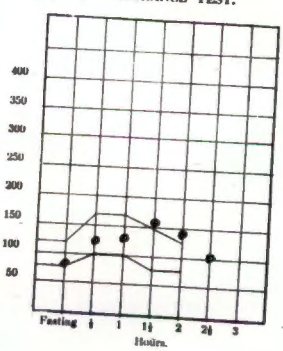
Serial No. 40323 - 324 Date 17.11.59.

Patient's Identification MARIA VAN RENSBURG (59/11885)

Ward A3D. Physician or Surgeon Dr. Benjamin.

GLUCOSE TOLERANCE TEST.

Test-hour	URINE SUGAR
1. 0	0
2. 0	0



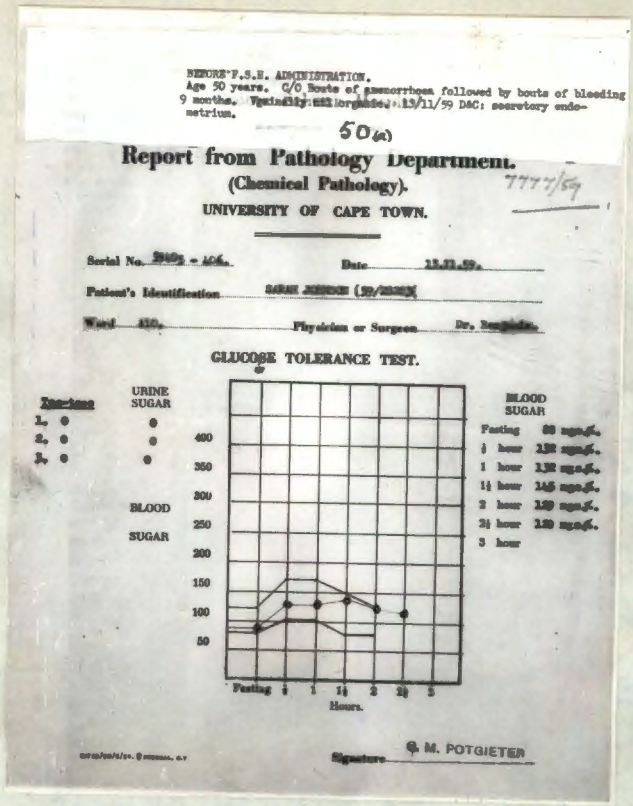
BLOOD SUGAR	
Fasting	84 mgs.%.
½ hour	185 mgs.%.
1 hour	134 mgs.%.
1½ hour	161 mgs.%.
2 hour	148 mgs.%.
2½ hour	104 mgs.%.
3 hour	

Signature G M POTGIETER

Glucose Tolerance Test After F.S.H. Administration.

Case 3:

S.J., aged 50 years, complained of bouts of amenorrhoea, followed by bouts of profuse bleeding, for 9 months. The cervix, uterus, and adnexa were normal. A curettage on the 13/11/1959 revealed a normal secretory endometrium. The blood sugar levels before F.S.H. administration were: fasting 88, ½ hour 132, 1 hour 132, 1½ hours 145, 2 hours 129, 2½ hours 120 mgs.% respectively; after F.S.H. injections the respective readings were: 83, 110, 159, 102, 101, 115 mgs.%.



BEFORE F.S.H. ADMINISTRATION.
Age 50 years. C/O Route of amenorrhoea followed by bouts of bleeding 9 months. Vaginally: [illegible] 13/11/59 D&C: secretory endometrium.

50(a)

Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

7777/59

Serial No. [illegible] Date 13.11.59.

Patient's Identification [illegible]

Ward 410. Physician or Surgeon Dr. Benjamin.

GLUCOSE TOLERANCE TEST.

Reactions	URINE SUGAR
1.	
2.	
3.	

BLOOD SUGAR

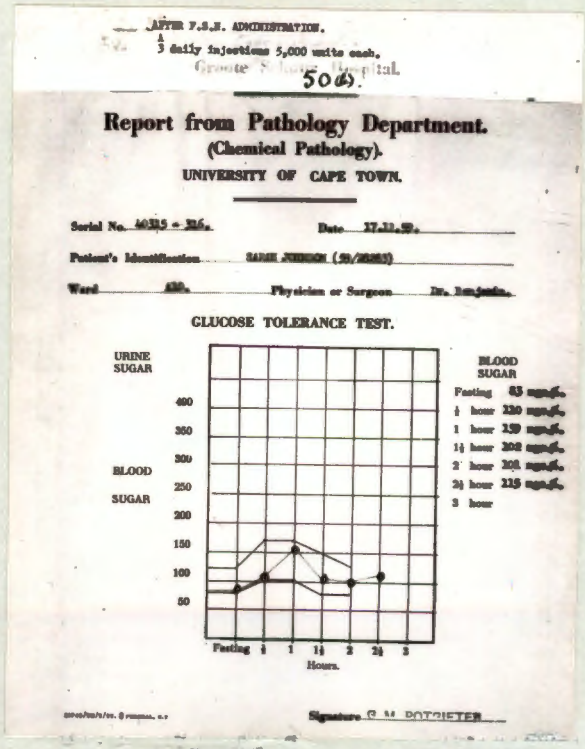
400 350 300 250 200 150 100 50

Fasting ½ 1 1½ 2 2½ 3
Hours.

BLOOD SUGAR
Fasting 88 mgm.%.
½ hour 138 mgm.%.
1 hour 138 mgm.%.
1½ hour 146 mgm.%.
2 hour 129 mgm.%.
2½ hour 129 mgm.%.
3 hour

Signature G. M. POTGIETER

Glucose Tolerance Test Before F.S.H. Administration.



AFTER F.S.H. ADMINISTRATION.
3 daily injections 5,000 units each.
Groote Schuur Hospital.

50(b).

Report from Pathology Department.
(Chemical Pathology).
UNIVERSITY OF CAPE TOWN.

Serial No. [illegible] Date 17.11.59.

Patient's Identification [illegible]

Ward [illegible] Physician or Surgeon Dr. Benjamin.

GLUCOSE TOLERANCE TEST.

URINE SUGAR

BLOOD SUGAR

400 350 300 250 200 150 100 50

Fasting ½ 1 1½ 2 2½ 3
Hours.

BLOOD SUGAR
Fasting 83 mgm.%.
½ hour 230 mgm.%.
1 hour 139 mgm.%.
1½ hour 202 mgm.%.
2 hour 202 mgm.%.
2½ hour 235 mgm.%.
3 hour

Signature G. M. POTGIETER

Glucose Tolerance Test After F.S.H. Administration.

In each of these cases, then, the opposite result to that expected, was obtained. Instead of aggravating glucose tolerance, there was a marked improvement. Indeed, in the case of Mrs. M. vd.W., an unquestionably diabetic curve was converted into a normal curve, and both the other curves were distinctly improved.

Obviously, similar studies on a much larger scale must be done before any definite conclusions can be reached. But it would appear from these isolated experiments that the presence of excess F.S.H. in cases of benign glandular hyperplasia is <u>not</u> the cause of the impaired glucose tolerance found in such patients. It is more likely that the disturbed glucose tolerance is the cause of or abnormality associated with the excess F.S.H. or other endocrine inbalance which is responsible for benign glandular hyperplasia.

4. <u>THE GROWTH HORMONE OF THE ANTERIOR PITUITARY:</u>

In addition to its protein-anabolysing action, the growth hormone influences carbohydrate

metabolism. The anterior pituitary influences carbohydrate metabolism in two general ways: (a) through the adrenocorticotrophic hormone stimulating the adrenal cortex, and to a lesser extent through the thyrotrophic hormone, stimulating the thyroid, and (b) by direct action, the directly acting hormone being the growth hormone. Some of the actions to be described have been studied with the use of crude preparations only, whereas others have been obtained recently with pure growth hormone preparations. Growth hormone lowers the respiratory quotient (Greaves et al., 1940), and maintains muscle glycogen ("glycostatic action") (Hering and Evans, 1943; Russell and Wilhelmi 1950). It appears very likely that the chief action in this connection consists in inhibition of carbohydrate utilization (Lukens, 1946). In the hypophysectomised animal the utilization of glucose is enhanced (Krahl, 1955; Renold, et al., 1956). This effect can be studied in balance experiments; it is reflected in the high uptake of glucose by the diaphragm of the hypophysectomised rat in vitro. Growth hormone treatment of the hypophysectomised rat returns the uptake to normal;

this effect is predicated upon a permissive action of adrenocortical steriods; experiments on hypophysectomised-adrenalectomised rats show that a full effect of growth hormone treatment can only be obtained if the animals also receive a small amount of adreno-cortical extract. The effect of growth hormone just described is an anti-insulin effect inasmuch as insulin enhances the glucose uptake and is capable of overcoming the effect of the growth hormone. Similarly, the two hormones have an opposing effect on glycogen synthesis in the diaphragm, inasmuch as the latter is greatly enhanced in the hypophysectomised animal and returned to normal only by continued treatment with growth hormone and cortisone (Stadie, 1954).

The hypophysectomised animal and the patient suffering from panhypopituitarism exhibit a greatly increased sensitivity to insulin. This is undoubtedly a complicated phenomenon, because a number of hormones counteract, by way of different mechanisms, the blood-sugar-lowering action of insulin. The two most important pituitary hormones in this regard are A.C.T.H. and growth hormone. It is, therefore, of

interest that treatment of hypophysectomised dogs with growth hormone abolishes the excessive insulin sensitivity, even in the absence of adrenals (De Bodo and Altzuler, 1955).

Much experimental work has been carried out since 1930 on the aetiology of diabetes by Houssay (1942) and his co-workers in the Argentine, and by F.G. Young (1951) in Cambridge. For a large number of years, mainly due to the work of Cushing, glycosuria was thought to be due to lesions of the posterior pituitary. In 1930 Houssay and Biasotti showed that total hypophysectomy ameliorated pancreatic diabetes, but that subsequent injection of anterior pituitary extract intensified it.

F.G. Young in Cambridge using a crude saline extract of anterior pituitary, produced severe diabetes in dogs, and subsequently produced it with purified extracts of anterior pituitary growth hormone. He explains, in the case of purified extracts, however, that he is uncertain whether the effect is not produced by slight contamination with other pituitary hormones. These diabetogenic growth-

producing extracts when given to puppies and kittens produce growth and not diabetes, but in the adult animal where body growth is already completed, injection of these extracts resulted first in an initial rise in body weight, and then in the development of diabetes. Young failed completely to induce diabetes with these extracts in pregnant animals. Instead they deliver themselves of a greatly overweight litter, somewhat reminiscent of the human pregnant diabetic and the large babies. Here, therefore, according to Way (1954), we have evidence of the anterior pituitary being at fault as one of the causative factors in carcinoma of the body of the uterus.

In the light of subsequent work it was believed that this diabetogenic action of anterior pituitary extracts was mediated through the adrenal cortex. Subsequently, however, Young et al. (Cotes, Reid and Young, 1949; Young 1949; Young 1938) showed that the diabetogenic effect of pituitary extracts may be obtained in the adrenalectomised dog, and that the diabetogenic action of growth hormone is due to inhibition of peripheral utilization of

carbohydrate. In the fasting rat, anterior pituitary extracts produce hypoglycaemia by diminishing the availability of protein for carbohydrate formation (Harrison et al., 1940; Milman and Russel, 1950; Pashkis, 1942).

The influence of the anterior pituitary on carbohydrate metabolism and the diabetogenic action of the anterior pituitary are exerted through several channels. Only the action of the growth hormone has so far been discussed. There remains to consider the action of the anterior pituitary mediated through stimulation of the adrenal cortex and through the thyroid.

It is indeed possible therefore, that the aberrations in the glucose tolerance curve and the condition of benign glandular hyperplasia, are both due to disburbances in the anterior pituitary gland. The growth hormone can cause diabetes and less severely disturbed glucose tolerance. Abnormal activity of the gonadotrophic hormones may well cause benign glandular hyperplasia. The improvement in the glucose tolerance curve in the patients to whom I

gave large doses of F.S.H. is very interesting. It would tend to show that F.S.H. per se does not cause the aberrations in the curve - but does not disprove the possibility of both the latter and benign glandular hyperplasia being due to anterior pituitary dysfunction.

5. THE ADRENAL CORTEX:

The corticosteriods ("sugar hormones", 11, 17 - oxysteriods, glucocorticoids) are produced by the zona fasciculata of the adrenal cortex and have widespread metabolic effects. Hydrocortisone (cortisol, compound F), the chief representative of this group, accounts for 80 per cent of the corticosteriods found in the adrenal venous blood, and can produce all the known physiological and metabolic effects resulting from adrenal cortical stimulation. (Rukes et al.,1955). These compounds influence carbohydrate metabolism by increasing gluconeogenesis, promoting hepatic deposition of glycogen, inhibiting the peripheral utilization of glucose, decreasing the sensitivity to insulin, elevating the threshold

for hypoglycaemic symptoms, and lowering the renal tubular resorption of glucose. (In addition corticosteroids are anti-anabolic, or possibly catabolic, and produce a negative nitrogen balance. They also mobilise peripheral fat stores, have important anti-inflammatory effects, alter certain enzyme functions and produce both Eosinopaenia and lymphopaenia).

The active adrenocortical steroids differ in their potency as "sugar hormones". Hydrocortisone is the most potent; the activity of cortisone is about four-fifths that of hydrocortisone. The activity of the compounds oxygenated only at C 11 (corticosterone, 11 - dehydro-corticosterone, and aldosterone) is much weaker than that of the C 11 - C 17 oxygenated steroids. Corticosterone (Compound B of Kendall) has about one half, and aldosterone about one third, of the activity of cortisone. 11 - Desoxycoticosterone is, for practical purposes, devoid of carbohydrate activity; in very large doses, entirely outside any conceivable physiological range, it is active, 100 mgm. being equivalent to 1mgm.

cortisone. The ratio of activity of the compounds secreted by the gland is approximately: Hydrocortisone: Corticosterone: Aldosterone = 1: 0.4: 0.26: (Noble, 1955).

Theoretically, adrenal cortical dysfunction could be responsible for the aberrations in the glucose tolerance curves in benign glandular hyperplasia. However, there is every reason to believe that the disturbed glucose tolerance in these cases was due to the ordinary pituitary-type of diabetes - clinically the frank diabetics were of this type, and follow-up of the less severely affected ones, showed that some of them became ordinary pituitary-type diabetics. It has been shown that the excretion of gluco-corticoids in diabetes mellitus is normal (Reed, 1955).

It is, therefore, most unlikely that the adrenal cortex can be implicated as the cause of the metabolic disturbance and the endometrial picture. But assays of adrenocortical hormones in benign glandular hyperplasia have not been reported on, and should be made.

6. THE THYROID GLAND:

In hyperthyroidism fasting blood sugar values are frequently normal, but in severe thyrotoxicosis they may be moderately elevated, seldom exceeding 150 mgm.%. Higher values suggest co-existing diabetes.

Oral glucose tolerance tests characteristically reveal a rapid sharp rise in blood sugar, the values in the first hour often being 100% or more above the fasting value. This is due in part to an increased rate of absorption from the intestine (Althausen et al., 1940), and in part to increased glycogenolysis in the liver. The curve, however, in contradistinction to the plateau type of curve seen in diabetes, falls rapidly, since peripheral utilization of glucose is increased as a result of increased oxidation. This type of blood sugar curve is seen in severe cases. The practical usefulness of the test is in differentiating glycosuria and mild hyperglycaemia due to thyrotoxicosis, from co-existing diabetes. The same type of curve may be found in the presence of hepatic disease and sympathicotonia,

amongst other conditions.

In hypothyroidism, fasting blood sugar levels are normal. Oral administration of sugar results in "flat" blood sugar curves, owing to a diminished rate of absorption. Intravenous administration of these sugars yields essentially normal curves. (Althausen et al., 1940).

The thyroid gland cannot be implicated in the abnormal glucose tolerance found in my cases of benign glandular hyperplasia. In none of these 50 cases was there clinical evidence of hyperthyroidism. In fact in one the patient was found clinically to be hypothyroidic, and special investigations confirmed this. Treatment with thyroid hormone cured the myxodema and the dysfunctional uterine bleeding. This is in keeping with the reports in the literature; the latter indicate that hypothyroidism is often associated with uterine bleeding of the benign glandular hyperplasia type, whereas hyperthyroidism is more likely to be associated with oligo, hypo, or amenorrhoea (Israel, 1959).

7. PROLACTIN:

Changes in carbohydrate metabolism have been noted to follow the injection of prolactin. This was found in 1949 by Houssay and Anderson, but their lactogenic preparation contained up to 30 per cent. of A.C.T.H. Foa and his co-workers (1954,1955) and Houssay et al. (1955) later showed that the administration of a purer form of prolactin isolated from animal pituitaries to intact dogs produces initial hypoglycaemia which is then followed by hyperglycaemia. Pancreatectomy eliminates the hypoglycaemic but not the hyperglycaemic phase. This suggests that the lowering of the blood sugar is produced by the release of pancreatic insulin, and eliminated the possibility that the subsequent hyperglycaemia is attributable to a release of glycogen or hyperglycaemic glucagon glycogenolytic factor which may be secreted by the alpha cells. Examination of the pancreas of animals following the first intravenous injection of prolactin reveals degranulation of beta cells suggestive of insulin release, but it may be related to the diabetogenic effect of oestrogens released in small amounts by the gonads in response

to prolactin.

No reports have appeared in the literature regarding the relationship, if any, of prolactin to benign glandular hyperplasia. It is most unlikely that this hormone plays any part in the disease.

8. CARBOHYDRATE METABOLISM IN THE ENDOMETRIUM:

Since there is a disturbance of glucose tolerance in such a high proportion of cases of benign glandular hyperplasia, it is necessary to discuss the physiology and pathology of carbohydrate metabolism in the endometrium itself, this latter being the site of the main histopathology.

Hughes (1950) studied the carbohydrate metabolism in the endometrium, and it is relevant to recapitulate much of his work on this problem. The metabolism of carbohydrate that takes place in the endometrium apparently passes through the many chemical changes found in other parts of the body where storage of glycogen occurs. The amount of sugar is probably determined by the type of arteriolar development. Glucose must be converted into glycogen,

and be stored in the endometrium as such. It must be phophorylyzed to glucose 6 - phosphate and then down through the various stages of polymerization into glycogen. The process of conversion and storage from glucose to glycogen occurs at ovulation time, and extends for several days thereafter. When implantation is about to take place, glycogen not being usable per se, is again changed back to simple monosaccharide, either glucose or fructose. Probably many enzymes are involved in this two way process and many of them have been isolated. It has been found that the phosphatase group plays an important role in this reaction, and it is probable that certain vitamins are also a part of the process (Hughes, 1950).

Alkaline phosphatase, as noted by histochemical methods, appears in the epithelial cells of the endometrial glands before glycogen is actually present. At about ovulation time, and shortly thereafter, this enzyme increases markedly in amount in the nuclei and the cytoplasm as glycogen is about to be stored. On the 26th day of the cycle

it is found in large quantities in the endometrium of the spiral arterioles and free in the lumen of the endometrial glands. It is difficult to understand why this enzyme is not found in the epithelial cells of the gland at this time, when it is noted in such quantity in the other 2 locations. The veins, lymphatics, and probably the basal arteries do not seem to contain this enzyme. Phosphatase is also found in large quantities in hyperplasia of the endometrium and in the non-ovulatory type of endometrium. The presence of alkaline phosphatase in these instances, when glycogen is not present, or is found in smaller amounts, is not easily explained. It could be conjectured that the amount of glucose brought to the endometrium is diminished due to the lack of arteriolar formation. The epithelial cells of the uterine glands at this time of the cycle furnish the enzyme, which is provided later by the endothelium of the arterioles.

Formation and storage of glycogen begin after the secretion of alkaline phosphatase in the epithelial cells of the endometrial gland. The glycogen accumulates at the bases of these cells at ovulation

time and appears as red granules when the material is stained with carmine. The accumulation causes the nuclei to be pushed up into the midportion of the cells at about ovulation time. This stratification of the nuclei remains until the beginning of the secretory phase. At nidation time, glycogen passes to the periphery of the cells, and the nuclei settle down again to their cellular bases. At the time of implantation, glycogen is released into the lumen of the glands and upon the surface of the endometrium. At this point it is again converted back to glucose or fructose by further enzymatic action. It has been found that glycogen is not present when ovulation does not occur or when there is hyperplasia of the endometrium. These are the very cases where I have found such a high incidence of disturbed glucose tolerance.

Many enzymes are probably needed for the process of glycogenesis just described. Not only are the phosphatase enzymes important in this respect, but glycogen-splitting enzymes have been detected and quantitated by a glycogen hydrolysis at various stages of the cycle. Samples of endometrium were

removed from the uterus on the various days of the month, and were quantitated for this enzyme (Hughes, 1950). It was found to be present in smaller amounts in the pre-ovulatory phase, and to increase markedly at the time of ovulation and during the secretory portion of the cycle. After the menopause the amount of enzyme was diminished. At this time glycogen is absent, or present only in small quantities.

The endometrium must proceed through the normal histological changes that are characteristic of each phase of the cycle in order to have normal glycogenesis take place. The entire process of glucose metabolism in the uterus observed histologically seems to be in keeping with the theories of glycogenesis observed elsewhere in the body. It has been stated that there must be a phosphorylated intermidiate, such as the Coyi ester, for this transformation. Such circumstances are seemingly present within the endometrium (Hughes, 1950; Wislocki and Dempsey, 1945).

Profound activity in the way of carbohydrate metabolism, therefore, takes place in the endometrium,

in the various stages of the menstrual cycle. Marked disturbances in this metabolism have also been demonstrated when there is endometrial hyperplasia or non-ovulation. It is understandable, then, that if the glucose tolerance of the body is disturbed the carbohydrate metabolism in the endometrium could be upset. Whether aberrations in glucose tolerance can cause the histological changes in the endometrium seen in benign glandular hyperplasia, and in endometrial carcinoma, is a very important question, and is indeed worthy of further investigation. It would seem to be a distinct possibility in view of my findings of the frequent presence of disturbed glucose tolerance in these two conditions.

SUMMARY.

In an attempt to detect the reason for the very high incidence of impaired glucose tolerance in cases of benign glandular hyperplasia, the literature was extensively studied, and several original investigations were done. The conclusion is reached that, in the present state of our knowledge, the association cannot be explained. Although there is much evidence that oestrogens, F.S.H., adrenal cortical hormones, thyroid hormone, and prolactin can influence carbohydrate metabolism, it is unlikely that they can be incriminated as the cause of the altered glucose tolerance curves in this condition. Evidence is adduced to show that the absence of progesterone, and a disturbed function of anterior pituitary gland may play an important role. The altered carbohydrate metabolism in the endometrium itself may be an important factor.

Lines of further research are indicated which may clarify this mystery. Such research may also throw more light on the association of benign glandular hyperplasia and cancer of the endometrium, and indeed may help to detect the causes of the latter type of malignant development.

CHAPTER 7.

INVESTIGATION INTO THE AGES OF THE MENOPAUSE IN WOMEN IN THE WESTERN CAPE PROVINCE.

CHAPTER 7.

INVESTIGATION INTO THE AGES OF THE MENOPAUSE IN WOMEN IN THE WESTERN CAPE PROVINCE.

The abrupt rise in the incidence of impaired glucose tolerance after the age of 45 years has been shown by my aforementioned investigations, and is summarised as follows:

TABLE 13. Glucose Tolerance in 100 Control women with normal endometria. 45 years and over.

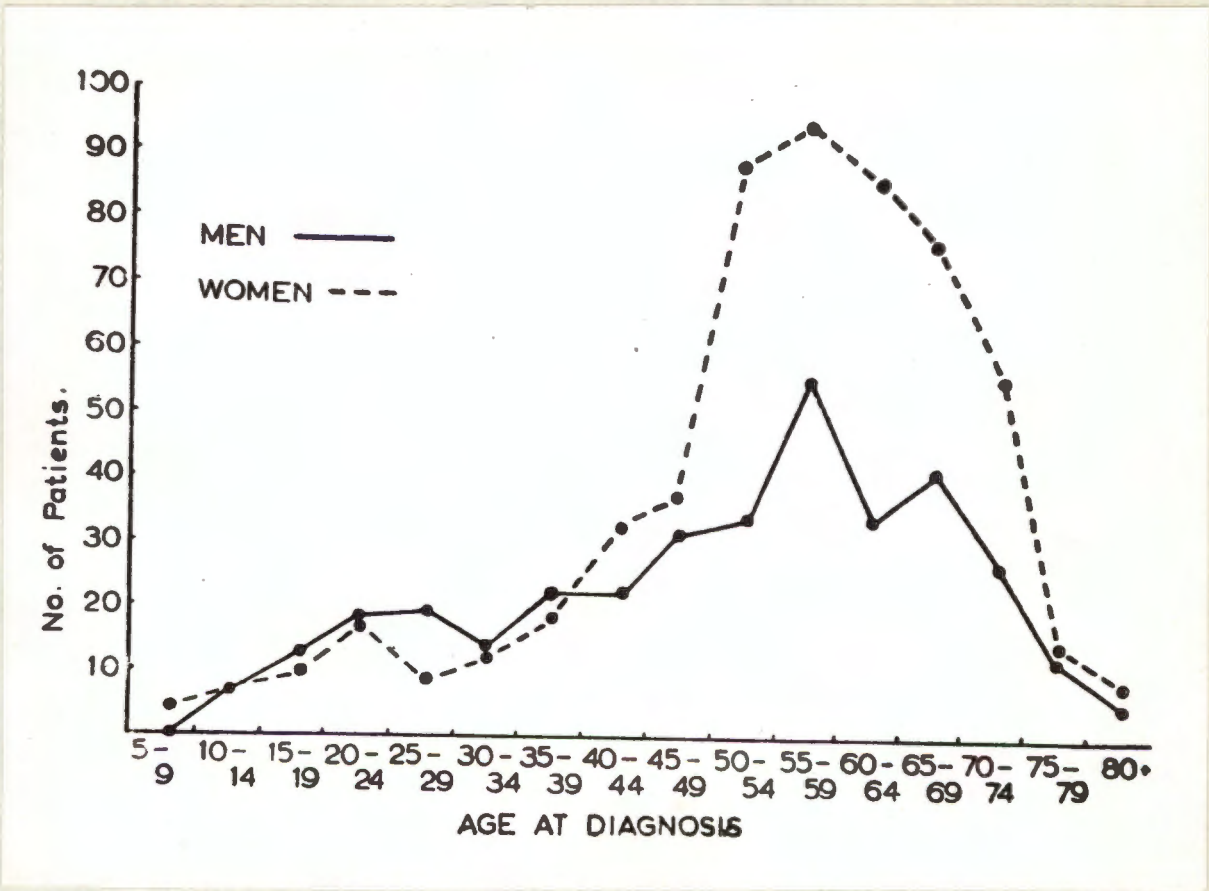
Normal Curves	Abnormal Curves.		
	Diabetics	Mildly Impaired Glucose Tolerance	Total.
78%	13%	9%	22%

TABLE 14. Glucose Tolerance in 50 Control Women with Normal endometria. Between 35 and 45 years.

Normal Curves	Abnormal Curves		
	Diabetics	Mildly Impaired Glucose Tolerance	Total
92%	2%	8%	10%

My own investigation results correspond very closely with the findings of Pyke (1959). The latter investigated the age and sex distribution of 953 diabetics attending the Radcliffe Infirmary, Oxford, England. Among the younger patients there were a few more men than women, but after the age of 40, and especially 45 years, women heavily outnumbered men. The incidence of diabetes in women showed a precipitous rise at the age of 45 to 50 years. This is demonstrated by Graph I reproduced from Pyke.

This sudden rise in incidence, being about the time of the climacteric, makes it possible that the



GRAPH 1.

AGE AND SEX DISTRIBUTION OF DIABETICS (Pyke, 1959.)

menopause and its associated change in ovarian and pituitary function may be a factor in causing impaired glucose tolerance.

It thus became necessary to determine what, in fact, was the menopausal age in the population from which the subjects of this investigation were drawn. A study of the literature showed that no such investigation had been carried out in South Africa.

I therefore proceeded to fill this gap by studying:

1. The ages of the menopause in 1,000 women who served as controls.

2. The ages of the menopause in 100 postmenopausal women attending the diabetic clinic of the Groote Schuur Hospital.

3. The ages of the menopause in 100 patients with carcinoma of the endometrium.

METHOD:

For the purpose of this analysis 1,000

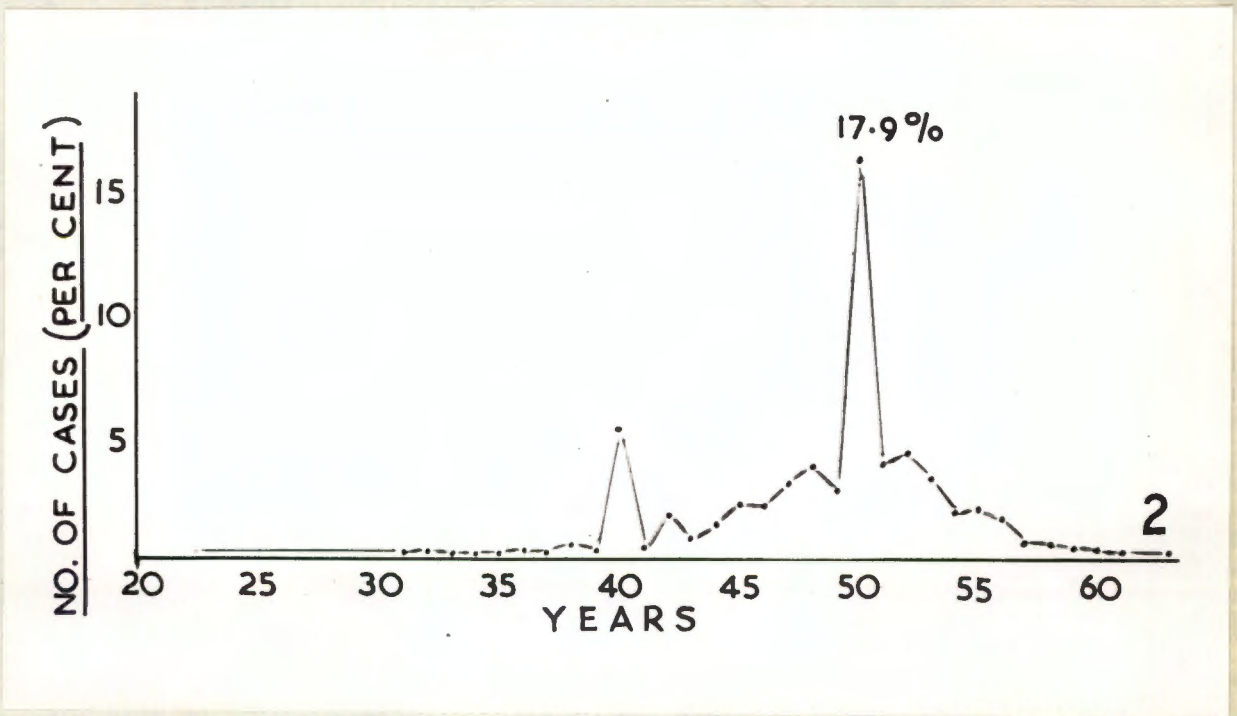
random women, who had already reached the menopause, were interrogated. These comprised white patients attending the Medical and Casualty Departments of the Groote Schuur Hospital, Cape Town, some of their visitors, and also patients who were seen at the Gynaecological Outpatient Department for conditions which would obviously not affect Menstrual function such as discharges, prolapse. In addition to these thousand women who served as controls, another 2 groups of patients were interrogated along the same lines, or the information was obtained from the records of the hospital. These groups were 100 post-menopausal women attending the diabetic clinic, and 100 postmenopausal women who had developed cancer of the endometrium.

The reason why the investigation was limited to white women, was that it was not possible to obtain accurate data from sufficient Bantu African women. Many of the latter were unaware of their age, let alone the age of the menopause. All patients who were uncertain about the age of the menopause were excluded; likewise only those who had lived in the Cape Province

for most of their lives were included. The women were questioned about the age of the menopause (as well as about the age of the menarche, the marital status and the number of children and abortions). Although in some cases menstruation ceased abruptly, there was more commonly an alteration in the cycle at the climacteric, with oligo-menorrhoea and bouts of amenorrhoea - for this analysis the menopause was only regarded as having occurred when a year had elapsed with freedom from bleeding; any bleeding thereafter was recorded as postmenopausal bleeding. In cases where the age was given in terms of years and months, the nearest year was taken, i.e. below 6 months was placed in the category of the preceding year, and above 6 months in the subsequent year.

THE AGE OF THE MENOPAUSE IN THE CONTROL GROUP:

The ages of the menopause in the 1,000 control women are shown in Graph 2. The average age of the menopause was 48.7 years, but there is a striking peak (17.9% of the total) at 50 years. The



GRAPH 2.

AGES OF THE MENOPAUSE IN 1,000 WOMEN IN THE WESTERN CAPE PROVINCE.

details are listed in Table 15. Although the peak incidence is at exactly the same age (50 years) as that found by other authors (Way, 1954), yet all the other evidence shows that the menopause tends to occur later in South African women than in women in other countries. Thus the Medical Women's Federation Investigation (1933) showed that of 966 women, 71% ceased menstruating before 50 years, and 29% thereafter. Of 536 patients Way (1954) found that 65% ceased to menstruate before 50 years, and 35% after 50 years. The figures for South African women, by comparison, are 45.2% and 54.8% respectively.

TABLE 15: AGES OF THE MENOPAUSE IN 1,000 WOMEN IN THE WESTERN CAPE PROVINCE.

No. of Cases	Age at Menopause
3	23
1	31
3	32
1	33
2	34
1	35
5	36
4	37
10	38
9	39
52	40
7	41
35	42
16	43
29	44

No. of Cases	Age at Menopause
41	45
41	46
60	47
75	48
57	49
179	50
74	51
83	52
64	53
38	54
40	55
31	56
13	57
11	58
6	59
6	60
2	61
1	63
AVERAGE AGE	48.7

AGE OF THE MENOPAUSE IN DIABETES MELLITUS:

Of 100 consecutive postmenopausal patients seen at the diabetic clinic, 39% ceased menstruating before the age of 50 years, 15% at 50 years, and 46% after 50 years of age. The diabetic, then, tends to have a later menopause than the non-diabetic. The detailed ages are shown in Table 16.

TABLE 16. DIABETES MELLITUS. (100 cases)

Age of Menopause	No. of Cases.
37	1
38	2
39	1
40	3
41	2
42	6
43	3
44	4
45	1
46	1
47	8
48	5
49	2
50	15
51	13
52	7
53	8
54	5
55	2
56	3
57	4
58	2
59	2
60	0
Before 50 years	39%
At 50 years	15%
After 50 years	46%

CARCINOMA OF THE ENDOMETRIUM AND THE AGE OF THE MENOPAUSE:

One hundred cases of carcinoma of the endometrium who were postmenopausal were analysed separately. The ages of the menopause are shown in Table 17. Only 26% ceased menstruating before the age of 50 years, whereas 30% were 50 years of age, and 44% over 50 years. Comparing this with the figures for the control group (42.2%, 17.9% and 36.9% respectively), the menopause is seen to occur later in cases who subsequently develop carcinoma of the body of the uterus. The comparisons are made in Table 18.

TABLE 17. AGES OF THE MENOPAUSE IN CASES OF CARCINOMA OF THE BODY OF THE UTERUS.

(100 Cases).

Age of Menopause	No. of Cases.
45	4
46	6
47	8
48	3
49	5
50	30
51	7
52	13
53	8
54	6
55	4
56	2
62	3
63	1
Before 50 years	26%
At 50 years	30%
After 50 years	44%

TABLE 18. AGE OF MENOPAUSE IN CONTROL AND OTHER GROUPS.

	Before 50 years	At 50 years	After 50 years
Control Series (1,000 women)	45.2%	17.9%	36.9%
Carcinoma of the Body of the Uterus (100 cases)	26%	30%	44%
Diabetes Mellitus (100 cases)	39%	15%	46%

SUMMARY AND CONCLUSIONS:

In the population of the Western Province where this investigation was carried out, the average age of the menopause is 48.7 years, with a peak at 50 years. This is later than the age in other countries. Women who develop carcinoma of the body of the uterus in the postmenopausal era, tend to have a later menopause than women in a control group; the same phenomenon was observed in a series of cases of diabetes mellitus, but this is not as marked as in patients with endometrial cancer.

The rising incidence of impaired glucose tolerance does, therefore, correspond with the age of the menopause. The explanation of this, however, is far from clear. The role of oestrogens in carbohydrate metabolism has already been discussed, (page 208), and the diminishing levels of oestrogen secretion after the menopause has been mentioned (page 204). If increased oestrogen activity were a factor in the production of cancer of the endometrium (see page 198), then the late menopause in this disease would be understandable.

However, if large doses of estrogens improve diabetes mellitus, it is difficult to understand why the menopause tends to occur late in diabetics. The problem is far from being resolved, and many complex factors must be involved in the increased incidence of diabetes in women after the age of 45 years.

CHAPTER 8.

SUMMARY.

CHAPTER 8.

SUMMARY.

The grossest forms of Diabetes Mellitus were recognised as long as 2,000 years ago. With the passing of the centuries a vast knowledge of the disorder has accumulated. Yet there is much that is still unknown.

In the past few decades mild forms of disturbed glucose tolerance have become recognised, and more and more manifestations of this metabolic disturbance have been discovered. This thesis is an attempt to advance a stage further our knowledge of disturbed glucose tolerance in the gynaecological sphere. It represents a study of the association of imparied glucose tolerance with certain pathological states of the endometrium, including cancer; and it brings to light a manifestation that has not been studied before, namely, certain benign dysfunctional endometrial bleedings. Evidence is produced to show that many of the dysfunctional uterine

bleedings, especially at the time of the climacteric, are associated with abnormal glucose tolerance curves; and these bleedings may be further manifestations of a "pre-diabetic", unrecognised diabetic, or frankly diabetic state.

EVOLUTION OF THE PRESENT INVESTIGATION.

In the ubiquitous search for the aetiology of cancer, metabolic factors have been widely studied. Much attention has been paid to carbohydrate metabolism and glucose tolerance. In 1934 Marble made studies to determine whether the incidence of all cancers is higher among diabetics than among non-diabetic individuals; in 1948, Jacobson reported on a similar investigation. The results of these studies were conflicting.

In the 2 decades that followed, however, increasing evidence appeared that, when it came to a study of cancer of the endometrium, the association of diabetes mellitus was much more significant. Several writers found that as many as one-quarter to

one-third of such patients had unquestionable diabetes, and a further one-quarter to one-third, an impaired glucose tolerance curve. However, other workers were unable to find this raised incidence of diabetes mellitus in their cases. I studied all these publications most carefully to determine why these reports were so contradictory, and was able to find the reasons for the discrepancies. Whenever the cases were investigated by complete glucose tolerance tests, a high incidence of diabetes mellitus was found. In each and every series where the incidence of diabetes mellitus was low, routine glucose tolerance tests had not been done - the disease had only been diagnosed when the patient was an obvious diabetic. I carried out investigations myself with the aid of glucose tolerance tests, and was able to confirm this view and show that the high incidence of diabetes mellitus and of mildly impaired glucose tolerance in these cases is incontrovertible.

However, even these proven high figures are not significant unless they are compared with suitable control subjects, i.e. women of the same age groups, and under similar conditions, who do not

suffer from cancer of the endometrium. An exploration of the literature revealed several studied, but none of these was suitable as a control group for this investigation. Not one of the bulk population diabetic surveys was done by routine glucose tolerance tests, and other investigations reported in the literature were not comparable groups. I therefore carried out full glucose tolerance tests on 100 random gynaecological cases of 45 years and over to serve as a suitable control group. Because of the well-known rise in the incidence of diabetes mellitus with age, a further control series of 50 women between 35 and 45 years of age were similarly studied.

Having obtained proper and suitable control groups, I was now in a position to compare these with the results obtained in cases of carcinoma of the endometrium. I went further, and made glucose tolerance studies on all cases of abnormal uterine bleeding, of both benign and malignant aetiology, that were encountered round about and after the climacteric. In each case the results

were correlated with the histological picture of the endometrium, because the latter is a mirror of pituitary - ovarian endocrine activity. Some interesting and significant findings came to light, which will be presented.

The following groups, then, were investigated by carrying out glucose tolerance tests in each case:

1. 100 random women of 45 years of age and over, who were not suffering from cancer of the endometrium or benign glandular hyperplasia.

2. 50 similar women between 35 and 45 years of age, to serve as further control subjects in this lower age group.

3. 50 cases of carcinoma of the endometrium.

4. 50 cases who were proven by histological examination of the endometrium to have glandular hyperplasia.

5. 10 consecutive cases in whom the endometrium was not a hyperplastic one, but where secretory changes were absent.

In certain of the above groups striking abnormalities of glucose tolerance were found (these will be described later). In an attempt to determine the cause of these abnormalities of glucose tolerance other investigations were made:

1. Follicle-stimulating hormone was administered to patients and the effect of this on the glucose tolerance test was studied.

2. Oestrogens were administered to post-menopausal women with normal glucose tolerance curves, to see whether this altered the curves.

3. Patients who had benign glandular hyperplasia with abnormal glucose tolerance, and who had undergone a hysterectomy, were followed up, and the glucose tolerance curves repeated after operation.

4. It is emphasized that many exogenous factors may influence the glucose tolerance curve. The same standard conditions were therefore applied when each test was carried out.
However, in view of the possibility of unknown exogenous factors being present and influencing

the curves, a number of cases, where aberrations were found, was submitted to a repeat test at a different time. The fact that all of these showed similar aberrations in the repeat tests, confirmed that these abnormalities in the curves were, in fact, due to true endogenous abnormalities of glucose tolerance, i.e. the findings were not caused by technical errors or extraneous influences.

5. The study of the abovementioned control groups revealed an abrupt rise in the incidence of abnormal glucose tolerance after the age of 45 years. In order to determine whether this coincided with the age of the menopause, it was considered necessary to establish the age of the menopause in Cape Town. This was done by studying a thousand random women in the Western Cape who had already reached the menopause, to determine the average age of the menopause in this group. Likewise, 100 postmenopausal women attending the diabetic clinic of the Groote Schuur Hospital, and 100 postmenopausal women with cancer of the endometrium were investigated along the same lines.

THE GLUCOSE TOLERANCE TEST:

Since the glucose tolerance test is the main test on which this investigation is based, the subject is discussed in detail. It was necessary to decide on a standard glucose tolerance test, and on a standard interpretation, and then to apply these identical conditions to the control and other groups.

For many reasons that are put forward, the "Standard" (50 G) oral glucose tolerance test was chosen, and was carried out under strictly laid down criteria in each subject investigated.

The interpretation of the glucose tolerance curve is a very difficult subject and is discussed in detail. Glucose tolerance curves can be divided into 3 groups, namely, normal, diabetic and an intermediate group. The last-mentioned group is the most difficult to interpret and reasons are submitted for labelling this group "mildly impaired glucose tolerance".

GLUCOSE TOLERANCE INVESTIGATIONS AND RESULTS:

Using the same glucose tolerance test on each subject investigated, and placing the same standard of interpretation on the results, the following groups were studied. In view of the known effect of age on the glucose tolerance, this factor was constantly taken into consideration. A photograph of each original glucose tolerance curve is shown, as well as a summary of the clinical findings and the histological picture of the endometrium.

1. Women who acted as Control Subjects;

(a) Subjects of 45 years of age and over:

One hundred consecutive women of 45 years of age and over, who were not suffering from cancer of the endometrium or benign glandular hyperplasia were submitted to the glucose tolerance test. In each case the endometrium was examined histologically. All these women had a normal endometrium - an endometrium which was either proliferative or secretory, or physiologically atrophic due to the menopause.

In this group 78% had normal curves, 13% diabetic curves, and 9% mildly impaired curves - the total of abnormal curves was, therefore, 22%.

(b) Control subjects between 35 and 45 years of age:

Fifty consecutive women with normal endometria, between 35 and 45 years of age, were also examined and subjected to the standard glucose tolerance test. The incidence of disturbed glucose tolerance was remarkably lower than in the over 45 year group, the results being as follows: normal curves 90%; diabetic curves 2%; mildly impaired curves 8%.

2. Cancer of the Endometrium.

The findings in 50 consecutive cases of cancer of the endometrium of 45 years and over, similarly studied, were as follows: diabetic curves 28%; mildly impaired curves 24% and normal curves 48%.

3. Patients with Benign Glandular Hyperplasia:

The definition and pathology of benign glandular hyperplasia are considered in detail, and the glucose tolerance studies in 50 consecutive such patients are presented. Photographs of each curve and photomicrographs of the endometrium in each case are presented, as well as the clinical picture. The findings were: diabetic curves 20%; mildly impaired curves 64% and normal curves 16%.

4. Patients with non-ovular cycles, and non-hyperplastic endometria.

Ten such patients were encountered and investigated. Only 1 of these had a normal glucose tolerance curve; 8 of the curves were of the mildly impaired type, and 1 was diabetic.

5. Other Research Studies:

Other studies included investigation of (a) glucose tolerance at various stages of the menstrual cycle (b) the effect of oestrogens on glucose tolerance (c) the effect of follicle

stimulating hormone on glucose tolerance (d) a follow-up study was made of cases with benign glandular hyperplasia which had been treated by total hysterectomy and bilateral salpingo-oophorectomy and the glucose tolerance tests were repeated.

DISCUSSION OF THE FINDINGS.

The findings are discussed in detail, and several interesting features come to light that have not been reported previously. In summary, these features are:-

1. In the random control group, the surprising finding was the high incidence of impaired glucose tolerance in older women, when compared with previous diabetic surveys of entire populations. Probable explanations for the discrepancies are given.

2. Comparison of the 2 age groups (35 - 45 years and over) demonstrated an abrupt and marked rise in the incidence of abnormal glucose tolerance curves after 45 years.

3. The investigation into cases of cancer of the endometrium showed that the increased incidence of impaired glucose tolerance in such cases (as compared with control groups) is indisputable.

4. The most striking findings were those seen in the cases of benign glandular hyperplasia. In this group it was found that as many as 84% of the curves were abnormal. Most of the aberrations in the curves were not gross - 22% were diabetic-type curves, and 62% showed less severely impaired glucose tolerance. This figure is considerably higher than that found in the control group (i.e. 84% compared with 22%), and is statistically most significant. The figure of 84% is also appreciably higher than that obtained even in cases of cancer of the endometrium (52%).

There is another reason why the findings in this group are most significant. The increased indidence of diabetes and mildly impaired glucose tolerance in older people has constantly been referred to in this thesis. Many of the patients with cancer of the endometrium were old women,

and this fact had to be taken into account. in that group. But in this series with benign glandular hyperplasia, the average age was low: 6 were under 35 years, 7 were between 35 and 44 years, 22 between 45 and 54 years, and 15 between 55 and 64 years (the oldest patient was 63 years of age).

5. Only one of 10 cases with non-ovular, non-hyperplastic endometria showed abnormal glucose tolerance.

Two important questions arise as an outcome of these findings:

1. What is the cause of the remarkably high incidence of abnormal glucose tolerance in cases of benign glandular hyperplasia?

2. Since a high incidence of abnormal glucose tolerance is a factor common to both benign glandular hyperplasia and cancer of the endometrium, are these two conditions related? And does this metabolic disturbance give a clue as to the cause of endometrial cancer and benign glandular hyperplasia?

These questions are discussed in detail in the thesis.

HYPOTHESES CONCERNING THE IMPAIRED GLUCOSE TOLERANCE IN BENIGN GLANDULAR HYPERPLASIA:

Having discovered that there was such a remarkably high incidence of impaired glucose tolerance in cases of benign glandular hyperplasia, an attempt was made to detect the reason for this association.

In conducting this enquiry several factors had to be considered. The hormones concerned in the development of benign glandular hyperplasia must come under discussion i.e. oestrogens, progesterone and follicle-stimulating hormone. The effect of these hormones on carbohydrate metabolism, and the effect of carbohydrate metabolism on the secretion of these hormones needs to be discussed. In addition, other hormones which are known to be concerned with carbohydrate metabolism and which may effect the pituitary-ovarian control of the endometrium, must be considered. These include

the growth hormone of the anterior pituitary, prolactin, the adrenal cortical hormones, and the thyroid hormone. And finally it is necessary to discuss the physiology of carbohydrate metabolism in the endometrium itself, the site where the main histo-pathology is evident.

All these factors are considered in detail, by a review of relevant literature, and personal investigations. This had lead to the following conclusions:-

1. The role played by oestrogens in the aberrations of carbohydrate metabolism is far from clear. Although there is evidence that glucose tolerance may be altered by oestrogens, it seems that in benign glandular hyperplasia it is not the altered oestrogens activity that causes the aberrations in the glucose tolerance curve - it is much more likely that benign glandular hyperplasia is, in a great proportion of cases, a manifestation of the disturbed metabolic state.

2. I have demonstrated clearly in this thesis the high incidence of impaired glucose tolerance in 3 conditions where progestational activity

of the endometrium is absent (viz. benign glandular hyperplasia, non hyperplastic non-ovular menstruation, and cancer of the endometrium). A careful search through the literature failed to disclose any studies on the effect of progesterone on carbohydrate metabolism. I wrote to Prof. Houssay of Buenos Aires and Prof. Marrian of Edinburgh, both of whom had made many studies on the effect of steroids on diabetes; they, too, had not investigated the effect of progesterone on carbohydrate metabolism. This is a subject worthy of investigation. I suggested to Dr. W.P.U. Jackson, who is in charge of the Diabetic clinic in the Groote Schuur Hospital, that he administer progesterone to diabetic patients to observe whether there is any amelioration in the diabetes. Because of my findings, he feels that this is a worth-while investigation, and will carry out such research.

3. Since there may be an excess F.S.H. secretion in cases of benign glandular hyperplasia, I considered it necessary to determine the effect of F.S.H. on glucose tolerance. Such an investigation

has not been done before. These isolated experiments appeared to show that the presence of excess F.S.H. in cases of benign glandular hyperplasia is not the cause of the impaired glucose tolerance found in such patients. It is much more likely that the disturbed glucose tolerance is the cause of, or an abnormality associated with, the excess F.S.H. or other endocrine inbalance which is responsible for benign glandular hyperplasia.

4. Evidence is submitted to show that the aberrations in the glucose tolerance curve, and the condition of benign glandular hyperplasia may both be due to disturbances in the anterior pituitary gland.

5. It is shown that it is most unlikely that the adrenal cortex can be implicated as the cause of metabolic disturbance and the endometrial picture in benign glandular hyperplasia. But assays of adrenocortical hormones in benign glandular hyperplasia have not been reported on, and should be made.

6. The thyroid gland cannot be implicated in the

abnormal glucose tolerance found in the cases of benign glandular hyperplasia that were studied.

7. Although changes in carbohydrate metabolism have been noted following the injection of prolactin, no reports have appeared in the literature regarding the relationship, if any, of this hormone to benign glandular hyperplasia. It is most unlikely that this hormone plays any part in the disease.

8. Profound activity in the way of carbohydrate metabolism takes place in the endometrium in the various stages of the menstrual cycle. Marked disturbances in this metabolism have also been demonstrated when there is endometrial hyperplasia or non-ovulation. It is understandable, then, that if the glucose tolerance of the body is disturbed, the carbohydrate metabolism in the endometrium could be upset. Whether aberrations in glucose tolerance can cause the histological changes in the endometrium seen in benign glandular hyperplasia and in endometrial carcinoma, is a very important question, and is indeed worthy of further investigation. It would seem to be a

distinct possibility in view of my finding the frequent presence of disturbed glucose tolerance in these 2 conditions.

SUMMARY.

In an attempt to detect the reason for the very high incidence of impaired glucose tolerance in cases of benign glandular hyperplasia, the literature was extensively studied, and several original investigations were done. The conclusion is reached that, in the present state of our knowledge, the association cannot be explained. Although there is much evidence that oestrogens, F.S.H., adrenal cortical hormones, thyroid hormone, and prolactin can influence carbohydrate metabolism, it is unlikely that they can be incriminated as the cause of the altered glucose tolerance curves in this condition. Evidence is adduced to show that the absence of progesterone, and a disturbed function of anterior pituitary gland may play an important role. The altered carbohydrate metabolism in the endometrium itself may be an important factor.

Lines of further research are indicated which may clarify this mystery. Such research may also throw

more light on the association of benign glandular hyperplasia and cancer of the endometrium, and indeed may help to detect the causes of the latter type of malignant development.

INVESTIGATION INTO THE AGES OF THE MENOPAUSE IN WOMEN IN THE WESTERN CAPE PROVINCE.

This separate investigation was carried out. It was considered necessary because: 1) the abrupt rise in the incidence of impaired glucose tolerance after the age of 45 years was proven earlier in this thesis; 2) this sudden rise being about the time of the climacteric makes it possible that the menopause and its associated change in ovarian and pituitary function may be a factor in causing impaired glucose tolerance; 3) since no such study had been made in South Africa it became necessary to determine what in fact was the menopausal age in the population from which the subjects in this investigation were drawn.

The following studies were made:-

1. The ages of the menopause in 1,000 women who served as controls.

2. The age of the menopause in 100 post-menopausal women attending the diabetic clinic of the Groote Schuur Hospital.

3. The ages of the menopause in 100 patients with carcinoma of the endometrium.

This investigation showed that in the population of the Western Province where this study was carried out, the average age of the menopause is 48.7 years, with a peak at 50 years. This is later than the age in other countries. Women who develop carcinoma of the body of the uterus in the postmenopausal era, tend to have a later menopaue than women in a control group; the same phenomenon was observed in a series of cases of diabetes mellitus, but this is not as marked as in the case of patients with endometrial cancer.

The rising incidence of impaired glucose tolerance does, therefore, correspond with the age of the menopause. The explanation of this, however, is far from clear. The role of oestrogens in carbohydrate metabolism has already been discussed, and the diminishing levels of oestrogen secretion after the menopause has been mentioned. If increased

oestrogen activity is a factor in the production of cancer of the endometrium, then the late menopause in this disease is understandable. However, if large doses of oestrogens improve diabetes mellitus, it is difficult to understand why the menopause tends to occur late in diabetics. The problem is far from being resolved, and many complex factors must be involved in the increased incidence of diabetes in women after the age of 45 years.

This study of glucose tolerance in patients with abnormal endometrial bleeding has added several facts to our knowledge of the subject. It has also brought to light some new data in related fields. By a repetition of previous investigations and a review of these, the controversy has been cleared up regarding the association of cancer of the endometrium and diabetes mellitus. The present investigation has led to the discovery of new facts about benign glandular hyperplasia, especially the very high incidence of abnormal glucose tolerance curves in control subjects before and after 45 years has

been shown, and the abrupt rise in the incidence of diminished tolerance at the climacteric has been clearly demonstrated. Similar detailed surveys have not been reported before. The findings indicate, inter alia, that progesterone may have an important effect on carbohydrate metabolism, a feature that has not been known - further research into this aspect is clearly necessary to fill this gap in our knowledge.

This and other studies may serve as a link in the chain of our understanding 1. the cause of benign glandular hyperplasia, 2. its association with cancer of the endometrium, and 3. the aetiology of this latter type of malignant disease.

BIBLIOGRAPHY.

BIBLIOGRAPHY.

Albanese, A.A., Higgone, R.A. Orto, L., Belmont, A. Dilallo, R. (1954): Metabolism, 3,154.

Althausen, T.L., Lockhart, J.C. and Soley, M.J. (1940): Amer, J.M. Sc., 199, 342.

Amatuzio, D.S., Stutzman, F.I., Vanderbilt, J.N. and Nesbitt, S. (1953): J.clin. Invest., 32,428.

Banting and Best (1921): Jour.Lab. and Clin. Med., 7,251.

Barnes, B.O., Revan, J.F. and Nelson W.O. (1933): Jour. Am. Med. Assn., 101, 926.

Barr, W. and Charteris, A.A. (1955): J. Obst.Gynaec.Brit. Emp., 62, 187.

Bastenie, P.A. Conard, V., Frankson, J.R.M., Kestens, J. and Novaks, L. (1953): Arch.int.Pharmacodyn., 93, 277.

Bastenie, P.A. Conard, V., and Frankson, J.R.M. (1954): Diabetes, 3, 205.

Beeler, C., Bryan, A., Cathcart, E.P. and Fitz, E. (1922): J. Meta. Res., 1, 549.

Blotner, H. (1945): Arch. intern. med., 75, 39.

Brewer, J. and Foley, P. (1953): Obstet. and Gynaec. 1, 67.

Brown, J.B., Kellar, R. and Matthew, G.D. (1959): J. Obst. Gynaec. Brit. Emp., 66, 177.

Cantilo, E. (1941): Endocrinology, 28, 20.

Chesro, E.J. and Bleyer, J.M. (1954): Geriatics, 9, 276.

Collens, W.S., Slo-Bodkin, S.G., Rosenbliett, S. and Boas, L.C. (1936): Jour. Am. Med. Assn. 106, 678.

Comesatti, G. (1907): Beitr.chem. Physiol. Path., 9, 67.

Conn, J.W. (1940): Amer. J. Med. Sci., 199, 555.

Cotes, P.M., Reide, E. and Young, F.G. (1949: Nature (London) 164, 209.

Debodo, R.C. and Altzuler, N. (1955): The Hypophyseal Growth Hormone, R.W. Smith, O.L.H.L. Goebler, and C.N.H. Long, eds., New York, Blakiston Division, Mcgraw-Hill Book Co.

Deren, M.D. (1937): J. Lab. and Clin. Med., 22, 1138.

Dische, S. Stamm, W.P. and Goudie, R.B. (1958): J. clin. Path., 11, 350.

Dolin, G., Joseph, S. and Gaunt, R. (1941): Endocrinology, 28, 840.

Duncan, G.G. (1952): Diseases of Metabolism, Philadelphia, W.B. Saunders Company.

Duncan, L.J.P. (1956): Quart. J. exp. Physiol., 41, 85, 453.

De Vigneaus, V. and Karr,W.G. (1925): J. biol. Chem. 66. 281.

Editorial (1952), Brit. med. J., 2,712.

Editorial (1959), Brit. med. J., p. 555.

Editorial (1959), Brit. med. J., p. 1340.

Engle, F.L. and Scott, J.L. (1950): J. clin. Invest. 29,151.

Evenson, O.K. (1942): Acta. med. scand. Suppl., 126,110.

Exton, W.G. and Rose, A.R. (1934): Amer. J. clin. Path. 4. 381.

Fajans, S.S. and Conn, J.W. (1954): Diabetes, 3, 296.

Fenton, P.F. (1945): Amer. J. Physiol., 144, 609.

Foa, P.P., Galansion, G., Weinstein, H.R. and Magill, A.M. (1954): Am. J. Physiol., 179, 636.

Foa, P.P., Galansino, G., Weinstein, H.R. and Magill, A.M. (1955): Am. J. Physiol., 180, 313.

Foglia, V.G., Penhos, J.C. and Cardoza, A.F. (1954): Compt. red. Soc. Biol., 148, 1656.

Foglia, V.G., Scuster, M. and Rodreguez, R.R. (1946): Endocrinology, 4, 428.

Franke, H. and Fuchs, J. (1955): Dtsch. med. Wschr., 81, 331.

Garnet, J.D. (1958): Amer. K. Obst. and Gynaec. 76, 11.

Gessler, C., Halstead, J.A. and Stetson, R.P. (1939): J. Clin. Invest., 18, 715.

Gilbert, J.A.L. and Dunlop, D.M. (1949): Brit. med. J., Jan. 4.

Gilbert, M. Schneider, H and Bock, J.C. (1926): J. biol. Chem. 67, 629.

Gitlow, S. and Kurschner, D.M. (1943): Arch. Int. Med. 72. 250.

Goldblatt, M.W. (1925): Biochem. J., 19, 948.

Goudie, R.B. Stamm, W.P. and Disch, S. (1958): J. clin. Path., 11, 428.

Greaves, J.D., Freiberg, I.K. and Johns, H.E. (1940): J. Biol. Chem. 133, 243.

Griffiths, M., Marks, H.P., and Young, F.C. (1941): Nature (London), 147, 359.

Hagedorn, Jensen, Krarup and Wodstrop (1936): Jour. Am.Med. Assn., 106, 177.

Hale-White, R. and Payne, W.W. (1926): Quart. J.Med.,19,393.

Harrison, H.C. and Long, C.N.H. (1940): Endocrinology, 26. 971.

Hering, V.V. and Evans, H.M. (1943): Am. J. Physiol., 140, 252.

Hertig, A.T., and Somers, S.C. (1949): Cancer, 2, 946.

Himsworth, H.P. (1932): Lancet, 2, 935.

Himsworth, H.P. (1933): Clin. Sci. 34, 1.

Himsworth, H.P. (1934): J. Physiol. (London), 81, 29.

Hinkle, L.E. and Wolf, S. (1952): Diabetes, 1, 383.

Hoest, J.P. and Lukens, F.D. (1954): Diabetes, 3, 1.

Hoet, J.J. Gommers, A. and Hoet, J.P. (1958) : 3rd Congress of International Diabetes Federation, Dusseldorf, July 24.

Houssay, B.A. (1942): Endocrinology, 30, 884.

Houssay, B.A. (1951): Brit. Med. J., 2, 505.

Houssay, Prof. (1959): Personal Communication.

Houssay, B.A. and Anderson, E. (1949): Endocrinology, 45, 627.

Houssay, B.A. Anderson, E., Bates, R.W. and Li, C.H. (1955): Endocrinology, 57, 55.

Houssay, B.A. and Biasotti, A. (1930): Arch. int. Pharmacodyn. Therap., 38,250.

Houssay, B.A. and Foglia, V.G. and Rodriguez, R.R. (1954): Acta. Endocrinol., 17, 146.

Hughes, E.C. (1950): Progress in Gynaecology Vol.2 Edited by Meigs J.V. and Sturgis, S.H., Heineman, London, p.88.

Hunter, R.A. and Greenberg, H.P., (1954): Lancet, 2, 58.

Hunter, R.A., Ludwig, K.V. Motley, J.F. and Oaks, W.M.(1954): Amer. J. Obst. Gynaec., 67, 121.

Ingle, D.J. (1941): Endocrinology, 29, 838.

Ingle, D.J. (1944): Endocrinology, 34, 361.

Ingle, D.J. (1954): J. clin. Endocr., 10, 1312.

Irving, E.M. and Wang, I. (1954): Glasgow. Med. J., 35, 275.

Israel, S.L. (1959): Diagnosis and Treatment of Menstrual Disorders and Sterility, 4th Ed. Hoever, New York, p.308.

Jackson, W.P.U. (1952): Brit. med. J., 2, 695.

Jackson, W.P.U. (1953): S.Afr. Med. J., 27, 795.

Jackson, W.P.U. and Woolf, N. (1957): Lancet, 1, 614.

Jackson, W.P.U. (1959): Postgrad. Med. J., 35, 293.

Jacobson, P.H. (1948): Milbank Mem. Fund Quart., 26, 90. Quoted by Vander, J.B. (1959).

Jones, H.O. and Brewerm J.I. (1941): Amer. J. Obst. Gynaec., 42, 207.

Joslin, E.P., Root, H.F., White, P.Marble, A., and Bailey C.C. (1948): The Treatment of Diabetes Mellitus, Lea and Febiger, Philadelphia, p.765.

Joslin, E.P. H.F., White, P. Marble, A. and Bailey, C.C. (1952): The Treatment of Diabetes Mellitus, 9th ed., Lea and Fibiger, Phidelphia, p.38.

Kajori, A.A., Crouter, G.Y. and Pemberton, R. (1925): J. Biol. Chem., 66, 89.

Krahl, M.E. (1955): The Hypophyseal Growth Hormone, R.W. Smith, O.H. Goebler, and C.N.H. Long, eds., New York, Blakiston Division, McGraw-Hill Book Co.

Kritzer, M.D. (1952): Med. clin. N. Amer., 36, 1151.

Langerhans (1869): Inaugural Dissertation, Berlin.

Laurence, R.D. and Buckley, O.B. (1927): Brit. J. exp. Path. 8, 58.

Laurence, R.D. (1947): Med. clin. N. Amer., 31, 289.

Laurence, R.D. and Madders, K. (1941): Lancet, 1, 601.

Lennox, W.G. (1927): Arch. intern. Med., 40, 128.

Leonards, J.R. and Free, A.H. (1945): J. Lab.clin. Med. 30, 1070.

Lewis, J.T., Foglia, V.G. and Rodriguez, R.R. (1949): Compt. rend. Soc. Biol. 143, 259.

Lewis, J.T., Foglia, V.G. and Rodriguez, R.R. (1950): Endocrinology, 46, 111.

Loughlin, W.C., Rosenthal, H.O. Halpern, R. (1943): J. Lab. clin. Med., 28, 1165.

Louw, J.T. (1958): Transactions 2nd. World Congress of Onst. and Gynaec. (F.I.G.O.-Montreal)

Loßner, E.L., Winkler, A.W., Taylor, F.H.L. and Peters, J.P. (1941): J. clin. Invest., 20, 507.

Lukens, F.D.W. (1946): Am. J. Med. Sci., 212, 229.

Lund, C.J. and Weese, W.H. (1953): Amer. J. Obst. Gynaec., 65, 815.

Marble, A. (1934): New England J. Med., 211, 339.

Marcus, H. and Glotzer, S. (1948): New York State J. Med., 48, 1461.

Marrian, Prof. G.F. (1959): Personal Communication.

Mazer, C.J. and Israel, S.L. (1937): Jour. Am. Med. Assn., 108, 163.

Mazolla, V.P., (1938): Amer. J. Obst. Gynaec., 36, 689.

McBride, J.M. (1955): J. Obst. Gynaec. Brit. Emp. 62, 574.

McCulloch, E.P., Beck, J.C. and Schaffenburg, C.A. (1952): Cleveland Clin. Quart., 19, 121.

McCullach, E.P., Beck, J.C. and Schaffenburg, C.A. (1955): Diabetes, 3, 13.

McCullagh, E.P. and Johnston, G.R.K. (1938): Amer. J. Med. Sci., 195, 773.

McLellan, W.S. and Wardlow, S.H. (1932): J. Clin. Invest., 11, 513.

Medical Women's Federation (1933): Lancet, 1, 106.

Merrivale, W.H.H. and Hunter, R.A. (1954): Lancet, 2, 939.

Milman, A.E. and Russel, J.A. (1950): Endocrinology, 47,114.

Mirsky, I.A. (1946): Prov. cent. Soc. clin. Res., 19, 74.

Morton, J.H. and McGavack, T.H. (1946): Ann. Int. Med. 25, 154.

Mosenthal, H.O. and Barry, E. (1946): Amer. J. dig.Dis., 13, 160.

Mosenthal, H.O. and Barry, E. (1950): Ann. intern. Med., 33, 1175.

Moss, W.T. (1947): Amer. J. Rosendgenol., 58, 203.

Moyer, J.H. and Womack, C.R. (1950): Amer. J. Med. Sci. 219, 161.

Nauyn (1906): Der Diabetes Mellitus, Wien, Alfred Holder, p.37.

Nelson, W.O. and Overholser, M.D. (1936): Endocrinology, 20, 473.

Nielson, O.J. (1928): Biochem. J., 22, 1490.

Noble, R.L. (1955): The Hormones, G. Pincus and K.V. Thimann, Vol.3, New York Academic Press, Ic.

Novak, E. and Martzloff, K.H. (1924): Amer. J. Obst. and Gynaec., 8, 385.

Novak, E. and Novak, E.R. (1958): Gynaecologic. and Obstric Pathology, 4th Ed. W.B. Saunders Coy. 163.

Novak, E. and Richardson, E.H. (1941): Amer. J. Obst. and Gynaec., 42, 564.

Novak, E. and Yui, E. (1936): Amer. J. Obst. and Gynaec. 32, 674.

Oakley, W.G. and Peel, J.H. (1949): Transactions of British Congress of Obstet. and Gynaec., p. 161.

Ogilvie, R.F. (1935): Quart, J. Med. 28, 244.

Opie, (1901): Bull, John Hopkins Hosp. 12, 263.

Orrahood, M.D. and Wyatt, J.P. (1953): J. Obst. Gynaec. Brit. emp. 60, 859.

Palmer, J.P., Reinhard, M.C., Sadugor, M.G., and Goltz,H.L. (1949): Amer. J. Obst. and Gynaec. 58, 457.

Pashkis, K.E. (1942): Am. J. Physiol., 136, 128.

Pashkis, K.E. Rakoff, A.E. and Cantarow, A. (1958): Clinical Endocrinology, Hoever, New York, 2nd Ed., p.433.

Purdie, A.W. (1945): J. Obst. Gynaec. Brit. Emp. 60, 859.

Pyke, D.A. (1959): Postgrad. Med. J., 35, 261.

Reed, J.A. (1955): Diabetes, 4, 377.

Reid, J., MacDougall, A.I. and Andrews, M.M. (1957): Brit. med. J., 2, 1071.

Renold, A.E., Ashmore, J. and Hastings, A.B. (1956): Vitamins and Hormones, 14, 140.

Rodriguez, R.R. (1950): Proc. Soc. Exper. Biol. and Med., 73, 317.

Rodriguez, R.R. (1951): Acta. Physiol. Latin., 1, 226.

Rodriguez, R.R. (1954): Brit. Med. J., 2, 505.

Ross, C.W. (1938): Arch. Dis. Childh., 13, 289.

Rukes, J.M., Orr, R.H. Forsham, P.H. and Galante, M. (1955): Ann. New York Acad. Sc., 61, 448.

Russell, J.A. and Wilhelmi, A.E. (1950): Endocrinology, 47, 26.

Scheffey, L. Thudium, W.J. and Farell, D.M. (1943):Amer J. Obst. and Gynaec. 46, 786.

Schneeberg, N.G. and Pineseone, I. (1952): J. Gerontol., 754.

Sletzer, H.S., Fajans, S.S. and Conn. J.W. (1956): Diabetes, 5, 437.

Silverstone, F.A. Brand, F.M., Shock, M.W. and Yiengst, M.J. (1957): J. clin.Invest., 36, 504.

Skipper, E. (1933): Quart, J. Med., 2, 353.

Smith, L.E. (1948): J. Gerontol., 3, 67.

Smith, L.E. and Shock, M.W. (1948): Am. J. Physiol., 155, 470.

Smith, L.E. and Shock, M.W. (1949): J. Gerontol., 4, 27.

Smith, G.V. (1941): New England J. Med., 225, 608.

Speert, H. (1949): Surg. Gynaec. Obst., 89, 551.

Spence, J.C. (1921): Quart, J. Med., 14, 314.

Spiegelman, A.R. (1940): Am. J. Med. Sci., 200, 228.

Spiegelman, A.R. and Marks, H.H. (1946): Am. J. Pub.Health, 36, 26.

Srinivasan. M. (1957): Lancet, 2, 317.

Ssobolew (1902): Virshow's Arch. f. Path. Anat. 168, 91.

Stadie, W.C. (1954): Physiol. Rev. 34, 52.

Strachan, G.I. (1936): J. Obst. Gynaec. Brit. Emp. 43, 749.

Sweeney, J.S. and Lackay, R.W. (1928): Arch. Intern. Med., 41, 257.

Thompson, R.H.S. and King, G.J. (1957): Biochemical Disorders in Human Disease, p.512.

Tolstoi, E. (1929): J. biol. Chem., 83, 747.

Turnbridge, E.R. and Allibone, E.C. (1940): Quart. J. Med., 9.11.

Turnbull, A.C. (1956): J. Obst. Gynaec. Brit. Emp., 63, 179.

Vander, J.B. (1959): Amer. J. Obst. and Gynaec. Feb., p.213.

Von Mering and Minkowski (1889): Arch. f. exp. Path. u. Pharm., 26, 371.

Walker, J.B. (1959): Postgrad. Med. J. 35, 302.

Way, S. (1954): J. Obst. Gynaec. Brit. Emp. 61, 46.

Wayburn, E. and Gray, H. (1942): Am. J. Med. Sci. 204, 823.

Weiss, T.E. Segaloff, A. Moore, C. (1957): Metabolism, 6. 103.

Wilkerson, H.L.C. and Krall, L.P. (1947): Jour. Am. Med. Assn. 135, 209.

Wilkerson, H.L.C., Kraal, L.P. and Butler, F.K. (1959): Jour. Am. Med. Assn. 169,910.

Wislocki, G. and Dempsey, E.W. (1945): Am. J. Anat. July,87.

Young, F.G. (1938): Biochem. J., 32, 513.

Young, F.G. (1941): Lancet, 1, 600.

Young, F.G. (1949): Acta. med. scandinav. 135, 275.

Young, F.G. (1951): J. clin. Endocrinol., 11, 531.

ACKNOWLEDGEMENTS.

ACKNOWLEDGEMENTS.

It is with much pleasure that I place on record my indebtedness to Professor James T. Louw, without whose encouragement this study would not have been made. It was at his instigation that this research was begun, for he visualised the importance of abnormalities of glucose tolerance in gynaecological disorders at the time of the climacteric. Not only did he arouse my interest in the subject, but he also placed at my disposal all the facilities necessary for carrying out this research and he gave me constant encouragement.

Professor J.E. Kench and his Department of Chemical Pathology at the University of Cape Town, carried out the overwhelming majority of the biochemical investigations. I am indeed grateful to him for his help in performing this massive amount of work, which was purely of a research nature, and would not have been necessary as part of his department's routine analyses.

Thanks are due to the Department of Pathology, University of Cape Town, for the hundreds of histological reports. The slides were all placed at my

disposal for examination and photomicrography. Dr. D. Uys was particularly helpful in this respect.

Dr. W.P.U. Jackson helped me to carry out a number of biochemical investigations. I am grateful to him for this, for helpful discussions in connection with glucose tolerance generally, and for advice on the statistical aspects of the results.

Mr. C. Constantine, of the Department of Gynæcology and Obstretics was of great help to me in the taking of the photomicrographs. Mr. B.Todt took the majority of the other photographs presented in this thesis. Mrs. Strydom assisted in collecting a large number of the blood and urine specimens, and helped to trace former patients for repeat tests. All this is gratefully acknowledged.

The investigation into the ages of the menopause of women in the Western Cape would not have been possible without the constant assistance of Sister T. Fox and the staff of the Gynæcological Out-Patient Department of the Groote Schuur Hospital. In this connection, again, Dr. Jackson kindly gave me permission to interview patients attending the Diabetic Clinic of the Hospital.

And finally, I wish to thank Dr. Burger, Superintendent of the Groote Schuur Hospital, Cape Town, for permission to carry out research on the patients. All the subjects investigated were cases from the Groote Schuur Hospital.