# EPIDEMIOLOGY AND MANAGEMENT OF DIABETES MELLITUS IN ZARIA URBAN AREA

BY

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A thesis submitted to the Postgraduate School, Ahmadu Bello University, in partial fulfillment of the requirements for the degree of Mater of Science

DEPARTMENT OF PHARMACOLOGY AND CLINICAL PHARMACY
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AHMADU BELLO UNIVERSITY, ZARIA

AUGUST, 1989

# DECLARATION

This is to certify that the work reported in this thesis was carried out by me in the Department of Pharmacology and Clinical Pharmacy, Faculty of Pharmaceutical Sciences, Ahmadu Bello University, under the supervision of Dr. A. A. Odutola and Dr. I. Abdu-Aguye. All sources of information are specifically acknowledged and referred to accordingly. I declare that no part of this thesis has been submitted elsewhere for a degree.



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# THESIS APPROVAL.

This Thesis entitled "Epidemiology and Management of Diabetes Mellitus in Zaria Urban Area" by Rabi Rafindadi Sanusi meets the regulations governing the award of the degree of MASTER OF SCIENCE (Pharmacology) of the Ahmadu Bello University, Zaria and is approved for its scholarly contribution to knowledge and literary presentation.

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# DEDICATION

To my father

Sanusi Muhammad Zayyan

and my husband

Muhammad Misbah Na'iya

## ACKNOWLEDGEMENT

I wish to express my sincere gratitude to Dr. A. A. Odutola and Dr. I. Abdu-Aguye, both of the Department of Pharmacology and Clinical Pharmacy, for making it possible for me to undertake this course in Pharmacology, for accepting to supervise the study, and for their pieces of advice, criticism and guidance throughout the period of this project. I also appreciate the useful advise and suggestions of Dr. T. Iyaniwura and Mr. I. S. Adagu. I appreciate as well Mrs Kwanashie's kind gesture for supplying some useful information.

I wish to acknowledge with thanks the contributions of staff of the Diabetic Clinic, medical wards, Medical Record Unit the Medical Library all in A.B.U.T.H., Zaria. The Chief Pharmacist and staff of the Quality Control and Industrial Pharmacy Department rendered valuable assistance. I wish to acknowledge Mr. Jimoh of Chemical Pathology Department, Mr. V. Amurawaiye of Immunology Unit, Miss M. T. Bakare of Pharmaceutical Chemistry Department, Mr. C. Ayewah and Mrs N. Uzonicha, both of Pharmacology Department, for their technical assistance.

I am particularly grateful to my husband for his love, patience, advice, moral support and prayers throughout the period of study. My appreciation also goes to my parents, brothers, sisters and relatives for their prayers, encouragement and moral support during the period of my studies.

I am grateful to the Health Services Management Board of Katsina state for granting me the fellowship to carry out the Master's Programme. The personal encouragement of the Chief Pharmacist of the Board, Alhaji M. Imam, the financial controller, A. Zayyad and the Hospital Secretary, H. Kurfi are gratefully acknowledged.

My appreciation goes to Dr. Abdu-Aguye's family particularly his wife, Aishatu, who handled the word processing on her *Joyce*.

Above all, I am most grateful to God Almighty for sparing my life and giving me the opportunity to conduct the study sucessfully and for His numerous Blessings on me.

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## GLOSSARY OF ABBREVIATIONS

ABUTH---Ahmadu Bello University Teaching Hospital

B.P ---British Pharmacopoiea

c ---Around (About)

°C ---Degree Centigrade

*et al*---and others

FDNB ---Fluorodinitrobenzene

Fig. ---Figure

GHb ---Glycosylated heamoglobin

GIT ---Gastro-intestinal Track

GM ---Gram(s)

h ---hour(s)

HCl ---Hydrochloric acid

L ---Liter

M ---Molar

Mg ---Milligram(s)

Min ---Minute(s)

Ml ---Milliliter(s)

MODY ---Maturity onset diabetes of the young

N ---Normal

NB ---Note well

nm ---nanometer

% ---Percentage

U.T.I---Urinary tract infection

U. V ---Ultraviolet

yr ---year(s)

ABSTRACT

Studies on the epidemiology end control of diabetes mellitus among the patients of ABUTH, Zaria were carried out. Patient understanding of the disease and compliance with treatment and other control measures were assessed through direct questioning, and by detecting the concentration of one of the oral hypoglycaemic agents (chlorpropamide) used by majority of the patients, in their blood. Samples of chlorpropamide tablets were obtained from some patients. The amount of active ingredients present were determined by standard assay techniques in order to determine the quality of drugs used by the patients.

The result6 of the study reveal that many patients lacked the understanding of their disease and the importance of proper management. This ignorance was attributed to the fact that many of the patien's had no formal education and also to the limited number of doctors at the clinic, a factor which limited the individual patient education. Appearance of some diseases and comnlications in some of the patients studied was

related to their ignorance and lack of patient compliance with treatment. It was observed that regular clinic attendance was variable. Those who were not regular were often readmitted with relapse of symptoms or complication of the disease.

The majority of the patients were not following a planned diet. This was because many of them were poor and so could not afford the ideal diabetic diet. This poverty and ignorance also limited the Individual control monitoring of diabetes by testing urine (and blood) for presence of sugar. For these reasons the patients were not able to adjust their diet and medication and so could not achieve good control.

The result of the tablet assay showed that 2 of the 5 brands of the tablets tested did not comply with official (B.P.) requirement. Failure to achieve good control in some of the patients studied could also therefore be attributed to consumption of substandard or faked drugs.

Serum chlorpropamide levels in 54 patients was detected using the method of Carmichael (1959) and the result shows that majority of the patients had levels within the therapeutic range. Interindividual differences as expected were

observed in the serum concentration of the drug in the patients used for the study. Differences in metabolising capacity and renal excretion were suggested to be responsible for this. Two patients had serum levels well in the toxic range while a few others achieved only subtherapeutic levels.

In conclusion, our data suggest that a significant number of diabetic patients in Zaria urban area lacked the understanding of their disease and this is related to their illiteracy, poverty and also to the inadequate number of medical personnel at the hospitals to educate the patients properly. The loss of control of diabetes in some patients was probably also due to non-compliance with treatment on the port of some patients and consumption of substandard drugs in addition to the above factors.

It is suggested that the Diabetic Clinic in Zaria needs the services of a dietician as well as more medical personnel. The Pharmacy Shop within the hospital should always stock antidiabetic drugs of adequate standards to reduce the risk of patients buying faked, substandard or expired drugs from other places. A booklet titled % Living with Diabetes' , and its Hausa translation, 'Rayuwa

da cutar sukar' , primarily directed at the diabetic has been prepared as part of this effort,

# CHAPTER ONE

## INTRODUCTION AND LITERATURE REVIEW

### 1.1. Introduction

In accounts of the history of diabetes, it is stated that in the Ebers papyrus (c. 1500 B.C) there was mention of polyuria (Papaspyros, 1964). The first good clinical description of the disease was made by Celsus, and the name 'diabetes mellitus' was introduced by Aretarus, another Roman Physician (both of who lived in the first century of the Christian era) (Best, 1960). The recognition of the central importance of surfiet of glucose in the urine, and, later, in the blood have led to a progressive expansion of the concept of diabetes mellitus to the point where it is now considered a common and widespread biochemical disorder of which the original descriptive, clinical entity occupies only a small part. The importance of the disease as a public health problem becomes even more apparent when one considers the complications of long-term diabetes. These complications, which are mainly vascular in nature, lead to marked disabilty due to

involvement of large vessels in the brain, heart, kidneys and the extremities, and, of small vessels especially in the eyes and kidneys. Diabetes, therefore poses a problem for many people and their families and more or less touches the practice of every physician regardless of his/her specialty. Moreso it provides the investigator (both in basic science and clinical study) an important field of research in which to work.

## 1.2 **Definition of Diabetes Mellitus:**

The term 'diabetes' comes from a Greek word meaning a siphon; it refers to polyuria characteristic of 2 pathological conditions namely diabetes insipidus in which the urine is dilute and relatively tasteless, and diabetes mellitus in which the urine has a sweet taste owing to its high glucose content. Diabetes mellitus therefore means 'passing out of a sweet substance through the body'. In medicine, diabetes mellitus is a term used for a group of chronic diseases in which the body fails to complete the conversion of certain foods into energy. (Colwell, 1978).

This clinical syndrome is characterized by hyperglycaemia due to deficiency or diminished

effectiveness of insulin. It affects the metabolism of carbohydrate, protein, fat, water and electrolytes, sometimes with grave consequencies. In a diabetic there is an overproduction of glucose and its under utilization and is primarily manifested as hyperglycaemia and glycosuria (Koda-Kimble, 1978)

The normal blood glucose level is between 60-120mg%. The heamostatic mechanisms of the body are aimed at maintaining a blood glucose level within this range at all times. Maintenance of blood glucose level is essential because some tissues such as the brain, rely solely on glucose for their metabolism. A rise in blood glucose produces osmotic diuresis and dehydration due to excessive micturation. This gradually leads to functional and structural changes in the body cells and those of the vascular system being particularly susceptible. The changes in turn lead to development of diabetic complications which most characteristically affect the eye, kidney and the nervous system.

## 1.3 Classification of Diabetes Mellitus

FitzGerald *et al* (1964) classified diabetes into the following:

A. Potential Diabetes :- Here a person has normal glucose response but has an increased potential risk of developing diabetes anytime in life. Examples of such include a person who has both parents as diabetics, or an identical twin, the other being a diabetic.

B. Latent Diabetes :- A person has normal glucose tolerance but is known to have glucose intolerance at some times such as during an infection, stress or pregnancy or when obese.

C. Asymptomatic Diabetes :- This is when a person has glucose intolerance but does not have any clinical obnormalities or symptoms.

D. Clinical Diabetes :- A person is said to have this when s/he presents with an abnormal glucose tolerance with symptoms and/or complications of diabetes.

The National Diabetes Data Group (1979) further classified the clinical diabetes into the following:

a. Idiopathic or primary diabetes.

b. Non-idiopathic or secondary diabetes.

c. Gestational diabetes.

1.3.1 Primary Diabetes:

This is further subdivided into the type I and type II diabetes.

The type I diabetes is also called the insulin-dependent diabetes (IDD). Patients with this type of diabetes are usually, but not always, young adults and children below the age of 35. They generally present with a serious and short onset of glycosuria (few days, weeks or few months). The symptoms are severe and the patient usually looks thin and emaciated sometimes with dehydration (Fajans *et al* 1976). The patients are either ketotic at diagnosis or are prone to it once insulin treatment is stopped. This hence defines their state of absolute insulin dependency. They have low insulin levels in plasma and there is little or no response to glucose challenge and are functionally characterized by insulinopenia (Genuth, 1973). Anatomically the islets are small and devoid of β-cells, though hyperplasia of other islet cells that produce glucagon, somastostatin and pancreatic polypeptide

was observed (Gepts *et al*, 1977). Patients with type I diabetes require prompt attention else they run into serious problems (Moran *et al*, 1984) and in some cases contribute to early death.

Type II diabetes is more common than type I. It is also referred to as non-insulin dependent diabetes (NIDD). It is usually, though not always, seen in patients who are above the age of thirty at the time of diagnosis. Many of the affected patients are usually obese. It is less abrupt in its onset and also less severe in its results. The patients with type II diabetes are usually not ketotic, although they may be so during an acute infection or surgical stress. Many of the affected persons are unaware of the disease and are only discovered during an unrelated illness, during the course of investigation for opthalmologic or gynaecological or cardiovascular complains. Patients with NIDD are hyperglycaemic because of a combination of insulin resistance and relative insulin deficiency. This resistance may be attributable to decreased number of plasma membrane insulin receptors and/or to post-receptor blocks in the insulin stimulated pathways of intracellular glucose metabolism. The latter may be induced by obesity as was reported by Archer *et*

*al*, 1975 and De Fronzo *et al*, (1978). However the work of Nagulesparam *et al* (1980) suggested that the resistance was independent of the obesity. The hyperglycaemia in the NIDD can well be controlled with diet alone, or with a combination of diet and oral hypoglycaemics. Insulin may be needed temporarily in such patients when medical or surgical stress causes acute hyperglycaemic decompensation or when obese patients are unable to control hyperglycaemia by other conservative measures. The type II, diabetes is usually a nonketosis-prone form.

Other rare form of primary diabetes include the maturity-onset type diabetes of young people (MODY). Tattersall and Fajans (1975) reported it and associated its symptoms more to the classical maturity-onset type diabetes of the middle age or elderly. They further reported that the stimulated-insulin release from the pancreas is retained though this may be delayed and diminished, and also that ketonuria and hyperglycaemia can be controlled without use of insulin. Additionally they suggested that both microvascular and macrovascular complications are rare and that an autosomal dominant mode of

transmission is most likely to underlie MODY (Tattersall and Fajans, 1975).

1.3.2 Secondary Diabetes :-

This defines a class of patients in whom hyperglycaemia is associated with another disease and may result from either or a combination of the following:

a. The destruction of the islet cells by surgical proceedure or pancreatic disease such as pancreatitis or carcinoma of pancreas. This gives rise to diabetes with symptoms very similar to those of type I diabetes.

b. Drug administration - for example the thiazide diuretics (Rapoport *et al*, 1964) which inhibit the secretion of insulin by the β cells, may induce diabetes similar in character to type I. The effect, is however reversible once the drug administration is stopped. The oral contraceptives and other steroids tend to precipitate the type II in character diabetes (Gharib, 1969).

c. Cushing's disease, acromegaly and glucagonomia produce diabetes that is type II

in character. In this case hormonally induced insulin antagonism is the prominent lesion Also in glucagonaemia the excess glucagon promotes conversion of glycogen (from liver and tissue) to glucose.

1.3.3. Gestational Diabetes :-

This, by definition, is a form of diabetes which appears during pregnancy and disappears or becomes subclinical when the pregnancy is over. Gestational diabetes may be attributable to the secretion of large amounts of human chorionic somatomammotropin (placental lactogen), during the 2nd and 3rd trimester. This substance is an insulin antagonist. Carbohydrate tolerance by the mother at this period is also reduced. Gestational diabetes is usually not very severe although it is associated with increased risk of congenital anomalies, high birth weights and other problems in the foetus. Therefore early detection and adequate treatment are important.

## 1.4. Aetiology of Diabetes Mellitus

The precise cause of diabetes mellitus is yet to be known. However, a number of contributing factors are known to be involved. They include the following:

### 1.4.1 Hereditary Cause :

The mode of inheritance of type I diabetes differs considerably from that of type II as was suggested by MacDonald (1974). He found an equal incidence of adult-onset type of diabetes among ancestors of juvenile diabetes and non-diabetics. Furthermore the study of Tattersal and Pyke (1972) suggested that genetic factors appear to be more predominant in the type II diabetes than type I. They found that after a long-term study of monozygote twins there was a 93% concordance rate for the adult onset diabetes (type II) and only 50% concordance rate for the type I, insulin dependent diabetes. These observations therefore suggest that the same pattern of inheritance cannot exist in all patients with diabetes, and that different genetic factors operate by and large in younger patients as opposed to older ones.

The work of Tattersall and Fajans (1975) provides further evidence of genetic heterogenicity in patients with diabetes. The results of their studies indicated that 85% of the cases with MODY had a diabetic parent, whereas only 11% of the cases with juvenile onset diabetes had a diabetic parent. Also their result showed that 46% of the families with MODY showed direct vertical transmission through three generations and all the affected individuals had non-insulin dependent type of diabetes, whereas only 6% of the families with IDD showed three generation inheritance.

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1.4.2 Viral infection:

There is evidence in man of an association between various viral infections and diabetes mellitus. The development of this disease shortly after mumps has been reported in case studies by Hinden (1962) and Kremer (1947). Insulin - dependent diabetes was reported in association with infectious hepatitis (Adl, 1974). Sultz *et al* (1975) also found an increased incidence of new diabetic cases some years after a community - wide out-break of mumps in New York. Menser *et al*

(1978) further reported on the clinical features of some patients with congenital rubella and diabetes mellitus.

The role of viral infections in the pathogenesis of diabetes mellitus was also reported by Yoon *et al* (1979). They recovered Coxsackie virus, group B type IV, from the pancreatic tissue of a child who died of meningoencephalitis and new-onset diabetes. This isolated virus was then injected into susceptible mice. The result was a β-cell destruction and diabetes.

1.4.3 Autoimmunity:

Many reports have suggested an association between IDDM and autoimmune diseases both of the endocrine and non-endocrine origins. Such diseases include hyperthyroidism (Perlman 1961), primary hypothyroidism (Ganz *et al* 1974) and pernicious anaemia (Munichoodapa *et al*, 1970). The presence of circulating pancreatic islet-cell antibodies in the plasma of patients with insulin dependent diabetes was reported by Bottazzo *et al* (1974) and MacCuish *et al* (1974). The latter suggested that due to the presence of these antibodies in the patients, insulin deficiency may be secondary to autoimmune disease of the pancreas.

## 1.5. Pathology of Diabetes Mellitus:

In a normal person, following the ingestion of food, blood glucose concentration rises thus stimulating the release of insulin. Insulin promotes the uptake of glucose, amino acids and fatty acids and their conversion to storage forms in the tissues. It also reduces the blood sugar level so that none is unneccessarily wasted in the urine.

In the normal fasting individual, low glucose level inhibits the release of insulin. In addition to this a number of other hormones eg growth hormone, glucagon and adrenaline are released to oppose the effect of insulin and provide an increase in the blood sugar level. Due to this insulin lack, several processes occur in the body which maintain and preserve a minimal blood glucose level for the brain to function. Glycogen in the liver is broken down to glucose by the process of glycogenolysis, amino acids are also converted to glucose by a process of gluconeogenesis. There will also be a diminished uptake of glucose by the insulin-dependent tissues to conserve as much as possible the glucose for the brain. Triglycerides are as well broken down

to free fatty acids which are subsequently used as fuel by the tissues and muscles.

In the diabetic patient, when high level of glucose occurs in the postprandial state, the absence of insulin or its action leads to unutilisation of the glucose by the muscles. As a result the level of blood sugar remains high and it is subsequently passed out along with the urine and taking along with it excess water by the process of osmosis, thus leading to polyuria and polydipsia. The lack of intracellular glucose is then interpreted by the body as starvation. As a result, fasting metabolism is initiated to provide for the cells some fuel to use. The free fatty acids are metabolised to ketone, proteins broken down to amino acids to provide substrate for gluconeogenesis and the liver glycogen broken down to glucose. All these processes result in the symptoms of muscle wasting, ketoacidosis and persistently high levels of blood glucose as is usually seen in the diabetic patient.

## 1.6. Signs and Symptoms of Diatetes Mellitus:-

The warning signs of diabetes mellitus are typical especially for the IDDM and are relatively easy to recognise. Some patients with the type II (NIDDM) may present with very mild signs that can even go unnoticed or sometimes ignored. Signs for type I often occur abruptly and include a dramatic and sudden increase in frequency of micturation and amount of the urine also increases. This is accompanied by excessive drinking of fluids. There is also sudden weight loss despite increase food intake. There may often be nausea, weakness, fatigue and irritability. The skin may be itchy, dry and often has sores or other skin infections which take a long time to heal.

The signs for type II diabetes often progress slowly and include extreme fatigue blurring of vision, numbness or tingling of the fingers or toes, drowsiness as well as itching and infection of the skin. In less severe diabetes there may be no signs at all.

## 1.7. Diagnosis of Diabetes Mellitus:

The decline of excess glucose in blood and urine after an injection of insulin or administration of tablets pointed to the key role that the measurement of glucose in blood plays in the diagnosis and treatment of diabetes. Once a patient presents with some of the signs and symptoms mentioned above, confirmatory laboratory tests are performed to establish a diagnosis of diabetes. There are 2 common tests done to monitor the blood sugar level. They are the urine and blood tests. The latter gives a more accurate amount of the blood sugar.

### 1.7.1 Urine Glucose Tests:

The presence of sugar in the urine (glycosuria) is in most but not all cases diagnostic of diabetes mellitus. A negative test for sugar in the urine however, does not eliminate diabetes. When glucose is found in the urine, a blood test is mandatory. There are 2 basic types of urine test currently in use. One is the glucose oxidase method in which testape, clinistix and/or ketodiatix are used. This method is specific for glucose although it has a high rate of

interpretation error (Leonards, 1957). Gifford *et al* (1961) also reported that vitamin C in therapeutic doses interferes with the test resulting in a false negative reading. Other drugs that intefere with the urine test and cause false positive results include the salicylates, sugars such as galactose, lactose and fructose in high doses, probenecid, streptomycin, methyldopa, levodopa, isoniazid and nalidixic acid, to mention a few. The other method of urine testing is the copper reduction method in which clinitest is used. This test is affected by any reducing agent in the urine. Though this is not specific for glucose in urine, it is more reliable in quantitating large amounts of sugar in the urine. Hence this is very useful in IDDM while the former is the test of choice for the NIDDM. In clinitest method the testing reagent is in the form of tablets. A patient using this method is required to observe the reaction while it is occuring since very high sugar content results in what is referred to as 'pass-through' phenomenon. This is demonstrated by a fleeting bright orange colour which occurs during the 'boiling and which fades to greenish brown once the reaction stops. This colour may be falsely interpreted as 0.75 - 1%,

when there is actually more than 2% glucose in the urine (Belmonte *et al* 1967). Patients should be taught how to test their own urine at home.

1.7.2. Blood Sugar Tests:

The diagnosis of diabetes mellitus demands that hyperglycaemia be demonstrated. The blood sugar criteria can be based on random blood sugar or fasting blood sugar or the Oral Glucose Tolerance Test (OGTT). Random blood glucose levels are more sensitive than fasting levels when seeking a diagnosis but elevated fasting levels are more specific.

Diagnosis of diabetes using fasting blood sugar (FBS) levels involves taking the blood sample in the morning before breakfast and high value is diagnostic of diabetes. (>6.5mmol/L).

The OGTT is a very reliable diagnostic test for diabetes (Ferrannini *et al*, 1985). It measures an individual's ability to handle a glucose load over a period of time. After an overnight fast, a morning fasting blood is drawn, and 100gm glucose load is given to the patient. Blood samples are then drawn at ½hr (30min) interval for 2hrs and then at 3hrs after the glucose ingestion. Urine

samples are taken simultaneously and tested for glucose in order to estimate the person's renal threshold for glucose. In normal person, blood glucose level returns to normal in about 2h or less. In the diabetic, the glucose level remains high and the peak occurs much later than in the normal, and, the level also declines at a slower rate as seen in Fig. 1.1 (Goodman and Gilman, 1980). Factors which affect the OGTT include some endocrine abnormalities, drugs, burns, malnutrition, infection, obesity, fever, neoplstic diseases, renal diseases, advanced age, stress, pancreatitis and pregnacy (Aguwa, 1986).

## 1.8. Management of Diabetes Mellitus:

The treatment objectives for all diabetics are essentially the same. They include making the patient well and keeping him/her so, as well as to avoid complications or at least postpone them. The plan of treatment is determined by a number of factors such as age, body weight, extent of the metabolic derrangement such as acidosis, fluid deficiency and electrolyte inbalance and the type of work the patient does (Drash, 1976). The aims of the treatment can be attained in part by

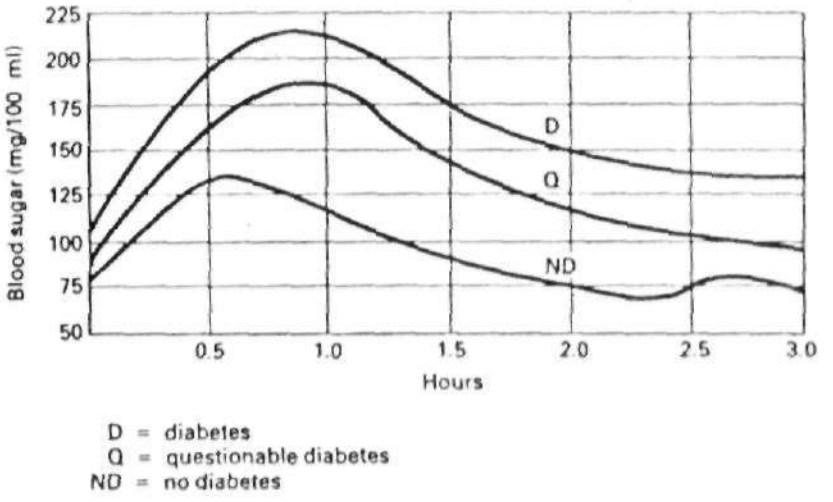



Figure 1-1 Interpretation of oral glucose tolerance test (OGTT)

adherence to a diet and sometimes by the administration of tablets or insulin. Educating patient and his/her relations about the disease and the importance of proper management is equally important.

1.8.1. Patient Education:

This begins when a patient is first seen by the physician and has been diagnosed as diabetic. Initially the patient is informed that she/he has diabetes and this is followed by information necessary for the immediate management. The importance of the patient's participation in the management is mentioned to the patient. After the patient has overcome the initial shock of the disease, an indepth education covering all aspects of the disease, its long-term management and complications if not properly controlled, will be mentioned. Thereafter every visit to the clinic by the patient should be a continuing educational experience and, the physicians should answer all immediate questions from the patient.

1.8.2. Diet:

To control diabetes mellitus, it is essential to follow a meal plan (Abramson *et al*, 1967; Bogardus *et al* 1984). The meal plan should be designed to suit the individual's needs and food preferences. The diet should be such that it is not monotonous and boring and should contain adequate amounts of vitamins, mineral salt, essential proteins, fats and carbohydrates.

A ban on certain food substances such as sweets, sugar, chocolates, jam, cakes, puddings and pastry should be encouraged by all diabetics. There is some evidence that diets which are high in fibre may reduce insulin requirement, postprandial fluctuations in blood glucose as well as in the amount of glycosuria (Jenkins *et al*, 1976; Kiehm *et al*, 1976 and Jenkins *et al* 1977). Dietary fibre has also been reported to reduce serum cholesterol by sequestrating bile acids and interefering with the enterohepatic circulation (Drug and Therapeutic Bulletin, 1979). Mcivor *et al* (1984) further concluded that high carbohydrate fibre therapy improves postprandial blood glucose acutely in patients with the NIDDM and that there is an additional improvement on basal carbohydrate tolerance From all these reports therefore, it is

of great advantage to the diabetic patient to eat foods which contain high fibre eg vegetables, wholemeal foods etc.

In the obese diabetic, low calorie and subsequent weight loss helps in lowering fasting blood sugar and also improves glucose tolerance. Davidson (1976) suggested 'short - term fasts' to initiate diet therapy in obese diabetics. He however further stated that such an approach should be initiated only in patients who:

a. are under constant medical supervision.
b. appreciate the severe complications as a result of starvation.
c. are capable of measuring urine ketones and seek medical advice when symptomatic (Davidson, 1977).

1.8.3. Insulin:

This is a hormone and a small protein molecule that is made up of 51 amino acids. It is synthesized in the pancreas by the β cells of the islets of Langerhans. The use of insulin in the mangement of diabetes mellitus dated back to 1922 when it was first administered to a 14 year old diabetic patient. (Sonksen, 1977).

The main action of insulin is to lower blood glucose level by enhancing the transport of blood glucose into the body cells (Felig *et al*, 1976,). In the muscles, the action of insulin leads to rapid uptake of glucose which is either converted to glycogen and lactic acid or oxidized to carbon dioxide and energy in form of heat. Insulin also enhances the protein synthesis in the muscles by increasing the movement of amino acids into the muscles and possibly other cells, and increasing the incorporation of the acids into protein. In man and other animals (mammals), the rate of synthesis of insulin was found to be highly dependent on glucose concentration (Parry and Taylor, 1966). Fasting was reported to lower the level of pancreatic insulin response. (Yalow *et al*, 1965). However refeeding was said to restore a normal response.

Indications for the use of insulin for the control of diabetes include acute illness, stress, infections, uncontrolled hyperglycaemia in the maturity onset type diabetes paticularly during stress, surgery or anaesthesia. Growth-onset diabetes (type I) is a classic indication for insulin.

Blood glucose standards for the insulin - requiring diabetic vary with age and degree of instability of the diabetes

1.8.3.1 Types of Insulin Preparations:

All forms of insulin can be conveniently classified into short or rapid acting type, intermediate acting and long acting. Most of the insulin sold in the pharmacy is obtained either as monospecies pork panacreas, beef pancreas or a mixture of the two (Galloway *et al*, 1978). The proportion of beef to pork in the mixed type does not affect its potency. The insulins are prepared usually in 3 concentrations - U-40, U-80 and U-100. The U-100 is more efficient in that it reduces error and confusion in adjusting the insulin dosages (Berne, *et al*, 1984). The properties of the various insulin preparation commonly used are tabulated below (see Table 1.1).

Table 1.1
Properties of Various Insulin Preparations.
From Goodman and Gilman (1980).

| Type of Insulin | Appearance | Protein Added | Approx time of onset (h) | Aprox time of acting (h) | Compatible/ mixed with |
|---|---|---|---|---|---|
| Fast acting | | | | | |
| Insulin Injection USP (Regular) | Clear | None | 1 | 6 | All preps |
| Crystalline zinc Insulin (CZI) | Clear | None | 1 | 8 | All preps |
| Semilente Insulin | Cloudy | None | 1 | 14 | Lente prep |
| Intermediate Acting | | | | | |
| Lente Insulin | Cloudy | None | 2 | 24 | Insulin Inj S/lente |
| NPH Isophane | Cloudy | Protamine | 2 | 24 | Insulin |
| Globin Zinc Insulin | Clear | Globin | 2 | 18 | - |
| Long Action | | | | | |
| Protamine Zinc Insulin | Cloudy | Protamine | 7 | 36 | Insulin Inj |
| Ultralente Insulin | - | - | 7 | 36 | Insulin Inj S/lente |

N. B.
Routes of administration
1. Regular : Intravenous, intramuscular, subcutaneous
2. CZI : " " "
3. Semilente: Subcutaneous
4. Lente : "
5. N. P. H. : "
6 All the long-acting ones are given subcutaneously.

1.8.3.2 Use of Insulin :

Insulin injection is used primarily in the insulin dependent diabetes. This type usually, though not always occurs in children and young adults. Other indications for the use of insulin have already been mentioned. Patients (particularly the IDD ones) are usually started with regular insulin which is an example of the short acting type. When glycosuria is well controlled, the patient is changed to an intermediate acting type of insulin. The 24-hour requirement for the regular insulin is determined and on the basis of this, the amount of the intermediate to be used calculated. About ⅔ to ¾ of the regular unsulin requirement is given using the intermediate. The insulin dose are usually given one before breakfast and the other before evening meal. In the Ahmadu Bello University Teaching Hospital, a diabetic patient is given 8 units of insulin for every positive (+) value obtained after a urine test.

1.8.3.3 Insulin Administration Technique:

There are various ways by which insulin is administered into the body. These include the use of special syringe and needle for the injection of the drug as well as use of insulin infusion pump (Tamborlane *et al*, 1979). Injection sites are usually rotated, in the case of those patients using syringe and needle. This is to avoid the atrophy of subcutaneous fat tissue at the site. Calibration of the syring used should correspond to the concentration of insulin used. Rolling of the insulin container before measuring out the drug is important for effective mixing especially of the cloudy insulin (Berne *et al*, 1984).

A variety of pumps are available for insulin delivery. Tambolane *et al* (1979) have developed and used a portable insulin pump over 24h in juvenile diabetics. Because the insulin is delivered into the peripheral instead of the portal circulation, the levels of circulating insulin were found to be higher than normal (Botz *et al*, 1976)

1.8.3.4 Side Effects of Insulin :

Insulin should always be administered with care as some of its adverse effects can be fatal.

The adverse effects of insulin therapy include allergy to insulin, lipoatrophy, hypoglycaemia, insulin resistance and refractive errors.

Insulin Allergy:- This can be local or systemic. The local allergies may occur within minutes after insulin injection. There may also be delayed local allergy which occur long after the injection. Systemic allergy to insulin and local allergy of the immediate type are mediated by IgE (Mattson *et al*, 1975; Bucholtz, 1976) while the delayed local allergy is IgG mediated (Galloway *et al*, 1978).

Atrophy :- of subcutaneuos fat tissue at the site of insulin therapy. Galloway *et al* (1978) reported that insulin local allergy and lipoatrophy occur in about 15% of cases. Rotation of injection sites helps in reducing it. Teuscher (1974) reported that injection of very highly purified monocomponent insulin directly into the affected area resulted in improvement. Kumar *et al* (1977) suggested a different method of treatment by use of steroids (dexamethasone) in form of injection directly into the areas.

Hypoglycaemia :- This is also referred to as insulin reaction or insulin shock, and it is one of the main problems with insulin therapy.

Initially the patient has sweating, tachycardia, blurred vision, feels hungry and becomes irritable. These signs may be due to adrenergic effects of the body trying to prevent the hypoglycaemia. In the severe form of hypoglycaemia the central nervous system effects begin to appear such as confusion, unconciousness or coma or even death. Hypoglycemia can be treated with sugar or sugary foods if patient can eat. If patient is unable to eat intravenous administration of glucose solution (in form of dextrose) is given. Glucagon may be injected in some cases.

Refractive Error :- due to insulin therapy occur in some patients. Such patients may complain of blurring of vision. This is due to change in shape of the lens (in the eyes) from fluid retension after cessation of osmotic diuresis in diabetes. It is usually temporary and will gradually subside. It is more common in patients that have been newly discovered of diabetes and are placed on insulin therapy.

1.8.4. Oral Hypoglycaemic Agents (OHGA):

The presently available oral hypoglycemic agents are the sulfonylurea agents and the

biguanides. The OHGA are useful in the management of the NIDD to which diet alone fails to control. The drugs are however contraindicated in severe diabetes mellitus regardless of age of patient or duration of the diabetes, pregnant women, during severe stress or infection, in patients who have had severe adverse reaction and patients with conditions such as renal failure or hepatic failure.

1.8.4.1 Biguanides :

Metformin is the only biguanide that is still clinically used in some parts of the world. It is used particularly in the obese diabetics, patients who are allergic to sulfonylurea agents and those who cannot be controlled by diet alone. Patients who are placed on this drug should have no predisposing factor for lactic acidosis. Metformin acts by increasing the peripheral utilisation of glucose, by decreasing absorption of glucose and inhibiting glyconeogenesis as well as enhancing anaerobic glycolysis (Nattrass *et al*, 1978). The main side effect of these drugs is lactic acidosis which is the result of the anaerobic glycolysis. This acid accumulates mainly in the liver and

muscle and, subsequently causes fatigue and inability of the muscles to function effectively. Accumulation of lactic acid leads to metabolic acidosis and coma, sometimes rapid in onset. Hence, although the risks of lactic acidosis can be effectively reduced by never prescribing any of these drugs to patients with chronic heart, lung, liver or kidney problems, phenformin can induce lactic acidosis even in diabetics who do not have any of these disorders, and, so should no longer be prescribed. (Gale and Tattersall, 1976).

1.8.4.2 Sulfonylureas :

The sulfonylurea drugs are reported to be useful in the management of type II (NIDD) in cases where the disease cannot be controlled with diet alone. (Kolterman *et al*, 1984). These drugs have the capacity to stimulate insulin release from the B cell (Michael *et al*, 1982). Hence they require the presence of a residual pancreas in order to lower the blood glucose effectively. It was also reported that treatment of diabetes with sulfonylurea agents caused a significant increase in insulin receptor sites in purified mouse hepatic cell membranes (Feinglos and Lebovitz, 1978).

Various other extrapancreatic effects of sulfonylureas have been reported. They include decreased hepatic insulin catabolism (Feldman *et al*, 1969), inhibition of insulinase enzymes (Mirsky *et al* 1956), reduction of intestinal glucose absorption (Teale and Love, 1972), inhibition of ketosis (Feldman *et al*, 1969). They have other effects which are not related to their hypoglycaemic action, for example their effects on water balance and inhibition of platelet aggregation.

When properly administered, the sulfonylureas are safe and need not lead to restrictions in working with machinery or driving a car. However they too are not without some side effects. These include gastrointestinal problems such as nausea, vomiting, and heartburn; rashes and other skin problems, and hypoglycemia. The latter may be due to administration of a large dose, failure to take adequate diet or, hypoglycaemia due to administration of other drugs together with the sulfonylurea eg aspirin. In a study conducted by Logie *et al* (1976), about 50% of the patients used for the study (709) were taking other drugs that have the potential to interact with their diabetic treatment.

The available sulfonylurea agents include tolbutamide, acetohexamide, tolazamide and chlorpropamide, all of which are the first generation drugs. The second generation sulfonylureas include glyburide, glipizide, glybornuride and gliclazide (diamicron®).

Tables 1.2 and 1.3 show the pharmacokinetics of the first and the second generation sulfonylurea agents.

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Table 1.2

Pharmacokinetics of the first generation sulfonylurea drugs. (Modified from Aguwa, 1986 and Larner, 1980).

| Drugs | Recommended Dose Range (gm) | Half-life (h) | Duration of Action (h) | Metabolism & Excretion |
|---|---|---|---|---|
| 1, Tolbutamide | 0,5 - 3 (divided dose) | 3,5 - 6 | 6 - 12 | Totally metabolised to inactive form, Inactive metabolites are excreted in kidney, |
| 2, Acetohexamide | 0,25 - 1,5 (single or divided dose) | 5,0 | 10 - 14 | Metabolite's activity equal to or greater than parent compound, Metabolite excreted via kidney, |
| 3, Tolazamide | 0,1 - 1,0 (single or divided dose) | 7,0 | 10 - 14 | Absorbed slowly, Metabolite active but less potent than parent drug, Excreted via the kidney, |
| 4, Chlorpropamide | 0,1 - 0,5 (single dose) | 35,0 | 24 - 72 | Previously thought not to be metabolised but recently found that metabolism may be quite extensive, Activity of these unknown, Significant percent excreted unchanged, |

Table 1.3
Pharmacokinetics of the second generation sulfonylurea drugs. (Modified from Aguwa, 1986 and Larner, 1980).

| Drugs | Recommended Dose Range (mg) | Half-life (h) | Duration of Action (h) | Metabolism & Excretion |
|---|---|---|---|---|
| 1, Glyburide | 1,25 - 30 (single or divided dose) | 10,0 | 24,0 | Metabolised to compounds (one of which may be weakly active). |
| 2, Glypizide | 2,5 - 4,0 (single or divided dose) | 4,0 | ( 24,0 | Metabolised to inactive compounds. |
| 3, Glibornuride | 12,5 - 75,0 (single or divided dose) | 8,0 | ( 24,0 | Metabolised to compounds which may be weakly active. |
| 4, Gliclazide | 80 - 320 | 12,0 | 24 | Metabolised to inactive compounds and excreted in the urine. |

1.8.4.2.1 CHLORPROPAMIDE : Review Of Literature.

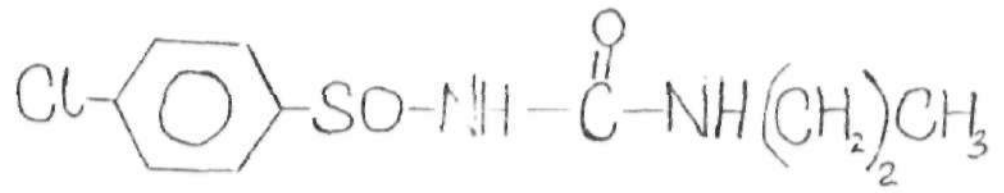
In 1942, two French investigators Janbon and Loubatieres studied the action of a group of thiadiazole derivatives in both animal and man, and in 1955 the sulfonylurea drugs were studied and used systemically in diabetic patients in Germany. Chlorpropamide one of the sulfonylurea drug, has been available since 1957.

Chemistry :

Chlorpropamide is a sulfonylurea drug whose structure contains a benzene ring, sulfonyl group and a urea.

The basic structure for the sulfonylurea agents is $R_1$-$SO_2$-NH-CO-NH-$R_2$, where $R_1$, is the aryl group and $R_2$ is the alkyl group. For the drug to have a hypoglycaemic action, $R_1$, and $R_2$, must have more than two carbon atoms. Changes in the alkyl group of $R_2$, or in an ion on the phenyl group leads to differences in metabolic disposition, blood level, therapeutic potency and toxicity of the various agents.

Chlorpropamide has the following formula and chemical name :



1-(4-chlorobenzenesulfonyl)-3-Propylurea.

Pharmacokinetics (Taylor, 1972 and Brotherton *et al*, 1969):-

Chlorpropamide is rapidly absorbed from the gastrointestinal tract. It appears in the blood within 1h after oral admimistration, attaining a maximum concentration from 7-10 days of daily doses. Duration of action of the drug may be as long as 60h.

Chlorpropamide is highly bound to plasma proteins. Hence there is competition for binding site with other drugs such as the oral anticoagulants and salicylates that are also highly bound to the plasma proteins. Chlorpropamide has a long biological half life (t½) of 35-40h. Only a small amount of the drug is metabolised in the body, and, this is extensively done by the liver. More than 99% of the drug is

thus excreted unchanged in the urine in single oral dose. The remaining drug is metabolised to p-chlorobenzenesulfonyl sulfonamide and p-chlorobenzene sulfonylurea which are both excreted in the urine (Brotherton *et al*, 1969) together with the unchanged drug.

Pharmacological and Mechanism of Action :

Chlorpropamide, like the other sulfonylurea agents, stimulates the islet tissue in the pancreas to secrete insulin. This stimulating effect is dependent on the functional state of the beta cell reserve. Hence the presence of a viable pancreas is necessary in order for chlorpropamide to lower the blood glucose level. Many extrapancreatic actions of chlorpropamide have been reported. They include inhibition of insulinase (Mirsky *et al*, 1956), decreased hepatic insulin catabolism (Feldman *et al* 1969), antidiuretic action (Miller *et al*, 1970). Miller *et al* further concluded that the mechanism of this action was potentiation of low levels of circulating antidiuretic hormone.

Uses of Chlorpropamide :

Chlorpropamide is a sulfonylurea drug that is orally active in lowering the blood sugar levels. It is used in the treatment of mild to moderately severe but uncomplicated maturity-onset type of diabetes mellitus, which may be unresponsive to diet alone. It is not suitable for patients with diabetic ketoacidoses or the juvenile onset type of diabetes. The drug is also useful in the management of diabetes insipidus (Miller *et al*, 1970).

The initial dose range for the management of diabetes mellitus is 250 - 500mg daily. This should be reduced to the maintenance level as soon as possible so as to avoid the hypoglycaemic attacks of the drug. The usual maintenance dose range is 100 - 375mg daily and should be taken with or before breakfast. The evening dose also, if taken twice daily, should be taken with meal or just before it. For the management of diabetes insipidus the dose range is 100-500mg daily.

Toxicity :

The rate of side effects with chlorpropamide has been estimated at 6% (Shen and Bressler, 1977). Significant side effects with

chlorpropamide include gastrointestinal - nausea, vomiting, heartburn, jaundice and abnormal liver function tests (Lebovitz *et al*, 1978). Skin reaction such as rashes and pruritis may also occur (Lebovitz *et al*, 1978). Other significant but rare side effect of the drug include its effect on the blood - red cell aplasia and bone marrow aplasia. Disulfiram-like reactions to alcohol and dilutional hyponatremia (due to its ADH-like effect) have also been reported (Lebovitz *et al*, 1978). Drug-induced hypoglycaemia can sometimes occur and it can be severe and prolonged, (Seltzer, 1972). It is often more common in the elderly and those patients with liver or kidney impairments.

Interaction with other Drugs

Chlorpropamide interacts with many drugs which will result in the potentiation or antagonism of the antidiabetic agent (Hansen *et al* 1977). These reactions are more commonly seen in the elderly who apart from the diabetes also have other common geriatric illnesses.

Antagonism of the hypoglycaemic activity of the drug is also a problem associated with hormone therapy including the use of oral contraceptives,

and glucocorticoids. Both of these types of hormones favor the hyperglycaemia by promoting peripheral insulin resistance. In addition, glucocorticoids generally oppose the metabolic effects of insulin by increasing the hepatic synthesis of glucose and decreasing peripheral glucose utilisation. There is the need therefore, to increase the dose of the antidiabetic drug when these drugs are administered concurrently.

Sulfaphenazole, a sulfonamide derivative displaces chlorpropamide from the plasma protein binding sites. This increases the concentration *of* free (active) drug and produces a more intense reaction that may result in hypoglycaernia.

Phenylbutazone is yet another drug that may enhance the hypoglycemic effect of chlorpropamide by increasing its half-life <tte). This drug appears to exert its effect by interfering with the renal excretion of chlorpropamide and its metabolites.

Aspirin and other salicylates when given in large doses for the treatment of inflammatory conditions can adversely effect the (hypoglycaemic) action of chlorpropamide.

Besides its inherent hypoglycaemic effect, alcohol interacts with chlorpropamide in some other ways. For instance, chronic consumption of alcohol by patients on chlorpropamide may diminish the hypoglycaemic response of the drug because of the induction of the hepatic microsomal enzymes responsible for the metabolism of the antidiabetic drug. Also, though not related to a change in blood glucose levels, there is another drug interaction between alcohol and chlorpropamide when the drug is taken at about the same time with alcohol or at a short interval. A large number of patients may experience a disulfiram-like effect consisting of headache, facial flushing, rapid heart rate and breathing,

1.9. **Complication of Diabetes Mellitus :**

Diabetic complications are numerous and occur frequently. The complications are usually caused by changes in the nerves (Engerman *et al*, 1977) and the blood vessels (Seperstein *et al*, 1977). The complications mostly involve the heart and the blood vessels, the kidneys, the eyes as well as the feet. It is however the belief of many workers that keeping the blood sugar under good control

can help delay or even prevent them (Engerman *et al*, 1977; Rossini, 1976). Diabetic complications usually take years to develop and patients with type II maturity onset type may be spared of them entirely.

Progressive damage to nerves called neuropathy, to the small vessels of the retina (retinopathy) and the blood vessels of the kidney (nephropathy) are common features of diabetes complications (Rundles, 1945, Howard-Williams *et al*, 1984; Deckrert *et al* 1981). The results of studies conducted by Gregerson in 1967 suggested that the prevalence and severity of diabetic neuropathy increased with increasing duration of the disease. Neuropathy may either be somatic or visceral type. The somatic type involves the sensory and motor function of the body. Sensory involvement produces signs such as anaesthesia, hypoesthesia or paresthesia, while the motor function abnormalities causes weakness of the lower limbs. Diabetes neuropathy has been reported to have occurred in patients with poor control of hyperglycaemia (Editorial, Lancet 1972).

Visceral neuropathy involves the autonomic nervous system. Sexual dysfunction in men may be the most common symptom related to autonomic

neuropathy. Urinary bladder function was also reported to be affected in impotent diabetic men (Ellenberg, 1971). This may be because of the fact that the penis, urinary bladder and the rectum innervation have a common source, with only the terminal nerve fibres being different.

Another important late complication of diabetes is the result of premature and severe hardening of the arteries usually referred to as atherosclerosis. The arteries especially affected are those supplying the heart (coronary) and those supplying the circulation to the lower limbs. In many cases the supply to the legs is adversely affected resulting in a condition that manifests itself by difficulty in walking, cramp pain on exertion, sometimes changes in the actual skin colour and poor wound healing.

## 1.10. Factors which may affect control of Diabetes :

The aims of controlling diabetes, as earlier mentioned, are to maintain good health and active productive life, as well as to delay or if possible prevent its complications. A number of factors have been reported to alter the control of diabetes. Such include body weight (Schmitz *et al* 1984), age, (Moran *et al* 1984) infections - acute infections are probably the most common single cause of deterioration in diabetic control - other concurrent diseases such as thyrotoxicosis and Cushing's syndrome, stress or surgery, pregnancy and even administration of other drugs. A good diabetic control is very important during pregnancy in order to avoid fetal abnormalities or even loss of the baby (Norbet *et al*, 1985, Rossini, 1976)

Exercise may also alter control of diabetes considerably. Diet and administration of proper doses of drugs also affect control of diabetes. This is particularly important to diabetic children who may omit their meals or drugs or fake urine tests sometimes just out of curiosity.

1.11 **Monitoring Management of Diabetes Mellitus:-**

There are various ways by which control management of diabetes mellitus can be monitored. Some of the parameters usually assessed clinically include the following:

a. Physical Improvement:

This includes amelioration of symptoms and weight control.

i. Amelioration of symptoms:- Patients are asked of any outstanding problems (or reappearance of symptoms of diabetes) during the clinic session. A patient who is physically fine and with no complains may be said to be in good physical control of disease. Patients are expected to be examined physically at each clinic visit. Blood pressure measurement, examination of the eyes, feet and chest as well as the skin are some of the parameters assessed by the clinician at clinic visits.

ii. Weight :- Regular weighing by patients either at home or in the clinic is important in assessing control of diabetes particularly with the obese

patients. A non-insulin dependent diabetic who is able to maintain an ideal body weight may be said to be in good control of the disease, while a young non-obese adult (on insulin) is expected too to maintain fairly constant weight. A growing child whose growth characteristics are within 95 percentile is doing well.

b. Laboratory Studies:

i. Urine and Blood Sugar tests:

These have already been discussed in pages 16-18. The urine sugar tests give an estimate of the blood sugar level, whereas the blood sugar test indicates the actual sugar level of the blood at the time of the test. It is important for every diabetic patient to perform his urine sugar test at least once daily so as to enable him to assess the control of his disease. High urine sugar indicates poor control of diabetes. Blood and urine sugar tests may be performed at home or in the clinic.

ii. Cholesterol Level:

Hyperlipidaemia may be a feature of established unregulated diabetes mellitus. Danowski *et al* (1971) reported that the serum

triglycerides are higher than normal even in the mild glucose intorance of the so called chemical diabetes type, provided the patient is in later years of life or obese. Therefore regular measurement of cholesterol level is important in the assessment of control of diabetes.

iii. Platelet function:

This is also assessed in diabetic patients. Abnormalities of platelet adhesiveness and platelet aggregation have been described in diabetes (Sagel *et al*, 1976). It has been shown that platelet stickiness is enhanced and that blood glucose concentration may be related to this phenomenon according to Colwell *et al*, (1978). So increased platelet stickiness serves as an index of loss of control.

iv. Glycosylated Hemoglobin (GHb):

Bunn *et al* (1976) reported that circulating glucose reacts with red cell hemoglobin to form glycosylated hemoglobins ($HbA_1$ a, b, c) and that the process is slow, non-enzymatic, irreversible and continues throughout the 120 days life-span of the red cells. Hence the levels of glycosylated hemoglobins reflect the time-average glucose level

seen within the erythrocyte during its life-span. So a single measurement of GHb levels gives an integrated measure of the glucose levels seen by the tissue (erythrocyte) over the preceeding several months. The study conducted by Gabbay *et al*, (1977) suggested that glycosylated hemoglobin measurement may be a valuable parameter for the evaluation of the adequacy of diabetic blood glucose control in the usual outpatient clinic setting. Their work also showed that plasma cholesterol levels correlates very well with the levels of glycosylate hemoglobin, hence indicating that hyperglycaemia is associated with and may contribute to hypercholesterolemia.

GHb estimation has been used mainly to monitor metabolic control in established diabetics (Gonen *et al*, 1977). The former suggested that measurement of GHbA, may serve as a screening test to identify those diabetics who are in poor control.

The GHb measurement has many advantages which include the use of small blood sample; glucose loading not necessary; test can be done at anytime of the day. There is also no need for confirmatory repetition of the test. However some of the setbacks of the method include instability of

samples on storage and various assay methods are used, so the absolute values of normal and abnormal ranges differ considerably and hence connot be directly compared.

v. Fructosamine levels in serum:

Glycosylated albumin (fructosamine) measurement is also becoming important in the monitoring of diabetic control. Its advantage over GHb measurement is principally related to the fact that it is a measure of glycaemic control within the preceeding few days, is relatively easier to measure and it does not suffer from storage problems outlined above for glycosylated haemoglobin (

c. Drug Level Monitoring :

This is an important parameter in assessing the control of diabetes. The pharmacokinetic parameters as well as the concentration of the drug in the patient's body can be assessed. Drug level monitoring helps in determining the extent of patient compliance with the management of his disease. Also, any abnormality in absorption distribution and excretion of the drug can be assessed and dosing may be altered where necessary.

OBJECTIVE OF THE STUDY.

Data from the medical record unit of the ABUTH, Zaria show an average of 1,926 diabetes mellitus patients very year for the past decade. The data further show an average of 70 new patients yearly from 1978 to 1988 in Zaria. (Table 1.4). Diabetes mellitus is at present a major contributor (world-wide) to other problems such as cardiovascular diseases, failure of the kidneys to function normally, blindness, pancreatic insufficiency and even death.

The incidence of this disease in Zaria was found to be 2.67% within the last decade (Table 1.4). This may not be unrelated to some enviromental factors such as stress, life-style such as drug and alcohol consumption, type of diet we eat which is mainly carbohydrate, genetic and certain disease states, for example hypertension, liver and pancreatic diseases and obesity to mention just a few. (West, 1972; Kempner *et al*, 1975).

Even though there is no known cure for the disease at present, proper control of the blood sugar level within physiological range helps considerably to reduce or even prevent the

complications of diabetes mellitus. The key to successful management of diabetes is continuing education of patient and also the relatives when the victim is geriatic or a small child.

Presently, diabetes is a life-long disease but with relentless efforts of the scientific researchers and the steady advances in knowledge of diagnosis and treatment, the altimate hope is that eventually the prevention or even cure of diabetes becomes a reality in the years to come.

The aims of this study are :

1. To find out how much the diabetic patient attending the ABUTH, Zaria knows about the disease, its management and complications.
2. To find out whether the patient actually complies with treatment and other control measures through direct questioning and by detecting the concentration of the drug in the blood.
3. To find out whether a sample of the drugs the patient uses is actually genuine or faked.
4. On the basis of the data gathered, to design an information booklet which shall provide the essential information about diabetes and its management for use of patients.

Table 1.4

Diabetic Data from the Medical Record Section of the ABUTH, Zaria (From 1978-1988

| YEARS | DIABETICS | | | ALL HOSPITAL PATIENTS | PERCENTAGE DIABETICS |
|---|---|---|---|---|---|
| | NEW | OLD | TOTAL | | |
| 1978 | 59 | 1540 | 1599 | 165904 | 0.967 |
| 1979 | 86 | 2183 | 2269 | 157605 | 1.437 |
| 1980 | 84 | 2132 | 2216 | 162643 | 1.362 |
| 1981 | 66 | 1199 | 1265 | 148429 | 0.852 |
| 1982 | 118 | 1874 | 1992 | 108955 | 1.828 |
| 1983 | 75 | 2045 | 2120 | 96437 | 2.198 |
| 1984 | 64 | 2505 | 2569 | 78727 | 3.263 |
| 1985 | 36 | 1358 | 1394 | 48473 | 2.876 |
| 1986 | 86 | 2094 | 2180 | 43515 | 5.010 |
| 1987 | 63 | 1688 | 1751 | 35141 | 4.983 |
| 1988 | 27 | 1808 | 1835 | 39799 | 4.611 |

N.B

--The data include both in- and out-patients.

--Average New cases per year = 69.5

--Average total diabetic cases seen in a year = 1,926.

--Clinic Session = 50 patients.

--Average attendance per week = 45.

--Mean per cent of diabetic cases per year = 2.672

# CHAPTER TWO

## MATERIALS AND METHODS.

### 2.0 Materials

### 2.1. Drugs

Chlorpropamide B.P. 250mg tablets. samples from five different brands of chlorpropamide tablets with the sources and batch numbers were collected from the patients directly during the clinic sessions (Table 2.1).

### 2.2. Equipments:

Analytical balance, type H51, AR Mettler Gallenkamp.

Flask shaker, Gallenkamp, England.

SP8--100 ultravoilet spectrophotometer (PYE UNICAM Ltd) York Street, Cambridge, England, CB1,2PX.

Thermometer, type E-mil®, Corning Limited, Science Products U.K. Stone, staffordshire ST15 OBG, England.

5-ml syringes and needles, Becton Dickinson and Co. Ltd, Ireland.

Centrifuge, Gallenkamp, England.

Hot plate, Shermono, Sussex, England.

2.3. **Reagents :**

Glycerin , May and Baker Chemical Laboratory, M&B Ltd, Dagenham, England.

Hydrochloric Acid, Analar grade, BDH Chemicals Ltd, Poole England.

Isoamyl Acetate, British Drug Houses Ltd, BDH Laboratory Chemicals Division, Poole, England.

Methanol, Analar grade, BDH Chemicals Ltd, Poole, England.

Methylated Spirit, May Baker Chemical Laboratory, M&B Ltd, Dagenham, England

1-fluoro-2, 4, --dinitrobenzene (FDNB Reagent), BDH Chemicals Ltd, Poole, England.

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ZARIA, NIGERIA.

Table 2.1.

Chlorpropamide Tablets Used in the Study.

| COMPOUND | MANUFACTURER | Lot/BATCH No | EXPIRY DATE | DESIGNATION |
|---|---|---|---|---|
| Diabenese | Pfizer Products Nigeria Ltd, Ikeja, Lagos, NIGERIA. | 724/7002 | May, 1990 | $C_1$ |
| Chlorpro-pamide | Neolab Industrial Pharmaceutical Centre, Denmark | F0405 | June, 1990 | $C_2$ |
| Diabenese | Pfizer Ltd, Thane-Belapur Road, Thane, 400613. | 820/17220 | August, 1991 | $C_3$ |
| Chlorpro-pamide | M.A. Steinford Ltd, London N.W.10.7UW Sigma England. | 70178 | October, 1989. | $C_4$ |
| Diabenese | Pfizer Products Nigeria Ltd, Ikeja, Lagos, NIGERIA. | 82417002 | September 1992 | $C_5$ |

## 2.4 Questionnaire

A questionnaire was prepared in English and consists of the following sections:

A--General questions (Demographic)

B--Questions about the disease.

C--Questions about medication, compliance with treatment and complications if any.

D--Questions about routine tests performed for the control of Diabetes mellitus.

E--Questions about diet and compliance with same.

F--Other questions related to management and control of Diabetes mellitus.

(See Appendix I).

The questionnaires were given to 120 diabetic out-patients who attend the diabetic clinic of the Ahmadu Bello University Teaching Hospital, Zaria. However 20 of these later dropped out from the study because they did not contain adequate information. Therefore 100 questionnaires were eventually used for the study.

40 questionnaires were also given to the in-patients of the medical wards of the same hospital. However only 30 were found to be useful for the study.

Majority of the patients (Both in the clinic and in the wards) did not understand English. Therefore the questions were interpreted to them in the Hausa Language.

## 2.5. Methods:

### 2.5.1 Chemical Assay of chlorpropamide:

The spectrophotometric method of assay in the British Pharmacopoea, 1980, was used as described below.

Twenty chorpropamide tablets of each sample, were accurately weighed and the average weight determined. These were then crushed and powdered using a motar and pestle. A quantity of the powder equivalent to 0.250gm (250mg) of chorpropamide was weighed. This was then added to 40ml of methanol in a flask with cover and was then attached to a flask-shaker and shaken for 20 minutes. Sufficient methanol was added to produce 50ml. The solution was then thoroughly mixed and filtered. 5ml of the filterate was diluted to 100ml with 0.1M HCl. 10ml of the resulting solution was further diluted to 250ml with 0.1M HCl. The absorbance of the final solution was measured at the maximum at about

232nm. The percentage content of chlorpropamide was calculated taking 598 as the value of A (1%, 1cm) at the maximum at about 232nm; formula:

$$\frac{\text{Absorbance}}{\text{Weight of the drug in 1gm} \times A} \times \text{Dilution factor}$$

$$A\ (1\%,\ 1cm) = A/cd.$$

Where A(1%, 1cm) is the specific absorbance which is also the absorbance of the 1cm layer of 1% w/v solution (1gm in 100ml) of the absorbing solute. C is the concentration of the absorbing solute expressed as 1% w/v, and d is the thickness of the absorbing layer in centimeter.

### 2.5.2. Determination of Serum Chlorpropamide Level

Colorimetic determination of chorpropamide in serum was carried out using the method of Carmichael (1959). 5ml of blood was taken from the antecubital vein of diabetic patient confirmed to be taking his/her medicine, into non-heparinised tube in the clinic. The blood was allowed to clot and serum was separated and de p frozen until analysed. Serum used for the calibration curve was pooled from 10 healthy volunteers (five males and

five females) and also the sera were deep frozen. Fifty four diabetic patients who were on chorpropamide treatmant alone, were used for the study. All samples were assayed within 20 days of collection. Chorpropamide has been shown to be stable in serum at - 20°C for at least 21 days. (Monro, 1974).

A stock solution of the FDNB reagent was prepared using isoamyl acetate and the pure FDNB in the ratio of 1 FDNB : 50 isoamyl acetate. This was stored in a dark coloured bottle and kept in the fridge. To prepare the working reagent of 0.1%, the above stock was diluted in the ratio 1:20 with isoamyl acetate.

0.5ml serum was transferred into a 15ml screw cap tube. 2.5ml of 0.02N HCl was added to the tube followed by the addition of 10ml isoamyl acetate. The tube was tightly stoppered and mixed by inversion for 10mins. The mixture was then centrifuged to separate the phases. 6ml of the top organic layer was transferred to another 12ml graduated centrifuge tube. To this, 1ml of 0.1% FDNB reagent was added and mixed well by inversion. A marble was placed on the tube and it was heated in glycerin bath at 125°C for 10mins. The tube was then removed and allowed to stand at

room temperature for 30mins. After cooling, isoamyl acetate equivalent to that lost by evaporation during heating was added to the tube. The resulting yellowish colour was then measured at 346nm, using visible region of the spectrophotometer. A serum blank treated in identical manner was used to adjust the zero setting of the instrument. The content of chlorpropamide per litre in the serum sample was then determined using the curve obtained from the serum standards.

2.5.3 Calibration Curve:

Standards of chlorpropamide were prepared in pooled serum. A standard chlorpropamide solution of 1mg/ml concentration was prepared by dissolving 10mg of chlorpropamide powder in 10ml methanol in a 10ml volumetric flask. 0.2, 0.5, 1.0, 1.5, 2.0, 2.5 and 3.0 ml of the standard solution were transferred into series of 10ml volumetric flasks. Enough serum was added to the flasks to make up to volume. Serum solution of various concentrations ranging from 2-30mg/100ml (or 20-300mg/L) were obtained. These were treated as mentioned in the assay. The ensuing yellowish colour was measured at 346nm against a serum blank that was treated in

a similar manner. The absorbances obtained were graphically plotted against the various concentrations.

2.6. **Data Presentation and Analysis:**

The results of the various studies were carefully recorded. Tables and graphical representations were drawn where appropriate. The results were statistically analysed where applicable.

# CHAPTER THREE

## RESULTS

### 3.1. Questionnaire Studies :

From the results of the questionnaires used for the study, it was observed that several of the patients lacked the understanding of their disease and the importance of proper control by use of drugs (injection or tablets), planned diet and the tests performed for the control of the blood sugar.

#### 3.1.1 Outpatients a. Age

The result indicated that many of the out-patients (46%) were of middle age, that is between the ages 41-50 years. 20% of the total patients interviewed were aged between 51 to 60yrs and 8% were above 60yrs. Only 20% of the population were between 10-20yrs, while 9% fell between 21-30yrs, and the rest of the patients (15%) between 31 - 40yrs. (Figure 3.1). 90% of the population interviewed were between the age of 21-60yrs.

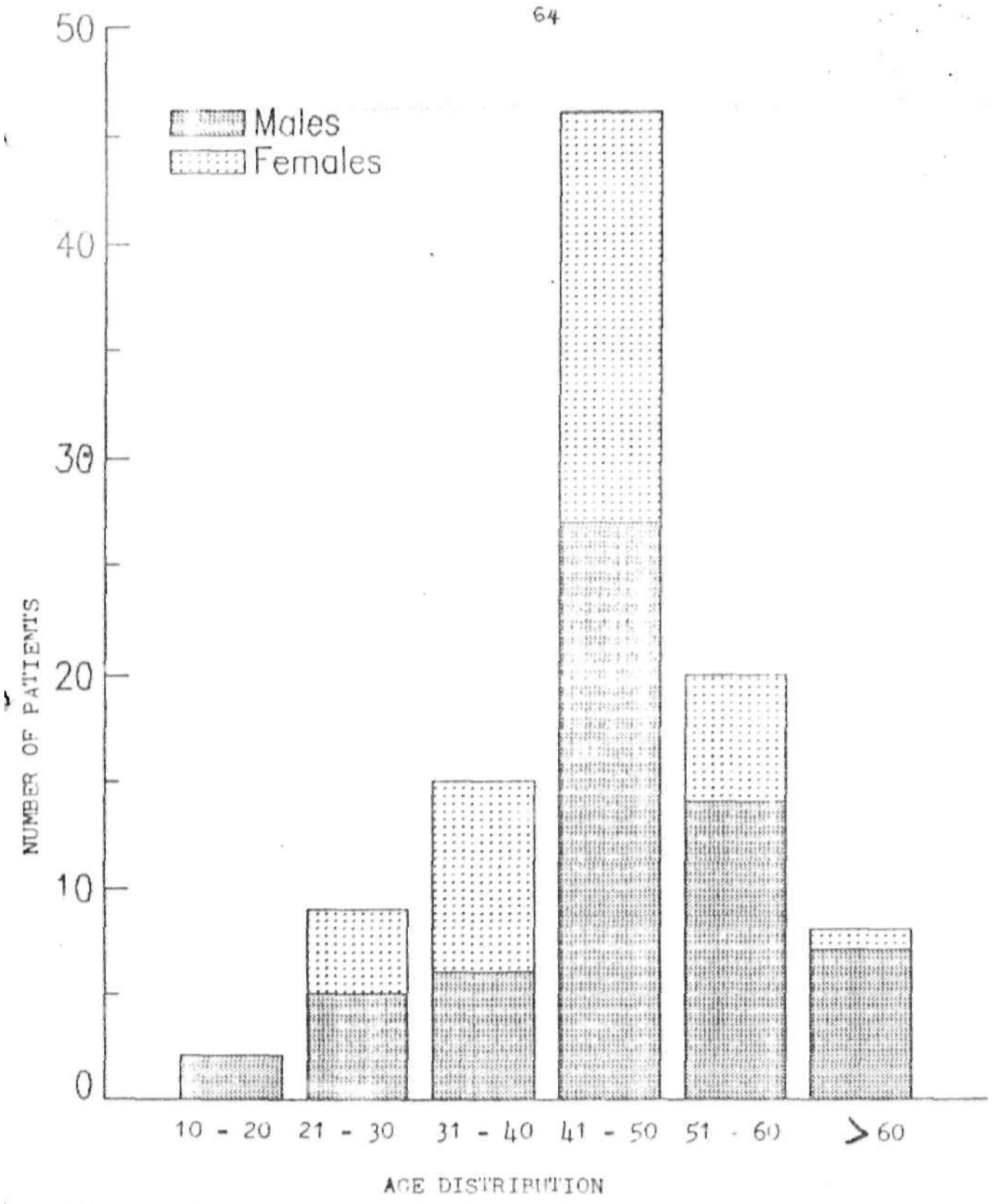



**Figure 3.1**

Age and Sex Distribution of 100 Diabetic Out-Patients of ABUTH, Zaria.

b. Education:

61% of the out-patients interviewed had not acquired formal western education. However about half of these (30%) understood the Arabic language which they had learnt in the Quranic schools. Hence 31% of the patients were total illiterates. Only 19% of the population had attended primary/adult education schools, while 20% had secondary to university education. (Figure 3.2)

c. Occupation:

The majority of the diabetic women interviewed were housewives whereas majority of the men were farmers or civil servants. A significant number of the men and women (23%) were engaged in petty trading, and the remaining patients (15%) were retired officers from both military and civil service, drivers, student and widows (Table 3.1)

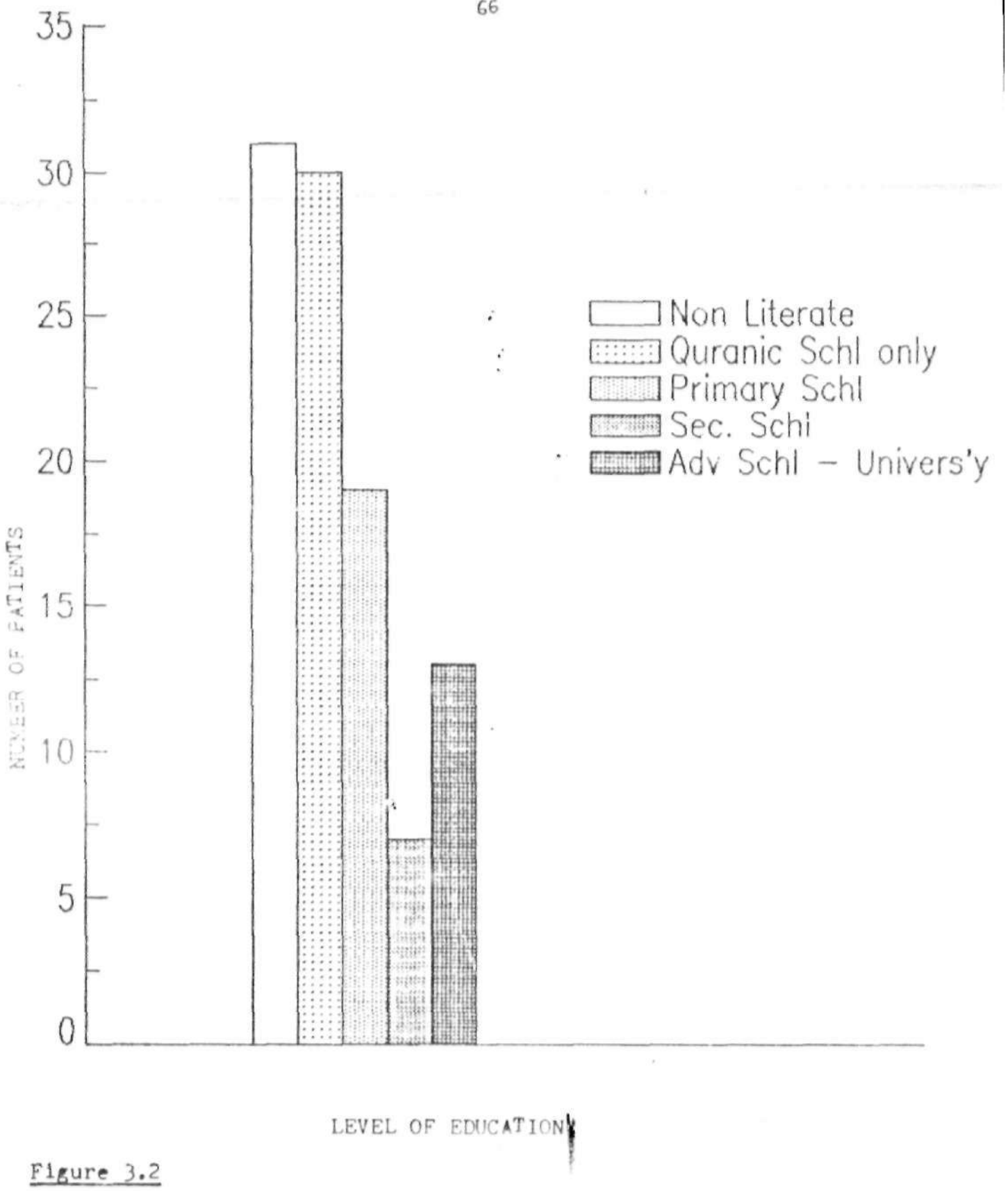



Figure 3.2

Level of Education Among 100 Diabetic Out-Patients of the ABUTH, Zaria.

Table 3.1

Occupation of 100 Diabetic Outpatients of the ABUTH, Zaria

| Occupation | Number | Percentage |
|---|---|---|
| Farming | 18 | 18 |
| Petty Trading | 23 | 23 |
| Housewife | 29 | 29 |
| Civil Servant | 15 | 15 |
| Others | 15 | 15 |
| Total | 100 | 100 |

d. Duration of Illness:

More than half of the patients interviewed had had the disease for more than 5 years. About 4% of the population had no idea of the onset of their disease. 7% had discovered the disease within the previous one year, while the in the remaining patients the duration of their disease was between 1½ - 5 years. (Figure 3.3).

Figure 3.3a shows good correlation between the percentage of the outpatients and the inpatients in terms of the duration of their disease.

e. Medication Profile:

Table 3.2 shows that 29% of the patients interviewed in the clinic received insulin injection, indicating that they had the Type I diabetes. All but one of these patients were on the long-acting insulin (Lente) alone. The remaining patients appeared to have had the Type II, insulin independent diabetes of whom only one patient was on diet alone. 85.9% of the patients with the Type II diabetes were on a sulfonylurea drug (chlorpropamide) only, while 7% of the Type II patients were on both the sulfonylurea and a biguanide (metformin) (Table 3.2)

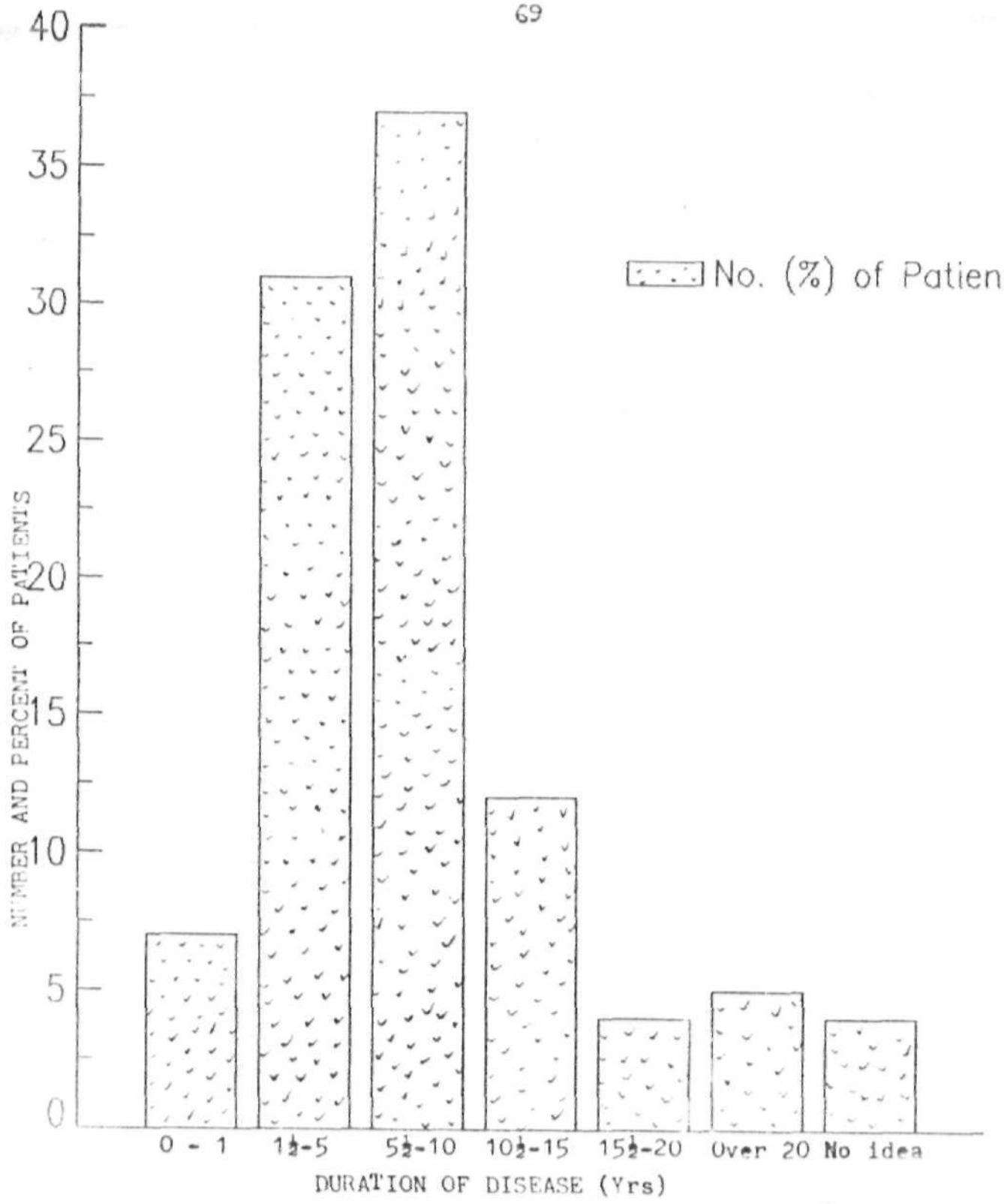



Figure 3.3

Duration of Diabetes Mellitus in 100 Diabetic Patients attending APUTH Zaria.

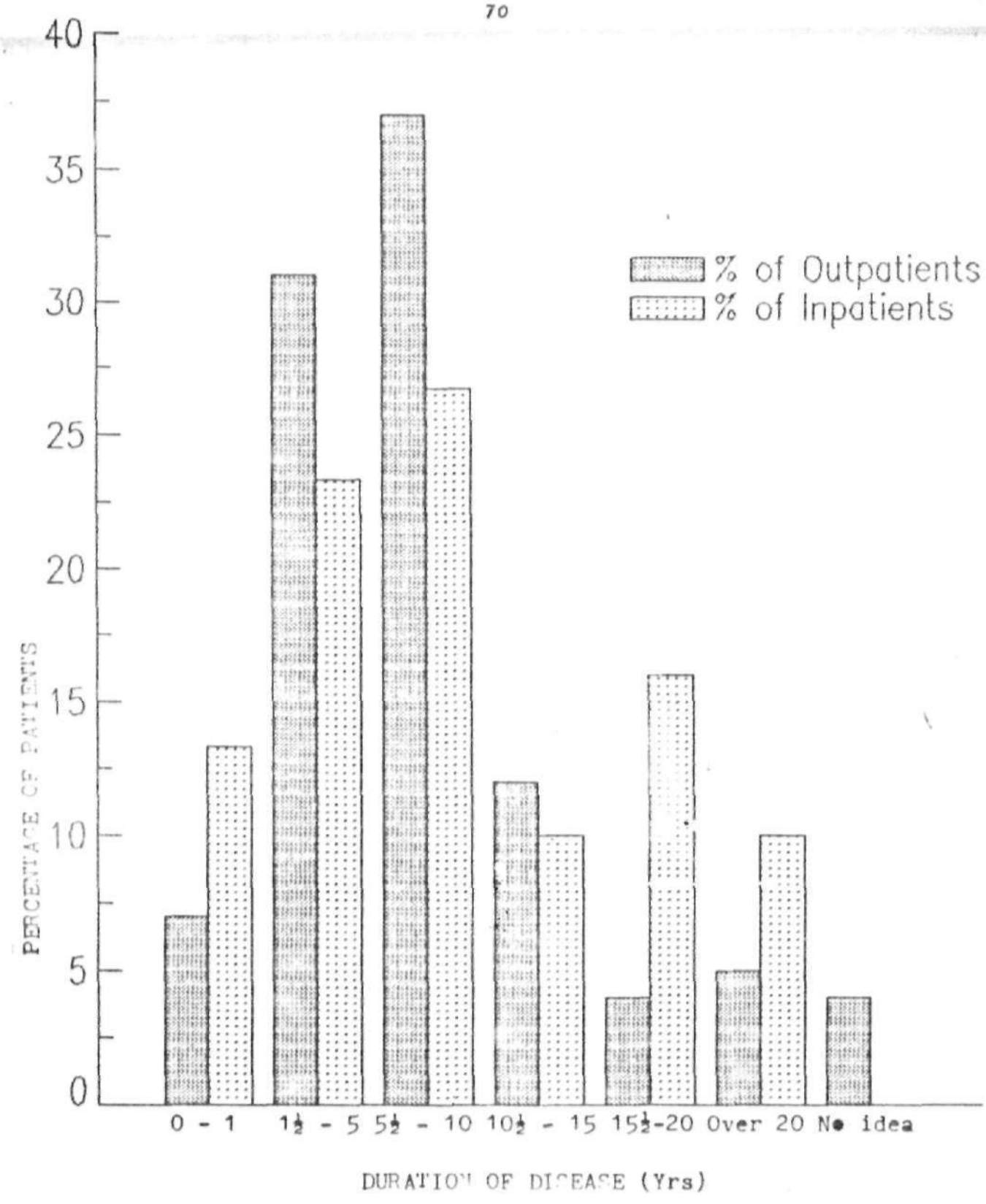



Figure 3.3a
Duration of Diabetes Mellitus in 100 Diabetic Patients attending ABUTH, Zaria, and 30 Diabetic Inpatients in the same Hospital.

Table 3.2

Medication Profile of 100 Diabetic Outpatients of the ABUTH, Zaria

| Type of Treatment | Number | Percentage |
|---|---|---|
| Diet only | 1 | 1 |
| Regular Insulin alone | - | - |
| Lente Insulin alone | 28 | 28 |
| Regular insulin + Lente Insulin | 1 | 1 |
| Sulfonylurea only | 61 | 61 |
| Biguanide only | 4 | 4 |
| Sulfonylurea + Biguanide | 5 | 5 |
| Total | 100 | 100.00 |

f. Complaints at the Clinic

The majority of the outpatients had one complaint or more to make at the clinic. Only 5% of the patients had no complaints at all, while more than 45% of them had one complaint and 22% presented with 2 complaints. The remaining patients complained of 3 or more problems at the clinic (Figure 3.4).

g. Compliance with management

It was observed that many of the patients took the management of their disease for granted. The results show that only 30% attended the clinic regularly. Only 19% followed a planned diet. 31% of the patient population studied could afford to test their blood in the hospital while only about 19% performed the urine sugar test at home (Table 3.3).

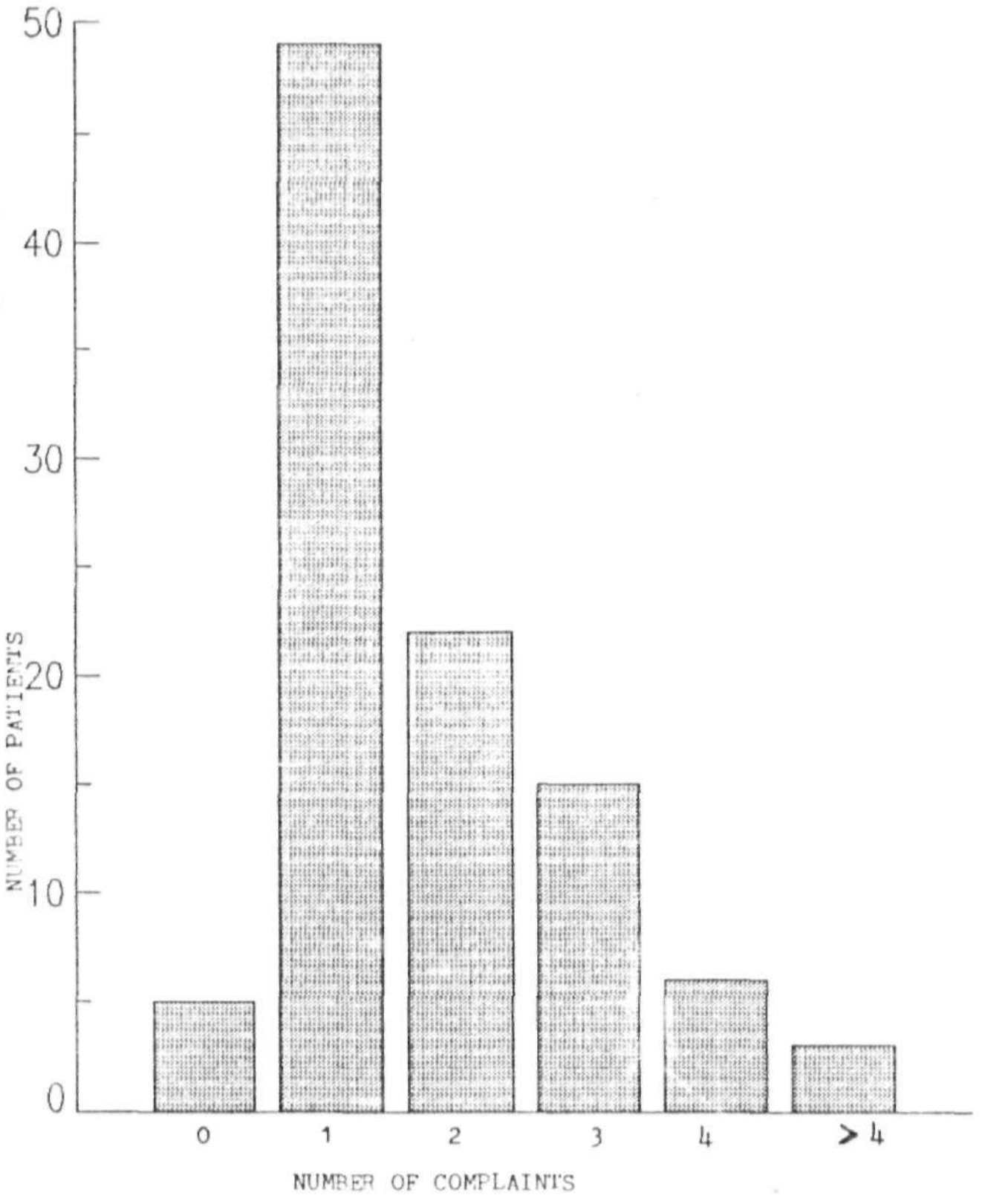



Figure 3.4

Number of Complaints Presented by 100 Diabetic Out-Patients of the ABUTH, Zaria.

Table 3.3

Compliance with Management And Control of Diabetes in 100 Diabetic Outpatients of ABUTH, Zaria.

| Parameter | Number | Percentage |
|---|---|---|
| Regular Clinic visit Following a Planned Diet | 81 | 30.68% |
| | 52 | 19.70% |
| Blood Sugar - Testing | 82 | 31.06% |
| Urine Sugar Testing | 49 | 18.56% |
| Total | 264 | 100.00 |

N.B:

Some patients complied with more than one parameter.

h. Concurrent Diseases And Complications:

A large proportion of the patients interviewed had developed one complication or the other. These included visual abnormalities such as cataract and blurred vision, parasthesia and other forms of neuropathy, and problems affecting the urinary tract. Many of the patients had more than one complication. This may not be unrelated to the fact that many of them did not comply with the rules of the management and control of their disease.

A number of patients also presented with one or more concurrent illness. A large proportion of the population had hypertension of whom more than half were males. Rheumatic disease and gastro intestinal disorders were common problems the patients presented with. Other problems included eye and skin infection, heart problems and some central nervous system diseases such as anxiety and headache (Tables 3.4 and 3.5)

Table 3.4

Concurrent Diseases Among 100 Diabetic Out-Patients of the ABUTH, Zaria

| Disease | Male | Female | Total Number | Percentage |
|---|---|---|---|---|
| Hypertension | 29 | 20 | 49 | 44,55% |
| Rheumatic Disease | 12 | 9 | 21 | 19,10 |
| G.I.T. Disorders | 5 | 7 | 12 | 10,91 |
| Respiratory Infection | 3 | 2 | 5 | 4,55 |
| Eye Infection | 3 | 1 | 4 | 3,64 |
| Heart disease | 2 | 1 | 3 | 2,73 |
| Others | 5 | 11 | 16 | 14,55 |
| Total | 59 | 51 | 110 | 100,03 |

N.B:

Some patients had more than one concurrent disease.

Table 3.5

Diabetic Complications Among 100 Diabetic Out-patients of the ABUTH, Zaria

| Complication | Male | Female | Total Number | Percentage |
|---|---|---|---|---|
| Visual Abnoraalities | 33 | 22 | 55 | 50,00% |
| Urinary Tract infection | 7 | 8 | 15 | 13,64 |
| Impotence (Neuropathy) | 13 | - | 13 | 11,82 |
| Paresthesia | 8 | 4 | 12 | 10,91 |
| Skin Problems | 4 | 3 | 7 | 6,36 |
| Kidney Problems (Nephropathy) | 2 | 1 | 3 | 2,73 |
| No obvious Problem | 2 | 3 | 5 | 4,55 |
| Total | 69 | 41 | 110 | 100,01 |

N.B:

Some patients presented with more than one complication.

3.1.2 In - Patients.

a. Age:

The analysis of the inpatients questionnaire showed that more than 36% of the patients were aged between 41-50 years. About 27% of the population were over the age of 50 and 20% were between 10 - 30 years. (Figure 3.5)

b. Occupation:

30% of the diabetic inpatients interviewed were housewives and about 16% farmers. A significant number of the inpatients (33%) were petty traders or civil servants. The remaining patients were either widows, students or retired officers. (Table 3.6).

c. Education: -

More than a third of the inpatients were found to be total illitrates while about 20% of the population had attended the Qur'anic school only. 36% of the patients went through primary to advanced schools while only about 6% had up to university education. (Figure 3.6).

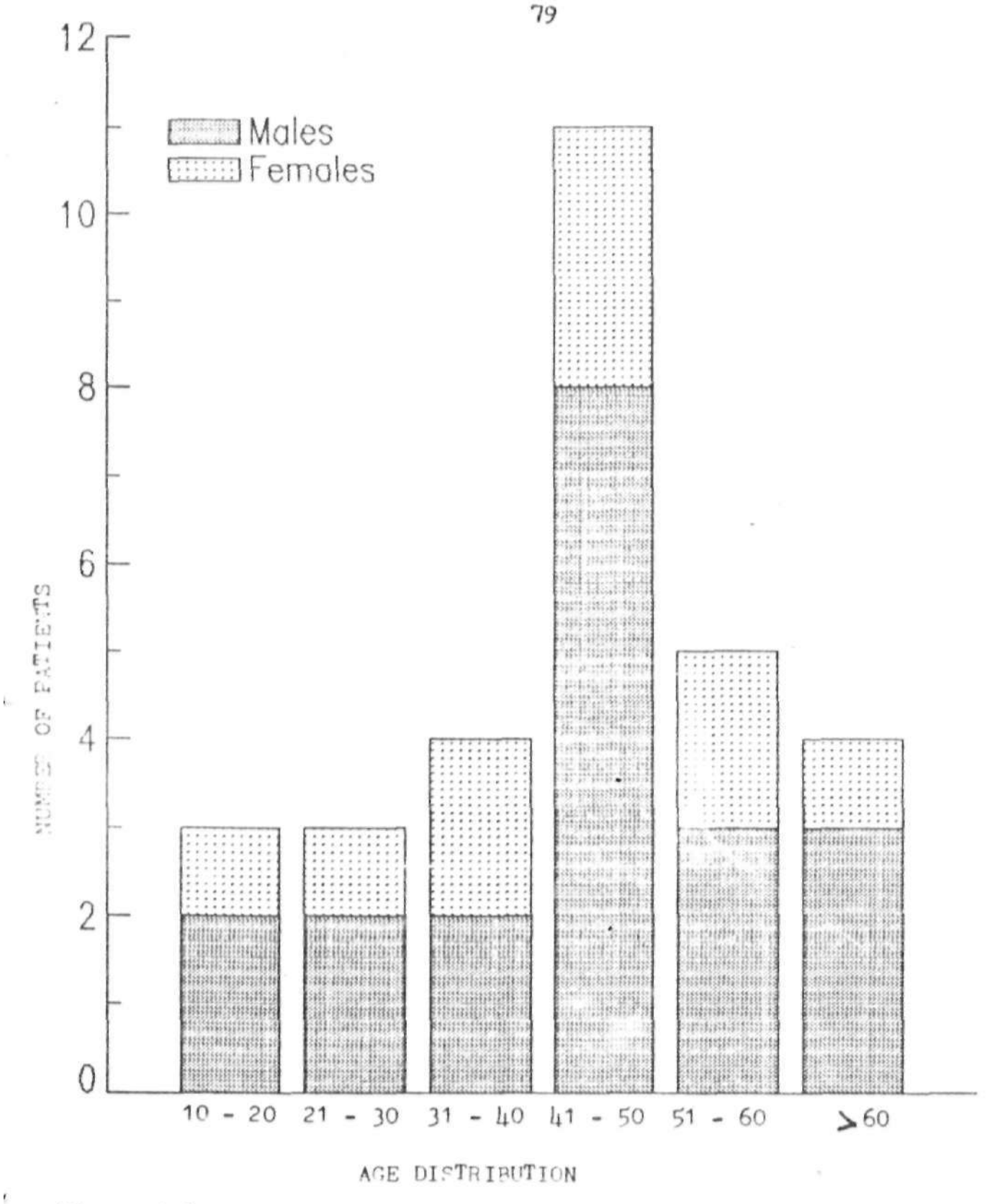



Figure 3.5

Age and Sex Distribution of 30 Diabetic In-Patients of the ABUTH, Zaria.

Table 3.6

Occupation of 30 Diabetic Inpatients of the ABUTH, Zaria

| Occupation | Number | Percentage |
|---|---|---|
| Civil Service | 6 | 20.00% |
| Farming | 5 | 16.67 |
| Housewife | 9 | 30.00 |
| Petty Trading | 4 | 13.33 |
| Others | 6 | 20.00 |
| Total | 30 | 100.00 |

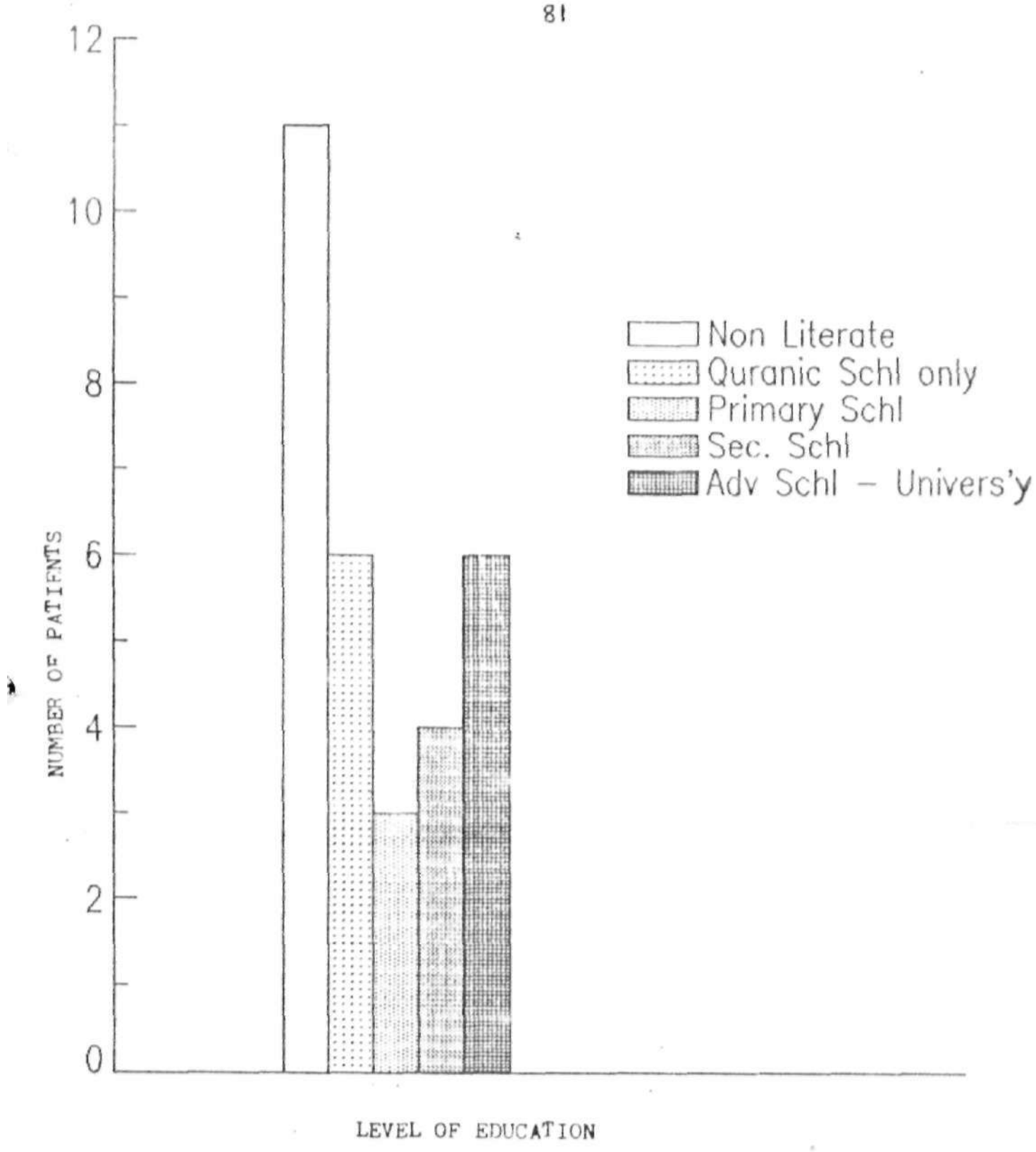



Figure 3.6

Level of education among 30 Diabetic In-patients of ABUTH, Zaria

d. Duration of Illness:

More than 50% of the inpatients interviewed had had the disease for more than 5 years but not above 20 years, while 10% of them had had it for more than 20 years and about 13% of the patients had discovered the disease within the preceeding 12 months. (Figure 3.7)

e. Medication Profile:

From the results, it can be seen that more than 50% of the inpatients interviewed were on injection of insulin even though not all of these were diagnosed to have the Type I diabetes. Of these about 43% were on the long-acting insulin (Lente) while about 13% of them were placed on both the long- and the short-acting insulin. 30% of the population were on a sulfonylurea agent (chlorpropamide) alone while 10% were on a biguanide alone and, about 3% on both agents (Table 3.7)

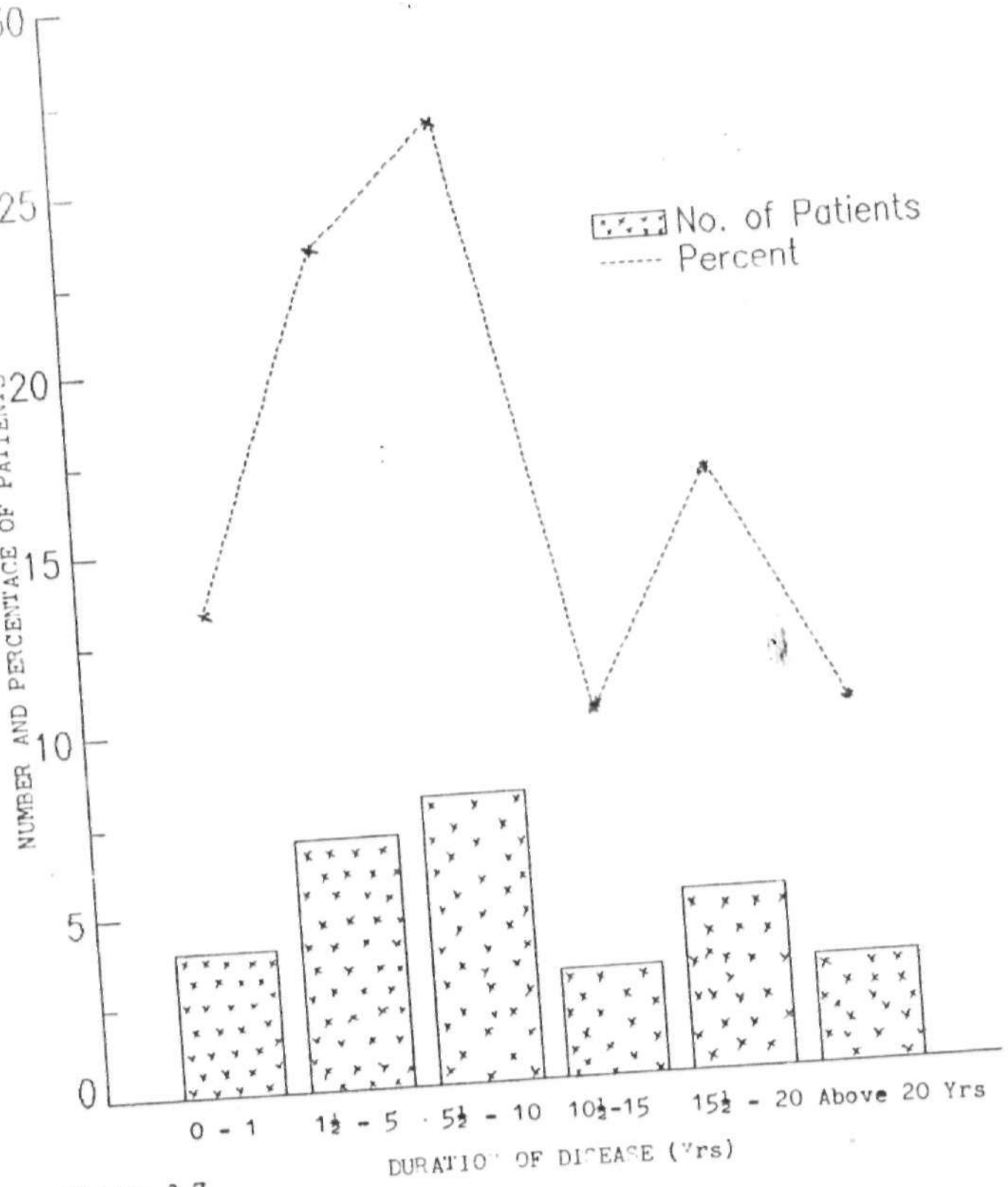



Figure 3.7

Duration of Diabetes Mellitus in 30 Diabetic In-patients of the ABUTH, Zaria.

f. Concurrent Disease and Complication:

All of the inpatients were found to have one illness or the other. However the common problems observed were cardio-vascular diseases e.g. hypertension and congestive heart failure; gastrointestinal disorders such as diarrhoea, abdominal pain and dysentery; and skin infection such as boils (Table 3.8)

Just as they presented with these illnesses so did some of them present with diabetic complications. Common among these were visual abnormalities (40%), urinary tract infection (20%) and neuropathic problems (31.4%). (Table 3.9)

Table 3.8

Concurrent Disease Among 30 Diabetic Inpatients

of the ABUTH, Zaria

| Disease | Male | Female | Total Number | Percentage |
|---|---|---|---|---|
| G.I.T. Disorders. | 5 | 3 | 8 | 24.24% |
| Cardiovascular disease | 4 | 2 | 6 | 18.18 |
| Skin Infection | 3 | 3 | 6 | 18.18 |
| Rheumatic disease | 3 | 2 | 5 | 15.15 |
| Respiratory disease | 2 | 1 | 3 | 9.09 |
| Others | 3 | 2 | 5 | 15.15 |
| Total | 20 | 13 | 33 | 99.99 |

N. B.

Some patients had more than one disease

Table 3.9

Diabetic Complications Among 30 Diabetic In-Patients of the ABUTH, Zaria

| Complication | Male | Female | Total Number | Percentage |
|---|---|---|---|---|
| Visual Abnormalities | 10 | 4 | 14 | 40.00 |
| Urinary Tract Infection | 4 | 3 | 7 | 20.00 |
| Parasthesia | 4 | 2 | 6 | 17.14 |
| Kidney Problems | 2 | 1 | 3 | 8.57 |
| Impotence | 2 | - | 2 | 5.71 |
| Other Neuropathic Problems | 2 | 1 | 3 | 8.57 |
| Total | 24 | 11 | 35 | 99.99 |

N.B:

Some patients had more than one complication.

3.2. **Chemical Assay:**

The results of the chemical assay of the five samples of chlorpropamide are shown in Table 3.10. Three of the five brands were found to comply with the official specification of B.P. (1980) for chlorpropamide tablets (ie., 92.0%-107.3%). The other 2 samples were found to contain less chlorpropamide than the official specification (Table 3.10).

Table 3.10

Quantitative Determination of Chlorpropamide

Tablets by Spectrophotometric Method

| Compound | %Content ± S.D. | Drug Content (mg) | Remark |
|---|---|---|---|
| $C_1$ | 97.94 (0.348) | 244.85 | Satisfactory |
| $C_2$ | 98.60 (0.916) | 246.50 | Satisfactory |
| $C_3$ | 94.88 (0.575) | 237.20 | Satisfactory |
| $C_4$ | 82.50 (0.510) | 206.25 | Unsatisfactory |
| $C_5$ | 83.33 (0.348) | 208.33 | Unsatisfactory |

S.D. = Standard Deviation.

### 3.3. **Serum Chlorpropamide Level:**

Linear calibration curve of chlorpropamide is shown in Figure 3.8 (r=0.9999462). Figure 3.9 shows the range within which the serum chlorpropamide levels of the patients lied. The majority of the patients, (79.6%) had levels within the acceptable therapeutic range of 75 - 250mg/L. 3.7% had levels in the toxic range (>200mg/L) while 16.7% had levels in the subtherapeutic range (>75mg/L).

********** RESULTS OF CALCULATION **********

SLOPE = 2.529692E-03
INTERCEPT ON Y AXIS = 7.787161E-04
PEARSON'S CORRELATION COEFFICIENT = 0.9999462
STANDARD DEVIATION OF POINTS FROM THE LINE = 3.129178E-03

SUM OF ERRORS SQUARED = 5.87505E-05

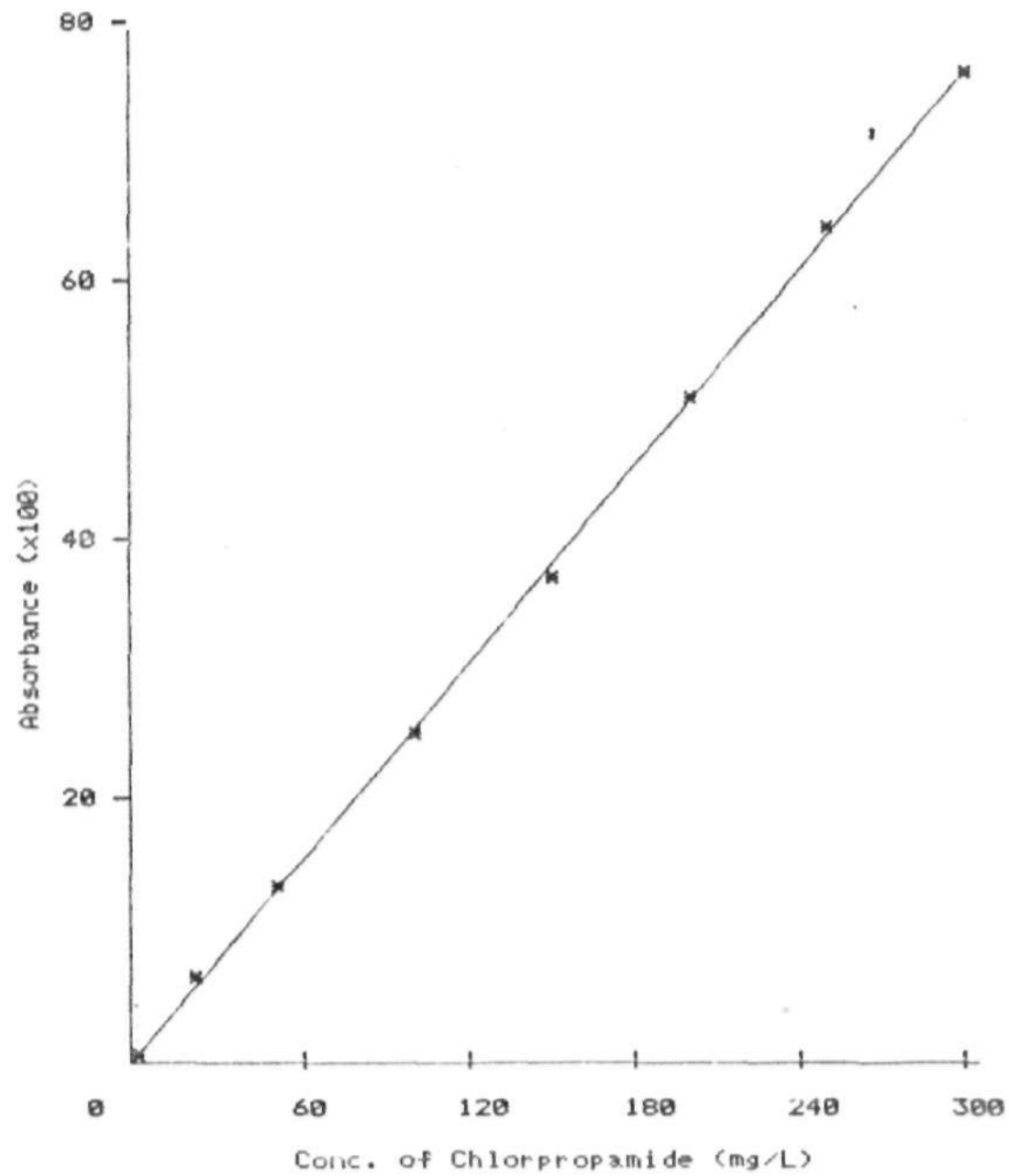



FIGURE 3.8

Calibration Curve of Chlorpropamide (r = 0.9999462).

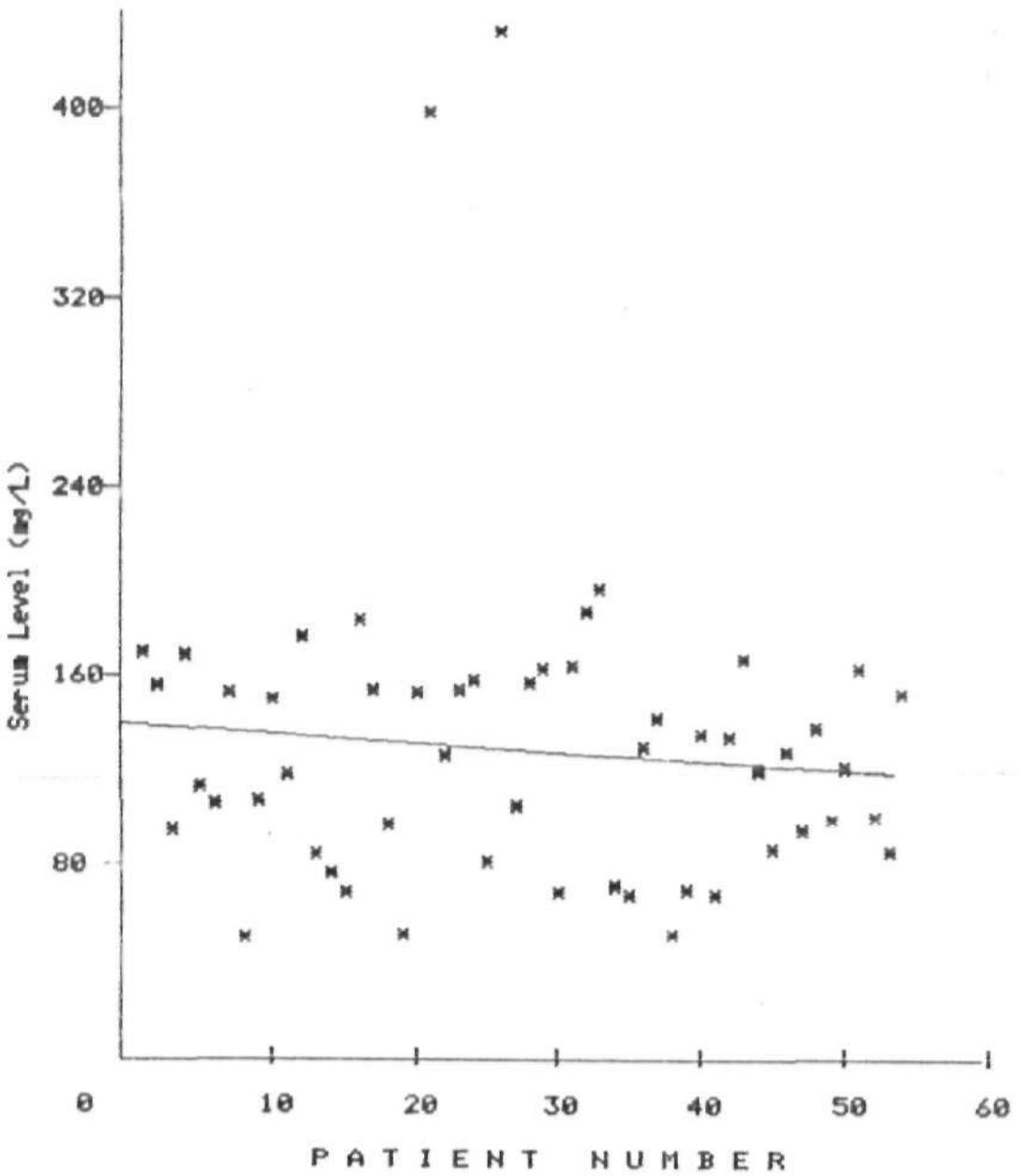



FIGURE 3.2

Serum chloropropamide levels in 54 diabetic outpatients attending UBTH, Zaria.

# CHAPTER FOUR

## DISCUSSION

The results of this study reveal that there were more male than female diabetics in Zaria urban area, similar to the findings of Osuntokun *et al.* (1971). This may be due to the fact that in Nigeria or Africa in general male patients attend the hospital more than the females. This may be attributed to the cultural background of the people (Osuntokun *et al*, (1971). These workers further revealed that it is only migraine as a general medical disease among Nigerians where females outnumber their male counterparts.

Like elsewhere most diabetics in Zaria urban area were found to be above the age of 40 years. This correlates well with the findings of Osuntokun *et al* (1971) who studied over 800 diabetic patients.

A significant number of patients were found to lack the understanding of their disease and the importance of proper management with diet and drugs. This lack of understanding was found to be more common among those patients who had no formal education including mostly the housewives and the

farmers who made up the majority of the patients. The ignorance may also be attributed to the fact that the doctors at the clinic see many patients during a short period of time (average clinic session is 50), a factor which limits the individual education of the patient (Simon *et al*, 1984). Studies conducted by Etzwiler (1962) on a group of juvenile diabetics demonstrated that they knew little about their disease and, tended to leave the management of their disease to their parents. Testing of the parents however disclosed a similar appalling lack of knowledge (Collier *et al*, 1971).

The lack of understanding of diabetes by most patients may in some way account for lack of good control of the disease, which could eventually lead to appearance of diabetic complications. It is however worth noting that teaching patients about the disease, and increasing their knowledge and understanding of management, does not fully guarantee improved control. Etzwiler *et al*, (1972) reported the findings of a study among some diabetic patients which showed that even though there was a significant increase of knowledge of the disease by the patients, there was no alteration in the high fasting blood sugar levels

or other physiologic parameters assessed. Hence patients' compliance with management of diabetes is also as important as the patient education.

The oral hypoglycaemic agents were found to be extensively used, particularly chlorpropamide accounting for more than 50% of the drugs prescribed for the patients studied. Chlorpropamide has many advantages enhancing its popularity in medical practice. Firstly the drug is taken orally, a significant advantage over injections. It has a long period (duration) of action so it can be taken just once or twice in a day. This may lead to greater patient compliance. It has minimal side effects with an incidence of about 6% (Shen and Bressler, 1977). Kinnear (1963) found the use of chlorpropamide in one of the Nigerian teaching hospitals very effective with only one patient that developed jaundice as a side effect. Secondary failure to this drug (and other oral hypoglycaemics) was found to be less common among Nigerians (Osuntokun *et al*, 1971). For these reasons chlorpropamide could be tried on all diabetics in the absence of infection and ketosis, except the young and insulin-dependent diabetic patient. However the economic problems and those of adequate production, supply and distribution in

this country are yet to be solved.

Most diabetic outpatients on insulin were found to self-administer their injections, and only a few depended on their relatives. In Nigeria where many patients live a long way from a hospital setting the importance of their learning the technique of self-injection of insulin cannot be exaggerated. As such the staff and management of a hospital usually take the pain of ensuring that these patients are independent.

Many of the inpatients were on insulin though not all of them had insulin-dependent diabetes. This is because some of them were admitted for severe illness, infection or ketosis. In these conditions, and many others, the use of oral hypoglycaemics is contraindicated (Shen and Bressler, 1977).

Regular attendance at the diabetic clinic was found to be variable. Those patients who were found not regular usually came back later with a relapse of symptoms or complication of the disease. A similar finding was reported by Osuntokun *et al*, (1971). These workers associated the poor attendance with the patients who were on the oral hypoglycaemics where they concluded that some of the patients who could afford to buy

tablets obtained them from a drug store and so remained away from the clinic for several months.

Treatment with diet was not satisfactory. It was discovered by questioning that only about 17% of the patients followed a planned diet. Even though diet is the conerstone of therapy in diabetes, it plays only a small part among the diabetics studied in this series. This is because many of the patients were poor (petty traders, housewives, subsistent farmers) and so could not afford the ideal diabetic diet. As the staple food in most Nigerian houses consists mainly of carbohydrates with little or no animal protein, rigid carbohydrate restriction and a change of dietary habit may be difficult and expensive to many patients. A peasant farmer or a labourer is not able to afford anything more than the cheapest diet which is necessarily high in carbobydrate content and low in protein and fat. As such dietary habits of the local population should always be put into consideration when planning a diabetic diet for the Nigerian diabetic particularly the illiterate ones. With the educated ones, list of food exchanges may be very useful.

Hypertension was observed to be the most common concomitant disease associated with diabetics in Zaria. However it was not severe in the majority of the patient because many were on drugs used for first stage treatment of hypertension (i.e diuretics). The inter-relationship between hypertension and diabetes among Nigerians has long been studied by Osuntokun (1972) where he found a close association between the 2 diseases. He further reported that majority of the patients with hypertention had the maturity-onset type of diabetes. The prevalence of hypertension among Nigerian diabetics increased with the duration of the disease, he concluded.

In this study, it was observed that only a small number of the patients monitored their control by testing their urine for the presence of sugar. This shortcoming indicates that many of them are unable to use this simple test for achieving good control. This is so because the patients, in the absence of the result of the urine sugar test are in no position to adjust their diet and medication. This may also likely account for the appearance of complications in the patients especially visual abnormalities and neuropathy. Constipation, diarrhoea, excessive

sweating, coldness of the extremities and impotence all indicate the evidence of autonomic neuropathy as was reported by Ellenberg (1960). Paresthesia which is a sign of peripheral neuropathy was also found to be common among the people in this study. Similar finding was reported by Greenwood and Taylor (1968) where they concluded that though it was rarely severe, the high incidence was due to the effect of not only diabetes but also of other toxic and nutritional factors acting on the population as a whole.

Regular blood glucose estimations at home was not carried out by any of the patients questioned. This is so because many of them did not know the significance of the test. Moreso a significant number of them could not afford the reagent. Accurate blood glucose monitoring is carried out using either meters or test-strips such as the BM Test-Glycemie (1-44 and 20-800) and the Visidex. The blood test allows fine tuning of the diabetic state. The blood sugar test was done only in the hospital on their clinic days. This shows that during clinic intervals patients are in no position to know the exact sugar level in their blood hence they may not be able to achieve good standard of control.

The presence and percentage of active ingredient in chlorpropamide tablets were quantitatively determined. Two brands (containing 82.50% and 83.33%) respectively of the labelled amount of chlorpropamide) did not comply with official requirement. This may be that the active ingredient used to make the tablets was not of suitable purity or the mixing proceedure was not very efficient as was reported by Wagner (1971). This worker went further to report controlled studies which have largely shown inequivalence rather than equivalence of marketed drug products containing the same drug in the same type of dosage form but made by different manufacturers. Failure to achieve good control of diabetes in some patients may therefore not be unrelated to patients' consumption of substandard or faked drugs which do not actually contain the exact amount of the medicament.

This result therefore suggests that different commercial brands of chlorpropamide may not all contain the actual amount of the stated official content of the drug. When patients take this (orally) it can give rise to markedly different pharmacokinetic profiles in the patients as was observed by Monro and Welling (1974). These

workers further suggested that during chronic administration, substitution of one generic brand for another might cause significant, even if only temporary, fluctuations in circulating blood levels of the drug. This in turn could concievably cause a loss of control of diabetes in a patient already stabilized on a particular formulation, they concluded.

Osuide (1988) blamed the incidence of faking on the high prices of drugs, scarcity, preferences for cheaper drugs, difficulty in detecting faked drugs. Ogunyemi (1988), on the other hand, blamed this problem on the absence of quality assurance in the pharmaceutical industry which causes many people to suffer physical and mental deformities or even death.

Serum chlorpropamide level in 54 patients indicated that more than 70% of the patients had levels within the acceptable therapeutic range of 75 - 250mg/L. However, as expected there are large interindividual differences in the serum concentration of the drug in the patients studied. Melander *et al* (1978) suggested that these variations may be due to interindividual differences in drug metabolising capacity. Differences in renal excretion may also add to

this variation as this drug is only partially metabolised (Taylor, 1972). Two of the patients had serum levels well into the toxic range (>250mg/L) while several seem to achieve only subtherapeutic levels. Study by Brotherton *et al* (1969) indicated that in diabetic patients receiving longterm treatment with chlorpropamide variations in the rate and type of metabolism might occur. These variations may be significant in understanding cases in which there is failure to respond to chlorpropamide therapy. The availability of assays like this one makes it possible to individualise dosage to promote therapeutic endpoints.

No serious attempts were made to relate the serum chlorpropamide levels with the control of the disease in individual patients because such assessment would have involved many other factors such as diet, insulin output response by the pancreatic β-cells etc.

## 4.2 CONCLUSION

About 2% of the patients seen in Ahmadu Bello University Teaching Hospital in Zaria annually are diabetics. The study also revealed that some of the diabetic patients who attend this Teaching Hospital, and who took part in this survey, lacked the understanding of their disease and the majority were illiterates. This could lead to loss of control of the disease which may also be attributed to the fact that majority of the patients were poor and so could not afford the type of diet suitable for diabetics. The cost of test reagents for urine (and blood) sugar also made it difficult for many of the patients to perform the tests for achieving good control of their disease.

The Diabetic Clinic in Zaria needs the services of a dietician as well as more medical personnel so as to educate the patients properly.

Some of the drugs used by the patients were found not to comply with the official requirement. As such the absorption and other pharmacokinetic parameters of the drug in the body would be adversely affected and, this may eventually lead to loss of control of diabetes. The pharmacy shop within the hospital should be made to stock enough

antidiabetic drugs of adequate standards. This will ensure that the patients receive the proper drugs and reagents and reduce the risk of them buying substandard drugs from other suppliers.

Diabetes is a chronic disorder whose proper management requires more than average wealth among patients. In view of the predominance of illiterates and housewives shown in this study it will help if the treatment of this potentially crippling disorder is made free or at least the drugs are sold at subsidized rates to patients.

Differences in blood levels of chlorpropamide as observed in this study suggests that blood of patients receiving longterm treatment with this drug should be assayed from time to time in order to adjust the dose and promote therapeutic efficacy.

There is need for educating the general public about diabetes mellitus including its management and complications. To fulfill some of this goal, a small booklet to be based on the modification of Appendix III of this write-up will be published, although the primary target is the diabetic patient. Efforts of medical personnel who attend to patients with other chronic diseases such as hypertension, epilepsy etc need to be assessed through this type of study in order to enhance therapeutic endpoints.

# REFERENCES


Abramson E. A. and Arky, R. S.: Treatment of the obese diabetic - A comparative study of placebo, sulfonylurea and phenformin. Metabolism 16 : 204, 1967.

Adi F. C. : Diabetes Mellitus associated with epidemic of infections hepatitis in Nigeria. Br. Med. J. 1 : 183, 1974.

Aguwa C. N. Diabetes Mellitus : In Therapeutic Basis of Clinical Pharmacy In the Tropics , Aguwa C. N. (ed) 1st edition, MacMillan Publishers, London, 1986, pp 198 - 216.

Archer J. A. Gorden, P. and Roth. J. : Defect in inssulin binding to receptors in obese man. Amelioration with calorie restriction. J. Clin. Invest. 55 : 166, 1975.

Belmonte M. M. Sarkozy E. and Harpur ER : Urine sugar determination by two-drop clinic-test method. Diabetes 16 : 557, 1967.

Berne C. Ericksson G. Lundgren P. : How accurate are insulin mixtures prepared by the patients. Diabetologia 27 (2) : 256A, 1984.

Best. C. H. Epochs in the history of Diabetes. In Williams R. H. (Ed): Diabetes. New York, Paul B. Hoeber (1960).

Bogardus C., Ravussin, E, Robbins, D.C., Wolfe, R.R., Horton, E. S. and Sims, E.A.H.: Effect of physical training and diet therapy on carbohydrate metabolism in patients with glucose intolerance and type II diabetes mellitus. Diabetes 33 (4) : 311, 1984.

Bottazzo F. C., Florin-Christensen A and Doniach D : Islet cell antibodies in diabetes mellitus with antoimmune polyendocrine deficiencies. Lancet. 2 : 1279 - 1283, 1974.

Botz C. K., Leibel B. S. Zingg W. Gander R. E. Albisser A. M. : Comparison of periphenral and portal routes of insulin infusion by a computer - controlled insulin infusion system (artificial endocrine pancreas). Diabetes 25: 619, 1976.

Brotherton P. M., Grievson, P. and McMartin, C.: A study of the metabolism of chlorpropamide in man. Clinical Pharmacol and Therap. 10:505, 1969.

Bucholtz H. K. : Insulin allergy: An approach to therapy. South. Med. J. 69 : 1118-1120, 1976.

Bunn, H. F., Hanney, D. N., Kamin, S., Gabbay, K.H. and Gallop, P. M.: The biosyntesis of human Haemoglobin$_{A_c}$: Slow glycosylation of haemoglobin *in vivo*. J. Clin. Invest. 57: 1652, 1976.

Carmichael R. H. : A method for the routine determination of chlorpropamide in plasma (or serum). Clin. Chem. 5 : 597-602, 1959.

Collier B.N. Jr., Etzwiler D.D., Dekalb, I. and Minneapolis, M.: Comparative study of diabetics and their parents. Diabetes 20(1): 51, 1971.

Colwell A. R. Jnr. : Understanding your diabetes. Thomas Publisher, Springfield illinois (1978).

Colwell, J.A., Halluska, P.V. and Sarji, K.E.: Platelet function and diabetes mellitus. Med. Clin. North Am. 62:753, 1978.

Coore H. G. and Randle P. J. : Regulation of insulin secretion studied with pieces of rabbit pancreas. Biochem. Journ., 63 : 66, 1964.

Danowski, T.S., Khurana, R.C., Jung, Y., Wingert, J.P., Aarons, J.H., Katz, C. and Corredor, D.G.: Serum cholesterol and triglycerides during mild glucose intolerance. Am. J. Clin. Nutr. 24:855, 1971.

Davidson J. K. : Controlling diabetes mellitus with diet therapy. Postgrad. Med. 59 : 114 - 122, 1976.

Davidson J. K. : Plasma glucose lowering effect of caloric restriction in obesity induced insulin treated diabetes mellitus. Diabetes 26 : 355, 1977 (Suppl. 1, Abstract No. 12).

Deckrert T. and Poulson J. E. : Diabetic nephropathy : Fault or destiny? Diabetologia. 21 : 178, 1981.

De Fronzo R. A. Soman, V. Sherwin R. S., Hendler, R. and Felig, P.: Insulin binding to monocytes and insulin action in human obesity, starvation and refeeding. J. clin. Invest. 62 : 204, 1978.

Drash A : The control of diabetes Mellitus : Is it achievable? Is it desirable? J. Peadiatric 88 : 1074, 1976.

Drug and Therapeutic Bulletin. Vol. 17 (22) oct. 26, 1979 : Can the diabetic diet be controlled?

Editorial : Diabetic neuropathy : A preventable complication. Lancet 2 : 583, 1972.

Eilenberg M. Diabetic neuropathy : A consideration of factors in onset. Ann. Itern. Med. 52 : 1967-1975, 1960.

Ellenberg M : Impotence in Diabetes. The neurologic factor. Ann. Intern. Med. 75 : 213, 1971.

Engerman R. L., Bloodworth, J. M. B. Jr. and Nelson, S : Relationship of microvascular disease in Diabetes to diabetic control. Diabetes 26 : 760, 1977.

Etzwiler D. D. : What the juvenile diabetic knows about his disease. Peadiatrics 29 : 1, 1962.

Etzwiler D. D. and Robb J. R. : Evaluation of programmed education among juvenile diabetics and their families. Diabetes 21 : 967-971, 1972.

Evaluation of drug interactions. (2nd Ed)by Washington A. American pharmaceutical Association, 1976.

Fajans S. S. Floyd J. C. Jr, Tattersal R. B., Williamson, J. R., Pek, S. and Taylor, C. I. : The various facets of diabetes in the young . Arch. Intern. Med. 136 : 194 (1976).

Feinglos M. N., Lebovitz H. E. : sulfonylureas increase the number of insulin receptors. Nature 276 : 184, 1978.

Feldman J. H., Lebovitz H. E. : Appraisal of the extrapancreatic actions of sulfonylureas Arch. Intern. Med. 123 : 314, 1969.

Felig, P.: Insulin, glucagon and somastostatin in normal physiology and diabetes mellitus. Diabetes 25: 1091, 1976.

Ferrannini, E. : The disposal of an oral glucose load in healthy subjects : a comparative study. Diabetes 34(6): 589, 1985.

FitzGerald, MG, and Keen, lt : Diagnostic - classification of diabetes. Br. Med. J. i : 1568, 1964.

Gabbay, K.H., Hasty, K., Breslow, J.L., Ellison, R.C., Bunn, H.F. and Gallop, P.M. : Glycosylated haemoglobins and long-term blood glucose control in diabetes mellitus. J. Clin. Endocrinol. Metab. 44: 859, 1977.

Gale E. A. M. and Tattersal, R. B. : Can Phenformin - induced lactic acidosis be prevented? British Med. J. ii, 972-975, 1976.

Galloway J. A., Bressler R : Insulin treatment in diabetes. Med. Clin. North. Am. 62 : 663-680, 1978.

Ganz K. Kozak G. P. Diabetes mellitus and primary hypothyroidism. Arch, Intern. Med. 134 : 430, 1974.

Genuth S. M. : Plasma Insulin and Glucose profiles in normal, obese and diabetic Persons. Ann. Intern. Med. 79 : 812, 1973.

Gepts W; De Mey, J; Marichal-Pipeleers, M. : Hyperplasia of 'Pancreatic polypeptide' - cells in the pancreas of juvenile diabetes. Diabetologia 13 : 27, 1977.

Gharib H. : Steroid diabetes. Minn. Med. 52 : 669, 1969.

Gifford H. and Bergerman J. : Falsely - negative enzyme paper tests for urinary glucose. JAMA. 178 : 423, 1961.

Gonen, B., Rubenstein, A.H., Rochman, H., Tanega, S. and Horwitz, D. L. : Haemoglobin A,: an indication of the metabolic control of diabetic patients. Lancet ii:734, 1977.

Greenwood B.M. and Taylor J.R. : The coplications of diabetes in Nigerians. Trop. Geog. Med. 20: 1-12, 1968.

Gregerson G. : Diabetic neuropathy : Influence of age, sex, metabolic control and duration of diabetes on motor conduction velocity. Neurology 17 : 972, 1967.

Grodsky G. M. and Bennett L. L. : Cation requirement for insulin secretion in the isolated perfused pancreas. Diabetes, 15 : 910, 1966.

Hansen J. M., Christensen L. K., : Drug interaction with sulfonylurea hypoglyceamic drugs. Drugs, 13 : 24, 1977.

Hinden, E : Mumps followed by Diabetes. Lancet 1 : 1381, 1962.

Howard-Williams J., Hillson, R. M., Bron, A., Awdry, P., Mann, J. I. and Hockaday, T. D. R. : Retinopathy is associated with high glycemia in Type II diabetes mellitus. Diabetologia 27 (2) : 198, 1984.

Jenkins D. J. A, Leeds A. R., Wolever T. M. S., Goff, D. V., Alberti, K. G. M., Gassul, M. A. and Hockaday, T. D. R. : Unabsorbable carbohydrate and diabetes. Decreased postprandial hyperglyceamia. Lancet 2 : 172-174, 1976.

Jenkins D. J. A., Wolever T. M. S., Hockaday T. D. R., Leeds, A. R., Howarth, R., Bacon, S., Apling, E. C. and Dilawari, J. : Treatment of diabetes with guar gum. Lancet 2 : 779 - 780, 1977.

Kempner W., Newborg B. C., Peschel R. L. and Skyler, J. S. Treatment of massive obesity with rice reduction program: An analysis of 106 patients with at least a 45kg weight loss. Arch. Intern. Med. 135 : 1575, 1975.

Kiehum T. G., Anderson J. W., Ward K. : Beneficial effects of high carbohydrate, high fibre diet on hyperglyceamic diabetic men. Am. J. Clin. Nutr. 29 : 895, 1976.

Koda-Kimble M. A. Diabetes. In Herfindal E T and Hirschman J. L. (Eds) : Clinical Pharmacy and Therapeutics. 2nd Edn 1979.

Kolterman O. G., Gray, R.S., Shapiro, G., Scarlett, J.A., Griffin, J. and Olefsky, J.M.: The acute and chronic effects of sulfonylurea therapy in Type II diabetes. Diabetes 33 (4) : 346, 1984.

Kremer H. U. : Juvenile diabetes as a sequal to mumps. Am. J. Med. 3 : 257 - 258, 1947.

Kumar D, Miller L. V., Mehtalia S. D. : Use of dexamethasone in treatment of insulin lipoatrophy. Diabetes 26 : 296 299, 1977.

Larner J. Insulin and Oral Hypoglyceamic Drugs In: Goodman L. S., Gilman A (Eds): The pharmacological Basis of Therapeutics 6th Ed. 1980, pp 1497-1521.

Lebovitz H., Feinglos M. N.: Sulfonylurea drugs : Mechanism of antidiabetic action and therapeutic usefulness. Diabetes Care 1 : 189, 1978.

Leonards J. R. : Evaluation of enzyme tests for urinary glucose. JAMA. 163 : 260, 1957.

Logie A. W., Galloway D. B. and Petrie J. C. : Drug interactions and long term antidiabetic therapy. British J. of Clin. Pharmacol. 3 : 1027-1032, 1976.

Loubatieres, A. : The hypoglyceamic sulfonamides: History and development of the problems from

1942 - 1955. Ann. N. Y. Acad. Sci. 71 : 4-11, 1957.

MacDonald M. J. : Equal incidence of adult onset diabetes among ancestors of juvenile diabetics and non-diabetics. Diabetologia 10 : 767 - 773, 1974.

MacCuish A. C. Jordan J. Campbell C. J., Duncan L. J. P. Irvine W. J. : Antibodies to pancreatic islet-cells in insulin - dependent diabetes with coexistent autoinmune disease. Lancet 2 : 1529, 1974

Mattson J. R., Patterson R., Roberts M, : Insulin therapy in patients with systemic insulin allergy. Arch. Intern. Med. 135 : 818-821, 1975.

Mcivor M. E., Cumming, C.C., Leo, T.A., Mendeloff, A.I. and Baltimor, M.D. : Acute and Chronic effects of high carbohydrate high fibtre food suppliments in patients with the Non-insulin-dependent diabetes mellitus. Diabetes 33 (Suppl. 1), 1984.

Melander, A., Sartor, G., Wahlin, E., Schersten, B. and Bitzen, P.O. : Serum tolbutamide and chlorpropamide concentrations in patients with diabetes mellitus. Br. Med. J. 1: 142, 1978.

Menser, M. A. Forrest J. M. Bransby R. D. : Rubella infection and diabetes mellitus. Lancet. 1 : 5760, 1978.

Michael S. : Effect of sulfonylurea treatment and *in vitro* insulin secretion and action in patients with Type II diabetes. Diabetes. 31 (4) : 307, 1982.

Miller M. and Moses A. M. : Mechanism of chlorpropamide action in diabetes insipidus J. Clin. Endocrinol. 30 : 488, 1970.

Mirsky I. A., Persitti G. Diengatt D : The inhibition of insulinase by hypoglyceamic sulfonamides. Metabolism. 5 : 156, 1956.

Monro A. M. and Welling P. G. : The Bioavailability in man of marketed brands of

chlorpropamide. Eur. J. Clin. Pharmacol. 7 : 47-49 1974.

Moran G. S. Fanagh P. Lindsay M. Kurtz A. B., and Brown R. : Childhood diabetes : Diabetic control and psychological disturbances. Diabetologia 27 (2) : 312 (A), 1984.

Moses A. M. And Miller M. : Diuretic action of 3 sulfonylurea drugs. Ann. Intern. Med. 78 : 541, 1973.

Munichoodappa C. Kozak G. P. : Diabetes mellitus and pernicious anaemia. Diabetes 19 : 719, 1970.

Nagulesparam M. Savage P. J. Mott D.M., Johnson, G.C., Unger, R.H. and Bennet, P.H. : Increased insulin resistance in obese glucose-intolerant south-west American Indians : Evidence for a defect not explained by obesity. J. Clin. Endocrin. Metab. 51 : 739, 1980.

National Diabetes Data Group : Classification and Diagnosis of Diabetes Mellitus and other

categories of glucose intollerance. Diabetes 28 : 1039 (1979).

Nattrass M. and Alberti, K.G.M. : Biguanides. Diabetologia 14 : 71, 1978.

Norbet F., Metzger, B.E., Phelps, R.L., Dooley, S.L., Ogata, E.S., Radvany, R.M. and Belton, A.: Gestational Diabetes. Diabetes 34(2): 1985

Ogunyemi, B. : Pharmanews 10(8): 1988.

Osuide, G. E., Pharmanews 10(7): 1988.

Osuntokun, B. O. : Hypertension in Nigeria diabetics : A study of 832 patients. Afr. J. Med. Sci. 3 : 257, 1972.

Osuntokun, B. O. Akinkugbe, F. M., Francis T. I., Reddy, S., Osuntokun, O and Taylor, G. O. L. : Diabetes Mellitus in Nigerians: A study of 832 patients. West Afr. Med. J. 20 : 295-312, 1971.

Papaspyros, N.S : The history of Diabetes Mellitus. Stuttgart Georg Thieme,, (1964).

Parry D. G. and Taylor, K. W. : Effects of sugar an incorporation of (3H) Lencine in insulin in vitro. Biochem. Journ. 110, 2c, 1966.

Pell S. and D'Alonzo, C. A. : Some aspects of hypertension in diabetes mellitus. JAMA. 202 : 104, 1967.

Perlman L. V. : Familial incidence of diabetes in hyperthyroidism. Ann. Intern. Med, 55 : 796, 1961.

Rapoport M. L. and Hurd H. F. : Thiazide induced glucose intolerance treated with potassium. Arch. Intern. Med. 113 : 405, 1964

Rossini A. A. : Why control glucose level? Arch. Surg. III : 229, 1976.

Rundles R. W. : Diabetic neuropathy: A general review with report of 125 cases. Medicine 24 : 111, 1945.

Sagel, J., Colwell, J.A. and Crook, L. : Increased platelet aggregation in early diabetes mellitus. Ann. Intern. Med. 82:733, 1976.

Schmitz, A. and Mogensen, C.E. : Obesity and mortality in Type II diabetes. Diabetologia 27(2):328A, 1984.

Seltzer H. S. : Drug induced hypoglyceamia: A review based on 473 cases. Diabetes 21 : 955, 1972.

Shen, S.W. and Bressler, R. : Clinical pharmacology of oral antidiabetic agents. Part I. New Engl. J. Med. 296:493, 1977.

Shen, S.W. and Bressler, R. : Clinical pharmacology of oral antidiabetic agents. Part I. New Engl. J. Med. 296:787, 1977.

Simon K., Bakos J., and Gimesi J. : Are general practitioners and Nurses suitable to educate diabetic patients? Diabetologia 27 (2) : 332, 1984.

Siperstein M. D., Foster D. W., Knowles H. C. Jr., Levine, R., Madison, L. L. and Roth, L. : Control of blood glucose and diabetic vascular disease. New. Engl. J. Med. 296 : 1060, 1977.

Sonksen P. H : The evolution of insulin treatment. Clinics in Endocrinol. Metab. 6 : 481-497, 1977.

Sultz, H. A. Hart B. A. Zielezny M. and Schlesinger, E. R. : Is mumps virus an aetiological factor in Juvenile diabetes mellitus? Preliminary report. J. Peadiatric 86 : 654, 1975.

Tamborlane W. V. Sherwin R. S. Genel M., Felig P : Administration of insulin to juvenile diabetics via portable pump. New Engl. J. M. 300 : 573, 1979.

Tattersal R . B. and Fajans S. S. : A difference between the inheritance of classical juvenile - onset and maturity - onset type diabetes of young people. Diabetes 24 : 44, 1975.

Tattersal R. B, Pyke D. A. : Diabetes in identical twins. Lancet 2 : 1120, 1972.

Taylor, J.A. : Pharmacokinetics and biotrans-formation of chlorpropamide in man. Clin. Pharmacol. Ther. 13:710, 1972.

Teale J. D. and Love A. H. : The effects of Tolbutamide and Glibenclamide on intestinal glucose absorption. Biochem. Pharmacol. 21 : 183a, 1972.

Teuscher, A : A treatment of insulin - lipoatrophy with monocomponent insulin. Diabetologia 10 : 211-214, 1974.

Volpe R. : The role of antoimununity in hypoendocrine and hyperendocrine function Ann. Intern. Med. 87 : 86 - 99, 1977.

Wagner T. G. : Generic equivalence and inequivalence in oral products. In Biopharmaceutics and Relevant Pharmaco-kinetics. Pd. Wagner. T. G. and Pernarowski M. 1st Ed. Drug Intelligence Publications. Hamilton Illinois. pp. 166-180.

West K. M. : Epidemiologic evidence linking nutritional factors to the prevalence and manifestation of diabetes. Acta Diabetologia (Suppl) 9 (1) : 405, 1972.

Wolff F. W. *et al* : A new form of experimental diabetes. 12 : 335, 1963.

Yalow R.S. : Plasma insulin and Growth hormone levels in obesity and diabetes. Ann. of New York Academy of Sci. 131, 137 : 1965.

Yoon JW. Austin M. Onodera T. and Notkins, A.L.: Virus induced Diabetes Mellitus. N. Eng. J. Med. 300 : 1173-1179, 1979.

APPENDIX I

SAMPLE QUESTIONAIRE USED IN THE STUDY.

SURVEY ON THE CONTROL OF DIABETES IN ZARIA LOCALITY.

Tick where applicable [/]

A. 1. Name: .....Hospital Number.....

2. Age:.....Sex:.....

3. State/LGA:.....Occupation:.....

4. Educational status: Literate....Non-literate.....

5. Level of Education:

- Quranic school only
- Quranic school and Primary education
- Secondary School
- Advanced school
- University Education

B. 1. When did you discover you had diabetes?.....

2. How did you discover it (signs)?.....

3. Where did you discover this?.....

4. (a)Do you have any relative with Diabetes Yes/No

(b)If yes, what is your relationship with the fellow?

- Grand father/mother/both.

- Father/mother/both.
- Others (Specify).....

C. 1. (a) What did you do when you first noticed this disease?
- Consulted a doctor and followed the doctor's treatment plan.
- Visited the herbalist.

(b) If you visited a herbalist, were you relieved? Yes/No

(c) If you did nothing about it, did the symptoms disappear? Yes/No

(d) If you consulted a doctor, which drugs are you placed on?
- Injectable preparation
- Oral preparation

(e) How many times a day do you have your medication
- Once
- Two times
- Three times

2. What is your reason for taking your antidiabetic drugs.
- To control the disease
- To cure your disease
- Because the doctor told you to do so.

3. How many different drugs are you currently taking.....
.....

4. (a) Do you always obtain your drugs from the hospital?

Yes/No

(b) If No, where do you obtain them?

- From a Pharmacy/Chemist shop.
- From a patent Medicine store.
- From the open market.

5. How do you store your drugs?

- In a refrigerator
- In cool pot of water
- In a dry place
- Anywhere possible for me.

6. (a) Do you always keep your appointment with your doctor?

Yes/No

(b) If yes, why do you do so?

- Because you are told to do so
- Because you want to control your disease

7. (a) Do you have problem with your eyes? Yes/No.

(b) Are you hypertensive? Yes/No.

(c) If yes (for a or b or both), is it before or after the detection of your diabetes?

- Before
- After

8. (a) If you become to ill. what do you do?

- Visit the Doctor
- Stay at home and continue medication
- Do nothing about it

9. (a) (If you are on injectable) How do you identify your insulin from its other forms?

- It is a cloudy preparation
- It is a clear preparation
- I cannot say

(b) Who injects the insulin for you?

- self
- Nurse
- Anybody around

(c) What parts of your body is the drug injected into

.....

(d) How many units of insulin do you take daily?.....

.....

(e) Do you make adjustment for the insulin injection? Yes/No

(f) If yes, when do you adjust it?

- After a hardwork/exercise.
- During an illness
- Anytime you feel like
- Others (specify).....

(g) Insulin can be taken oraly. True/False.

(h) Have you had a reaction with your insulin? Yes/No

(i) If yes, what did you do?

- Went to see the doctor
- Stayed at home and continue the medication
- Did nothing about it

(j) Who at home can give insulin when you could not do s
yourself?

(k) What usually happens during an insulin reaction?

- Sweating
- Trembling
- Feeling of hunger.
- Blurred vision and dizziness
- Excitation and happiness.
- I have no idea

10. (a) Are you pregnant? Yes/No

(b) If yes, did you inform your doctor? Yes/No

(c) Any change in your medication? Yes/No

(d) If yes, what is the change? (specify).....
.....

(e) Have you been diabetic before the pregnancy? Yes/No

(f) Do you attend ante-Natal clinic regularly? Yes/No

(g) How many children have you?.....
Living .....dead.....

(h) Have you had any miscarriage before? Yes/No

(i) If yes, how many?.....

D1. (a) Do you test your blood? Yes/No

(b) If yes, where?

- At home
- At the hospital only

(c) Do you keep record of this? Yes/No

If yes, how has it helped you to control your disease

- Positively
- Negatively
- No change

2. (a) Do you test your urine Yes/No

(b) If yes, where

- At home
- At the hospital only

(c) Do you keep record of this? Yes/No

If yes, how has it helped you to control your disease

- Positively
- Negatively
- No change

3. What times of the day do you test the urine or blood

E. 1 (a) Have you ever been given a diet plan in the hospital? Yes/No

(b) If yes, do you follow it closely? Yes/No

(c) If yes, what do you always avoid?

- Vegetable containing foods
- Proteins
- Sugary (sweet) foods and carbohydrates.

(d) Why do you have to follow a diet plan?

- Because the doctor told me to do so.
- It helps me to control my disease
- I have no idea

F. 1(a) Do your friends and colleagues at work know that you are diabetic? Yes/No

(b) Do you carry anything that indicates you are a diabetic fellow? Yes/No

2. (a) Do you cut your nails? Yes/No

If yes, how?

- Cut them just broadly

- Cut them down to the extreme

3. Do you have any wound? Yes/No

If yes, for how long?.....

4. (a) Do you smoke/drink alcohol? Yes/No

(b) If yes, how many sticks/bottles in a day/.....

5. (a) Do you have any special difficulties? Yes/No

(b) If yes, please what sort (specify)?.....

.....

6. Is there any area in the management of diabetes you feel you need more information (specify).....

.....

7. Is there anything you want to comment on?.....

.....

.....

This is the end of the survey and thank you very much for your cooperation

## APPENDIX II

Diabetic Patients from the ABUTH, whose Blood samples were used in the Study of Serum Chlorpropamide Levels.

| S/No. | NAME | HOSPITAL No. | DAILY DOSE(mg) | SERUM LEVEL(mg/L) |
|---|---|---|---|---|
| 1. | R. A. | 504867 | 500 | 170.04 |
| 2. | S. U. | 23089 | 500 | 156.26 |
| 3. | I. U. | 850493 | 250 | 95.41 |
| 4. | O. A. | 770475 | 500 | 168.75 |
| 5. | H. S. | 896061 | 250 | 113.05 |
| 6. | M. A. | 564139 | 250 | 106.31 |
| 7. | M. A. | 720590 | 250 | 152.59 |
| 8. | S. U. | 156078 | 500 | 49.03 |
| 9. | R. A. | 803944 | 250 | 106.91 |
| 10. | M. S. | 378166 | 500 | 149.72 |
| 11. | A. A. | 581165 | 250 | 117.81 |
| 12. | A. T. | 781523 | 500 | 176.28 |
| 13. | K. T. | 922858 | 500 | 83.73 |
| 14. | J. M. | 371675 | 375 | 76.38 |
| 15. | A. A | 433634 | 250 | 68.46 |
| 16. | Z. A. | 421936 | 250 | 182.63 |
| 17. | H. M. | 416074 | 500 | 154.08 |
| 18. | M. A. | 927127 | 500 | 97.39 |
| 19. | A. D. | 766278 | 500 | 50.02 |
| 20. | L. N. | 868546 | 500 | 152.70 |

| S/No. | NAME | HOSPITAL No. | DAILY DOSE(mg) | SERUM LEVEL(mg/L) |
|---|---|---|---|---|
| 21. | O. M. | 59283 | 500 | 397.69 |
| 22. | D. H. | 746851 | 500 | 126.19 |
| 23. | A. J. | 936117 | 250 | 153.89 |
| 24. | U. I. | 738043 | 500 | 158.05 |
| 25. | A. Z. | 738967 | 250 | 81.34 |
| 26. | H. I. | 668700 | 500 | 432.44 |
| 27. | A. G. | 128174 | 500 | 104.53 |
| 28. | U. A. | 927350 | 250 | 147.35 |
| 29. | B. MK. | 544394 | 500 | 163.21 |
| 30. | A. B. | 344381 | 250 | 67.66 |
| 31. | G. M. | 829718 | 500 | 163.80 |
| 32. | B. H. | 511714 | 500 | 187.38 |
| 33. | L. G. | 141849 | 500 | 197.10 |
| 34. | G. B. | 710809 | 250 | 71.03 |
| 35. | K. O. | 652165 | 250 | 66.67 |
| 36. | H. T. | 044906 | 500 | 130.14 |
| 37. | Y. A. | 789228 | 500 | 142.40 |
| 38. | S. G. | 800954 | 250 | 49.50 |
| 39. | D. Y. | 568537 | 250 | 69.27 |
| 40. | A. E. | 928429 | 250 | 134.89 |
| 41. | F. D. | 877868 | 500 | 66.67 |
| 42. | E. Y. | 782761 | 500 | 133.58 |
| 43. | K. W. | 18042 | 500 | 166.62 |
| 44. | L. B. | 608288 | 250 | 119.72 |
| 45. | M. U. | 884428 | 250 | 85.69 |
| 46. | M . M. | 059667 | 500 | 128.43 |
| 47. | S. A. | 766394 | 250 | 94.00 |
| 48. | E. K. | 171594 | 250 | 137.53 |
| 49. | K. B. | 132168 | 250 | 98.94 |
| 50. | E. C. | 175854 | 500 | 120.91 |
| 51. | M. D. | 896314 | 250 | 163.06 |

| S/No. | NAME | HOSPITAL No. | DAILY DOSE (mg) | SERUM LEVEL (mg/L) |
|---|---|---|---|---|
| 52. | H. M. | 168696 | 250 | 99.54 |
| 53. | M. A. | 802299 | 250 | 85.49 |
| 54. | A. A. | 830506 | 250 | 151.58 |

3.7% - Toxic
79.6% - Normal Therapeutic Range
16.7% - Subtherapeutic

100%

# APPENDIX III

## GLOSSARY

The following is a glossary of medical terms used in Appendix III.

| | |
|---|---|
| Aetiology | Cause of a disease |
| Allergy | An altered or exaggerated reaction to various foreign substances or physical agents which may be harmless to some other people. |
| Atrophy | Wasting |
| Bronchitis | Disease of the air pathways. It may be sudden or of long standing. Signs include coughing, breathlessness and the production of much phlegm. |
| Calorie | Energy (available as fuel or heat) found in food. |
| Carbohydrate | Starchy foods like yam, bread, sugars, potatoes and the like. It is usually eaten to provide energy. |

| | |
|---|---|
| Chronic | Lasting or lingering for a long time. |
| Complication | Another disease or sequelae arising in the course of one disease. |
| Fibre | A thread-like structure which is a component of many foods, e.g. vegetables and starchy foods. |
| Gangrene | Death (of part) of tissue or organ of the body. It is usually the result of inadequate blood supply but it may also be due to direct injury or infection. |
| Gestation | Period of pregnancy |
| Hyperglycaemia | Excessive sugar in the blood. |
| Hypertension | High blood pressure. |
| Hypoglycaemia | Too low a sugar in the blood. It is usally accompanied by symptoms like sweating, trembling and fainting. |

| | |
|---|---|
| Insulin | A liquid substance produced by an organ called *pancreas*, located in the abdomen which is released into the blood stream. It regulates the amount of sugar in the blood. It is also prepared commercially in various forms from cows and pigs and bacteria. |
| Metabolism | Series of changes in ingested food that take place in the body through which their valuables are released to maintain life and their unwanted parts gotten rid of. |
| Microorganism | A very small living thing, e.g. germs. |
| Obesity | Overweight. |
| Palor | Paleness |
| Quadrant | One quarter of a whole. |
| Therapy | Treatment. |

# APPENDIX III

## LIFE WITH DIABETES

1.0 INTRODUCTION:

This booklet is an information handbook for patients suffering from diabetes mellitus. It provides, in a nutshell, the essential information about diabetes mellitus, its management with diet, tablets and insulin in form of injection, as well as other precautionary measures that help you to keep a good control of your blood sugar level. It only supplements but does not substitute the instructions and pieces of advice that you receive from your doctor.

1.1 What is Diabetes Mellitus:

Diabetes mellitus is a progressive chronic disease which is characterised by an abnormally high level of sugar in the blood, and the excretion of that sugar in the urine. The basic defect is an absolute or relative lack of insulin which leads to abnormalities of metabolism not only of carbohydrate but also of other dietary components such as fat and protein. Insulin a

substance (hormone) secreted by the pancreas, is necessary for the regulation of blood sugar level in the body. The pancreas, a gland which lies behind the stomach, is not producing enough insulin, or in some cases none is produced at all. If there is little or no insulin the sugar from (the digested) foods cannot be stored by the body or used for energy and therefore the amount of sugar in the blood rises.

## 1.2 Causes of Diabetes Mellitus

The causes of diabetes mellitus are complicated and still not clearly known. In the great majority of cases there seems to be no single cause of the condition. This is not surprising since diabetes itself is not a discrete and specific disorder, in which a person either has or does not have, rather it is a departure from normality.

In some few cases where the pancreas is removed or diseased there is a simple and direct explanation for the diabetes, but in majority of cases possible causes may be suggested. For example, some viral infections and a process where by the body fights its own cells (autoimmunity)

can result to diabetes. Overweight (obese) can also lead to diabetes. Inheritance (genetic factor) as well has a role to play in the aetiology of diabetes. Operations and some injuries particularly those involving the pancreas can cause diabetes mellitus.

1.3 Types of Diabetes Mellitus:

There are two fairly distinct types of diabetes mellitus. They are:

a) Type I Diabetes Mellitus:- This is also called growth-onset-type or insulin dependent diabetes mellitus (IDDM). It is usually more severe and mostly, though not always, occurs in childhood and young adults. It requires daily injections of insulin in order to maintain life and prevent serious problems. The pancreas of such patients produces very little amount of insulin or even none at all. If left untreated it can lead to severe body wasting and unconciousness (coma).

b) Type II Diabetes Mellitus: - This is also called the maturity-onset type or Non-Insulin Dependent Diabetes Mellitus (NIDDM). Onset is usually gradual. It occurs mostly in the

middle aged and elderly. Very few cases may be seen in some juveniles and young adults.

In this type, there is a relative insulin deficiency or the body does not respond adequately to the insulin produced. Insulin is rarely used for the management of this type of diabetes. It is instead managed by a combination of a proper diet, weight reduction in some cases, and tablets.

Other forms of diabetes mellitus include that which occurs during pregnancy in some women. This sometimes disappears after childbirth. It is often called gestational diabetes. Diabetes can also occur as a result of damage to the pancreas either by some chemicals or medicines or due to disease of the pancreas or some other glands. Some people have their blood sugar level between normal and diabetic levels and usually have an impaired glucose tolerance. Such people are said to be latent diabetics and have a high risk of developing it.

1.4 Signs and Symptoms of Diabetes:

The main signs and symptoms of diabetes mellitus include increase frequency in urination which is usually of large volume. This is

accompanied by excessive drinking of water or other fluids to quench the abnormal feeling of thirst, general tiredness, loss of weight and strength, failure to grow and thrive in children, blurring of vision, itching especially of the vulva, infections of the skin and sometimess numbness or pain in the feet.

The onset of the symptoms is more rapid in the case of the Type I diabetes. This is usually measured in days or few weeks. In the case of Type II diabetes however, the signs are milder and more gradual in onset. In general the symptoms in both cases may become intensified or apparently precipitated by infection.

2.0 Management of Diabetes:

So far there is no known cure for diabetes mellitus. The main aim of treatment is to make the patient well and keep him/her so. This is acheived by controlling the blood sugar level within the normal range, restoration of normal weight in adults and maintenance of normal growth and development in children.

The Type I diabetes is treated with daily injections of insulin, well planned diet that restricts total calorie intake but meets the patients requirements, and regular exercise. The amount of insulin needed to keep the blood sugar level as near normal as possible will vary from person to person and will need to be adjusted for each person from time to time.

The Type II diabetes is usually managed with a diet plan, exercise and weight reduction in case of the obese, and when all these fail to control the blood sugar level, the oral hypoglyceamic agents in form of tablets are used.

2.1 Diet Control:-

Diet is a very important factor in the successful management of diabetes mellitus in general. Hence all diabetics should be on a planned diet. When someone without diabetes eats a meal, an organ in the body called the pancreas (which lies behind the stomach) produces just the right amount of insulin to deal with the food eaten. The carbohydrates (sugar and starchy foods) are digested and move into the blood as sugar. The insulin then allows this sugar to be used for energy or stored in the body. In this way the level of blood sugar is kept normal.

When you suffer from diabetes mellitus you must keep the level of sugar in the blood as near normal as possible. To do this, it is neccessary to balance the amount of carbohydrate (or food) you eat with the amount of insulin you are injecting or that which your body produces through administration of tablets orally. It is important to eat the kinds of carbohydrate that will not make the blood sugar rise too quickly. You must therefore STOP taking the sugary kinds of carbohydrate such as sugar itself, sugar-cane, sweets, chocolates, jam, sweet biscuits, tinned fruits in syrup, and sweet drinks eg lucozade,

pepsi, coca-cola. ribena, kunu, fruit juice to which sugar has been added. Wines and liquors should also be stopped. You should eat the starchy kinds of carbohydrates particularly those with alot of fibre, for example whole meal bread, whole grain cereals. Fibre is very important in the diabetic diet as it slows down the movemant of sugar in the blood, hence helps in the control of diabetes. It is for this reason that diabetics are advised to take alot of vegetable foods.

It is important that diabetics eat regularly. If you eat too much carbohydrate, the blood sugar may go too high, if you eat too little you may have low blood sugar - what is usually referred to as hypoglycaemia. It is however advisable for an overweight diabetic to have a low carbohydrate and low-energy diet to which she/he sticks to for a long period of time. By avoiding exess fat you may lessen the risk of damaging your blood vessels and it will also protect you from putting on too much weight.

The calorie intake in a diabetic should be distributed so as to match the pattern of insulin activity. A common way of doing this is to take 2 parts out of seven of the total food for the day at each of three meals and the remaining 1 part

when going to sleep. This may however be modified to meet individual demand, for example, a snack at mid-afternoon may relieve the low blood sugar level (hypoglycaemia) that often occurs at this time in patients taking the intermediate-acting insulin.

As a diabetic 40% of the total calorie of your diet should be carbohydrate like *acca*, wheat and other cereals, 20% protein like soya beans, egg, milk, meat, fish and the remaining should be from unsaturated, low-cholesterol fat like palm-oil and other vegetable oils. These percentages may be translated into weights of the actual items of diet by means of an exchange system. In this system, an exchange unit of food comprises of fixed weights of carbohydrates, protein and fat or a combination thereof. By so doing, a diabetic can then choose an appropriate dietary item from a list of exchanges which add up to the allowances for that particular meal. Details of how to plan your diet are provided in the next few pages.

## CONSTRUCTION OF A DIABETIC DIET

The first step in constructing a diabetic diet is to assess the dietary energy requirement (measured as calories). Calories obtained from food constituents are estimated as follows:

a) Carbohydrate - 4 cal/gm.

b) Protein - 4 cal/1gm.

c) Fat - 9 cal/1gm.

Total energy requirement for a non-obese diabetic is the same as that for normal individual of the same age, sex, height and activity. For the obese diabetic, the food should be designed to promote weight loss while the diet for a growing child should be such that it promotes normal growth. Table A below shows calories per kilogram of ideal body weight considering activity level and present weight.

Depending on your weight you will be able to calculate the details of your diet based on the energy needs. For example: if you are an adult with normal weight say 69kg and moderate activity, your total energy need will be 35x69 = 2415 Kcal.

Table A

Total daily energy requirement (per kilogram) of individuals based on their weight and activity.

| Activity --> | Sedentary | Moderate Activity | High Activity |
|---|---|---|---|
| Weight | Kcal | Kcal | Kcal |
| Over weight | 20-25 | 30 | 35 |
| Normal " | 30 | 35 | 40 |
| Under " | 35 | 40 | 45-50 |

From this amount 40% is to come from carbohydrate,

i.e $\frac{2415 \times 40}{100} = 966$ Kcal.

Another 40% of the total energy is to come from fat, which is also 966 Kcal.

The remaining 20% is best obtained from protein, thus; $\frac{2415 \times 20}{100} = 483$ Kcal .

Converting these energies to weights is done as follows:-

(a) Total Carbohydrate: $\frac{966}{4}$ = 241.5gm ≅ 242 gm.

(b) Total fat = $\frac{966}{9}$ = 107.3gm ≅ 107 gm.

(c) Total Protein = $\frac{483}{4}$ = 120.75gm ≅ 121gm.

These total weights are then spread throughout the day into about 7 portions. During each of the three main meals 2 portions are consumed and the last portion taken at bed-time.

**Food Exchange system**

Food exchange system is a simplified system of diet calculation for diabetics using various food groups. The food groups in the system are useful to you in that you can alter your dietary patterns according to preference without affecting the total calorie intake. It also allows you to tailor your dietary needs to fit local staples. Table B below gives the average protein, fat and carbohydrate as well as energy value for each exchange group.

Table B

Nutrient composition of Food exchanges

| Group | Amount | Weight (gm) | Protein (gm) | Fat (gm) | Carbohydrate- (gm) | Energy (Kcal) |
|---|---|---|---|---|---|---|
| Milk, whole | Mpt(8oz=240ml) | 240 | 8 | 10 | 12 | 170 |
| V/table Grp,A | As desired | - | - | - | - | - |
| " " B | ½ cup | 100 | 2 | - | 7 | 35 |
| Fruit Exchanges | | | | | | |
| (cereal) | Varies | - | - | - | 10 | 40 |
| Bread Exchanges | " | - | 2 | - | 15 | 70 |
| Meat Exchanges | 1oz | 30 | 7 | 5 | - | 75 |
| Fat Exchanges | 1tsp | 5 | - | 5 | - | 45 |

The following tables give the lists of some of the common foods included in each exchange. (Modified and adapted from Mitchell H.S, Rynbergen H.J. Anderson L. and Dibble M.V. Nutrition in Health and Disease. 6th Edn. J.B. Lippincott, Philadelphia. 1976. pp 330-331).

Foods that need not be measured:

| | | |
|---|---|---|
| Tea | Vinegar | Pepper |
| Coffee | Garlic | Ginger and other spices. |
| Saccharin | Onion | |
| Pepper | Salt (assuming normal blood pressure) | |

List 1

Milk Exchanges: One exchange of milk contains 8gm of protein, 10gm of fat, 12gm of carbohydrate and 170 calories. This list shows the different types of milk to use for one exchange.

| Type of milk | Amount to use |
|---|---|
| Whole milk | 1 cup (240ml or 8oz) |
| Evaporated milk | ½ cup. |
| Powdered whole milk | ¼ cup. |

One type of milk may be used instead of another.

List 2

Vegetable Exhanges Group A

Group A contains very little protein, carbohydrate or calories 1 cup at a time may be used without counting it. The types include the following:

Cabbage

Eggplant (garden egg)

Cucumbers

Lettuce

Okro

Spinach

Pepper (green)

Tomatoes

List 3

Vegetable Exchanges Group B

Each exchange contains 2 gm of protein, 7gm of carbohydrate and 35 calories. ½ cup of vegetable equals 1 exchange. Examples include:

Carrots

Onions

Greenpeas

Pumpkin

Turnip.

## List 4

Fruit Exchanges

One exchange of fruit contains 10 gm of carbohydrate and 40 calories. This list shows the different amounts of fruits to use for one fruit exchange:

| Fruit | Amount to use |
|---|---|
| Banana | ½ small |
| strawberries | 1 cup |
| Dates | 2 |
| Figs (fresh) | 2 large |
| Figs (dried) | 2 small |
| Grapes fruit | ½ small |
| Grapes | 12 |
| Mango | ½ small |
| Orange | 1 small |
| Orange juice | ½ cup |
| Pineapple | ½ cup (4oz) |
| Tangerine | 1 large |
| Water melon | 1 cup (8oz) |

List 5

Bread Exchanges

One exchange contains 2gm of protein, 15gm of carbohydrate and 70 calories. This list shows the defferent amounts of food to use for one bread exchange.

| Bread, cereal, etc. | Amount to use |
|---|---|
| Bread/Biscuit roll/cornbread | 1 slice |
| Cereals cooked | ½ cup |
| Cereal dry, flake, puff types | ¾ cup |
| Rice, cooked | ⅓ cup |
| Macaroni, spaghetti, cooked | ⅓ cup |
| Flour | 2½ tablespoon |
| Vegetables: | |
| Beans and peas, dried, cooked | ⅓ cup |
| Baked beans | ¼ ✓ |
| Corn (Maize/Guinea corn | ⅓ cup |
| Corn on the cob | 1 small |
| Popcorn | 1 cup |
| Irish potatoes | 1 small |
| Sweet potatoes or yams | ¼ cup (2oz) |

N B

These foods are measured carefully because they contain significant amount of carbohydrate.

List 6

Meat Exchanges

One meat exchange contains 7gm of protein, 5gm of fat and 75 calories. This list shows some of the different amounts of foods to use for one meat exchange:

| Meat | Amount of use |
|---|---|
| Meat and Poultry (medium fat) | 1oz cooked |
| (beef, lamb, liver, chicken etc) | |
| Egg | 1 |
| Fish: Fresh or Frozen | 1oz |
| Salmon, crab, lobster | ¼ cup (2oz) |
| Shrimp, oysters | 5 small |
| Sardines | 3 medium |
| Cottage cheese, dry | ¼ cup |

List 7

Fat Exchanges

One fat exchange contains 5 gm of fat and 45 calories. This list shows some the different foods to use for one fat exchange:

| Fat | Amount to use |
|---|---|
| Butter or Margarine | 1 tsp |
| Oil or Cooking fat | 1 tsp |
| Cream or Cream Cheese | 1 tbsp |
| (All) Nuts | 6 small |
| Olives | 5 small |

Note: tbsp = tablespoon; tsp = teaspoons

Although this exchange system and any other reflect average and not specific energy and nutrient values, the therapeutic successes that result when the values are used to calculate diet paterns demonstrate that the method is accurate enough to serve this purpose. Example of the use of the Exchange system to calculate dietary pattern is demonstrated in the 2 tables below:

Table C - 1:

Nutrient composition of 1500 calories Diabetic Diet

| Food Exchanges | AMOUNT No | AMOUNT Household measure | Protein (gm) | Fat (gm) | Carbohydrate (gm) | Energy (cal) |
|---|---|---|---|---|---|---|
| Milk | 2 | 1pint(500ml) | 16 | 20 | 24 | 340 |
| Veg, Group A | As desired | - | - | - | - | - |
| Veg, Group B | 2 | 1 cup | 4 | - | 14 | 70 |
| Fruit | 4 | varies | - | - | 40 | 160 |
| Bread (cereal) | 5 | " | 10 | - | 75 | 350 |
| Meat | 6 | 6oz | 42 | 30 | - | 450 |
| Fat | 3 | 3tsp | - | 15 | - | 135 |
| Totals | | | 72 | 65 | 153 | 1505 |

Table C - 2.

1500 Calories Diabetic Pattern (75gm Protein, 65gm fat and 150gm carbohydrate)

| Meal | Food Exchanges | Amount | Protein (gm) | Fat (gm) | Carbohydrate (gm) | Energy (cal) |
|---|---|---|---|---|---|---|
| Morning | | | | | | |
| Milk | 1 | 8oz | 8 | 10 | 12 | 170 |
| Fruit | 1 | varies | - | - | 10 | 40 |
| Meat | 1 | 1oz | 7 | 5 | - | 75 |
| Cereal | 1 | varies | 2 | - | 15 | 70 |
| Fat | 1 | 1tsp | - | 5 | - | 45 |
| Total | | | 72 | 20 | 37 | 400 |
| Lunch | | | | | | |
| Meat | 2 | 2oz | 14 | 10 | - | 150 |
| Cereal(bread) | 2 | varies | 4 | - | 30 | 140 |
| Fruit | 1 | varies | - | - | 10 | 40 |
| Fat | 1 | 1tsp | - | 5 | - | 45 |
| Total | | | 18 | 15 | 40 | 375 |
| Supper | | | | | | |
| Meat | 2 | 3oz | 21 | 15 | - | 225 |
| Cereal(bread) | 2 | varies | 4 | - | 30 | 140 |
| Vegetable B, | 2 | 1 cup | 4 | - | 14 | 70 |
| Fruit | 1 | varies | - | - | 10 | 40 |
| Fat | 1 | 1tsp | - | 5 | - | 45 |
| Total | | | 29 | 20 | 54 | 520 |
| Bedtime | | ≈ 250ml) | | | | |
| Milk | 1 | 8oz | 8 | 10 | 12 | 170 |
| Fruit | 1 | varies | - | - | 10 | 40 |
| Total | | | 8 | 10 | 22 | 210 |
| Overall TOTALS | | | 72 | 65 | 153 | 1505 |

2.2. Exercise:

Regular excercise helps in the control of diabetes this is particularly important to the obese diabetics. Extra food may be needed before or after the exercise depending on the individuals blood sugar level. Extra food may not be neccessary however in the case of the obese diabetic.

2.3 Drugs:

Drugs used in the management of diabetes are either the oral hypoglcaemic agents in the form of tablets or the different types of insulin for injection.

The tablets are either of the sulfonylurea group or the biguanides. Any of these group of tablets or both may obviate the need for injection. Majority of the patients with the Type II diabetes are placed on either or both of these tablets. Examples of the sulfonylurea agents include chlorpropamide (Diabinese®), glibenclamide (Daonil®), among many others. As for the biguanides metformin is tha only commonly used one. The sulphonylurea tablets stimulate the

release of insulin and increase tissue sensitivity to its actions. The biguanides on the other hand may reduce appetite and intestinal absorption (of digested foods). Thus they are good for the obese diabetics. Both group of drugs are used in the management of the type II diabetes whose blood sugar cannot be controlled by diet alone. However they are not effective in the presence of severe infection. When such happens the tablet is replaced with insulin injection. A patient who is on any of these tablets may experience some abdominal pain, nausea, vomiting, and some feeling of weakness. These however may be overcome by taking the tablets in a full stomach. It is advisable for a patient on tablets to avoid alcohol as this may adversely affect their activity.

2.4 Insulin Therapy:

Management of diabetes with insulin is indicated in the Type I diabetes or in severe infection in both Types I and II diabetes, or in a diabetic who is to undergo surgical operation, or in an illness or during pregnancy. There are many different types and makes of insulin available.

The type of insulin you are given is chosen for you by the doctor to suit your needs. It is very important that you know the type you use, and you keep to the same brand as different brands may vary in their action. You will probably need two injections of insulin everyday - one before breakfast and the other before the evening food (supper). Insulin therapy is usually started in the hospital. When the patient has been reasonably well controlled, s/he is taught how to inject him/herself, as s/he is going to be on a 'permanent insulin regimen'.

A specific syringe and needle are used for the injection. These (special) syringe and needle can be used several times before they are changed. It is advisable for the patient to sterilise the syringe and needle at least once every other day. You do this as follow:

1. Separate plunger from syringe
2. Place the syringe, plunger and needle in a small, clean pot or bowl, and add clean water to cover them, then cover the container.
3. Put this on fire and bring to boil; allow it to continue boiling for about 10 -15 minutes.
4. Remove them and put in a clean container and store in a cool place.

It is important that at each time of injection, the hands are thoroughly washed. The insulin bottle should be rubbed between palms (in the case of the cloudy type) for proper mixing. When filling the syringe, make sure that you get rid of the air bubbles by gently tapping the side of the syringe. This makes them to rise to the tip of the side of the syringe. From there they are expelled by pushing the plunger upward. if the bubbles persist, expell all insulin back into the bottle and withdraw again.

It is important that the injection sites are changed from day to day. This is because using the same site repeatedly can produce thickening at the site and interfere with insulin absorption. Suitable sites for insulin injection are:
-the upper, outer thigh and the front of the thigh
-the upper, outer arm; -the abdomen or
-the upper, outer quadrant of the buttocks.

The dose of insulin must be correct before injecting it. The needle should not be touched and there is no need to swab the injection site. At the time of injecting the drug, the needle should be pushed into the skin vertically. The plunger is to then be withdrawn slightly, just to be sure that it is not in a vein, and then pressed firmly

to release all the insulin in the syringe after which the needle is withdrawn. It is advisable to mark the syringe clearly, eg with a sticky tape or water proof ink, to indicate the volume of insulin required. A patient's relative or a close friend should learn how to give the injection so that s/he can offer a helping hand to the patient in times of need. All insulin takers should have spare syringe and needle in case of breakage.

Common side effects associated with the use of insulin include allergy, fat atrophy at the injection site, and hypoglyceamia (ie low blood sugar).

Hypoglyceamia is when there is too little sugar in the blood. It may be due to any of following:

-taking too much insulin

-being late for a meal, missed a meal or eating too little.

-after having performed some unusual exercise or hard work.

-Hypoglyceamia can also occur without any obvious reason.

Common signs of hypoglyceamia are sweating, intense feeling of hunger, pallor, trembling, blurring of vision, slurring of speech,

irritability, difficulty in concentration.

The remedy to hypoglyceamia is to take some easily digested sugar, for example, a table spoonful of honey or jam, 2 or more heaped teaspoonful of sugar or glucose or about half a tumbler of lucozade or kunu, some dates or sweets. It is important for patients (on insulin) to always carry some form of sugar with them, and take it immediately they start feeling the signs of hypoglyceamia. Do not wait to see if it will pass off as untreated hypoglyceamia can lead to unconciousness. It is equally important that you try to discover the reason for your hypoglyceamia, so that you can aviod it happening in the future. If you have several 'hypos' at the same time of the day, it might mean that your insulin dose needs changing, and it will be advised that you see your Doctor. It is important also that your relatives, friend(s) and at least one person you work with know that you are diabetic on insulin, so that they can help treat these insulin reactions when neccessary.

Insulin preparation should ideally be kept in a refridgerator, not in the freezer compartment, or, where a fridge is not available, in a cool pot of water placed on a sand.

It is advisable that a diabetic patient on insulin avoids alcohol as it interferes with the action of the drug.

2.5 Hyperglyceamia and illness.

Hyperglyceamia means a high blood sugar level. The cause of this may be :

I. You are not keeping to your diet and you are eating too much carbohydrate, or

II You are taking too little insulin or you do not take your tablets regularly, or

III You have an infection or illness.

If you do not take your medication and you allow your blood sugar to remain high, other signs and symptoms occur. They include an increased thirst, frequency in urination, a feeling of sickness, drowsiness and stomach pain. If this is left untreated it can result to unconciousness.

During illness, it is important never to stop taking your medicine particulary those on insulin. Do not make the mistake that if you are ill and cannot eat your food well, then you should not take your insulin. Infact very often during an illness or infection, the blood sugar rises and

you will need more insulin. (Those on the tablets may sometimes have to be changed to the injection) If you cannot eat your normal solid food, then take the equivalent in liquid form e.g. some milk, lucozade, 'Kunu', 'fura'. Remember that anytime you feel sick, do not hesitate to go the hospital.

3.0 Monitoring blood sugar control:

There are various ways by which to monitor the control of diabetes. They include:

3.1. Questioning: e.g.:-

How many days you are sick?

How many 'hypos' in a day or week?

Any morning headaches or lethargy?

How about the number of times of urination and/or thirst during a night?

If the answers to these questions are all negative or tend towards that, then you should know that you are likely to be in good control. However this is not confirmatory.

3.2 Regular weighing :- If an obese patient is reducing weight or if an insulin dependent patient maintains his/her weight then the control is good. This is also not confirmatory.

3.3 Urine testing:- It is important that all diabetics carry out this. Urine test should generally be made either before meals, at bed time, or anytime when signs suggest hypoglyceamia or hyperglyceamia. Urine testing is important in

that it helps you to check the control and make any neccessary alteration in food habits. It also serves as a constant remainder of the necessity of following your doctor's instruction. Above all urine testing is done for safety so that you do not run into more serious difficulties.

There are several tests for determining the presence of sugar in the urine. The tests mentioned below are easy to perform at home.

Method one: Clinistix® or Tes-tape® is used. Clinistix usually comes in strips in a dark bottle container, whereas Tes-tape comes in a form of a tape in a special container. Whichever one you use, make sure you follow the manufacturer's instructions. A strip from the bottle of clinistix is removed and dipped into the urine specimen, after which the colour obtained on the moistened strip is compared with the colour chart on the container. When tes-tape is used a tape about the length of an index finger is torn from the container and gently dipped into the urine specimen. Whatever colour is obtained on the tape is compared with the colour chart on the container.

Method Two: Here clinitest in form of tablets is used. A tablet is dropped into the specimen and the colour is read immediately after the 'boiling' has ceased.

Whichever reagent you use for the urine testing, it is important that you store it in a cool, dry ,and dark place, and, away from children.

4.0. Other Precautions;

4.1. Foot care:

After people have had diabetes for some time they may experience a slight loss of sensation in their feet due to poor blood supply to the feet. It is therefore important that you take good care of your feet. To avoid problems remember the following:

a) Keep your feet and legs clean and dry always. When washing them make sure the water is not too hot.

b) Do not put your legs too close to fire.

c) Do not walk barefoot. Wear well fitting shoes - i.e. not too tight for your feet,

d) Be very careful when cutting your (toe) nails. If you notice any problems report to the docter at once.

e) If you cannot see well, ask a spouse, friend or a nurse to check the feet for you regularly.

4.2. Smoking and Diabetes:

Smoking is harmful to the body even when not diabetic. If you are diabetic and you smoke, there is an increased risk of causing damage to your blood vessels. This can eventually lead to diabetic complications. There is also the risk of having bronchitis and even lung cancer."

4.3. Driving and Travelling

Diabetics on tablets or diet alone can generally drive a car without taking any special precautions. However the case is different for those diabetics on insulin. If you are on insulin, do not drive unless you can recognise the signs of hypoglycaemia and to prevent and treat it. Diabetics on insulin should carry some means of identification especially those who drive. They

should also always carry some sugar or other form of easily digested carbohydrate such as sweet, bread, sweet potato, sugar cane etc, with them and in the car. Remember not to miss meals. If neccessary, stop for a meal (or a snack). Never take insulin injection and miss a meal. When driving and you feel the sign of hypoglyceamia coming on, stop the car, switch off the ignition, leave the driver's seat, then take some food (or sugar) and wait outside the car for some time until the symptoms disappear. When travelling, remember to carry all equipments in your hand bag or luggage. This will include your medicines (tablets or injectable), urine sugar testing kit, and some sugar or form of it (to be taken when necessary). It will be good for you if someone around knows that you are diabetic especially if you are on insulin.

4.4 General Hygiene:

Keeping yourself always clean and fit especially the injection sites (if on insulin) cannot be overemphasized. Be careful when cutting your nails (on fingers and toes). Do not cut them down to the extreme as this may create some

problems for you. If you have any cuts and grazes, make sure that they are kept clean and adequately protected. Try to wear clothes that do not rub or restrict blood circulation.

4.5 Pregnancy:

For your own good, try and get your diabetes well controlled before you are pregnant and moreso throughout the gestation period. Inform your doctor as soon as you realise that you are pregnant. This is important because there may be changes in your medication. Also problems can be avioded throughout the pregnancy and you have a safe delivery with a healthy baby.

5.0 Long-term Problems of Diabetes:

The long-term complications of diabetes are usually as a result of certain changes in the nerves and the blood vessels. This gradually affects such organs as the eyes, kidneys, heart, feet and other important organs in the body. These complications are most common in those patients with poor blood glucose control, that is those having blood sugar almost always higher than

normal. Keeping good control of the blood sugar always, can help to prevent or at least delay complications. Diabetes complications manifest in the form of poor eye sight, which can eventually lead to blindness, heart attack, kidney failure, numbness or pain in the legs and foot infection which can lead to gangrene. Hypertension may also present itself as a result of inefficient function of the heart and the kidneys. Urinary track infection is also a common feature in uncontrolled diabetes. This is because the excess sugar that is passed in the urine serves as a good medium for the growth of microorganisms along the urinary pathway.

Complications in diabetes take many years to develop but once they have occured they are usually difficult to reverse. Prevention is better than cure, so keep good control of your blood sugar by following the diet plan and your medication in order to reduce, if not prevent, the long-term problems caused by diabetes. Remember that good control of your diabetes not only relieves you of the symptoms and prevents the development of complication, but it also enables you to lead a relatively normal life.

# APPENDIX IV

## RAYUWA DA CUTAR SUKARI.

1,0 Gabatarwa;

Wannan dan littafi takaitaccen bayani ne a kan cutar sukari. Littafin yayi takaitaccen bayani a kan muhimman abubuwan dake tattare da cutar, da yadda za'a iya rage kaifinta ta cin abincin da ya da ce da shan magani da yin allura, da kuma sauran han yoyin da za su taimaka wajen rage yawan sukari a cikin jini. Saidai littafin fa kari ne kurum ga shawarwarin da likitoci suke badawa, ba an yi shi ne domin ya maye gurbin shawarwarinba.

1,1. Menene Cutar Sukari?

Cutar sukari wata matsananciyar rashin lafiya ce wada ake gane ta ta hanyar samun sukari fiye da kima a cikin jinin mutum, da kuma fitar da wannan zakin a yayin da ake fitsari. Babbar illar wannan cuta shine karanci ko kuma ma rashin wani abu a cikin jini da ake kiransa 'Insulin' da turanci, kwata-kwata. Wannan kuma shi ke jawo sukurkucewar lafiyar jiki saboda rashin abinci mai bada karfin jiki da ma masu sa lafiya kamar su bitaminda furotain. Insulin wanda jiki ke fitarwa wajibi ne ga kayyade adadin sukarin da ake dashi a cikin jiki. Wata sa'a, wata tsoka a jiki wadda take a bayan ciki bata fitar da isasshen insulin, a wata sa'ar ma bata ma fitar da ko kalilan, wato idan mutumin yana cutar sukari. Idan babu isasshen insulin ko kuma babu shi kwata-kwata a jiki to sukarin da ake

fitarwa daga abinda aka tace ba zai samu zuwa warinda jiki zai adana shiba, ko kuma a yi amfani dashi domin gina jiki, Soboda haka sai sukarin dake cikin jini ya hauhawa,

1,2 Abubuwan da ke haifar da Cutar Sukari;

Dalilan dake haifar da cutar sukari a dukunkuna suke, kuma ma ba saninsu aka yi ba sosai, Ga mafi yawan masu yin cutar dai ba'a iya samun abu daya daya haifar da ita, Sai dai wannan ba wani abin mamaki bane, tunda cutar sukari ba wata taka maimiyar cuta ce ba, ta yadda za'a iya cewa mutum na fama da ita ko kuma bai da ita, a'a ita dai sabawa ka'ida ce kurum,

A wasu 'yan lokutan inda aka cire tsokan nan ta bayan ciki ko kuma cuta ta ci karfinta to a nan kai waye an samu dalilin da ya haifar da cutar sukari, Amma a mafi yawan lokuta, sai dai ayi mamunga a ce ga dalilin cutar, Alal misali wasu 'yan halittu masu sa cututtuka, da kuma wasu 'yan tsiton gurabu na jiki da jiki yake yaka da kansa suna iya haifar da cutar sukari, Hakama matsananciyar kiba ma na iya sa wannan cutar ta sukari, Hakanan kuma ana gadon ita wannan cutar, Hakannan ma wajen yi wa mutum aiki (Tiyata) da kuma raunuka musamman wadanda suka tabo sashen jikin nan na bayan ciki duk suna haifar da cutar sukari,

1,3 Ire-iren cutar sukari;

Akwai manyan nau'o'in cutar sukari guda biyu, Sune;-

a) Ta farko ita ce wadda ake kira mai bunkasa, A mafi yawan lokuta ita ce tafi tsanani, Ana samunta a lokacin yarintaka da kuma na kuruciya, Tana bukatan ayi allura da insulin a ko wacce rana, idan har ana son a rayu a kuma kauce wa shiga halin ni 'yasu, Tsokar mai wannan cutar wadda ke bayan

ciki bata tatso insulin da dama, a wata sa'a ma bata fito da kome, Idan ba'a yi maganin cutar ba, to zata raunana jikita kuma sa a kai halin da marar lafiyar zai fita daga hayyacinsa da kuma hankalinsa,

b) Na'u'in cutar na biyu shine wanda ake kira kammalalle ko kuma wanda bai amfani da insulin, A mafi yawan lokoci kammalallar cutar sukarin sannu a hankali take shiga jikin mutum, Kuma anfi samunta a wajen wadanda suka girma (manya) da kuma tsofaffi, Yara da matasa 'yan kadan ne ake samu masu irin wannan cutar, A irin nau'in wannan cutar ba safai ake samun karancin insulin ba, ko kuma a ce jiki bai amfani da insulin da ake tatsowa, ba, Saboda haka ba'a cika yin amfani da insulin ba domin magance irin wannan nau'in cutar, Ita cin abinci na musamman da rage kiba, wata sa'a, da kuma shan kwayoyin magunguna ke maganinta,

Wasu nau'o'in cutar sukari sun hada hadda irin wadda ke aukuwa a lokacin da mace ke da juna-biyu, Wata sa'a irin wannan nau'in sai ya bace da zarar an haihu, Ana ma kiranta da sunan cutar sukari ta masu juna-biyu, Kazalika cutar sukari na iya aukuwa saboda wata illar da ta sami wannan tsoka ta ba/an ciki, ko wani magani, kokuma saboda wata cuta da ita tsokar tayi, ko kuma da wasu sassan jiki sukayi, Adadin matsayin sukari da ke jinin wasu mutane tsaka-tsaki ne tsakanin daidai wa daida da kuma cutar sukari, Kuma a mafi yawan lokuta zakin da ke cikin jininsu ba daidai wa daida yake ba, Ana kiran wadannan mutane da sunan boyayun masu cutar sukari, kuma suna da alama ta kukut ta kamuwa da cutar,

1,4 Alamu da sifofin cutar sukari;

Muhimman alamu da sifofin cutar sukari sune karuwar yawan yin fitsari kuma da fitsari mai yawa, Wannan kuma na haifar da shan ruwa kamar anale, ko kuma wasu abu masu ruwa-ruwa domin a yi maganin jin matsanan ciyar kishirwa, Sauran alamun cutar sune; sanya lalaci da ramewa da kuma rashin karsashi, da tsumburewa da dundumi da jin kaikayi musamman a wajen farji, da kuraje da kuma kaikayi a kafa a wata sa'a,

Fitowar alamun cutar yafi kamari ainun a nau'i na farko, Anan kamun cutar ana auna shine da kwanaki ko makonni, Sai dai walau a nauin cutar na farko ko na biyu, cutar kan kankama ko kuma tana kara ruruwa ta hanyar hayayyafa,

2,0 Yadda za'a magance cutar;

Har ya zuwa yau babu wani maganin rabuwa da cutar sukari, Makasudin yin maganin dai shine do min marar lafiya ya sami sauki, Ana kuwa yin wannan neta hanyar dakatar da hauhawan zakin da ke jikin jinin marar lafiya,

Ana maganin nau'in cutar na farko ta hanyar yin alluran insulin da cin abinci wanda aka tsara domin ya kayyade yawan sukari a cikin jikin mai ciwon, amma kuma zai iya bashi karfi da kuma kuzarin da yake bukata, Adadin yawan insulin din da ake bukata domin ya daidaita sukari da ake so a cikin jini ya zama kadaran-kadahan ya bambanta daga wanan mutum zuwa wancan, haka kuma sai ana yi ana duba shi ga kowane matum akai a kai,

Yawanci ana magance nau'i na biyu na cutar ta hanyar tsara abinci (ya kumshi dukkan abubuwan da jiki ke so amma adadin sukarin sa kadan), da

motsa jiki da kuma rage kiba, Idan duk wadannan sun gaza biyan bukata wajen rage hauhawan sukarin a jini, to sai a sha kwayoyin magunguna,

2,1 Kayyade Abinci;

Abinci muhimmi ne ainun a wajen magance cutar sukari, Saboda haka lalle dukkan masu cutar su kasance suna bin tsarin abinci na musamman, Idan mutumin da bai da cutar sukari ya ci abinci, sai wata tsoka wadda ke bayan ciki ta samo insulin daidai yawan yadda ake bukata ta narkar da abincin da aka ci ta kuma rage yawan abincin daga cikin jini zuwa ga ma'ajin jiki, Abinci masu sa karfi kamar su sukari da sauransu ana nike su, sa'an nan aja su su shiga jini su zama zaki, Sai insulin yasa a rika amfani da sukarin domin samun karfi ko kuma a adana shi a jiki, Ta wannan hanyar ce ake kayyade matsayin yawan sukari a jini ya zama daidai wa daida,

Idan mutum yana da cutar sukari to dole ne ya hana yawan zakin dake cikin jininsa wuce gona da iri, Yadda za'a yi haka kuma shine a daidaita yawan abinci mai bada karfi da insulin din da ake sa wa jiki ko kuma wanda shi da kansa jikin yake yi ta hanyar shan kwayoyin magunguna, Saboda haka wajibine a daina shan ko cin abinci mai zaki alal misali shi sukarin dakansa, da rake, da alewa da madi, da gurasa mai zaki da kayan zaki na gwangwani da lemun zaki irinsu lucozade da coca-cola da fanta da 'ya'yayen itatuwa wadanda akahada da sukari, da kuma da dai sauransu, Dama ita barasa da burkutu haramun ne ko ga mai lafiya (balle kai da baka da lafiya), To in ma ana sha lalle ne a daina, Ya kamata kuma a rika cin abinci masu ganye cikinsu, alal misali burodin da akayi da alkama da ba'a surfe ta ba ko kuma abincin da aka yi da ba'a surfe hatsin ba, ko acca, kowake alayyafo, kozogale tafasa, ko rama da sauransu, Abinci na ganyaye suna amfani matuka

ga mai ciwon sukari, domin suna takaita zaryar sukari a cikin jini, saboda haka suna bada gudummuwa wajen sauwaka ciwon sukari, Wannan ne dalilin da yasa ake ba masu cutar sukari shawarar su rika cin abinci masu ganyaye a ko da yaushe,

Yana da mahimmanci kwarai mai cutar sukari ya rika cin abinci a kan kari, Idan mara lafiyar yaci abinci mai sa karfi da yawa to sukarin da ke cikin jinin sa yana iya hauhawa, idan kuma yaci dan kadan to yana iya yin kantar sukari a cikin jinin - wannan halin ne kuwa masana kimiyya da cututtuka ke kira karancin sukari, Ana ba mai ciwon sukari wanda kuma yake da kiba shawarar da ya rika cin abinci mai bada karfi kadan kuma yayi ta yin haka na lokaci ma tsawo, Idan ka kauce ma abu mai maiko, to zaka tsinaka wajen rage yiwuwar lalata kafofin jininka, wannan kuma zai kare ka daga yin kibar nan marar kan gado,

## HARHADA ABINCIN MAI CIWON SUKARI

Abu na farko wajen hada abincin mai ciwon sukari shine a san kiman karfin da abincin zai bayar wanda awonsa ake kira da turanci 'calories', Calories da ake samu daga nau'uin abinci daban-daban an yi kimansa kamar haka;-

(a) Nau'in abinci mai bada karfi - Calories 4 a ko wane awon nauyi daya,

(b) Nau'in abinci mai gina jiki - Calories 4 a ko wane awon nauyi daya,

(c) Nau'in abinci mai maiko - Calories 9 a ko wane awon nauyi daya,

Yawan kiman karfin da mai ciwon sukari marar kiba yake bukata daidai yake da na mutumin da bashi da wannan ciwon wanda yake shekarunsu daya, tsawonsu daya, kuma irin aikin da suke yi ya kasance kusan daya ne, To amma abincin da mai ciwon sukarin da ya kasance yana da kiba, za'a tsara shi ne yadda zai zama yana rage wannan kibar, Shi kuwa yaro mai ciwon sukari abincinsa zai kasance ya kunshi komai da komai wanda zai sa yaron ya girma cikin koshin lafiya,

Jadawali da aka yi anan kasa ,ya nuna yawan karfin da mutun ke bukata domin samun nauyin jiki wanda yake kadara-kadahan kuma gwargwadon irin aikin da mutumin yake yi,

Jadawali na daya

| Irin Aiki ------> | Daga Zaune | Motsa jiki kadan | Motsa jiki sosai |
|---|---|---|---|
| Nauyi | ( Kimar karfi awon kilocalories ) | | |
| Mai kiba | 20 - 25 | 30 | 35 |
| Mai nauyi daidai | 30 | 35 | 40 |
| Kyamusashe | 35 | 40 | 45 - 50 |

Dangane da irin nauyin jikinka yanzu kana iya yin lissafin abincin da ya dace da kai gwargwadon irin karfin da jikinka ke bukata. Alal misali;- Idan ya kasance nauyinka kadaran-kadahan ne (misali kilogram 69) kuma aikin da ka ke yi baya motsa ma jiki da yawa, to zai kasance cewar kana bukatar yawan karfi kamar haka; 35 x 69 = 2415 kilocalories. A cikin yawan wannan karfin kashi 40 daga cikin 100 za'a same shine daga nau'in abinci mai bada karfi. Lissafin zai kasance kamar haka;

$$\frac{2415 \times 40}{100} = 966 \text{ kiolcalories}$$

Hakanan kuma kashi 40 daga cikin 100 za'a same shine daga nau'in abinci mai maiko, shine shi ma lissafin zai bada 966 kilocalories.

Sauran kashi 20 daga cikin 100 na yawan dukkan karfin za'a same shine daga nau'in abinci mai gina jiki. Shi kuma lissafin zai kasance kamar haka;-

$$\frac{2415 \times 20}{100} = 483 \text{ kilocalories.}$$

Yanzu kaga an sami yawan karfi da kake bukata daga duk nau'oin abincin nan uku. Sai kuma yanzu a yi lissafin juya wannan yawan karfi zuwa nauyin abincin da zaka ci, kamar haka;

(a) Abinci mai bada karfi $\frac{966}{4} = 241,5 = 242gm$

Wannan ya nuna zaka auna nauyin gram 242 kafin ka sami karfi awon kilocalories 966 na binci mai ba da karfi,

(b) Abinci mai maiko $\frac{966}{9} = 107\ gm,$

Shi ma wannan lissafin ya nuna cewa awon nauyin gram 107 ne na binci mai maiko zai bada karfin misalin kilocalories 966,

(c) Abinci mai gina jiki $\frac{483}{4} = 121$

Watau awon nauyin gram 121 ne na abinci mai mai gina jiki zai bada karfin kilocalories 483,

Wadannan nauyin abinci iri-iri da aka yi lissafin su sai a kasa su kashi 7, A ci kashi 2 a lokacin kalaci, kashi biyu da rana, kashi 2 da yamma, Sauran kashi 1 na karshe kuma a ci shi yayin da mutum ya gama komai zai kwanta barci da dare,

Jadawalin musayar abinci yana da amfani wajen lissafin abincin da mai ciwon sukari zai ci, Duk nau'oin abinci an kasa shi kashi daban daban ta yadda mai ciwon sukari zai ji saukin sauya abincinsa a kowane lokaci ba tare da takura ba, Shi wannan kuma zai ba mai ciwon sukari daman cin abynda aka fi samuwa a inda ya kasance da zama, Ga yadda aka kasa duk nau'oin abincin zuwa kashi 7;

| Kashin Abinci | Adadi | Yawan nauyi | Yawan gina jiki | Abinci Maiko | Mai (gram) sa karfi | Yawan karfi (Kilocalories) |
|---|---|---|---|---|---|---|
| Madara | kofi 1 | 240 | 8 | 10 | 12 | 170 |
| Ganyaye kashi 1 | Ba adadi | - | - | - | - | - |
| Ganyaye kashi 2 | Rabin kofi | 100 | 2 | - | 7 | 35 |
| 'Ya'yan itace | Ya danganta da iri | - | - | - | 10 | 40 |
| Hatsi | Ya danganta da iri | - | 2 | - | 15 | 70 |
| Nama da makama-ncinsa | Dan gutsure | 30 | 7 | 5 | - | 75 |
| Kayan maiko | Karamin coka-li 1 | 5 | - | 5 | - | 45 |

A nan kasa kuma za'a bada misalan irin abinci da ke a kowane kashi, Amma kafin nan ga misalan wasu abincin da ba sai an auna su ba, Watau ana iya amfani da su a koda yaushe, Sune kamar su;

Shayi ko koffi

tashi

gishiri (ga wanda ba shi da hawan bugun jini)

lemun tsami

albasa

tafarnuwa, citta da sauran kayan yaji,

Kabewa da

Jimbiri,

4. Kashin Ya'yan itace

Kana iya amfani da daya daga cikin misalan da za'a bayar a nan wajen sauya abincinka, 'Ya'yan itace suna ba wa jiki karfi, Yana da muhimmanci ka kiyaye adadin da aka bayar a nan, Misalan sun hada har da;-

| | |
|---|---|
| Ayaba | Rabin babba (ko karama daya |
| Dabino | 2 |
| Baure (danye) | 2 manya |
| Baure (busashe) | 1 karami |
| Lemun tattaka | Rabi |
| Inubi | 12 |
| Mangoro | Rabin guda |
| Lemun zaki | Daya karami |
| Ruwan lemun zaki | Rabin kofi |
| Abarba | Gutsure karami |
| Tanjirin | Daya babba |
| Kankana | Gutsure |
| Gwanda | Gutsure |

5. Kashin Hatsi da Makamantansu

Ko wane daga cikin abubuwan da aka lissafa a nan ya kunshi abin gina jiki da kuma na sa karfi a cikinsa. Misalan wannan kashi sun hada da;-

| Misali | Adadi |
|---|---|
| Burodi | Yanka daya |
| Biskit | Daya mai tsawo |
| Dafaffen hatsi | Rabin kofi |
| Gurasa ko cipati ko kanzo | Kashi 3 daga 4 na kofi |
| Shinkafa dafaffa | Rabin kofi |
| Dafaffen taliya, makaroni da makamantansu | Rabin kofi |
| Garin hatsi ko hilawa | Babban cokali 2 da rabi |
| Wake dafaffe | Rabin kofi |
| Tumun gero ko dawa | Misalin duntsa daya |
| Masara (dafaffa ko gasassa) | 1 karama |
| Sayayyen masara | Kofi daya |
| Dankalin turawa | 1 karami |
| Dankalin Hausa | Rabin guda |
| Doya | Daya bisa uku na karama |

Yana da muhimmanci ka ga cewa ka bi awon da aka kiyasta a nan saboda wadannan kashin abinci suna sawa jiki zaki da yawa,

6. Kashin Nama da Makamancinsa

Wannan kashi yana kunshe da abubuwan dake gina jiki dumi, Ga misalan wannan kashi kamar haka;-

| Misali | Adadi |
|---|---|
| Nama (na rago, tunkiya, akuya, shanu kaza da sauran tsuntsaye) | Yanka 3 makimanta |
| Kwai | 1 |
| Kifi - danye da sandararre | 1 |
| Kifi - busashshe | 1 karami |
| Tarwada | 5 kanana |
| Kifin gwangwoni - sadin | Yanka 3 |
| Cuku (busashshe) | 1 makimanci |

7. Kashin Kayan Maiko

Wannan kashi yana da mai a cikinsa, kuma ko wane daga cikinsu zai iya ba jiki karfi a misalin calories 45. Kana iya amfani da ko wane a adadayin sauran wajen sauya abincinka.

| Misali | Adadi |
|---|---|
| Man shanu | Karamin cokali 1 |
| Manja, ko na gyada, ko na auduga ko na bi rana, ko na kade | Karamin cokali 1 |
| Aparari ko cukunsa | Babban cokali 1 |
| Gyada, yazawa da makamantansu | 6 kanana |

Kamar yadda aka yi bayani baya, wadannan kashi kashin abinci suna da muhimmanci sosai wajen lissafin abincin da mai ciwon sukari zai ci ba tare da abincin ya gaza da wani abu ba, Ga misalin yadda za'a yi amfani da wadannan kashi kashin abinci a wani biyan bukata,

Idan misali kana so ka hada abincin da zai bada karfin calories 1500 kuma ya kunshi duk kashin abincin da aka ambata, gashi;-

Abincin mai ciwon sukari da zai bada karfin calories 1500

| Kashin Abinci | A d a d i | | Karfi (Calories) |
|---|---|---|---|
| | Yawan Musaya | Awo | |
| Madara | 2 | Kofi 2 | 340 |
| Ganyaye na farko | Babu Adadi | - | - |
| Ganyaye na biyu | 2 | Kofi 1 | 70 |
| 'Ya'yan itace | 4 | Ya danganta da iri | 160 |
| Hatsi da makamantansa | 5 | Ya danganta da iri | 350 |
| Nama da makamantansa | 6 | Babbar tsoka | 450 |
| Kayan maiko | 3 | Karamin cokali 3 | 135 |
| Dukan Adadi (Karfi) | | | 1505 |

Ga kuma bayani a jadawali yadda za'a yi lissafin abin da za'a ci ko wane lokacin cin abinci,

| Kashin Abinci da lokacin cinsa | Yawan Musaya | Awo | Karfi (Calories) |
|---|---|---|---|
| Da Safe | | | |
| Madara | 1 | Kofi 1 | 170 |
| Dan icce | 1 | Ya danganta da iri | 40 |
| Nama | 1 | Yanka 1 | 75 |
| Hatsi ko makamantansa | 1 | Ya danganta da iri | 70 |
| Kayan maiko | 1 | Karmin cokali 1 | 45 |
| Dukkan adadi | | | 400 |
| Da Rana | | | |
| Nama | 2 | Yanka 2 | 150 |
| Hatsi ko makamantansa | 2 | Ya danganta da iri | 140 |
| Dan icce | 1 | Ya danganta da iri | 40 |
| Kayan maiko | 1 | Karamin cokali 1 | 45 |
| Dukkan adadi | | | 375 |
| Da Yamma | | | |
| Nama | 2 | Yanka 3 | 225 |
| Hatsi da makamantansa | 2 | Ya danganta da iri | 140 |
| Kashin ganyaye na biyu | 2 | Kofi 1 | 70 |
| Dan icce | 1 | Ya danganta da iri | 40 |
| Kayan maiko | 1 | Karamin cokali 1 | 45 |
| Dukkan adadi | | | 520 |
| A lokacin kwanciya | | | |
| Madara | 1 | Kofi 1 | 170 |
| Dan icce | 1 | Ya danganta da iri | 40 |
| Dukkan adadi | | | 210 |
| Jimlar Adadi baki daya | | | 1505 |

2,2 Motsa jiki:

Motsa jiki akai a kai yana taimakawa wajen saukaka cutar sukari, Babu ma inda wannan yafi alfanu kamar ga wanda yake gashi da matukar kiba bayan civon sukari. Za'a iya bukatar kara abinci kafin a motsa jikin ko kuma bayan an motsa, koda yake wannan ya danganta ga matsayin hauhawar sukarin a cikin jinin mutum.

2,3 Magunguna:

Magunguna da ake amfani da su wajen sauwaka cutar sukari sune kwayoyi ko kuma ire-iren insulin da ake allura da su.

Kwayoyin magungunan sun kasu biyu, kuma ko wane nau'i daga cikinsu, ko kuma dukkansu, suna iya amfani a madadin allura. Mafi yawa mutu cutar sukari nau'i na biyu ana basu daya daga cikin ire-iren wadan nan magungunan ne ko kuma ma dukkansu. Misalin nau'in kwayoyin na daya wato wadanda ake kira da turanci "sulfonylurea" sune "diabinese®" da "Glibenclamide" wanda shi ma ake fi sani da "daonil®" da "tolbutamide" ko "artosin®" da dai sauransu. Dan gane da kwayoyi nau'i na biyu kuma wato "biguanide" a turance, "metformin®" ne kawai ake fi amfani da shi. Magungunan sulfonylurea sune ke sawa tsokan nan kaimin samar da insulin su kuma su ma sauran jiki kuzari. Shi kuma maganin biguanide shine ke sa a rage kaimi wajen cin abinci da kuma takaita yadda 'yan hanji ke wuce wa da abinci zuwa ga jini. Watau dai kwayoyin suna da kyau ga mai cutar sukari wanda kuma yake da matsananciyar kiba. Ana amfani da dukkan nau'o'in kwayoyin biyu domin sauwaka nau'i na biyu na cutar sukarin, wadda ba a iya tsagaita hauhawar zakin da ke cikin jinin mai fama da ciwon sukarin ta hanyar

kayyade abinda zai ci, Sai dai wadannan magunguna basa iya tsinana wani abu idan cutar tayi kamari, Idan cutar takai ga wannan matsanan cin hali to babu abin yi illa a maye gurbin kwayoyin da allurar insulin, Marar lafiyar dake shan daya daga cikin wadnnan kwayoyin zai rika jin abubuwa kamar su ciwon ciki, alamar jin amai, da kuma kasala, To amma ana iya shawo kan wadannan idan aka hadiyi magnin bayan an cika ciki da abinci, Wata shawara ga mai shan maganin ita ce kuma cewa lalle ya yi nesa-nesa da giya idan ma har yana shanta domin ita giyar tana iya bata amfanin maganin a cikin jiki,

2,4 Allurar Insulin;

Ana amfani da insulin a wajen samun saukin cutar sukari nau'i na daya, ko kuma irin wanda ta kai intaha a nau'i na daya ko na biyun ko kuma a wajen wanda za'a yi wa hida, ko kuma ga mai rashin lafiya ko mai juna biyu, Akwai nau'o'in insulin iri-iri, Likita ne ke zabar maka irin insulin din da ya dace da bukatarka, Yana da mahimmanci kuwa ka san irin insulin din da kake amfani dashi, kuma yana da kyau ka dauwama da yin amfani da irinsa domin wani nau'in dabam amfanisa na iya banbanta da wani, Mai yiwuwa kana bukatar allura biyu na insulin a kowace rana, daya da safe kafin ka karya kumallo, dayar kuma kafin kaci abincin dare, Yawanci dai a asibiti ne ake fara yi wa mutum allurar insulin, Da zarar mara lafiya ya murmure sai kuma a koya masa yadda zai shiga 'tarkon insulin na "mutuka raba"',

Akwai sirinji da allura na musamman da ake amfani da su wajen allura insulin, Kana iya amfani da su sau da yawa kafin a musanya su da wasu, Ana baka shawarar ka rika dafasu akalla sau daya cikin kwana biyu, Ga kuma yadda zaka dafa su;

1. A cire sirinji daga bota,
2. A sanya sirinji da bota da allurar a cikin 'yar tukunya ko kwano mai kyau, a zuba ruwa mai kyau a lullube su sa'annan a rufe tukunyar,
3. A aza a kan wuta kuma barsu su tafasa a bar su suyi ta tafarfasa har na tsawon kimanin minti goma zuwa shabiyar,
4. A ciresu a zuba a cikin wani abu mai sanyi,

Yana da mahimmanci ya kasance an wanke hannaye fes a kowane lokacin da za'a yi allurar, A murmuza kwalbar insulin sosai domin a cuda ta, Idan za'a cika sirinji kuma sai a tabbatar da cewa an fitar da dukkan iskan da ke cikinsa ta lalatsa sirinji sannu a hankali, Ta haka idan aka tura botar sama, sai dukkan iskan dake cikin sirinji ya fita, Idan iskan yaki fita, to a mayar da insulin din cikin kwalbar sa'annan a sake cika sirinjin,

Hakanan kuma muhimmin abu ne a rika sauya wajen da za'a yi allurar a kai a kai, Domin kuwa idan aka yi ta yin allurar a wuri daya zai sa wajen yayi kulu, ya kuma hana insulin din bin jiki, Wuraren da suka dace a yi allurar insulin sune;-

- Saman cinya da bayanta da kuma gabanta,
- Saman dantse da gabansa,
- Saman duwawu da kuma gefensa,
- ciki,

Tilas ne a tabbatar da cewa yawan insulin da za'a yi allurarsa adadin daidai yake, Kada a taba allurar kuma ba sai an goge wajen da za a yi allurarba, Idan an zo yin allurar sai a soka ta tsaye ba tare da an karkatata ba, Daga nan sai a jawo botar sannu a hankali, domin a tabbatar da cewa ba a kan jijiya take ba, daga nan sai a dannata domin a saki

dukkan ruwan maganin, daga nan kuma sai a fitar da allurar, Shike nan an gama. Wata shawara itace, yana da kyau a yi alama a jikin sirinji da wata takarda da aka sava danko ko tauwada, wadda ruwa baya wanketa, domin a san yawan insulin din da ake bukata. Ya kamata wani dan' uwan marar lafiyar ko kuma abokinsa ya san yadda ake yin allurar, domin ya agaza masa in ya shiga halin da yake bukatar taimako. Haka nan kuma lalle duk wadanda suke amfani da allurar insulin sukasance suna da wani sirinji da kuma allurar ko da wadanda suke amfani dasu sun kare ko sun lalace.

Wasu fitattun matsalolin da ke tattare da allurar insulin sune; wasu mutane idan suka yi ko akayi musu allurar jikin su ba zai amince da itaba, saboda haka sai su shiga wata wahala, wata sa'a ma har sai an sake masu wani nau'in allurar. Wata matsalar kuma itace kulu a wajen da ake yin allurar, da kuma karancin sukari a cikin jini.

Karancin sukari a cikin jini yana faruwa ne a sanadiyya daya daga cikin wadannan abubuwa;-

- Amfani da insulin mai yawa.
- Cin abinci a makare ko kin cin abincin ko kuma cin kadan.
- Bayan an motsa jiki ko kuma anyi aiki mai wahalar wa.
- Haka kuma ana iya samun karancin sukari a jini haka kawai ba tare da wani dalili ba.

Fitattun alamun karancin sukari kuwa sune; yin zufa da jin matsananciyar yunwa da bushewar jiki musamman a fuska da makyarkyata da dundumi (rashin gani sosai) da in'ina da rashin natsuwa.

Ana sauwaka wadan nan alamu ta hanyar shan wani abu mai zaki kuma mai saurin narkewa, alal misali cokali daya na zuma ko na madi, ko cokula biyu ko fiye da haka na sukari ko gulucodi ko rabin tambilan daya na lukozad ko 'ya'yan dabino ko halawa ko kunu ko surkin da aka sa wa sukari, Abu ne mai kyau ga mai cutar sukari ya rika tafiya da wani abu mai zaki (misali daya daga cikin abubuwan da aka zana a sama) a duk inda yake kuma ya sha shi da zarar yaji duriyar alamar karancin sukari a jikinsa, Kada ace bari in bari in gani ko alamun sa lafa, domin rashin yin maganin (alamun karancin sukarin a jiki) na iya kaiwa ga suma, Hakanan kuma yana da muhimmanci mutum yayi kokari ya san dalilin daya ke haifar da rashin sukarin a jininsa domin a yi maganinsa a gaba, Idan mutum yana samun bugun rashin sukari a jini da dama a rana daya to mai yiwuwa wannan na nufin sai an sauya yawan ruwan allurar insulin da mutumin ya ke yi, To anan shawara ita ce mutum ya hamzarta ya tafi ya ga likita, Yana da muhimmanci sosai cewa ko dai dan'uwa ko kuma wani wanda kuke tare kusan ko da yaushe (a kasuwa ne ko a gida ko wurin aiki) ya san cewa kana da ciwon sukari kuma kana amfani da allurar insulin, domin ko da wani abu yaka faru (kamar karancin sukari a jini bayan allurar) su taimakeka ko su kaika wajen likita har in ya zama dole,

Da san sanu ne da sai mai yin allurar insulin ya aje ruwan allurar a cikin gidan sanyi (firiji) amma ba a cikin gidan kankarar ba, Inda kuma babu firiji, to sai a aje shi a cikin randa wadda ke kan yashi, Shawara mai amfani itace cewa kada mai ciwon sukari da ke kan allurar insulin ya sha barasa domin zaka hana shi ruwan maganin yin aiki sosai a cikin jiki,

2.6 Tsananin yawan sukari ajini ga mai cutar sukari da kuma yayin da ciwo ya kamasa ;-

Tsananin sukari a jini yana faruwa ne a sanadiyyar daya daga cikin wadannan abubuwa ;

- Rashin bin wani tartibin tsarin cin abinci da kuma cin abinci mai sa karfi fiye da kima,
- Rashin amfani da isasshen insulin din da jikinka ke bukata, ko kuma rashin shan kwayoyin magani a kai a kai yadda ya kamata
- Yayin da wata rashin lafiya (cuta) ta kama mutum,

Idan mutum bai shan maganinsa ko yin allurarsa a kan kari, kuma ya bari yawan sukari ya hauhawa a jikinsa to sai wasu alamun tsananin sukari a jini su bayyana a garesa, Wadan nan alamu su ne yawan jin kishi da yawan yin fitsari da kuma jin alamar rashin lafiya, da jin juwa da kuma jin alamun ciwon ciki, Idan ba'a yi maganinsu cikin hamzari ba to fa abin yana iya kaiwa ga somewa,

Idan ciwo ya kama mai cutar sukari kada yace zai dena shan magani ba, tunba dai wadanda ke kan allurar insulin ba, Kada kayi kuskuren cewa ai tunda ban lafiya kuma ban iya cin abinci sosai to bai kamata inyi allurar insulin ba, Hasali ma mafi yawan lokuta, a lokacin rashin lafiya ko wani ciwon sai yawan sukari a cikin jini ya karu, kaga kuma dole a bukaci insulin, (Haka ma wadanda ke shan kwayoyi wata sa'a sai an sauya musu magani sun koma ga allura kafin a shawo kan rashin lafiyar tasu), Idan baka iya cin abin cinka na yau da kullum, to sai ka sha kwatan kwacinsa mai ruwa-ruwa kamar su kunu, fura, madara, lukozed da dai sauran ire-irensu, Ka

tuna cewa a duk lokacin da kaji alamun yawan sukari ko rashin lafiya to ka garzaya zuwa asibiti domin likita ya duba ka sosai,

3,0 Yadda za'a rika lura da matsayin sukari a jini ;

Akwai hanyoyi da dama da mai ciwon sukari zai iya gane ko yana samun saukin ciwon, Wadan nan hanyoyi sune:

3,1 Tambayoyi (a tambayeka ko kuma kai ka tambayi kanka) alal misali;

- Saunawa ne ka sami rashin lafiya a sati ko a wata?
- Sauna ka sami alamun karancin sukari a rana ko a mako?
- Akwai ciwon kai ko kasala bayan ka farka da safe?
- Sau nawa ka ke fitsari a rana kuma sau nawa ka ke tashi shan ruwa da dare, Idan amsoshin dukkan wadan nan tambayoyi ya kasance a'a ne ko kuma dai wani abu makamancin a'an to ga alama dai kana samun sauki, Sai dai fa wannan zance ba nassi ne ba, yana iya tashi,

3,2 Auna Nauyi ;

Idan mai cutar sukari kuma mai kiba yana rage nauyi, ko kuma idan wanda yake yin allurar insulin ya kasance nauyin sa ba raguba kuma bai karuba, to a alama ya shawo kan cutar kenan, Sai dai fa shima wannan zance ba nassin bane,

3.3 Auna Fitsari :

Yana da mahimmancin gaske ga dukkan masu ciwon sukari da suyi wannan awo, Yawanci dai ana so ne a yi awon ko dai kafin aci abinci ko fafin a kwanta barcin dare, ko kuma a dukkan lokucin da ake jin duriyar hauhawar sukari ko karancinsa a jini, Auna fitsari muhimmi ne ga mai ciwon sukari domin yana taimakon mai cutar ya san inda ake kwana kuma ya san yadda zai shawo kan al'amarin ta hanyar cin abincin da ya dace. Haka kuma bita-da-kulli ne ga cewa a bi shawarwarin likita, Dadin dadawa ma ana yin awon fitsari ne domin a tsare kai daga fadawa halin kiki-kaka,

Akwai hanyoyi da dama na auna adadin sukari a fitsari, Ga wasu nan masu saukin yi ko da a gida;

Hanya ta daya itace ta amfani da wani abu da ake kira da turanci 'clinistix' ko kuma 'tes-tape', Shi clinitix ana saumunsa a cikin wata kwalba mai duhu, shi kuwa tes-tape tamkar igiya (ko tef) na awo ne a cikin wani gida na musamman, Kowane dai za ai amfani dashi to a tabbata cewa an bi ummur ni da aka rubuta a jikin kwalbar ko gidan da suke, Idan clinistix ne za a yi amfani da shi to sai ka dauki warka daya sa'an nan a tsoma shi cikin fitsarin da aka tanada, Bayan nan launin da aka samu a kan warkar sai a kwatanta shi da irin launikan dake jikin gidan maganin, Idan test-tape ne za'a yi amfani dashi, to sai a yago tef (ko igiyar) mai kimanin tsawon dan yatsa, sa'an nan a tsoma shi sannu a hankali a cikin fitsarin, Duk irin launin da aka gani a jikin tef din sai a kwatanta shi da irin launukan dake jikin gidan tef din,

Hanya ta biyu; A wannan hanyar ana amfani ne da wasu kwayoyi da ake kira "clinitest", Za a saka kwayar ne a cikin fitsarin da aka tanada, Sa'an nan a duba irin launin da ya koma da kwayar ta gama 'tafarfasa-tafarfasa',

Ko dai wace hanya aka bi ta auna fitsarin, muhimmin abu shine a ajiye abun aunawar (watau clinistix ko testape ko clinitest) a wuri busa sshe, mai sanyi, mai duhu kuma inda yara ba za su taba shiba,

4.0 Matakan kare kai;

4.1 Kula da kafa;- Bayan mutum ya dade da cutar sukari a rayuwarsa, zai ji tamkar kafarsa ba tasa bace saboda rashin jini a kafar. Saboda haka muhimmin abu ne mai ciwon sukari ya kula da kafarsa da kyau. Domin ayi maganin wasu matsaloli da ke iya tasowa, sai a kula da wadannan shawarwari :

a, A kodayaushe kafarka da tafinta su kasance cikin tsafta, kuma ba a jike ba. Idan za'a wanke su to a tabbata cewa ruwan ba mai zafi kwarai bane,

b, Kada ka aje kafarka daf da wuta (idan kana jin dumi),

c, Kada kayi yawoba takalmi. Sa'an nan ka sanya takalman da suka yi maka daidai,

d, Kabi a hankali wajen yanke akaifa. Idan ka lura da alamar fashewar jiki to garzaya ka sheda wa likita,

e, Idan kuma baka iya gani tangaran to kace wa matarka, ko aboki ko malamar asibiti (nas) ta rika duba maka kafa funka akai a kai,

4.2 Shan taba da Cutar Sukari ;

Shan taba abin cutarwa ne a jiki ko da ma mutum baya da cutar sukari, Idan kana da cutar sukari kuma kana shan taba, to akwai hatsarin

cewa zaka yi wa kafofin da jini ke bi illa, Wannan kuma yana iya kaiwa ga jagulewar cutar, Haka ma wannan na iya haifar da ciwon da hanyoyin da iska ke bi a jiki kai har ma kansar huhu,

4,3 Tukin mota da tafiye-tafiye ga mai cutar

sukari ;

Mai cutar sukari wanda yake shan kwayoyi ko kuma wanda yake bin tsarin abinci don magance cutarsa, yana iya tuka mota shi kadai abinsa ba tare da yayi kaffa-kaffa da wani abu ba, Sai dai al'amarin ya sha banban ga mai cutar wanda ya ke kan allurar insulin, Idan kana karbar insulin ne to kada ka yi tukin mota sai fa idan za ka iya gane alamun karancin sukari a jini da kuma yadda za a magance su, Duk wani mai cutar sukari mai yin allurar to ya rike wata sheda ko alama da za ta nuna cewa yana cutar (sukari) a duk lokacin da zai yi tuki ko wata tafiya mai nisa, Kuma lalle ayi guzurin sukari ko gulukos ko dai wani nau'in abinci mai saurin narkewa a jiki kamar su burodi mai zaki, dankalin hausa ko rake a tare da mai ciwon, Ka tuna kuma kada lokacin cin abinci ya zo ba ka ciba, Idan mata kana, sai ka tsaya ka ci abincin sa'an nan kaci gaba da tukin ko tafiya, Doka itace kada ka kuskura ka yi allurar insulin kuma ka ki cin abinci, Idan kana tuki kaji alamar karancin jini a jikinka, to sai ka kashe motar, ka tashi daga kujerar matuki, ka ci wani abinci da ka tanada ko sukari, ka kuma jira a wajen motar har sai kaji alamun sun sun lafa, Idan zaka yi wata tafiya mai tsawo, kada ka manta ka riko dukkan kayan allurar a wata 'yar jaka tare da kai, Wadan nan kayayyaki kuma sun hada da magunguna da kayan auna fitsari, Haka nan zai yi ma amfani idan wani nan kusa da kai

(lokacin tafiyar) ya san cewa kana da ciwon sukari musamman ma dai idan kana yin allurar insulin ne,

4.4 Tsafta :

Ka kasance fes-fes a kodayanshe musamman ma dai a wajen da kake (ko ake yi ma) allura, Kabi a hankali a lokacin da kake yanke farce, Kada ka yanke su har can makura da fata, domin kar su zame maka jangwangwan, Idan ka sami yanka ko sara a jiki to ka tabbata cewa an tsabtace su an kuma kare su daga kura da kasa, Kayi kokarin sa tufafin da basu matse ka kwarai ba domin kar su hana zagayar jini a jikinka,

4.5 Juna-biyu :

Domin lafiyarki yi kokari kiga cewa ciwon ki ya lafa sosai kafin ki sami juna biyu, da kuma tsawon lokacin da kike tare da cikin, Ki shaida wa likita da zarar kin lura cewa an sami ciki, Yin haka yana da muhimmanci domin a iya sauya magungunan da ki ke sha, Haka kuma ana iya maganin matsalolin da ke iya tasowa kuma ki haihu lami lafiya tare da jariri mai lafiya,

5.0 Cututtukan da wannan ciwo ki kawo wa sune wadanda sauye-sauye a jijiyoyi da kafofin jini ke haifarwa, A hankali sai wadannan sauye-sanye su shafi sassan jiki irin su ido da koda da zuciya da kafa da sauransu, An fi samun irin wadannan cututtuka ga mutanen nan wadanda a kusan koda yaushe yawan sukarin dake cikin jininsu ya wuce kima, Idan ana kula da hauhawar sukarin to za a iya magance al'amarin, ko a kalla a rage kaifisa, Cututtukan da ciwon sukari ke kawowa suna bullowa ta alamun dundumi (rashi

gani) wanda ke iya kaiwa ga makanta, da ciwon zuciya, da lalacewar kafa, abinda ke jawo macewar kafar gabadaya. Mai ciwon sukari ma zai iya zama ya sami hawan jini saboda rashi aikin zuciya da koda yanda ya kamata. Haka ma cutar sukari na iya haifar da ciwon mafitsara, domin yawan sukarin da ake fitarwa daga jiki na iya jawo hayayyafar wasu kwayoyin cuta a mafitsarar.

Illolin da ciwon sukari ke kawowa sukan yi shakara da shekaru kafin su bayyana, sai dai da zarar sun fito to fa maganinsu sai hali, sai Allah. Rigakafi ance yafi magani, saboda haka ka kula da adadin sukarin dake jinika, ta hanyar cin abincin da likita ya tsara maka da kuma shan magani ko yin allura a ciki lokaci. Hakanan kuma da bin duk wata shawara da malaman asibiti suka baka (ko likitanka). Ta yin haka sai a sauwaka ko ma a magance illolin dake tare da wannan ciwo na sukari. Ka tunafa idan ka shawo kan cutar ba ma alamunta da sifofinta kurum ne ka tsira da ga wajen suba, a'a, ka ma yi wa kan ka kiyamul laili daga illolin da takan haifar. Haka kuma sai ka rayu cikin jin dadi da lafiya ka wala kanar kowa.