

**EDUCATION MATERIAL
FOR
DIABETIC DIET COUNSELLING**

BY

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B.Sc. (H.Sc.)

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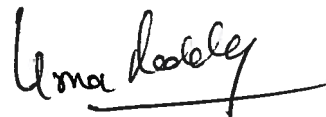
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Ms.B. JANAKI has satisfactorily prosecuted the course of research and that the thesis entitled EDUCATION MATERIAL FOR DIABETIC DIET COUNSELLING submitted is the result of original research work and is of sufficiently high standard to warrant its presentation to the examination. I also certify that the thesis or part thereof has not been previously submitted by her for a degree of any university.

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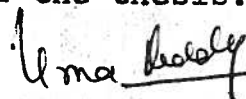


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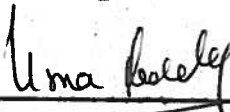
No part of the thesis has been submitted for any other degree or diploma. The published part has been fully acknowledged. All assistance and help received during the course of the investigation have been duly acknowledged by the author of the thesis.

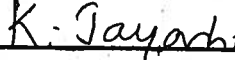


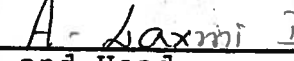
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Janaki

(JANAKI)

DECLARATION

I, B. JANAKI hereby declare that the thesis entitled EDUCATION MATERIAL FOR DIABETIC DIET COUNSELLING submitted to Andhra Pradesh Agricultural University for the Degree of MASTER OF SCIENCE IN HOME SCIENCE (Foods and Nutrition) is a result of original research work done by me. I also declare that my material contained in the thesis has not been published earlier.

Date: 16th nov '92

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ABSTRACT

Present study was planned to investigate the impact of education on the dietary management of diabetic subjects. This study was carried out for a period of 6 months on 32 NIDDM subjects attending out patient clinic of Endocrinology Department, Osmania General Hospital, Hyderabad. General information and disease information was collected through a schedule developed. Before the study, a pre-knowledge test was done by non-formal method with the help of knowledge test questions developed. Twenty four hour recall method was followed to collect dietary information and to prescribe diet to each individual, based on their disease condition, drug usage, height, weight, age, work, likes and dislikes. Along with regular counseling, subjects were monitored for blood sugar values, body weight and dietary intake with the help of a folder titled "your management goals" and food record titled "your meal management". Special education material such as a book-let on diabetes ' - 'For victory over diabetes' with appropriate illustrations for each instruction, charts - (a) 'For your victory over diabetes', showing areas of importance in disease management; (b) 'How are you?' showing graphic representation of blood sugar values of hyper, hypo and normoglycemic patients; (c) 'Avoid' showing restricted foods; (d) 'Free foods' showing some of the fruits and

vegetables that can be liberally consumed by diabetics. Subjects were exposed to diet displays, slide shows and demonstrations of food measurement and urine testing for sugar. After a period of 15 days, after education intervention, post knowledge test was conducted in non-formal method.

The findings of the study showed a mean decrease of 76.41 ± 45.74 FBS and 135.72 ± 83.03 PPBS, significant at 1 per cent level ($P = >0.001$). Change in body weight showed a significant result at 1 per cent level ($P = >0.001$). A mean increase of 1.5 ± 1 in under weight and 1.9 ± 1 in over weight subjects was observed. No significant change was seen in BMI, indicating a long term study for a substantial decrease in body weight. A mean increase of 6.56 ± 2.45 score was observed in knowledge test (max. score = 15). Regularity in meal timings, daily consumption of leafy vegetables, decreased consumption of deep fried foods and restricting to prescribed calories was observed in the sub-sample of newly diagnosed patients.

By the end of the study 6 per cent stopped taking OHD, 12 per cent decreased insulin dosage, number of visits with hypo/hyperglycemia decreased. Discussions and conversations generated during diet displays demonstration and slide shows lead to an increased follow up and patients expressed comfort in the knowledge that they were not alone in dealing with diabetes. Majority of the subjects were prone to risk factors like lower socio-economic status, sedentary work, smoking, alcohol, tobacco chewing and age. The mean age at which disease occurred was 43.1 ± 7.94 years. Half of the subjects were diagnosed as diabetics on routine check up.

From the findings of the present study, it is inferred that the material developed for counselling diabetic patients were effective in improving the patient compliance in the management of the disease. A need for effective screening methods and an on going intensive educational programmes in the out patient clinics of diabetes was observed.

...

LIST OF ABBREVIATIONS

| | | |
|------------------|---|--|
| AHD | : | Atherosclerotic heart disease |
| BMI | : | Body mass index |
| Ca ⁺⁺ | : | Calcium |
| EUR NIDDM PG | : | European Non-insulin dependent diabetes policy group |
| FBS | : | Fasting blood sugar |
| FCPD | : | Fibro-calculous pancreatic diabetes |
| GDM | : | Gestational diabetes mellitus |
| IDD | : | Insulin dependent diabetes |
| IGT | : | Impaired glucose tolerance |
| K ⁺ | : | Potassium |
| Mg ⁺⁺ | : | Magnesium |
| MRD | : | Malnutrition related diabetes |
| NIDD | : | Non-insulin dependent diabetes |
| OGTT | : | Oral glucose tolerance test |
| OGH | : | Osmania general hospital |
| OHD | : | Oral hypoglycemic drugs |
| CHA | : | Oral hypoglycemic agents |
| PPBS | : | Post prandial blood sugar |
| WHO | : | World Health Organisation |

INTRODUCTION

CHAPTER I

INTRODUCTION

Diabetes mellitus is most common serious metabolic disorder of humans. The term refers to the host of syndromes which differ in their clinical manifestation, pathophysiology and etiology. They all have in common the primary hyperglycemic state. The treatment for this disease is a matter of on going debate, however it is clear that nutritional management is the corner stone of the treatment. The nutritionist plays a central role in designing diets to maintain optimum metabolic state in the diabetic persons.

WHO (1985) defined diabetes mellitus as a chronic elevation of the glucose concentration in the blood, sometimes accompanied by symptoms of severe thirst, profuse urination, weight loss and stupor, culminating in coma and death in the absence of effective treatment.

The incidence of diabetes mellitus is increasing world wide, so also in India. Though at present the prevalence is lower in India as compared to western countries (India - 2%; Western countries - 5%) epidemiological studies indicate four undiagnosed persons for every diagnosed diabetic. By applying dynamic statistical model the number of diabetic

subjects in India will be 15 million (Ajgaonkar, 1986), out of which 98 per cent of them are non-insulin dependent diabetics.

The chances of getting diabetes increases with genetic suceptability, viruses, obesity, aging, diet, stress, hormones and medication or drugs. Many studies indicate these as risk factors triggering the onset of diabetes. Eventhough all these factors are inevitable, some of them like diet, stress, obesity can be dealt positively, where education has an important role to play.

Long before the discovery of insulin, the disease diabetes mellitus, means, "the production of large quantities of sweet urine," and therefore people could not eat sugars. Since then diet in diabetes has undergone major changes. Originally the word "diet" is derived from the Greek word 'diaita' which means 'habitual manner of living', especially that of eating, which implys more than just 'Food'. In 12th century Roman empire legislature was called as 'diet' who take decisions for the good of the state, so also is the 'diet' which is prescribed for the restoration of good health. Oxford dictonary described 'diet' as the customary allowance of food and drink to meet specific requirements of the individual.

Recommendations of the diet therapy for diabetes contained in the earliest description of the disease in the Papyrus Ebers in 1500 BC. In ancient India Sushruta and Charaka recognized the value of dietary therapy for diabetes. They recommended a reduction in total quantity of food consumed and the avoidance of animal fats which are accepted even today by many diabetologists and nutritionists. Later on in the modern era of diabetes i.e. starting from insulin discovery in 1922, liberalisation of restrictions in diet led to experimentation in specific nutrients, especially that of carbohydrates and fibre. Rawlegeylin (1929) first started experimenting with high carbohydrate diets, many scientists thereafter tried out diets with different levels of carbohydrates and finally concluded that 60 per cent of calories be met by carbohydrates alone. Discovery of hypoglycemic and hypocholestrimic properties of fibre lead to even more experimentation with diets and many scientists concluded that high carbohydrate and high fibre diets lead to a good glycemic control (Anderson, 1979 and 1982; Jenkin et al., 1976 and 1978; Ward et al., 1982 and Mohan, 1989).

While there is no firm evidence that physical fitness itself directly prevents complications of diabetes, it is known that in people without diabetes,

exercise reduces the risk of vascular problems such as heart disease and peripheral vascular disease. It is reasonable to assume that a person with diabetes would also enjoy the same benefits and moreover exercise also helps in blood glucose metabolism, not only by lowering the blood levels, but also by improving the body's sensitivity to the effects of insulin. Lingrade (1983) reported that glucose balance was affected significantly by physical fitness.

All is said well, if we cannot put it in practice. Presenting the subject matter in a form that allows concrete, external actions forms the basis for the education programme. Thus the results of educational programme are defined in terms of what a patient can do at the end of a learning period that they could not do before. Need for diabetic nutrition education has been stressed by National Diabetes Advisory Board(1982). Campbell (1990) in their study reported intensive educational approach as a means to establish good dietary management. Muhlhauser et al. (1983), WHO Committee (1985) reported that comprehensive education and training of the patient in self care has lead in certain situations to a 78 per cent decrease of hyperglycemic coma and 75 per cent decrease of below knee amputations.

The relatively high rate of hospital use by persons with diabetes makes in-hospital teaching a logical focus. The out patient nutrition education is one such tool that can be implemented and utilized to give the patient an opportunity to concentrate on their prescribed diet more intensely. Considering this and the fact that very scarce studies are available in this area specially in Andhra Pradesh, present study was conducted in the out patient clinic of Endocrinology department, Osmania General Hospital, Hyderabad, to cultivate positive approach in patients regarding their dietary management with the following objectives -

1. To elicit information from diabetic patients regarding their present knowledge and management practices of the disease.
2. To develop material for educating and counselling diabetic patients.
3. To counsel the patients with a series of personal contacts with the above developed material.
4. To monitor the behaviour modification and to test their gain in knowledge.
5. To study the impact of education intervention on management of diabetes.

REVIEW OF LITERATURE

CHAPTER II

REVIEW OF LITERATURE

Few decades ago diabetes mellitus was defined quite simply as a disease of metabolism resulting from the inability of the cell to utilize glucose. Since the discovery of insulin and its use in the treatment of diabetes, the great complexity of this disease has become more apparent and is now commonly defined not only in terms of high blood glucose levels and spilling of sugar in the urine, but also in terms of metabolic abnormalities in fat, protein, carbohydrates and disorders in the structure and function of blood vessels. Friedman (1980) described diabetes mellitus as a hereditary disease of metabolism in which there is an inadequate supply of effective insulin, characterised by disturbances of carbohydrates, fat and protein. It is in this context that giving a nutritional prescription to patient and making him to learn, to balance it with his daily activity and insulin in the body, presents a major challenge to nutritionists, doctors, nurses and pharmacologists who work as a team in the patient care.

It was Hippocrates 'the father of the medicine' who advanced the idea that sickness could be understood only if one considered the whole patient and his environment, successful treatment could be expected

only if one used the beneficial experience of similar cases.

2.1 PREVALENCE

Ahuja et al. (1991) have reported that diabetes mellitus today affects over 50 million people in the world, with about one half of them living in the developing world. While in the west, epidemiological studies indicated one unknown diabetic for every known case, the situation in developing countries seem to be vastly different, for every known diabetic there may be upto 4 others who are undiagnosed.

A study on 9950 rural population from parts of India based on post-glucose load capillary blood glucose based on WHO criteria, brought out the prevalence rate of 1.3 per cent. In the recent select urban studies in India, prevalence of diabetes have indicated a phenomenal increase over a decade. First study based on a questionnaire in an affluent population of Daryaganj Delhi (Total 3253) stated 3.1 prevalence. The other study was on hospital registered population in south India Kundermukh, Karnataka, stated 5 per cent prevalence, in the same study a staggering figure of 21 per cent of above 40 years age was found (Ramaiya et al., 1991). Similar results

were reported by Ramachandran et al. (1988), on south Indian population and Rao (1992) on Libiyan population.

Ajgaonkar (1986) expressed that considering the arbitrary prevalence of 2 per cent, the number of diabetic subjects in India could be 15 million.

2.2 ETIOLOGY

It is now apparent that diabetes is not a single clinical entity, but rather a heterogeneous group of disorders with different underlying causes. Several key factors which have been identified in the etiopathogenesis of insulin dependent diabetes mellitus as well non insulin dependent diabetes mellitus were broadly classified into genetic and environmental factors.

2.2.1 Genetic factors

Susceptibility to both insulin dependent diabetes mellitus and non-insulin dependent diabetes mellitus is determined to a substantial extent by genetic factors. These probably interact with an environmental trigger to induce expression of the disease state (Hitman and Niven, 1989).

The concordance rate for insulin dependent diabetes mellitus in identical twins was about 50 per cent while for non-insulin dependent diabetes mellitus

was approximately 90 per cent (WHO, 1985). Non-insulin dependent diabetes mellitus of young people i.e. maturity onset diabetes mellitus of the young, is inherited as a dominant trait and evidence is accumulating to suggest that non-insulin dependent diabetes mellitus susceptibility may also be conferred by a dominant gene (Tattersall, 1974).

Vishwanathan et al. (1985) found high prevalence of diabetes among off spring of conjugal non-insulin dependent diabetes mellitus parents in India. Diabetes was present in 50 per cent of offspring and 12 per cent had impaired glucose tolerance (IGT) according to the National Diabetes Data group criteria (1985).

2.2.2 Environmental factors

2.2.2.1 Over nutrition - obesity

Obesity can induce resistance to the action of insulin in several ways. In some instances, it reduces the number of insulin receptors on target cells, but in most cases it produces insulin resistance through post receptor changes eg; decreasing glucose transport or impeding intracellular glucose metabolism (WHO, 1985).

Obesity can be caused due to increased intake of fat or physical inactivity or hormonal disturbances.

In the western world, two-thirds or more of patients with diabetes are obese as compared with non-insulin dependent diabetes mellitus subjects in India where obesity is not common (Ramaiya, 1991).

Everhart et al. (1992) reported duration of obesity was inversely associated with fasting serum insulin concentration. Steven (1991) reported a stronger relationship between central adiposity and non-insulin dependent diabetes mellitus incidence in women than in men, the reason for this sex difference was related to body fat distribution.

Lundgren et al. (1989) in their longitudinal study from 1968-69 to 1980-81 concluded that fat distribution to the abdominal region as well as the total amount of fat per se as important risk factor.

2.2.2.2 Under Nutrition

Recent inclusion of malnutrition related diabetes mellitus by WHO committee indicates the high prevalence of diabetes in developing countries like India where population has lower body mass index, lower mean serum cholestrol value, low mean blood pressure and shorter life span.

Venkataraman et al. (1990) reported 20 per cent MRDM subjects in their study done on 16800

diabetic subjects registered in the diabetic clinic of Madras Medical College from 1979-89. Samal et al. (1990) reported protein deficient diabetes mellitus as 27 per cent in the childhood onset diabetes and 55 per cent in maturity onset diabetes in Orissa state.

2.2.2.3 Consumption of specific food

Diet varies from population to population depending upon their socio-cultural factors. NIDDM prevalence rates were higher on the coast of Andhra Pradesh and within the state, the rice eaters of Eluru and Tenali were having higher prevalence compared to urban and rural Hyderabad where wheat preparations (roti) form the basic staple food (Ramaiya, 1991).

In a long term study conducted by Raheija et al. (1989), on dietary habits of Indian population, showed that groups consuming an increased portion of diet from fast, fatty, refined, processed or preserved foods have increased prevalence of NIDDM and heart complications.

In the present day of modern life alcohol seems to be one of the major addiction most commonly seen in members of the society. Hirata (1988) suggested that alcohol intake aggravates blood glucose control and increases the complications of diabetes,

further more it may reduce the effects of diet treatment and drug treatment.

2.2.2.4 Physical activity

West (1978) stated exercise as a probable protective factor against the development of diabetes, supported by Lingarde et al. (1983) who stated that glucose balance was affected significantly by physical fitness, which was likely to be important in the pathogenesis of type 2 diabetes as well as its treatment. Taylor et al. (1984) in their study in migrant Indians in Fiji showed an association of NIDDM with sedentary physical activity independent of obesity.

2.2.2.5 Stress

No generally recognized definition exists for the term stress and even more difficult is its measurement. Hence it is very difficult to assess its contribution in precipitating diabetes. Stress can be physical (pain, dehydration, fever, illness, accident, hypoglycemia) or emotional (anger, fear, worry etc.). During a stressful situation certain 'stress' hormones (adrenalin, glucogon, growth hormone and cortisol) are released in person's body leading to raise in the blood sugar in response to the stressful situation. This extra-glucose may raise the blood sugar level leading

to hyperglycemia and in some insulin dependent diabetics ketoacidosis (Milchovich and Dunn-long, 1991). Cruickshank et al. (1980) described that urban stress is more marked in ethnic minorities than in local populations. In migrants, the changes in diet, customs, physical activity, and socio-economic status bring potential stress of varying degrees of magnitude.

2.2.2.6 Age

The prevalence of diabetes increases with age in India as elsewhere, maximum incidence being in third, fourth and fifth decades. In most studies the prevalence doubles if the results of population below 30 years are excluded. Ramachandran et al. (1988) showed 21 per cent prevalence of diabetes in age group over 40 years and 41 per cent in age group of 55-64 years in south India.

An analysis of the data from diabetic subjects between the age of 15 and 30 years in the age-standardized multicentre study in India showed significantly higher numbers in the rural areas than in urban areas. If we look at the recent studies (1986) from North India, New Delhi and Eluru, South India, 9.1 per cent of > 40 years; 8.6 per cent of 40-64 years and 6.1 of > 40 years were found respectively (Ramaiya, 1991).

2.2.2.7 Sex

Population studies show that diabetes is more frequent in men than in women in India (Gupta et al., 1978). However in recent studies (Omar et al., 1985; and Ramachandran et al., 1988) showed female preponderance in South Africa and South India respectively. They attributed these findings to increased frequency of obesity in females as compared with males. Vinke et al. (1959) also suggested greater obesity of women as predominant factor of diabetes.

2.2.2.8 Parity

High parity has been suggested as a causative or an important provoking factor in the development of diabetes (Jackson, 1961 and Hoet, 1954). However Rao et al. (1986) concluded that parity has no independent effect on glucose tolerance, but increasing parity may be associated with being increasingly over weight as seen in migrant Indians in Tanzania (Swai et al., 1990) and consequently with increasing glucose intolerance.

2.2.2.9 Diseases of Pancreas

Inflammatory, neoplastic and other disorders of the pancreas such as cystic fibrosis and haemochromatosis as well as pancreatectomy may lead to different degrees of insulin deficiency and hence in

some cases leads to diabetes mellitus. Damage to the islets of langerhans appears to be the basis of malnutrition-related diabetes mellitus (WHO, 1985).

Abraham and Geevarghese reported high prevalence of fibro-calculeus pancreatic diabetes (FCPD) (19%) in below 20 years age group. Similarly 26 per cent prevalence in 55 per cent of maturity onset diabetics and 12.2 per cent in childhood onset diabetes was observed in Orissa and all of the subjects belonged to poor socio-economic status with low BMI (< 14) (Samal et al., 1990).

2.2.2.10 Viral infections

Epidemiological studies provide circumstantial evidence for a possible causative role of viral infections in IDDM. Among the viruses that have been implicated are rubella, mumps and human coxsackie B₄ virus.

Hazra et al. (1980) reported significantly high frequency of coxsackie B₄ virus antibodies in young ketosis resistant group with acute onset of diabetes. Gamble and Taylor (1969) and Gamble et al. (1973) suggested an association between coxsackie B₄ viral infection and type 1 diabetes mellitus.

2.3 METABOLISM IN DIABETES

The underlying cause of diabetes is insulin deficiency, which is absolute in IDDM and partial in NIDDM. Metabolic changes that are observed in diabetes reflect the degree of insulin deficiency.

2.3.1 Metabolic effects of Insulin

Many tissues require insulin for the transport of glucose across cell membranes but some do not require eg. liver, kidney, tubule, intestinal mucosa, lymphnodes and erythrocytes.

Glucose for its utilization need to be converted to glucose 6 phosphate by the enzyme hexokinase and insulin is essential for activating this enzyme.

Enzyme glucose 6 phosphatase converts glucose phosphate to glucose when insulin levels are low therefore whatever glucose 6 phosphate is formed is converted back to glucose during insulin lack.

Decreased activity of glycogen synthetase is seen in the absence of insulin (leads to accumulation of excess glucose in blood).

Insulin deficiency leads to activation of glyconeogenic enzymes which results in increasing the glucose synthesis.

Target sites of metabolic action of insulin are liver, muscle and adipose tissue.

Several suggestions have been offered as to how insulin acts at the molecular level in achieving its many integrated effects. It has been proposed that the hormone acts by initiating a disturbance at the cell membrane as it attaches to a specific receptor site and then transmits information through the cell by various intra cellular messenger molecules. Ions such as K^+ , Ca^{++} and Mg^{++} have also been proposed as possible messengers (Bennion, 1979).

2.3.2 Insulin relation to adult onset diabetes

- Decreased synthesis of polypeptide chain (precursor of insulin)
- Inefficient conversion of polypeptide chain to pro-insulin.
- Improper conversion of pro-insulin to insulin.
- Time of release of insulin into blood stream.
- Decreased utilization of glucose.
- Decreased entry of glucose into cells.
- Increased concentrations of insulin antagonists.

The net result of altered metabolism in Diabetes mellitus leads to --

Glucose accumulation in blood - hyperglycemia
mobilization of fat, oxidation for energy and
deposition in the vessel walls occur.

Depletion of tissue protein occurs

Glycosuria

Dehydration due to high osmotic pressure of
plasma extracting water from the tissues with
resulting diuresis.

Acidosis (as described above), level of B-
hydroxybutyric acid and acetoacetic acid may
rise from 1 MEq/litre to may be 10 MEq/lit
(Bennion, 1979).

2.4 CLASSIFICATION OF DIABETES MELLITUS (WHO, 1985)

A. Clinical classes

Diabetes Mellitus (DM)

Insulin dependent diabetes mellitus (IDDM)

Non-insulin dependent diabetes mellitus
(NIDDM)

(a) Non-obese

(b) Obese

Malnutrition related diabetes mellitus (MRDM)

Other types of diabetes associated with certain conditions and syndromes

(1) Pancreatic disease; (2) disease of hormonal etiology; (3) drug-induced or chemical induced conditions; (4) abnormalities of insulin or its receptors; (5) certain genetic syndromes; (6) miscellaneous.

Impaired glucose tolerance (IGT)

(a) Non-obese

(b) Obese

(c) Associated with certain conditions and syndromes

Gestational Diabetes mellitus (GDM)

B. Statistical risk classes (subjects with normal glucose tolerance but substantially increased risk of developing diabetes)

Previous abnormality of glucose tolerance

Potential abnormality of glucose tolerance

2.5 DIAGNOSTIC CRITERIA

Until a few years back no consensus existed as to what should be the cut off values of blood sugars above which a person could be labelled as diabetic.

The 1979 National Diabetes Data Group classification was a step in the direction of establishing criteria to bring about some order in the chaos. Under the classification, a fasting plasma glucose of 115 mg/dl was considered normal. However, after much discussion and debate WHO group committee on diabetes mellitus in 1985 formed the following criteria.

A single blood glucose estimation in excess of the diagnostic value of 180 mg/dl of venous whole blood or 200 mg/dl of capillary whole blood or 200 mg/dl venous plasma or 220 mg/dl of capillary plasma establishes the diagnosis of diabetes irrespective of signs and symptoms. Venous whole blood and capillary whole blood when the values are below 80 mg/dl, diagnosis of diabetes is unlikely. Same holds good when venous plasma and capillary plasma is below 100 mg/dl. Only if blood glucose values lie in uncertain range (i.e. between the levels that establish or exclude diabetes) need an oral glucose tolerance test (OGTT). It is often sufficient to measure the blood glucose values only after fasting and at 2 hours after a 75 g oral glucose load. The other values measured in the full OGTT are not required to establish diagnosis by may serve to confirm it (WHO, 1985).

Table I : The diagnostic values for oral glucose tolerance test.

| Diabetes mellitus | Glucose concentration mg/dl | | | |
|----------------------------|-----------------------------|-----------|--------|-----------|
| | Whole Blood | | Plasma | |
| | Venous | Capillary | Venous | Capillary |
| Fasting value | > 120 | > 120 | > 140 | > 140 |
| 2 hours after glucose load | > 180 | > 200 | > 200 | > 200 |

2.6 SIGNS AND SYMPTOMS

Type I diabetes is characterised by many clinical symptoms. In type II, symptoms are less obvious or not apparent, necessitating a diagnosis by lab testing. In fact many NID diabetics are found on routine check up without any symptoms.

Hyperglycemia results in glycosuria or sugar in urine, which increases the osmotic pressure of the urine, preventing water reabsorption and results in excessive amounts of water being excreted, which is seen as polyuria. In polyuria along with large amounts of water sodium and potassium are also excreted from extracellular and intracellular fluids respectively which results in body fluid depletion causing polydipsia or increased thirst.

Insulin deficiency causes starved state in cells (due to lack of utilization of glucose) resulting in lack of energy (asthenia) and increased appetite or polyphagia. Additionally, muscle protein and adipose will be broken down to supply energy needs. Catabolization of muscle tissue results in weight loss and negative nitrogen balance and fatty tissue catabolization results in ketosis. Tiredness, fatigue, delayed wound healing, dizziness or giddiness, chronic and un-resolving infections are some of the other symptoms (Bennion, 1979). Patients and their relatives need to be educated in this area so that they do not suffer from diabetes unnoticed.

2.7 APPROACHES IN MANAGEMENT OF DIABETES

Different therapeutic approaches are required for the two major types of diabetes. In type 1 diabetes, administration of insulin is the primary therapy, the diet needs to be adjusted to maintain good nutrition and in children, to promote growth. The timing and content of meals must be co-ordinated with the schedule of insulin administration to prevent extreme oscillations of blood glucose concentration. In type 2 diabetes, most diabetologists believe that diet therapy is the fundamental management tool and that drugs should be employed only when diet therapy is inadequate to control hyperglycemia (American Diabetes

Association, 1979; British Diabetes Association, 1982). The recommended approaches to dietary management depend on resources for intensive dietary counselling (Zifferblatt and Wilbur, 1977). During the past decade, new approaches to diet therapy have been introduced in order to address some of the problems inherent in conventional methods. The education of health professionals, patients and their close associates has been emphasized, attempts have been made to individualize and simplify diets (West, 1973). Newer methods of improving compliance have been initiated such as behaviour modification. So also the recommended amounts of carbohydrates, fibre and fat have changed although simple sugars are still restricted. Food is talked in terms of type and amount of carbohydrates and fibre, biological value of protein, saturated, polyunsaturated and mono unsaturated fats. Franz (1987) emphasised exercise in the management of diabetes (especially in NIDDM patients) by stating that euglycemia is achieved during exercise through the precise regulation of hepatic glucose production and the utilization of glucose at the cellular level.

There are four main objectives of the management of Diabetes

- to preserve the life of the diabetic patient and relieve the symptoms of the disease.
- to enable the patient to lead as normal a social life as possible.
- to establish and maintain good metabolic control and
- to avoid the complications of diabetes mellitus.

These objectives can be achieved only if the patient is educated and motivated to anti diabetic programme which can be made possible through educating and counselling the patients.

2.7.1 Nutritional prescription

When we look at the metabolic implications of the insulin lack, the picture we get is, blood with high amounts of glucose and acidic compounds such as B-hydroxybutyric acid and acetoacetic acid, all highly alter blood composition and cause classic symptoms of diabetes mellitus. Therefore it is very appropriate that adjustments in food and fluid intake be made.

Prescribing a diet has its origins from 1550 BC and has undergone vast changes since then (see Appendix 11). Discovery of insulin and oral hypoglycemic drugs has brought about new dimensions to dietary prescription. Maintenance of meal timings with quantity and quality in accordance with the insulin or OHD has become important to prevent extreme oscillations in blood glucose levels.

The American Diabetes Association has given the following nutritional recommendations for individuals with DM (1987).

- 55-60 per cent of calories should be met by carbohydrates.
- 15-20 per cent of calories should be met by protein.
- less than 30 per cent of calories should be met by fat.
- upto 40 g of fiber should be met by the whole day diet.
- Cholesterol should be limited to under 300 mg per day.
- A low sodium diet may be recommended.

Extensive research has been done in formulation of diabetic diets. Following is a brief account of literature regarding each nutrient.

2.7.1.1 Carbohydrate and fibre

Carbohydrates forms a major source of calories in diet therefore should be selected carefully. Carbohydrates are of two types simple and complex and yield 100 per cent glucose. Considering the fact that simple sugars are absorbed quickly and complex sugars are absorbed slowly, many scientists recommended complex carbohydrate diets for diabetics to avoid sudden increase in blood sugar level.

Anderson et al. (1977) reported that there was little difference between isocaloric diets containing 20 per cent or 44 per cent carbohydrates whereas 75 per cent carbohydrate diet lowered the fasting glucose and improved glucose tolerance. Simpson et al. (1979) also supported the above. While Cooper et al. (1986) reported that there were no metabolic contraindications if sucrose was used as sweetner upto 28 g (simple carbohydrate) in the diabetic diet. Abaira et al. (1988) have reported that sugar did not aggravate hyperglycemia in type II diabetes, but in high carbohydrate diets have increased fasting triglycerides. Nise et al. (1989) concluded that

sucrose added to snacks in amounts supplying upto 7 per cent of total calories did not adversely affect short term blood glucose control. Simple carbohydrates have different affects on blood glucose according to their type and modalities of absorption (liquids, isolated or mixed with meals). Therefore, it is possible that variety of different levels of total carbohydrates are consistent with good diabetic control provided that the types of carbohydrates used are based on glycemic effect. The glycemic index of foods rich in simple sugars like milk products, oranges, apples is lower than that of starchy foods.

Many recent studies have shown that glycemic response varies enormously after ingestion of equal amounts of starch, according to the type of starch absorbed. The glycemic responses to white and whole bread were similar, but the response to white spaghetti was found to be only about two-thirds to that of bread (Jenkin et al., 1983). The glycemic responses to nutrients also appear to depend on the other foods with which they are mixed. Bantle et al. (1983) reported that simple sugars produced no greater increase in glucose or insulin concentrations than that of wheat or potatoes when they were ingested as part of a mixed meal. These studies of the acute glycemic affects of various types of food are an important beginning in

increasing our understanding of responses to dietary constituents, specifically carbohydrates. However the application of the glycemic index concept to clinical practice awaits further evaluation and data gathering.

Addition of fibre to the diabetic diet has been shown to blunt post-prandial increases in glucose concentration in type 1 and type 2 diabetes (Jenkin et al., 1976) and to allow reduction of the insulin dose in diabetic men (Anderson and Ward, 1979).

Dietary fibre appears to produce benefits by delaying both gastric emptying and the absorption of carbohydrates from the intestine. In view of the limited insulin secretory capacity of many type 2 diabetics, such delay in carbohydrates absorption is theoretically advantageous. In practice however, major limitations of high fibre diets are the lack of palatability and production of abdominal discomfort (Williams et al., 1980), flatulence and increased stool bulk and frequency (Kelsey et al., 1978) side effects decrease the long-term patient acceptance.

It has been found that the dietary fibre intake of diabetics with vascular disease was significantly lower as compared to diabetics without vascular disease. Serum cholesterol values decreased with fibre supplements in 4 out of 5 studies by

Anderson and Bryant (1986). Mohan (1989) carried out studies with high carbohydrate high fibre diets on over 25,000 diabetics and concluded that high carbohydrate high fibre diets decreased cholesterol and triglycerides levels (risk factors of atherosclerosis) and improved insulin sensitivity.

These prudent, health-promoting diets offer good benefits to diabetic persons. Most individuals alter their diet best in step-wise manner when nutrition counsellor negotiates a weight goal and daily energy intake outlining good food choices thus encouraging them to take more fresh fruits and vegetables (Anderson and Gustaffson, 1989).

2.7.1.2 Proteins

It has been recognised for many years that excessive protein intake can be deleterious to the diabetic because of its association with animal fat and excess amino acids which can be converted to glucose. Brenner et al. (1982) postulated that excess protein intake by diabetics can increase the rate of deterioration in renal function. Cohen et al. (1987) reported that protein restriction had a beneficial effect on micro-albuminuria independent of changes in glucose concentration and arterial blood pressure. When the diabetic presents with diminished renal

function protein restriction seems warranted. Adults with chronic renal failure require atleast 0.6 mg protein/kg of body weight/day. Restriction of protein even to this extent may lead to a reduction in the intake of calcium, phosphorus, sodium, potassium and vitamins. However, to prevent protein malnutrition and mineral depletion, supplementation with amino acids or their ketoanalogs are often required to prevent muscle wasting (Bennion, 1979).

2.7.1.3 Fats

One rationale for the recommendations is the high incidence of coronary artery disease in individuals with diabetes and association between diets more in fat and coronary heart disease. The rate of death from coronary artery disease accounts for 1/3 of all deaths among persons with type II diabetes. Individuals with type I diabetes are 8 times more likely to suffer from coronary artery disease than their age and sex matched peers. In diabetes many factors may affect blood lipid levels and complicate formulating an effective dietary prescription, eg. low calarie diets and alcohol intake.

The most prominent effect on blood lipid levels of poorly controlled or untreated diabetes is an increase in VLDL, often associated with decreased

levels of HDL. Several epidemiological studies strongly prove that fat and cholesterol restriction in diet improves plasma lipid levels (Raheija, 1990). Daly et al. (1986) reported that low fat (25% of daily energy) diet was associated with decrease in several cardiovascular risk factors, systolic and diastolic pressure, LDL cholesterol, serum triglyceride values and also the glycemc control improved.

Quality and quantity of fat consumed is important. Higher saturation of fatty acids predisposes higher blood cholesterol thereby increasing cardiovascular events. Raheija et al. (1989) in their long term study on dietary habits showed population groups consuming increased protion of fast foods, fatty foods refined and processed or preserved foods had increased prevelence of NIDDM and AHD.

The American Diabetes Association (1987), the American Heart Association (1982) and the National cholesterol education program (1987) recommended that fat make up be less than 30 per cent of total calories, with saturated fat less than 10 per cent of total calories, polyunsaturated fat less than or equal to 10 per cent and the balance of fat calories (10 to 5 per cent) as monounsaturated fat and cholesterol intake less than 300 mg/day.

2.7.1.4 Vitamins and Minerals, Trace elements

Diabetics and non-diabetic persons have the same requirements for vitamins, minerals and trace elements.

An originally bound form of chromium called "glucose tolerance factor" appears to be required for optimal glucose absorption and utilization. Stress and refined foods including simple sugars increase chromium losses. Supplemental chromium was associated with improvements of risk factors associated with maturity on-set diabetes and cardio-vascular diseases (Anderson,, 1986).

Ginter and Vathova (1983) reported that several points of contact exist between the metabolism of glucose and vitamin C. Therefore deficiency of vitamin 'C' provokes disorders in glycoregulation reminiscent of diabetes mellitus. And diabetes brings about disorders in ascorbic acid metabolism which may lead to a local vitamin 'C' deficiency in some tissues. This vicious circle can be cut by increased supply of vitamin 'C'.

Free radicals were implicated in the pathogenesis of DM and its long term complications, suggesting that oxidative stress generated by free radicals as a common factor in the diverse

pathophysiological mechanisms leading to the development of DM as well as its long term complications. Sadikot and Raheija (1991a) in their study found that vitamin 'E' supplementation decreased the free radicals significantly and considered it as a part of diabetic management. Similarly vitamin 'C' supplementation also decreased oxidant stress and free radical damage in diabetics (Sadikot and Raheija, 1991b).

2.7.1.5 Water and electrolytes

Diabetics and non diabetics have the same requirements for water and electrolytes. When complications like hypertension and renal insufficiency arise, the dietary treatment is identical to that prescribed for non diabetics.

2.7.1.6 Energy :

The most important objective in nutritional care of the diabetics is total energy intake. The energy intake should be sufficient to maintain ideal body weight and if the patient is a child or pregnant women providing allowance for growth and development.

Recently many studies have shown that therapeutic intervention can produce major improvements in insulin secretion and action. Fukuda (1989)

reported improved insulin resistance, weight reduction and good glycemic control after weight reduction with very low calorie diets on obese subjects.

Rudnick and Taylor (1965); Hadden et al. (1975); Doar et al. (1975); and Kosaka et al. (1980) reported that hypocaloric diet therapy of type II obese patients for relatively short duration can maintain or enhance autoregulation. (Autoregulation is the ability to secrete hormones in response to change in nutrient concentration and the ability of these hormones to act on target tissues which will be impaired in type II diabetics). Similar results were reported by Nagulesparan et al. (1980); Stanik and Marcus (1980); Savage and Nagulesparan (1983).

American Diabetes Association (1979) and British Diabetes Association (1982) recommended restricted energy intake and believed to be the single most important therapeutic intervention in the overweight type II diabetics. Hadden et al. (1975) in a six month out patient study with calorie restricted (40% carbohydrates) diet reported reduced fasting and post challenge plasma glucose concentration and post-challenge plasma insulin increase. Wales (1982) also reported similar results. Making the patients to learn to adjust their daily calorie intake according to their

requirements and recommendations is an important component of nutrition education.

2.7.2 Exercise

Exercise has been proven to be extremely important in the control of blood sugar. In fact, for the majority of NIDDM exercise in combination with a weight loss diet is all that is needed to control the blood sugar.

During exercise fall in plasma insulin and rise in counter-regulatory hormones such as glucagon, catecholamines, growth hormone and cortisol plays an important role in maintaining glucose homeostasis, by producing glucose to support augmented muscle in response to exercise. The main effect of glucagon is the stimulation and maintenance of increased hepatic glucose production, increasing both glycogenolysis and gluconeogenesis in order to prevent hypoglycemia. Hanson et al. (1983) demonstrated that glucagon is responsible for 75 per cent of the increase in hepatic glucose production during exercise (Appendix 12). Lingrade (1983) concluded that glucose balance was affected significantly by physical fitness, which was likely to be important in pathogenesis of NIDDM as well as its treatment.

To recommend exercise to diabetic patients, first they should be educated and counselled regarding positive and negative effects of exercise and then slowly worked out into daily lives to reach their targets and stay there.

2.7.3 Oral hypoglycemic drugs

Oral anti-diabetic drugs have been developed during recent years. The action of oral anti-diabetic drugs may be due to (Sadikot, 1990) -

1. Stimulation of insulin production from B-cells of the islets of pancreas.
2. Making the circulating insulin more effective.
3. Their action directly on peripheral muscle tissue increasing utilization of glucose.

OHD are broadly divided into -

1. Sulphonylureas :

eg. glipizide (glynase), tolbutamide (rastinon), cerbutamide and chlorpropamide (copamide, diabinese), glibenclamide (euglucon, daonil, betanase).

2. Biguanides :

eg. metformin, phenformin

2.7.3.1 Drug interaction

Diuretics, glucocorticoids, estrogen compounds and diphenylhydantoin compounds impair the hypoglycemic action of the oral agents, while drugs such as salicylates, quinine (and alcohol) can indirectly potentiate the hypoglycemic effect of the oral agents (Sadikot, 1991).

2.8 COMPLICATIONS

Researchers say that high blood sugars over periods of time damage certain tissues of the body causing the complications of diabetes involving the eyes, kidneys, nerves, heart and blood vessels.

2.8.1 Heart and blood vessel disease

For both men and women, there is evidence that diabetics to have a greater incidence of heart and blood vessel disease-gangrene blindness etc. Estimates of an increased risk for the development of atherosclerosis vary from 1.2 to 6 times in diabetics as compared to controls (non-diabetics) vascular changes leading to gangrene occurred 53 times more frequently in diabetic men and 71 times more frequently in women compared with controls (Lele, 1990).

2.8.2 Nerves

A great majority of diabetics have some changes in the peripheral nerves. Clinical complications are manifest in excessive stroke damage, permanent impairment in brain function from hypoglycemia and a mild chronic encephalopathy (Anthony, 1992).

2.8.3 Eyes

The widespread association between retinopathy and many forms of diabetes is well recognised. Retinopathy of different degrees of severity was documented in all 14 national samples commissioned for the WHO multinational study of vascular disease in diabetics (WHO, 1985).

2.8.4 Kidneys

Progressive impairment of renal function accompanied by urinary protein loss and culminating in end-stage renal failure, threatens the health and life of upto half of those patients who develop IDDM in youth and a substantial amount of renal failure in NIDDM persons (WHO, 1985). Urinary tract infections, damage to delicate filtering capillaries of the renal glomerulus (nephropathy) are some of the other problems.

2.8.5 Hypoglycemia

Hypoglycemia is a common side-effect of insulin therapy. Its symptoms include weakness, profuse sweating, irritability, confusion, unconsciousness and convulsions. Consumption of alcohol, insufficient food intake and interaction with other drugs produces hypoglycemia in patients dependent on OHD. Severe Hypoglycemia is rare and is seen mainly in patients taking oral hypoglycemic drugs with prolonged action (WHO, 1985).

2.8.6 Severe Hyperglycemia (diabetic coma)

Severe stresses like heart attack and trauma may precipitate ketoacidosis even in mild diabetics through increased secretion of hormones that oppose insulin action. In diabetics already undertreatment, ketoacidosis most often results from failure to take adequate insulin and may point to poor education, psychological factors and inadequate provision of health care (WHO, 1985).

2.9 ROLE OF EDUCATION AND DIET COUNSELLING IN THE MANAGEMENT OF DIABETES

During the past decade, new approaches to diet therapy have been introduced in order to address some of the problems inherent in conventional methods.

Education of health professionals, patients and their close associates have been emphasized.

Pioneers of modern diabetology, Joslin and Lawrence put forward firm and well founded recommendations for patient education. Krall and Beaser (1989) in their book Joslin diabetes manual said "education is not a part of treatment - it is treatment".

However it has taken almost 50 years for the beneficial effects of patient education to be recognized generally.

WHO (1985) have put forward the biological, psychological and social effects of education as follows.

Biological effects

- more efficient short-term and long-term metabolic control
- prevention of acute loss of metabolic control
- better diabetes control during unusually strenuous physical activities or illness.

Psychological effects

- decreased fear of diabetes and its complications

- decreased sense of helplessness
- improved acceptance of diabetes
- better participation by the patient in prevention strategies and therapy.

Sociological effects

- decreased sense of isolation as the diabetic learns to adapt management to his/her social needs
- less absence from work as acute and chronic complications lessen
- decreased fear of diabetes as information is transmitted to the patients family, school, teachers, employers and society in general.

Patient education in treatment of chronic illness is centred on changing the patient's behaviour and on selfcare and thus requires the active participation of the patient. Etzuiler (1976) said that the key to successful diabetes control is patient participation.

The participatory approach to teaching, includes, open questions which should help the diabetic person acquire the necessary knowledge and know-how, physicians have the responsibility in promoting

integration of the biomedical, educational and psychosocial aspects of diabetes (Assal et al., 1985).

Changing the nutritional habits for better is a main challenge to nutrition education, which puts us to another important aspect of nutrition education is not simply to change nutrition habits formed generation ago, but also to prevent the acquisition of other irrational attitudes towards nutrition which results from new, pseudo-scientific information.

Education and nutrition counselling are synonymous in nature, a nutritionist can educate a patient to learn as much as they can about, what is being attempted on their behalf through counselling.

Counselling process is conducted in 5 stages -

1. developing a co-operative alliance
2. Information gathering (understanding the present state)
3. setting goals (constructing a realistic desired state)
4. interviewing (techniques for leading to the desired state)
5. maintaining the change.

Thus it helps patients to identify some desired state and to help them move in stages from their present state to that desired state.

Williams (1984) concluded in the proceedings of the Nutrition Society, U.K., that Nutrition education is appropriate for the long-term management of diabetes in the community.

Diabetes education can occur in a variety of settings. Material used will make all the difference in imparting the education to the patients. Wedman (1987) studied the effect of using graphic aids and models of various foods, eye, kidney, arteries, nerves and human cells along with counselling and found that patients who received counselling along with aids registered highest rates of compliance, then their counterparts who received only counselling. Patient acceptance was found to be good when a poster containing visible options of different foods was given to patients to paste it on their kitchen walls or boards (Power, 1973).

Kendall and Jensen (1990) in their study concluded that nutrient based (diet guide) method was more effective in planning balanced menu to exchange list method by patients. Lorini and Ciriaco (1990) concluded from their study that education has improved

the diabetes management. Korhonen et al. (1987) showed a significant weight loss and improvement in the control of diabetes of newly diagnosed NIDDM patients through education regimens. Mani et al. (1989) reported that intensive diabetes education programme for a period of one month resulted in a significant decrease in both fasting and post-prandial blood sugar. Patients showed a decrease in carbohydrate and fat intake and an increase in fibre intake.

In a study done by Cohen and Zimmet (1983) 79 per cent of the overweight patients decreased their weight, 40 per cent ceased oral therapy, 17 per cent decreased their dosage and blood glucose values nearly improved in all patients and also dietary compliance was better after education programme.

Running into elevated lipid levels is a common problem of diabetics, intensive education and dietary counselling were recommended for dietary management of this kind. Campbell et al. (1990) reported that intensive educational programme established good dietary management and improvements in total cholesterol but not necessarily improve glycemic control in NIDDM.

Translating the prescription into life-style changes and monitoring metabolic changes present a

major challenge and requires good communication between the health professional and patient and in particular patient participation and support from peer group and family members. Anderson and Gustaffson (1989) demonstrated that good communication between patient and health professional increased the adherence (70%) to high carbohydrate and high fibre diets in IDD and NIDD patients. Mount et al. (1991) demonstrated group participation as a method to achieve weight loss and blood glucose control and results showed significant reduction in weight and haemoglobin values. Pratt et al. (1987) observed significant weight reductions in persons who participated in the combined nutrition education and peer support programme, several psychosocial variables were significantly correlated with weight reduction and improvement in glycemc control.

Sociologist, Dechamp et al. (1990) in their study with IDD and NIDD patients concluded that patients etiological beliefs may contribute to the knowledge or behaviour gap, correct information about a more rational etiology for diabetes could improve patient compliance. Because patients knowledge was often satisfactory, while behaviour was inadequate.

Educators believe that the suffering and economic burden of diabetes can be reduced through training, this was supported by Wood (1989) in her

study on 53 patients who attended out patient education programme after discharge reported reduced blood glucose levels, fewer hospitalizations and regularity in meal timings. Muhhauser et al. (1983), WHO Committee (1985) reported that comprehensive education and training of the patient in selfcare has lead in certain situation to a 78 per cent decrease of hyperglycemic coma and to a 75 per cent decrease of below knee-amputations. Miller and Goldstein (1972) reported reduced rates of hospitalization for acute complications of diabetes in a setting with an integral diabetes education programme which were reinforced by Koplan and Devis (1986).

Education is a process of communication where meeting of minds takes place, for this many methods are followed, Schreiber and Amos (1983) in their study of class room instruction and self instruction education programme on 20 patients for each method, concluded that both the instructional approaches were viable. Mazzuca et al. (1986) found blood glucose control and a reduction in body weight for obese patients participating in an education programme which stressed both knowledge and self help behaviours. Piagessi et al. (1989) conducted individual teaching as a first-step intervention for the education of diabetic subjects and results showed significantly higher

control on study group compared to age gender matched control group, regarding diet, physical exercise, OHD or insulin.

Learning is a fundamental part of diabetic management, and education material that is developed should meet the criteria. Hosey and Freeman (1990) designed and evaluated diabetes education book-lets targeted to 5th and 7th grade level and concluded that 62 per cent of the target audience understood the messages in the book-lets. Garber et al. (1977) gave pre and post knowledge quizzes to the patients after an education programme and an overall improvement in patient compliance was found.

Further communication between nutrition educator and patient occurs only when patient follows the given instructions at home. Data collected from 1977 through 1979 showed the response rate of 41 per cent; 86.5 per cent wished to contact the educator again for doubts; 59.6 per cent had instructional materials and 62.5 per cent wanted another appointment (Bell, 1981). Ney et al. (1983) in their research found that subjects will keep accurate food records using a check list system for upto 30 weeks and the described compliance system was found to be a valuable educational tool and can assist in the interpretation

of clinical data used in diabetes management and the assigned compliance grades showed correlation with quantitative parameters of diabetic control.

All the research reviews indicate that quality of diabetic control can be improved by educating the diabetic patients. The need for education was stressed in all the areas of diabetes management, namely diet, drugs, exercise and complications of diabetes.

MATERIALS AND METHODS

CHAPTER III

MATERIALS AND METHODS

The two educational approaches being used in teaching of diabetes are knowledge based educational interventions and behavioural based interventions, both types assume a causal path from learning to changed patient performance and from altered performance to improved medical or psychological outcomes.

Based on the earlier studies, the present study is based on these two approaches. Knowledge based - booklets and charts were developed, behavioural based - self help skills like urine testing for sugar, food selection, food measurement and management during the occurrence of hypoglycemia were demonstrated through patient participation. Individual diet counselling with the help of patient monitoring folder, food intake record book and developed printed material (Appendix I) was carried out.

Materials and methodology followed in this study are as follows :

3.1 SELECTION OF THE SUBJECTS

Thirty two non-insulin dependent diabetic patients were selected from the out patient ward of Department of Endocrinology of the Osmania General

Hospital, Hyderabad. Osmania General Hospital is the legal post-graduate teaching hospital of Osmania Medical College and follows a similar protocol of EUR-NIDDM PG (European NIDDM policy group) in the management of Non-insulin dependent diabetes mellitus (Appendix-2).

All patients were from the twin cities of Hyderabad and Secunderabad suffering from non-insulin dependent diabetes mellitus (NIDDM). Duration of diabetes after diagnosis ranged from 15 days to 15 years with the age group ranging from 30-68 years. Their income level ranged from Rs.1000-5000/- per month with majority of them doing sedentary work.

3.2 CASE HISTORY

A schedule was developed to elicit information regarding each case. Data collected included age, sex, anthropometric measurements, occupation, income level, personal habits, history of diabetes, duration, drug history, symptoms, family history, associated disorders and dietary history (Appendix 3). Dietary modification according to the patients requirements were given in the subsequent sessions of the counselling.

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3.3 KNOWLEDGE TEST QUESTIONS

To know the knowledge level of the patients questionnaire comprising of 15 questions was formulated

and pre-tested by an amalgam of nutritionists, extension educators, doctors, dietitians and a student (other than nutrition student), a house-wife, an employee and a diabetic patient (Appendix 4).

Pre and post knowledge test was done on the selected sample before and after education programme and the scores were tabulated. The non-formal method was followed for testing patient's knowledge.

3.4 PATIENT MONITORING

Patient monitoring was done through diet counselling for dietary and behaviour modification with the help of a folder and a record book designed to keep a record of blood sugar levels, body weight and daily food intake. Each patient was exposed to five individual counselling sessions, group discussions, demonstrations and slide shows. Group discussions were between patients and nutrition educator, between patient and doctors and between patients and hospital dietitians.

A folder titled "Your Management Goals" was maintained by the nutrition educator through out the study period consisting of normal values of fasting and post prandial blood sugar levels. Desirable body weight (to be reached) and BMI was calculated during the initial counselling session based on height and weight

of the patient. The type of treatment a patient was undergoing was noted and monitored throughout the study period (Appendix 5a).

A record book titled "Your Meal Management" was maintained by each patient in which he/she was asked to keep a daily record of food intake along with the quantity, timings and name of the recipe. The patient was also asked to write how he/she felt during the day, with time specification so that occurrence of hypoglycemia could be traced and talked about during the subsequent counselling session (Appendix 5b).

3.5 DEVELOPMENT OF EDUCATION MATERIAL

Based on the following expected outcome of the study, education material was developed.

| Topic | Expected outcome |
|------------------------|--|
| Definition of Diabetes | Patient should be able to tell acceptable definition. Should easily tell the signs and symptoms of diabetes. |
| Diet | Patients should be able to tell the importance of controlled portion size of foods, meal timings, meal measurement and proper selection of food. |

Contd..

| Topic | Expected outcome |
|----------------|--|
| Weight control | <p>Should differentiate between refined and complex carbohydrates with examples.</p> <p>Should know the importance of fibre in diet.</p> <p>Should identify restricted foods.</p> |
| Exercise | <p>Patients should be able to calculate their ideal body weight.</p> <p>Should know the benefits of exercise.</p> |
| Complications | <p>Patients should be able to differentiate between hypo and hyperglycemia.</p> <p>Should be able to tell the steps to be taken during occurrence of hypoglycemia or to avoid hypoglycemia.</p> <p>Patients should know that uncontrolled sugar levels leads to complications of the eyes, heart, kidney and nervous system.</p> |

Contd..

| Topic | Expected outcome |
|----------------|---|
| Clinical tests | <p>Patients should be able to test sugar in urine by Benedicts method and monitor their sugar level.</p> <p>Should be able to tell the normal values of blood glucose.</p> <p>Should know the tests done to evaluate blood glucose level.</p> |

3.5.1 Book let

A book-let titled "For Victory Over Diabetes" was developed with main emphasis on dietary control with appropriate illustrations for each instruction (Appendix 6). This was translated into Telugu and Urdu for the benefit of the patients and was distributed to all the patients after they were exposed to diet displays, charts, slide-shows and demonstrations. The programme was conducted step wise starting with diet-displays and at the end book-let was distributed to each patient to give a comprehensive view of the disease and its management. After a gap of 15 days post-knowledge test was conducted.

3.5.2 Charts

Four charts were developed (Plate 1; Plate 2; Plate 3; Plate 4) and displayed during the food displays.

Plate 1: 'For your victory over diabetes' - the areas of importance in diabetes management.

Plate 2: 'How are you?' - a graphic representation of blood sugar levels of hyper, hypo and normoglycemic patients.

Plate 3: 'Avoid' - foods to be avoided by the diabetic patients.

Plate 4: 'Free foods' - some of the fruits and vegetables that can be consumed in liberal amounts.

All the above material was screened by experts, pre-tested and standardised before educating the patients.

3.6 SLIDE SHOW

A set of slides which were previously developed by Foods and Nutrition Department, Andhra Pradesh Agricultural University were shown to the patients twice with a gap of 7 days (one week) in between. They contained figures of - location of

pancreas, insulin production and transfer of glucose to cells, a normal cell and diabetic cell, signs and symptoms of diabetes, restricted foods and a balanced diet. During the show, formal class room method was followed, while informal teaching was followed during discussions.

3.7 FOOD DISPLAY

To have a first hand experience of what, how much and when to take food, two food displays were conducted on two different days with a gap of 2 weeks.

3.7.1 1st Food display

A diabetic diet of 1800 KCals for whole day along with timings was displayed. Meal planning was in accordance with the recommended dietary allowance i.e. carbohydrates - 60 per cent; protein (high biological value) - 20 per cent and fat (low cholesterol) - 20 per cent with high fibre. The menu was planned keeping in view that food intake must be spread throughout the waking part of the day (Appendix 7 and Plate 5).

Restricted food items were displayed, which the patients could identify as foods to be restricted by diabetics (Appendix 8 and Plate 6).

Inbetween meal snacks were displayed providing maximum 50 Kilocalories which were mainly from fruit and vegetable group (Appendix 9 and Plate 7).

3.7.2 IInd Food display

Eight traditional break-fast items providing maximum of 325 kilocalories were displayed. This display was clubbed with food measuring demonstration (Appendix 10 and Plate 8).

Food display was a continuation of what patients were being advised regarding diet. Some vegetable and fruit wax models were also used during displays (Plate 9).

3.8 Demonstrations

To promote active participation and confidence in patients, food measuring and urine testing demonstrations were also given.

3.8.1 Food measuring demonstration

Standard measuring cups (Plate 10) that are available in market were used for measuring rice, dal, vegetable (cluster beans were used) green leafy vegetable (amaranth), curds, milk and oil. Daily allowances of cooked food was shown with the help of dietary prescription given to each patient. A house

hold measure (a glass (Plate 10) of 200 ml volume) was standardised (equivalent to 200 ml volume of measuring cup) and used in the demonstration to help patients measure their food at home. Raw and cooked weight of all foods demonstrated were also shown with the help of a food weighing balance.

3.8.2 Urine testing demonstration

Testing of urine for sugar level using Benedict's solution was organised to encourage daily home testing to provide an opportunity for the patients to test their own urine sugar levels.

3.9 FOOD AND NUTRIENT INTAKE

A sub sample of 9 patients who were not using oral hypoglycemic drugs or insulin and who were only on diet control was selected to know the dietary modification being practised after education programme as a follow up measure. A 24 hour food intake (cooked food) was carried out by weightment method. A control group of 6 patients who did not receive the present education programme was taken to compare the results of food and nutrient intake. Nutrients in cooked food were calculated as per the method of Pushpamma et al. (1981) and Gopalan et al. (1991).

3.10 BLOOD SUGAR TESTING

Blood sugar levels were recorded once in two weeks i.e. on each counselling session to monitor the blood glucose levels.

Sugar levels were tested by biochemist using the Glucometer (Reflectance Photometer), which is a portable, battery operated instrument used with Ames Dextrostix reagent strips to measure the amount of glucose in whole blood.

Principle : Dextrostix reagent area reaction is based on the action of the enzyme glucose oxidase which is specific for glucose. Glucose oxidase catalyzes the oxidation of glucose in blood by oxygen in the atmosphere producing gluconic acid and hydrogen peroxide. In the presence of peroxidase, chromogens in the reduced form are oxidized by the hydrogen peroxide producing gray to blue-purple colours. When the test chamber lamp shines on the reacted reagent pad, light is reflected. This reflected light is measured electronically and amount of glucose in blood is displayed.

Procedure

From the prick of the finger a large drop of blood was pressed generously to cover the reagent pad.

Test chamber lid was closed gently and timer was pressed. At the sound of the timer, blood was applied to reagent pad quickly and allowed to react for 60 seconds (i.e. at the sound of second buzzer). Immediately blood was washed off with a sharp stream of water and blotted gently on a lint-free paper, then reagent strip was put in test chamber closing the lid gently with reagent strip pad facing down and read button was pressed. Glucose concentration value was read on the display within seconds.

Accuracy of instrument was checked on each day of its use by running a standard test with a known concentration of glucose solution which reacts in a similar manner in the blood stream.

3.11 DATA ANALYSIS

Paired 't' test was carried out on data obtained to find out the significant difference, before and after education intervention (Rao N G, 1983).

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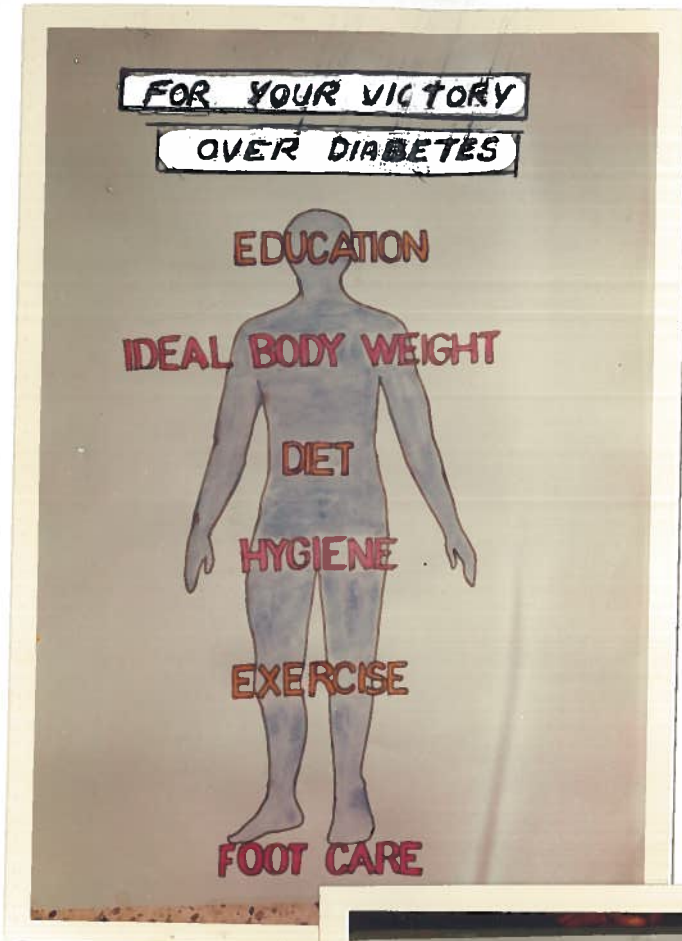


PLATE . 1

FOR YOUR VICTORY
OVER DIABETES

PLATE . 2
HOW ARE YOU

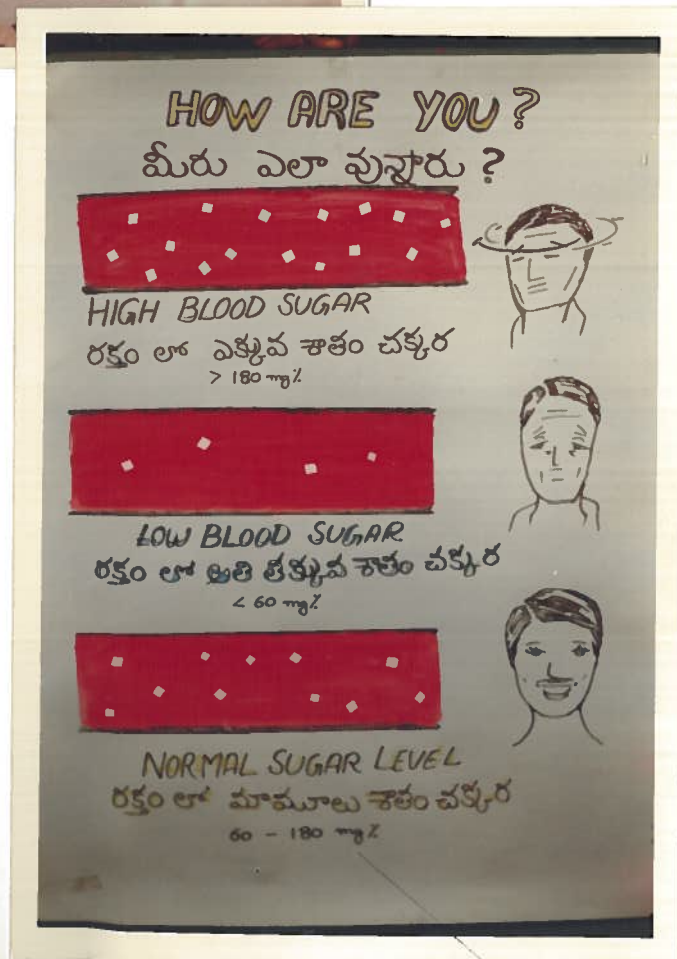




PLATE . 3

AVOID



PLATE . 4 FREE FOODS



PLATE . 5 DIABETIC DIET



PLATE . 6 RESTRICTED FOODS



PLATE . 7 INBETWEEN MEAL SNACKS



PLATE . 8 BREAKFAST ITEMS



PLATE . 11 DEMONSTRATION MATERIAL



PLATE . 12 PATIENT'S FAMILY PARTICIPATING
IN A DEMONSTRATION



PLATE . 13 PATIENTS DURING A FOOD DISPLAY





PLATE . 14 BLOOD SUGAR TESTING



PLATE . 15 PATIENTS UNDERGOING
HEALTH CHECK UP

RESULTS

CHAPTER IV

RESULTS

Diabetes is an important disease, not only because of its high prevalence, but also because many diabetics are diagnosed at an early age. One of the factors influencing the likelihood of complications developing is thought to be the quality of diabetes control and the better this is, the fewer complications develop in the long run. Dietary intake and education of the patient plays a role in the quality of control that is being employed. Present study was carried out to investigate the importance of educating the patients. The education of patients was done through the material developed and its impact was assessed by monitoring the patients with the help of daily food intake, periodic blood sugar values and body weight records of the patients.

A brief account of the results obtained are presented below :

4.1 GENERAL PROFILE OF THE SUBJECTS

Out of 32 subjects, 59 per cent were males and 40 per cent were females with age ranging from 30 years to 68 years. Majority of the patients were between 36-45 years age group followed by 46-55 years age group patients.

The mean age of the subjects was 46.1 ± 10.1 years. Ninety per cent of the subjects were doing sedentary work which included house wives, retired personnel and office executives (desk work) while only 9 per cent of them could be categorized as moderate workers engaged as car mechanic, factory worker and hospital nurse. Income level of the families ranged from Rs.1000-Rs.5000/- month. Thirty eight per cent belonged to the income group of Rs.1000-2000/- month, 31 per cent belonged to Rs. > 2000-3000/- month, 22 per cent belonged to Rs. > 3000 - 4000/- month and lastly only 9 per cent belonged to the income group of Rs. > 4000-5000/- month . Majority of the subjects i.e. 75 per cent did not present any known history of diabetes in their families while 25 per cent subjects had a known history of diabetes in the family. Data regarding the personal habits revealed that 50 per cent of the subjects did not smoke/chew or eat tobacco in pan or drink alcohol. While 3 per cent of the subjects had the habit of smoking, drinking and eating tobacco in pan. About 22 per cent including 6 per cent female subjects consumed tobacco in pan and 9 per cent had the habit of smoking and drinking while 3 per cent did smoke. Three per cent of the respondents had the habit of only drinking alcohol.

Table 2 : General profile of the subjects.

(n = 32)

| S.No. | Variable | Number | Per cent |
|-------|----------------------------|-----------------|----------|
| 1. | Age range : | | |
| | 30 - 35 years | 6 | 18.8 |
| | 36 - 45 years | 11 | 34.4 |
| | 46 - 55 years | 9 | 28.1 |
| | 56 - 65 years | 5 | 15.6 |
| | 66 and above | 1 | 3.1 |
| 2. | Sex | | |
| | Males | 19 | 59.4 |
| | Females | 13 | 40.6 |
| 3. | Occupation | | |
| | Sedentary work | 29 | 90.6 |
| | Moderate work | 3 | 9.4 |
| 4. | Income level | | |
| | Rs. 1000 - 2000/month | 12 | 37.5 |
| | Rs. > 2000 - 3000/month | 10 | 31.3 |
| | Rs. > 3000 - 4000/month | 7 | 21.9 |
| | Rs. > 4000 - 5000/month | 3 | 9.4 |
| 5. | Family history of Diabetes | | |
| | Present | 8 | 25.0 |
| | Absent | 24 | 75.0 |
| 7. | Personal habits | | |
| | Smoking | 1 | 3.1 |
| | Alcohol | 1 | 3.1 |
| | Tobacco/pan | 7 | 21.9 |
| | | (2 are females) | |
| | All the above | 4 | 12.5 |
| | None of the above | 16 | 50.0 |
| | Alcohol + smoking | 3 | 9.4 |

Mean age = 46.1 ± 10.1

Mean age at which disease occurred = 43.1 ± 7.94

Mean age of females at which disease occurred = 42.7 ± 7.52

Mean age of males at which disease occurred = 43.37 ± 8.41

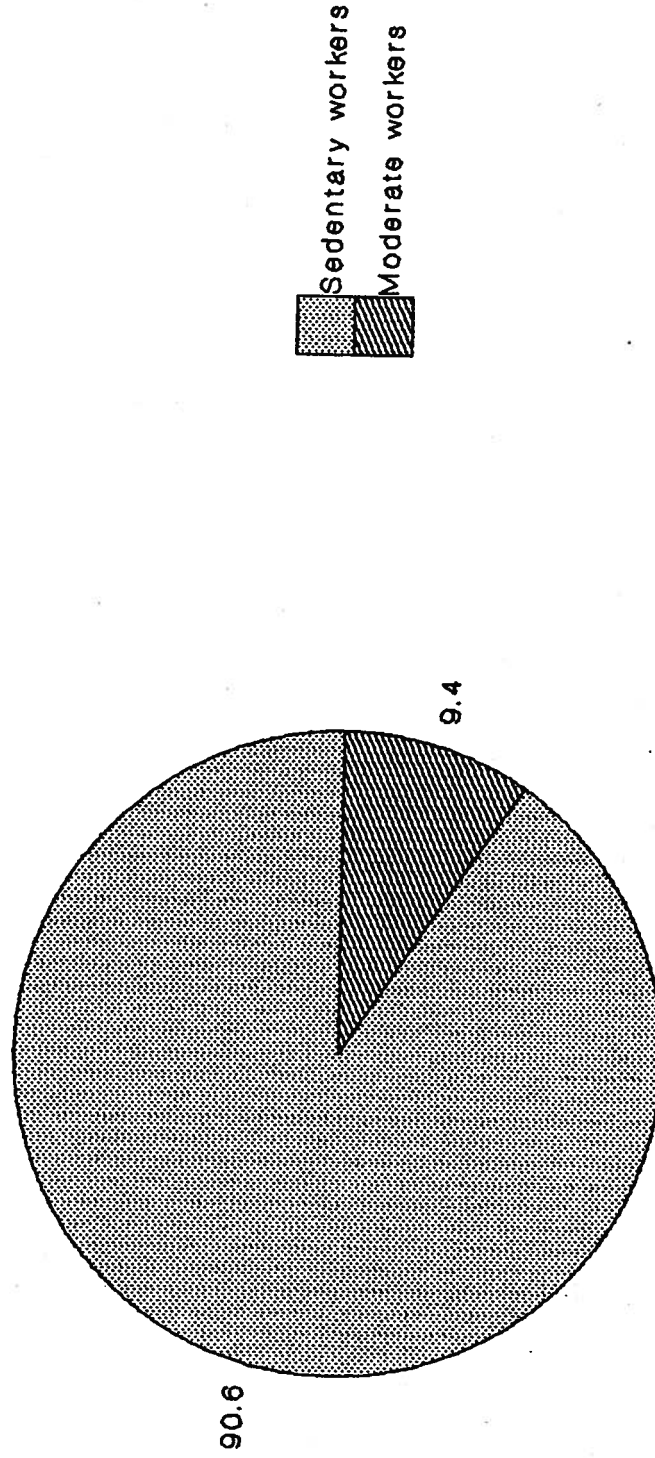


FIG.1: OCCUPATION OF THE SUBJECTS (%)

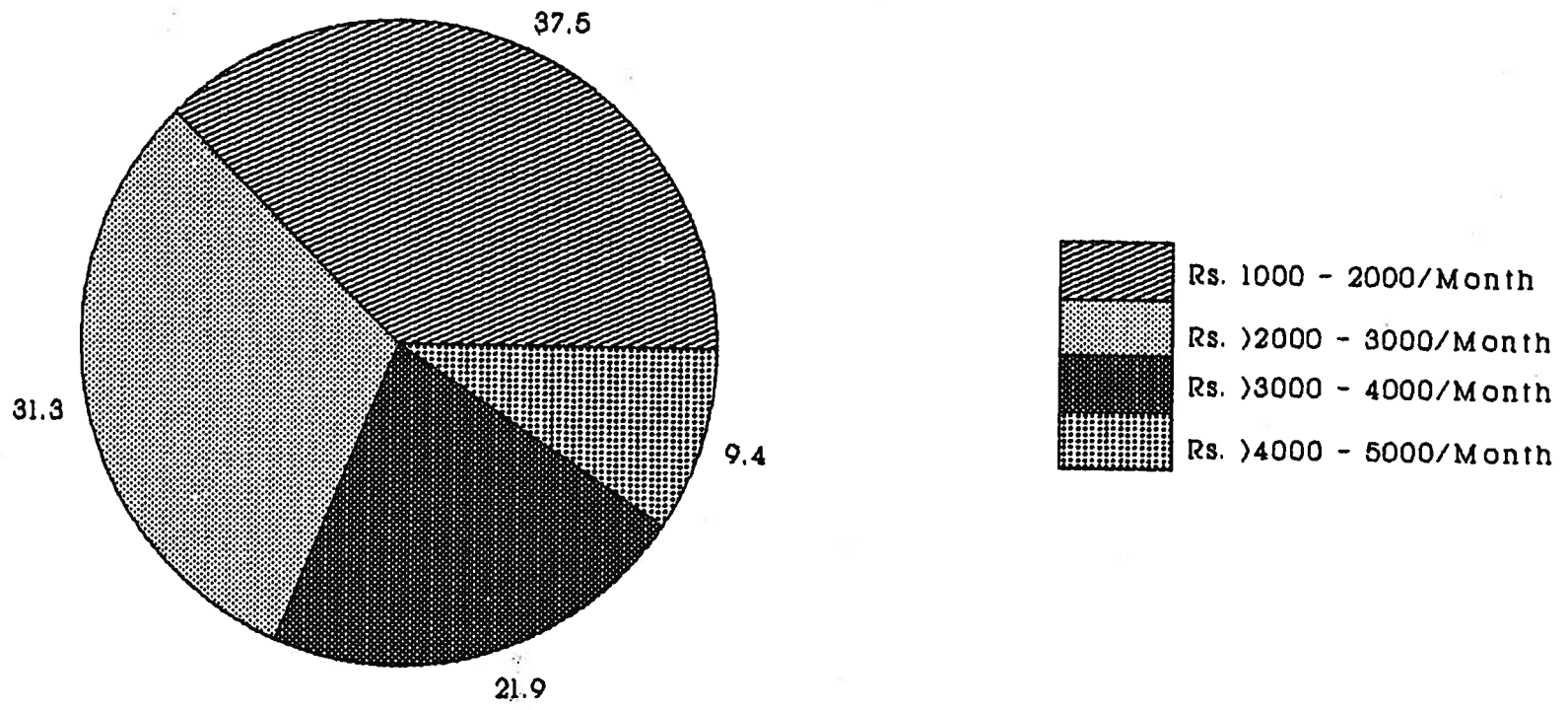


FIG.2: INCOME LEVEL OF THE SUBJECTS (%)

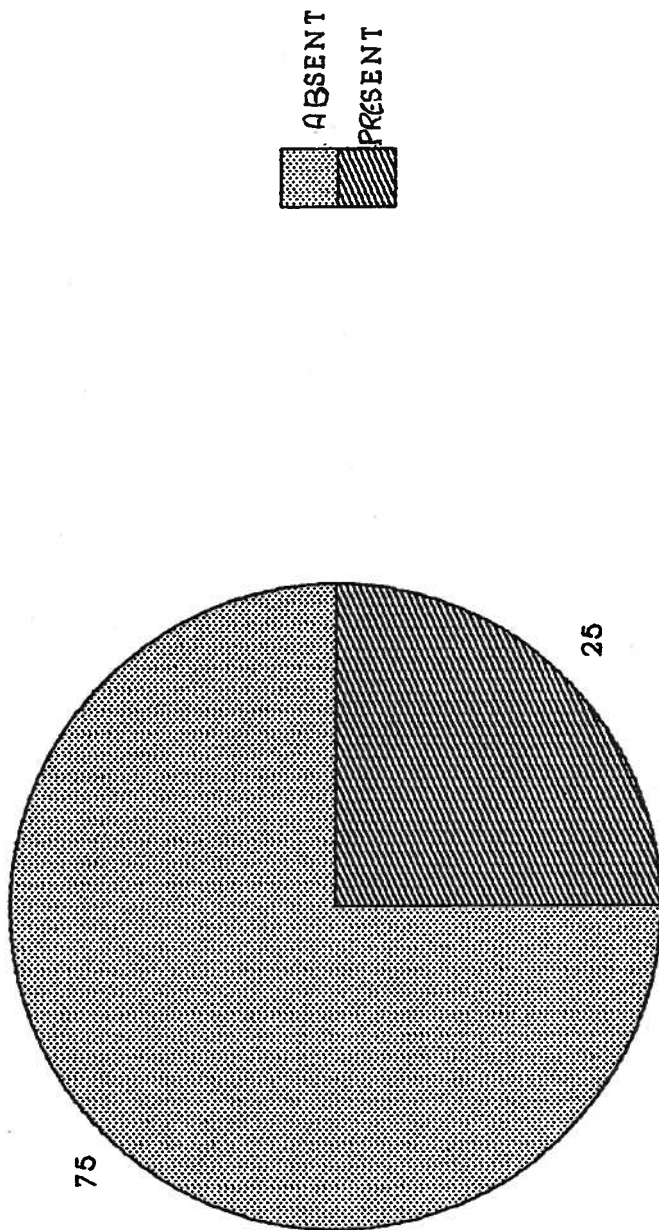


FIG.3: FAMILY HISTORY OF DIABETES (%)

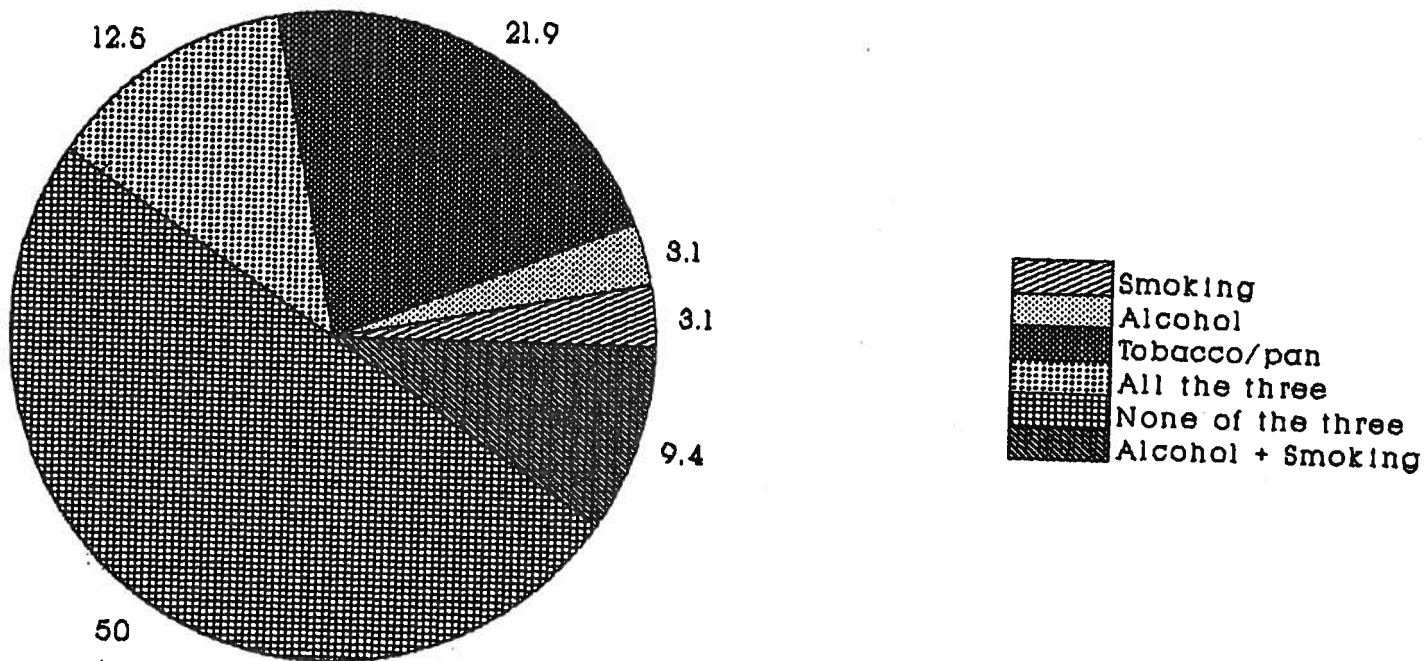


FIG.4: PERSONAL HABITS OF THE SUBJECTS (%)

4.2 DISEASE PROFILE OF THE SUBJECTS

Fifty per cent of the subjects were diagnosed as diabetic by routine check up while 47 per cent presented the symptoms such as polyphagia, polyuria, polydipsia and only 3 per cent were diagnosed by delayed wound healing. Duration of the disease after diagnosis ranged from < 6 months to > 5 years with majority of them (34%) belonging to < 6 months group indicating an increase in number of new cases being registered very recently. This is followed by > 5 years group (28%). And next large number was followed by < 1 year group (19%). Forty one per cent of the patients complained of the classical symptoms of polyphagia, polyuria and polydipsia while only 3 per cent had showed only polyphagia, ' other 3 per cent only polyuria and polydipsia. Weight loss was seen in 3 per cent while 6 per cent showed weight loss as well as polyphagia, polyuria and polydipsia which is an indication of the severity of hyperglycemia. Sixteen per cent complained of tingling and numbness of toes while only 3 per cent complained of polyphagia, polyuria, polydipsia, weight loss, tingling and numbness of toes and fingers.

Fifty per cent of the subjects were on oral hypoglycemic drugs and 22 per cent were exclusively on diet and were newly diagnosed, 6 per cent of newly

diagnosed diabetics were on diet as well as exercise. Sixteen per cent of the subjects were on insulin and diet, who comprise of mainly elderly patients and 6 per cent were on insulin, oral hypoglycemic drugs and diet. By the end of the study period 6 per cent totally stopped taking oral hypoglycemic drugs while 12 per cent decreased insulin dosage as well as oral hypoglycemic drugs. Number of visits to the hospital with complaints of hyperglycemia/hypoglycemia also decreased. Fifty nine per cent of the patients were given dietary advice by a qualified dietitian while 41 per cent were not given any dietary advice at the time of initial data collection which also included 28 per cent newly diagnosed patients. History of hypertension was present in 6 per cent which was under control at the time of the initial data collection as well as through out the study period.

The mean age of the subjects at which disease occurred was 43.1 ± 7.94 . Much difference was not seen in occurrence of disease for males and females with respect to age, as the mean age at which disease occurred for males was 43.37 ± 8.41 and females was 42.7 ± 7.52 years.

Table 3 : Disease profile of the subjects.

(n = 32)

| S.No. | Variable | Number | Per cent |
|-------|--|-------------------|----------|
| 1. | Duration of disease after diagnosis: | | |
| | < 6 months | 11 | 34.4 |
| | < 1 year | 6 | 18.8 |
| | < 2 years | 4 | 12.5 |
| | < 3 years | 2 | 6.3 |
| | 5 years or more | 9 | 28.1 |
| 2. | Symptoms | | |
| | Polyphagia | 14 | 43.8 |
| | Polyuria | 14 | 43.8 |
| | Polydipsia | 14 | 43.8 |
| | Tingling & numbness | 6 | 18.8 |
| | Weight loss | 6 | 18.8 |
| 3. | Mode of treatment | | |
| | Diet alone | 7 | 21.9 |
| | Diet + Exercise | 2 | 6.3 |
| | Diet + Oral hypoglycemic drugs | 16 | 50.0 |
| | Diet + Insulin | 5 | 15.6 |
| | Diet + Insulin + Oral hypoglycemic drugs | 2 | 6.3 |
| 4. | Dietary advice | | |
| | Given | 19 | 59.4 |
| | Not given | 13 | 40.6 |
| 5. | Associated disorders | | |
| | Hypertension | 2 (under control) | 6.3 |
| | Others | - | - |
| 6. | Diagnosed by | | |
| | Routine check up | 16 | 50.0 |
| | Polyphagia, polyuria, polydipsia | 15 | 46.9 |
| | Delayed wound healing | 1 | 3.1 |

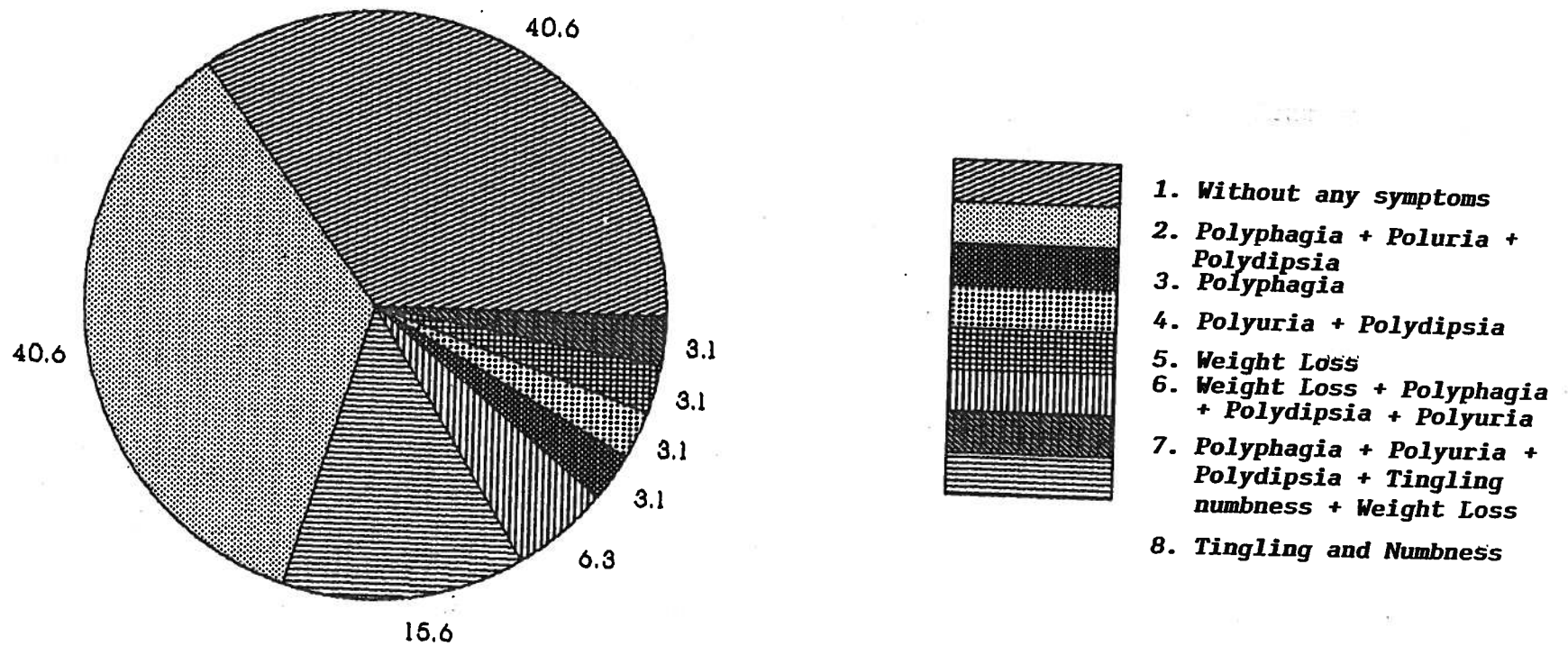


FIG.5: DISTRIBUTION PATTERN OF THE SYMPTOMS (%)

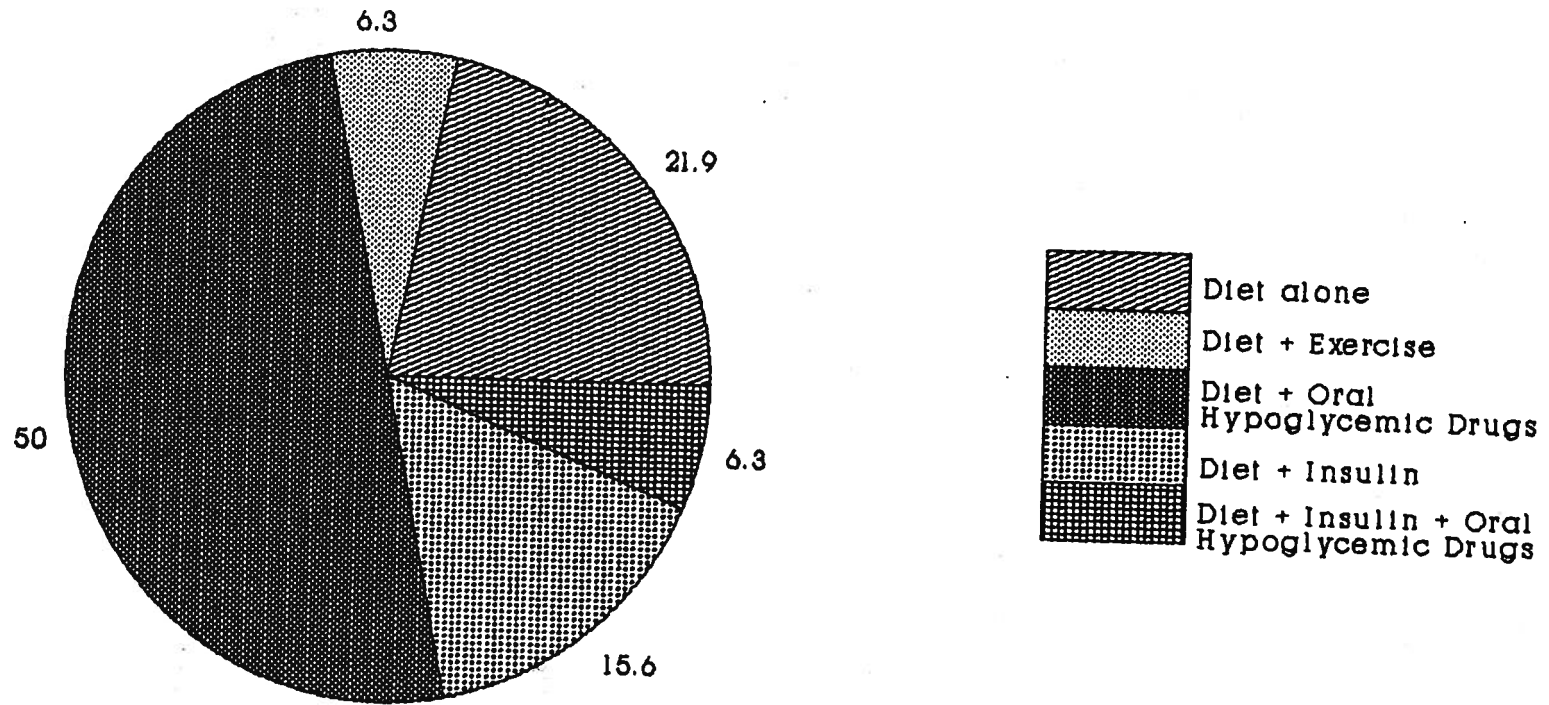


FIG.6: MODE OF TREATMENT (%)

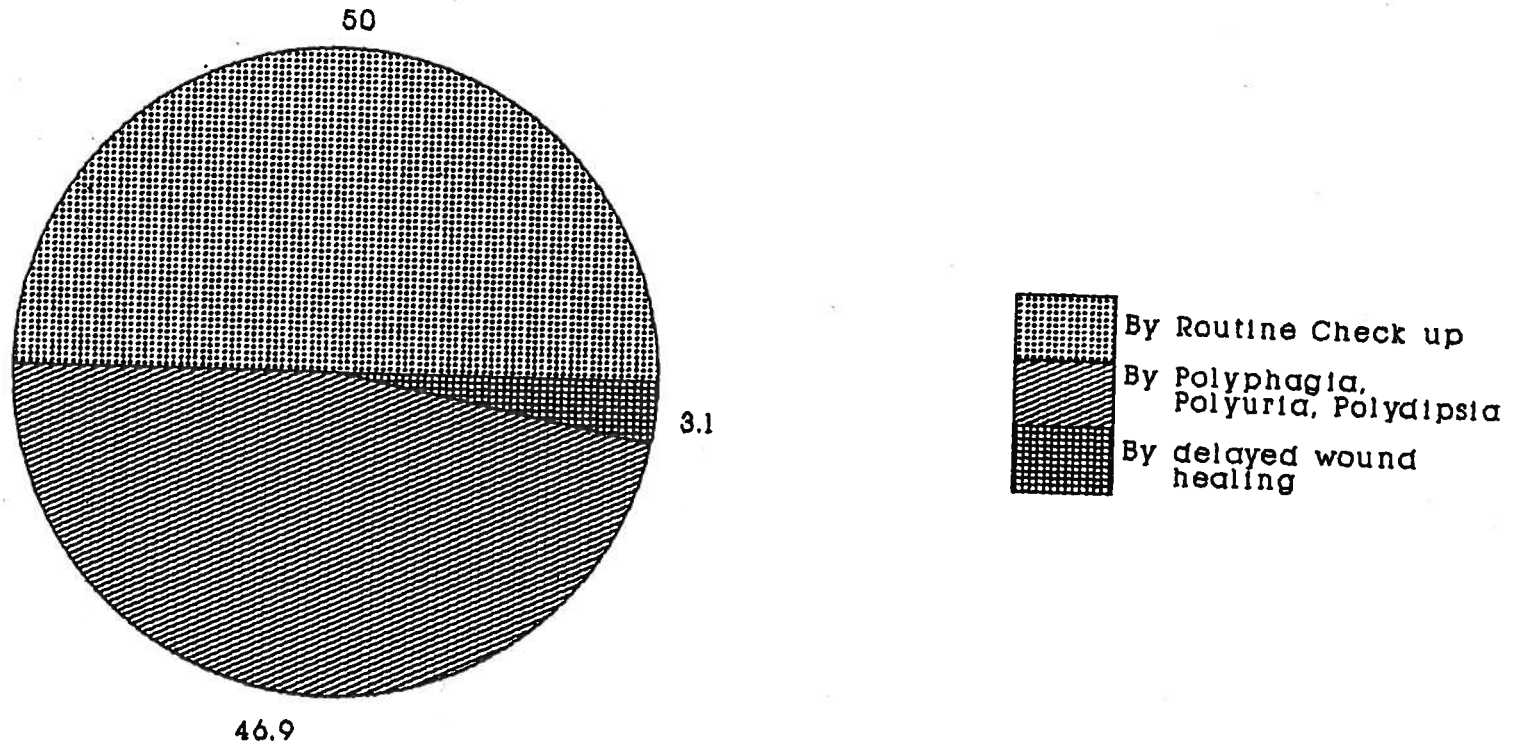


FIG.7: DIAGNOSIS OF THE DIABETES (%)

Table 4 : Distribution pattern of symptoms among the selected subjects

| S.No. | Variable | Number | Per cent |
|-------|--|--------|----------|
| 1. | Without any symptoms | 13 | 40.6 |
| 2. | Polyphagia + polyuria + polydipsia | 13 | 40.6 |
| 3. | Polyphagia | 1 | 3.1 |
| 4. | Polyuria + polydipsia | 1 | 3.1 |
| 5. | Weight loss | 1 | 3.1 |
| 6. | Weight loss + polyphagia + polyuria + polydipsia | 2 | 6.3 |
| 7. | Polyphagia + polyuria + polydipsia + tingling numbness + weight loss | 1 | 3.1 |
| 8. | Tingling and numbness | 5 | 15.6 |

4.3 BLOOD SUGAR VALUES OF THE SUBJECTS

Before education intervention, out of 32 selected subjects only 9 per cent were having their fasting blood sugar level within the normal range (60-120 mg%). The fasting blood sugar values of 91 per cent of the patients were more than normal. Among them, 63 per cent of the patients were having their fasting blood sugar levels ranging from 125-200 mg % and rest of the 28 per cent had values more than 200 mg % to 280 mg %. After education intervention 81 per cent of the patients were having normal fasting values while 19 per cent were having more than normal values ranging from 125-180 mg %.

Before education intervention except 3 per cent of the patients, rest of the 97 per cent had more than normal post prandial blood sugar values. Majority of the patients (53%) had their post-prandial blood sugar values ranging from 168-300 mg % while rest of the 44 per cent had high values ranging from 300-399 mg %. After education intervention the number decreased and a total of 63 per cent showed normal post-prandial blood sugar values by the end of the study, while 37 per cent of the subjects still had more than normal post-prandial blood sugar values ranging from 165-240 mg %.

The mean initial fasting sugar levels of the patients was 180 ± 48.75 mg % while post-prandial blood sugar level was 297.6 ± 84.68 mg %. Final mean fasting blood sugar level was 103.13 ± 24.52 mg % while mean post-prandial blood sugar level was 155.88 ± 35.997 . The mean decrease in fasting blood sugar value was 76.41 ± 45.74 mg % and that of post-prandial blood sugar value was 135.72 ± 83.03 mg %. A significant decrease in blood sugar levels was found at 1 per cent when paired 't' test was applied, which indicates that present education intervention was quite effective in regulating blood sugar values.

Change in body weight and body mass index of the subjects

Change in body weight and body mass index are presented in table 5 and 6 respectively. Body weight of the patients ranged from minimum 40 to maximum 80 kgs before education intervention and 42 kgs to 79 kgs after education. Body mass index of the patients ranged from minimum 1.719 to maximum 3.672 before education intervention and 1.805 to 3.626 after education. According to the criteria of classifying 20 per cent above ideal body weight as over weight and 20 per cent below ideal body weight as underweight (Raghuram, 1991), 25 per cent belonged to over weight category excluding 9 per cent who were more than 35 per

cent above over weight category. Rest of the patients were in the acceptable category. However, 6 per cent were very near to under weight category (i.e. 20 per cent less than their ideal body weight). Same pattern was seen in body mass index of the patients, 25 per cent belonged to over weight category (over weight starts from 2.4 body mass index onwards) and 9 per cent obese patients (obesity starts from 3 body mass index onwards and is an indication for increased mortality rate). Rest of the patients belonged to acceptable range excluding 26 per cent who belonged to under weight category (under weight starts from 1.8 body mass index downwards Bray, (1978)

In the present study patients were divided into 2 categories - over weight (more than desirable body weight) and under weight (less than desirable body weight). Patients requiring decrease in weight were 69 per cent and patients requiring increase in weight were 25 per cent while remaining 6 per cent were already maintaining desirable body weight and does not require any change. Out of 69 per cent requiring decrease in weight, 31 per cent required to decrease more than 10 kgs, while 9 per cent needed to decrease 1-2 kgs weight and 28 per cent of the subjects required to reduce their weight by 3-9 kgs. Patients requiring increase in weight needed to increase about 5 to 11 kgs.

The mean weight of patients before education intervention was 60.91 ± 9.46 kgs and after education intervention was 59.83 ± 8.65 kgs. A mean decrease in weight by 1.932 ± 1.003 kgs weight and a mean increase of 1.5 ± 1 kg was observed by the end of the study. The mean body mass index of the patients was 2.391 ± 0.44 before education intervention and 2.315 ± 0.4 after education intervention. A mean decrease of 0.164 ± 0.26 in body mass index and a mean increase of 0.063 ± 0.020 was observed by the end of the study. However patients belonged to same categories of body weight and body mass index as before, after education intervention. Paired 't' tests on overall change in body weight towards positive side (decrease/increase) gave a significant result at 1 per cent level (4.92^{**}) while increase in body weight was significant at 5 per cent level (4.24^*) and decrease in body weight was significant at 1 per cent level (9.034^{**}).

Overall body mass index towards positive side was not significant when paired 't' test was applied, even though there was a slight decrease/increase in the body mass index of the patients.

Table 5 : Blood sugar values of the subjects before and after education intervention (mg/100 ml)

| Patient S.No. | Before education intervention | | After education intervention | |
|------------------|----------------------------------|-----------------|---------------------------------|---------------|
| | FBS | PPBS | FBS | PPBS |
| 01 | 230 | 360 | 90 | 124 |
| 02 | 120 | 168 | 86 | 176 |
| 03 | 140 | 200 | 80 | 125 |
| 04 | 125 | 225 | 80 | 115 |
| 05 | 210 | 300 | 100 | 135 |
| 06 | 140 | 210 | 100 | 135 |
| 07 | 160 | 200 | 100 | 140 |
| 08 | 100 | 200 | 90 | 120 |
| 09 | 190 | 232 | 130 | 174 |
| 10 | 140 | 220 | 90 | 140 |
| 11 | 152 | 224 | 100 | 134 |
| 12 | 200 | 399 | 136 | 240 |
| 13 | 220 | 399 | 100 | 160 |
| 14 | 156 | 240 | 100 | 130 |
| 15 | 140 | 260 | 60 | 156 |
| 16 | 138 | 226 | 140 | 210 |
| 17 | 200 | 399 | 110 | 164 |
| 18 | 110 | 158 | 74 | 143 |
| 19 | 200 | 399 | 120 | 184 |
| 20 | 200 | 360 | 125 | 200 |
| 21 | 225 | 399 | 69 | 180 |
| 22 | 260 | 380 | 90 | 100 |
| 23 | 280 | 399 | 180 | 240 |
| 24 | 165 | 285 | 90 | 112 |
| 25 | 194 | 296 | 100 | 165 |
| 26 | 138 | 194 | 98 | 158 |
| 27 | 200 | 339 | 105 | 128 |
| 28 | 280 | 399 | 120 | 172 |
| 29 | 151 | 340 | 140 | 194 |
| 30 | 210 | 374 | 80 | 100 |
| 31 | 248 | 399 | 112 | 180 |
| 32 | 133 | 198 | 115 | 154 |
| Mean | 180.0 ± 48.8 | 297.6 ± 84.7 | 103.1 ± 24.5 | 155.9 ± 36 |

Normal levels : F.B.S. : 60 - 120 mg %
P.P.B.S. : 110 - 160 mg %

Mean decrease of F.B.S. : 76.4 ± 45.7
Mean decrease of P.P.B.S.: 135.7 ± 83.0

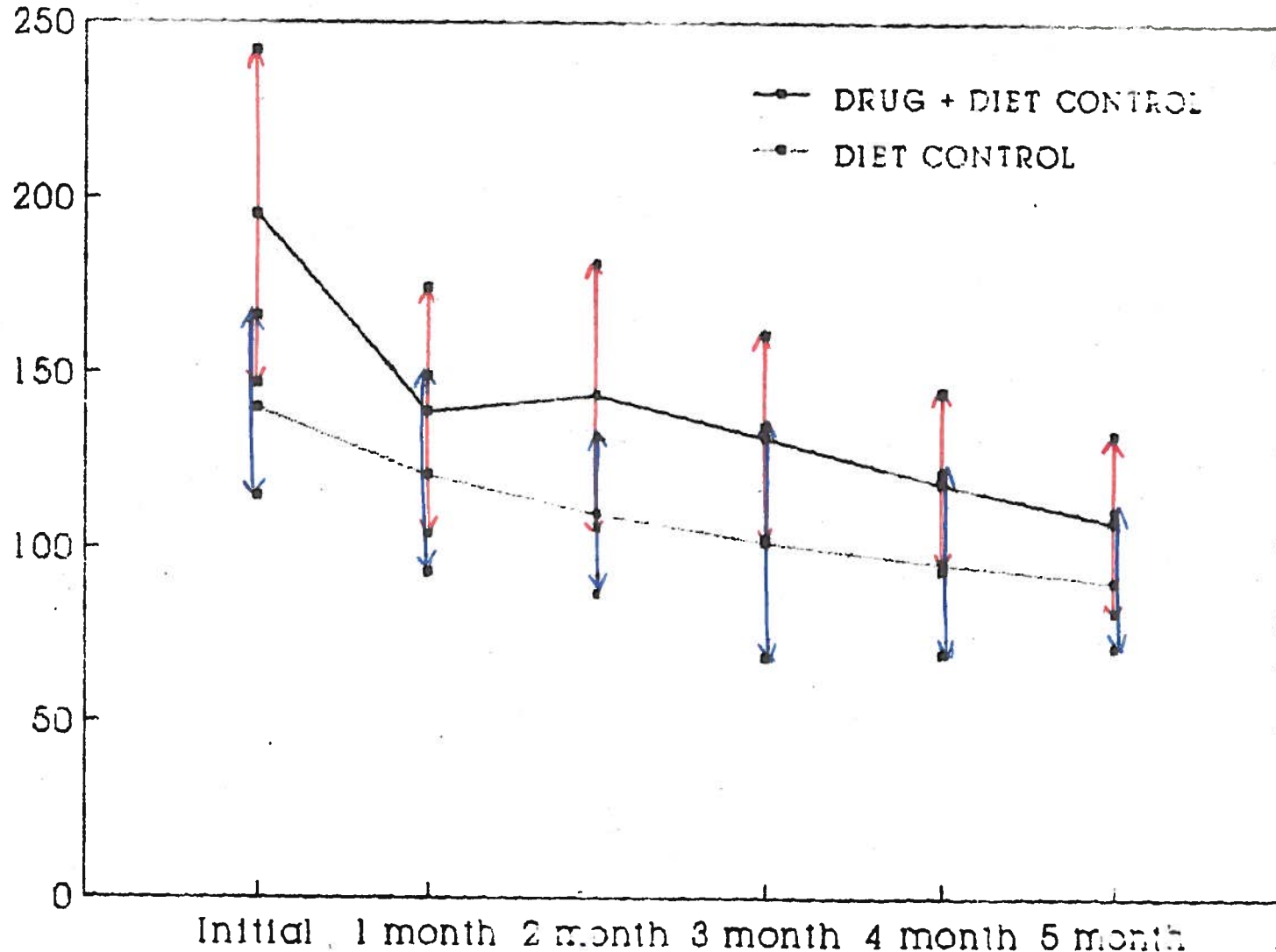


FIG. 9 FASTING BLOOD SUGAR VALUES OF THE DIABETIC SUBJECTS WITH DRUG + DIET AND DIET CONTROL TREATMENT

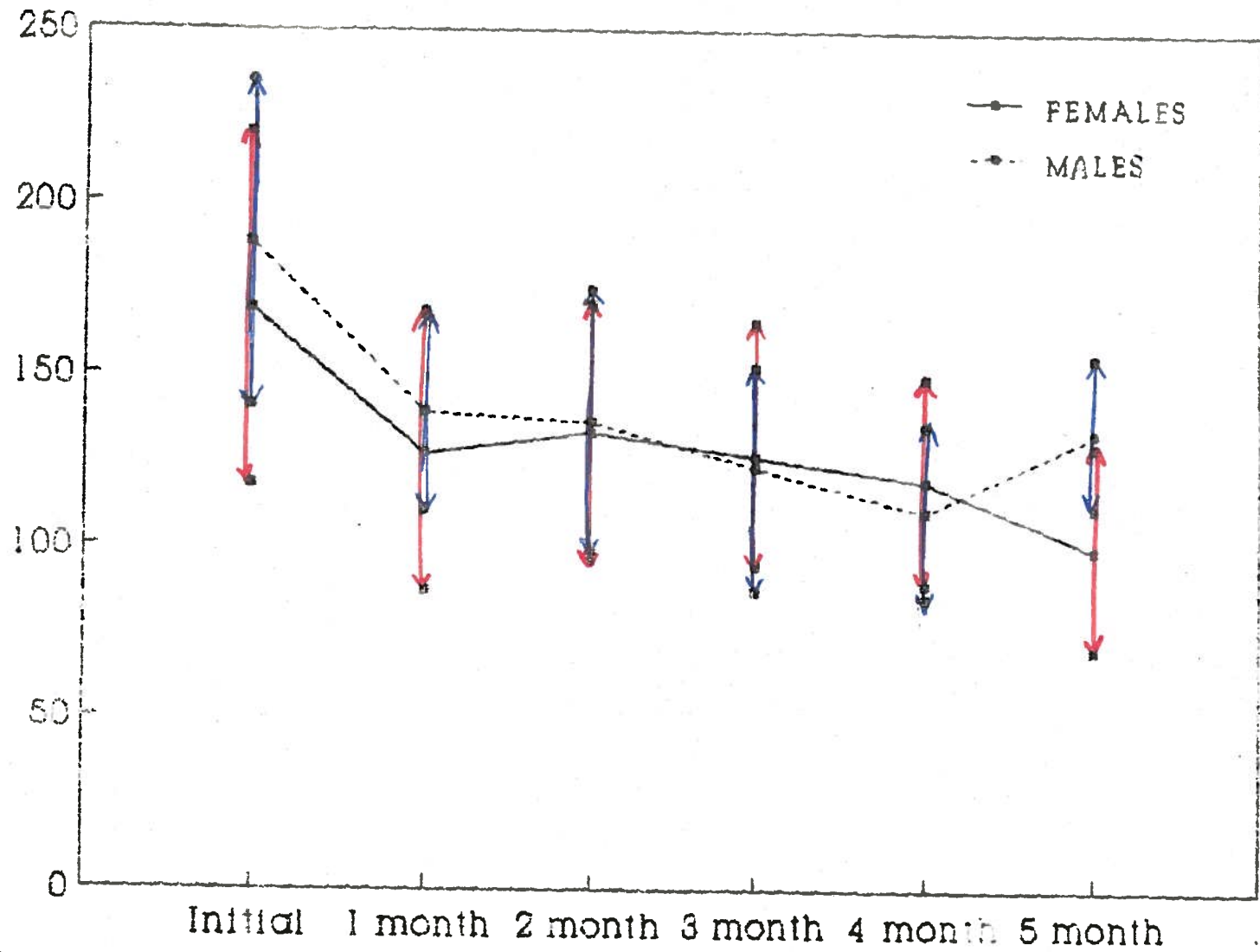
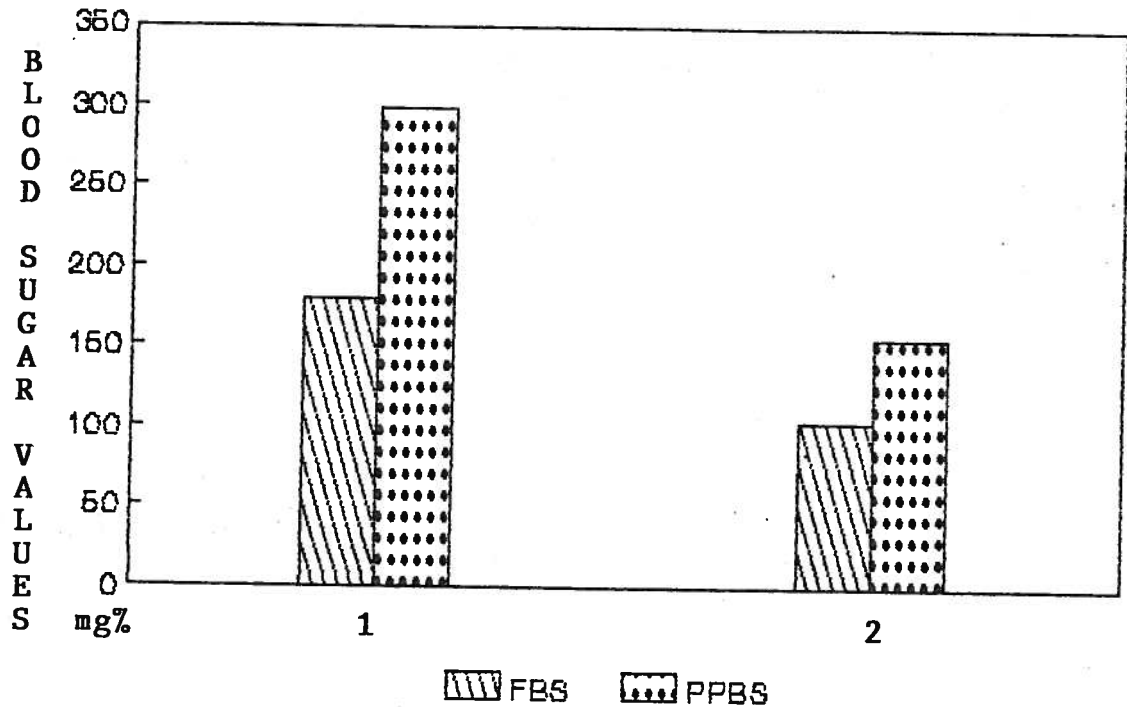


FIG. 10 FASTING BLOOD SUGAR LEVELS OF FEMALE AND MALE SUBJECTS



- 1. Before Education Intervention
- 2. After Education Intervention

FIGURE : 8 : MEAN BLOOD SUGAR VALUES OF THE SUBJECTS BEFORE AND AFTER EDUCATION INTERVENTION

Table 6 : Change in body weights of the subjects before and after education intervention.

| Patient S.No. | Height (cm) | Weight(kg) before education intervention | Weight (kg) after education intervention | Desirable weight (kg) |
|---------------|-------------|--|--|-----------------------|
| 01 | 162 | 50 | 53 | 55.8 |
| 02 | 172 | 70 | 68 | 64.8 |
| 03 | 165 | 60 | 59 | 58.5 |
| 04 | 165 | 75 | 72 | 58.5 |
| 05 | 155 | 51 | 51 | 49.5 |
| 06 | 148 | 55 | 54 | 42.8 |
| 07 | 165 | 52 | 53 | 58.5 |
| 08 | 160 | 58 | 56 | 54.0 |
| 09 | 158 | 66 | 64 | 52.2 |
| 10 | 158 | 73 | 71 | 51.3 |
| 11 | 173 | 54 | 56 | 65.3 |
| 12 | 180 | 79 | 76 | 72.0 |
| 13 | 163 | 55 | 55 | 56.3 |
| 14 | 148 | 80 | 79 | 42.8 |
| 15 | 150 | 50 | 48 | 45.0 |
| 16 | 153 | 68 | 64 | 47.7 |
| 17 | 160 | 58 | 55 | 54.0 |
| 18 | 160 | 63 | 63 | 54.0 |
| 19 | 153 | 74 | 72 | 47.7 |
| 20 | 152 | 61 | 58 | 46.8 |
| 21 | 153 | 40 | 42 | 47.3 |
| 22 | 173 | 60 | 62 | 65.3 |
| 23 | 163 | 55 | 55 | 56.3 |
| 24 | 150 | 48 | 46 | 45.0 |
| 25 | 160 | 60 | 58 | 54.0 |
| 26 | 143 | 65 | 63 | 38.3 |
| 27 | 163 | 70 | 69 | 56.3 |
| 28 | 164 | 52 | 54 | 57.6 |
| 29 | 168 | 60 | 60 | 60.3 |
| 30 | 150 | 58 | 55 | 45.0 |
| 31 | 172 | 64 | 64 | 64.8 |
| 32 | 170 | 65 | 64 | 63.0 |
| Mean : | | 60.9 ± 9.5 | 59.8 ± 8.7 | |

Mean decrease in weight : 1.9 ± 1.0

Mean increase in weight : 1.5 ± 1.0

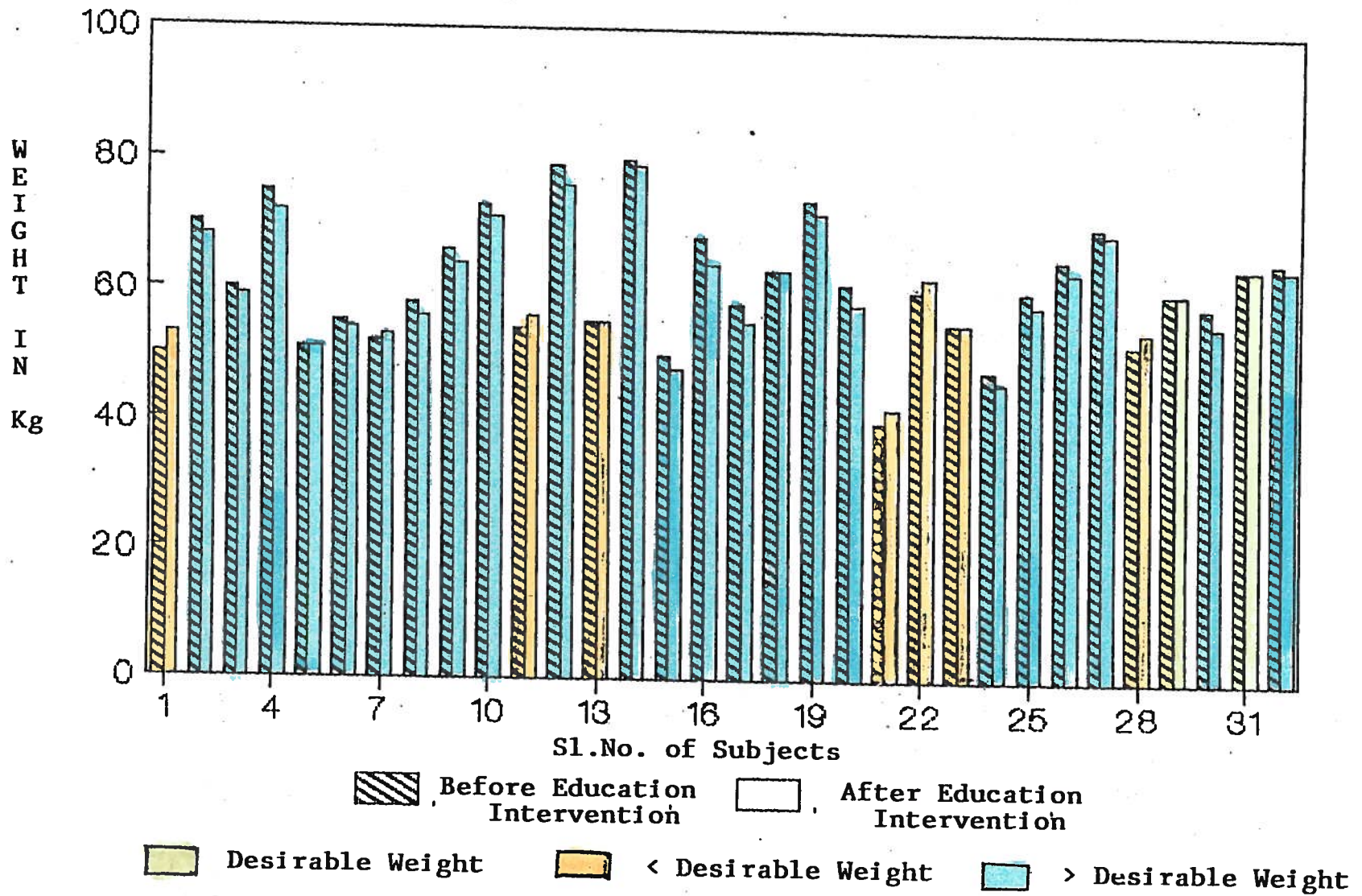


FIGURE : 11 : CHANGE IN BODY WEIGHT OF THE SUBJECTS

Table 7 : Body Mass Index of the subject before and after education intervention

| Patient S.No. | Height (cm) | Initial weight (kg) | Before education intervention (BMI) | After education intervention (BMI) | State of the BMI at the end of the study |
|---------------|-------------|---------------------|-------------------------------------|------------------------------------|--|
| 01 | 162 | 50 | 1.905 | 2.019 | acceptable |
| 02 | 172 | 70 | 2.366 | 2.298 | acceptable |
| 03 | 165 | 60 | 2.203 | 2.167 | acceptable |
| 04 | 165 | 75 | 2.754 | 2.644 | over weight |
| 05 | 155 | 51 | 2.122 | 2.122 | acceptable |
| 06 | 148 | 55 | 2.528 | 2.482 | over weight |
| 07 | 165 | 52 | 1.91 | 1.947 | acceptable |
| 08 | 160 | 58 | 2.265 | 2.187 | acceptable |
| 09 | 158 | 66 | 2.643 | 2.563 | over weight |
| 10 | 158 | 73 | 2.942 | 2.821 | over weight |
| 11 | 173 | 54 | 1.814 | 1.881 | under weight |
| 12 | 180 | 79 | 2.438 | 2.345 | acceptable |
| 13 | 163 | 55 | 2.08 | 2.08 | acceptable |
| 14 | 148 | 80 | 3.672 | 3.626 | obese |
| 15 | 150 | 50 | 2.222 | 2.133 | acceptable |
| 16 | 153 | 68 | 2.904 | 2.733 | over weight |
| 17 | 160 | 58 | 2.265 | 2.148 | acceptable |
| 18 | 160 | 63 | 2.460 | 2.460 | over weight |
| 19 | 153 | 74 | 3.161 | 3.075 | obese |
| 20 | 152 | 61 | 2.640 | 2.510 | over weight |
| 21 | 153 | 40 | 1.719 | 1.805 | under weight |
| 22 | 173 | 60 | 2.016 | 2.083 | acceptable |
| 23 | 163 | 55 | 2.082 | 2.082 | acceptable |
| 24 | 150 | 48 | 2.133 | 2.044 | acceptable |
| 25 | 160 | 60 | 2.343 | 2.265 | acceptable |
| 26 | 143 | 65 | 3.2 | 3.102 | obese |
| 27 | 163 | 70 | 2.650 | 2.613 | over weight |
| 28 | 164 | 52 | 1.933 | 2.007 | acceptable |
| 29 | 168 | 60 | 2.138 | 2.174 | acceptable |
| 30 | 150 | 58 | 2.577 | 2.444 | acceptable |
| 31 | 172 | 64 | 2.163 | 2.095 | acceptable |
| 32 | 170 | 65 | 2.249 | 2.214 | acceptable |
| Mean : | | | 2.391 ± 0.44 | 2.315 ± 0.4 | |

Mean decrease in body mass index of over weight persons : 0.164 ± 0.26
Mean increase in body mass index of under weight persons: 0.063 ± 0.020

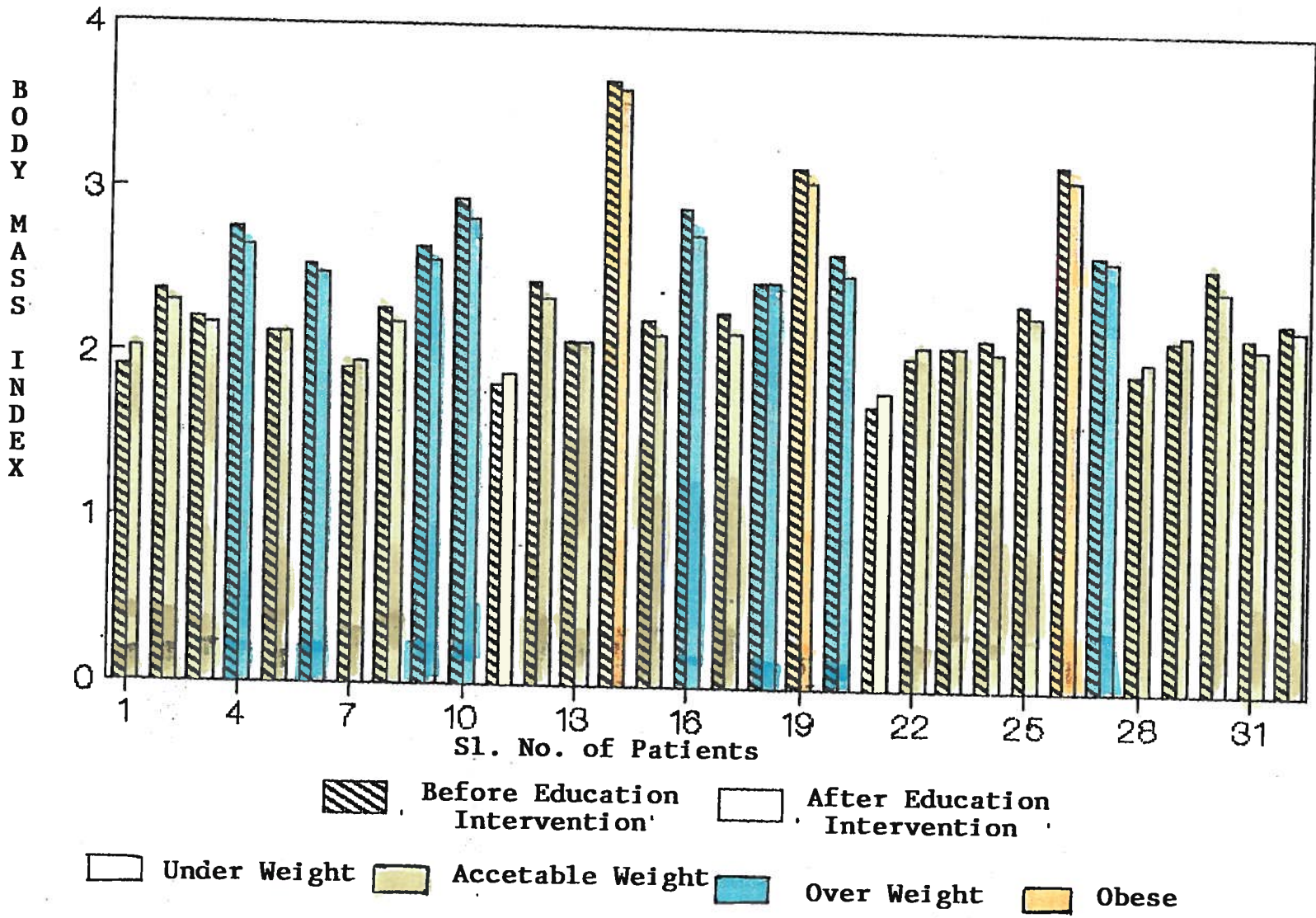


FIGURE : 12 : CHANGE IN BODY MASS INDEX OF THE SUBJECTS

4.5 FOOD AND NUTRIENT INTAKE OF THE CONTROL AND EXPERIMENTAL GROUPS (SUB SAMPLE)

Cooked food was measured by weight method, from which nutrients were calculated and tabulated (Table 10).

Food intake of the control group reveals a maximum intake of cereals comprising of rice, wheat and ragi followed by vegetables and curds. Flesh foods were used sparingly while leafy vegetables, roots and tubers were not used at all. Milk, pulses and fruits were used in moderate amounts.

In experimental group also similar results were observed, with cereals forming the major contributor to total energy, followed by vegetables. Milk, curds, leafy vegetables and pulses were used in moderate amounts followed by fruits. Flesh foods, roots and tubers were used sparingly. Intake of deep fried foods was 76 grams in control group whereas the experimental group consumed 25 grams.

Both the experimental as well as control groups showed an average intake of 65 gms protein per day. Carbohydrate and fat intakes were slightly greater in control group, when compared with experimental group. Fibre contribution was slightly

greater in experimental group compared to control group. However, the amount of fibre contributed by food in both the groups was less when compared to recommended allowance of 40 gms intake of fibre per day.

Total energy contribution was more in control group compared to experimental group.

Per cent of calories derived from carbohydrates were similar in both the groups. The contribution of proteins to calories was slightly more in experimental group. Fat contribution towards calories was more in control group when compared with experimental group. Both the groups however, were below the recommended contribution.

4.6 KNOWLEDGE TEST-SCORE

By the end of the study almost all patients were able to identify restricted foods, fibre rich foods and were able to tell importance of controlled portion size and meal timings. They were also able to measure food accurately and differentiate between refined and complex foods. In addition patients were able to do urine testing by Benedicts method and were able to tell the steps to be taken during an occurrence of hypoglycemia. Sixty three per cent of the subjects

were able to tell that blood, urine, sugar testing and oral glucose tolerance test are done to diagnose diabetes while 38 per cent could not tell about oral glucose tolerance test. All patients could relate uncontrolled diabetes to complications of the eye, heart, kidney and nervous system.

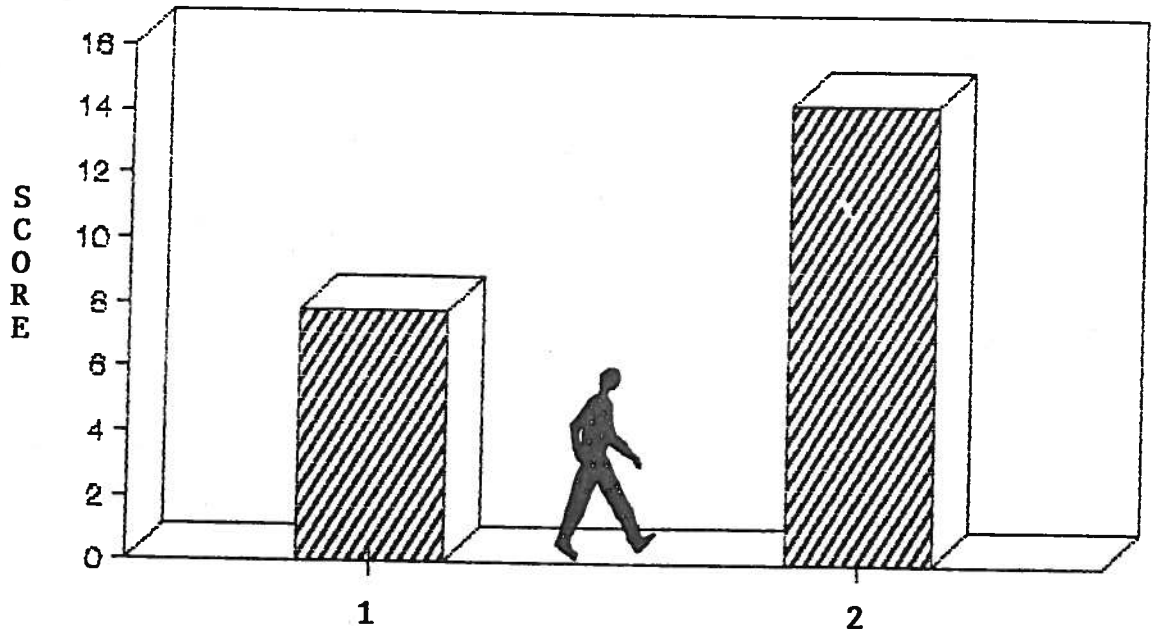
Before education intervention, the knowledge scores of the subjects ranged from 3-11 marks while after education intervention subjects scored higher marks ranging from 10-15 marks. The mean score observed before education was 7.78 ± 2.15 whereas the mean score obtained after education intervention was 14.28 ± 1.17 . A mean increase of 6.56 ± 2.45 was observed. Paired 't' test was applied and a highly significant difference was found at 1 per cent level indicating almost 100 per cent gain in knowledge of the patients.

...

Table 8 : Knowledge scores of the subjects before and after education intervention

| Patient S.No. | Score before education intervention | Score after education intervention |
|---------------|-------------------------------------|------------------------------------|
| 01 | 7 | 14 |
| 02 | 5 | 15 |
| 03 | 11 | 15 |
| 04 | 11 | 15 |
| 05 | 4 | 15 |
| 06 | 5 | 10 |
| 07 | 7 | 12 |
| 08 | 7 | 15 |
| 09 | 4 | 15 |
| 10 | 9 | 15 |
| 11 | 10 | 14 |
| 12 | 8 | 15 |
| 13 | 7 | 15 |
| 14 | 6 | 15 |
| 15 | 9 | 13 |
| 16 | 8 | 14 |
| 17 | 11 | 14 |
| 18 | 9 | 14 |
| 19 | 9 | 15 |
| 20 | 8 | 15 |
| 21 | 7 | 15 |
| 22 | 9 | 15 |
| 23 | 9 | 15 |
| 24 | 8 | 15 |
| 25 | 7 | 13 |
| 26 | 8 | 15 |
| 27 | 10 | 13 |
| 28 | 10 | 13 |
| 29 | 8 | 13 |
| 30 | 10 | 15 |
| 31 | 3 | 15 |
| 32 | 5 | 15 |
| Mean | : 7.78 \pm 2.15 | 14.28 \pm 1.17 |

Mean increase in knowledge score : 6.56 ± 2.45



- 1. Before Education Intervention
- 2. After Education Intervention

FIGURE : 13 : MEAN INCREASE IN THE KNOWLEDGE SCORE OF THE SUBJECTS

Table 9 : Average intake of cooked foods (sub sample).

| Food groups(g) | Control group | Experimental group |
|-----------------------------|-------------------|--------------------|
| Cereals | 727 \pm 235.5 | 520.6 \pm 102.1 |
| Pulses | 95 \pm 55.8 | 77.3 \pm 50.5 |
| Leafy vegetables | 0 | 61.1 \pm 33.3 |
| Vegetables | 181.8 \pm 152.6 | 181.1 \pm 51.6 |
| Roots & tubers | 0 | 13.3 \pm 15.73 |
| Fruits (raw weight) | 93.3 \pm 84 | 85.6 \pm 69.9 |
| Flesh foods (raw weight) | 25 \pm 38.7 | 26.7 \pm 28.3 |
| Milk | 131.7 \pm 92.8 | 152.2 \pm 81.5 |
| Curds | 183.3 \pm 93.1 | 128.9 \pm 51.4 |
| Deep fried foods | 76.7 \pm 68.1 | 25 \pm 55 |

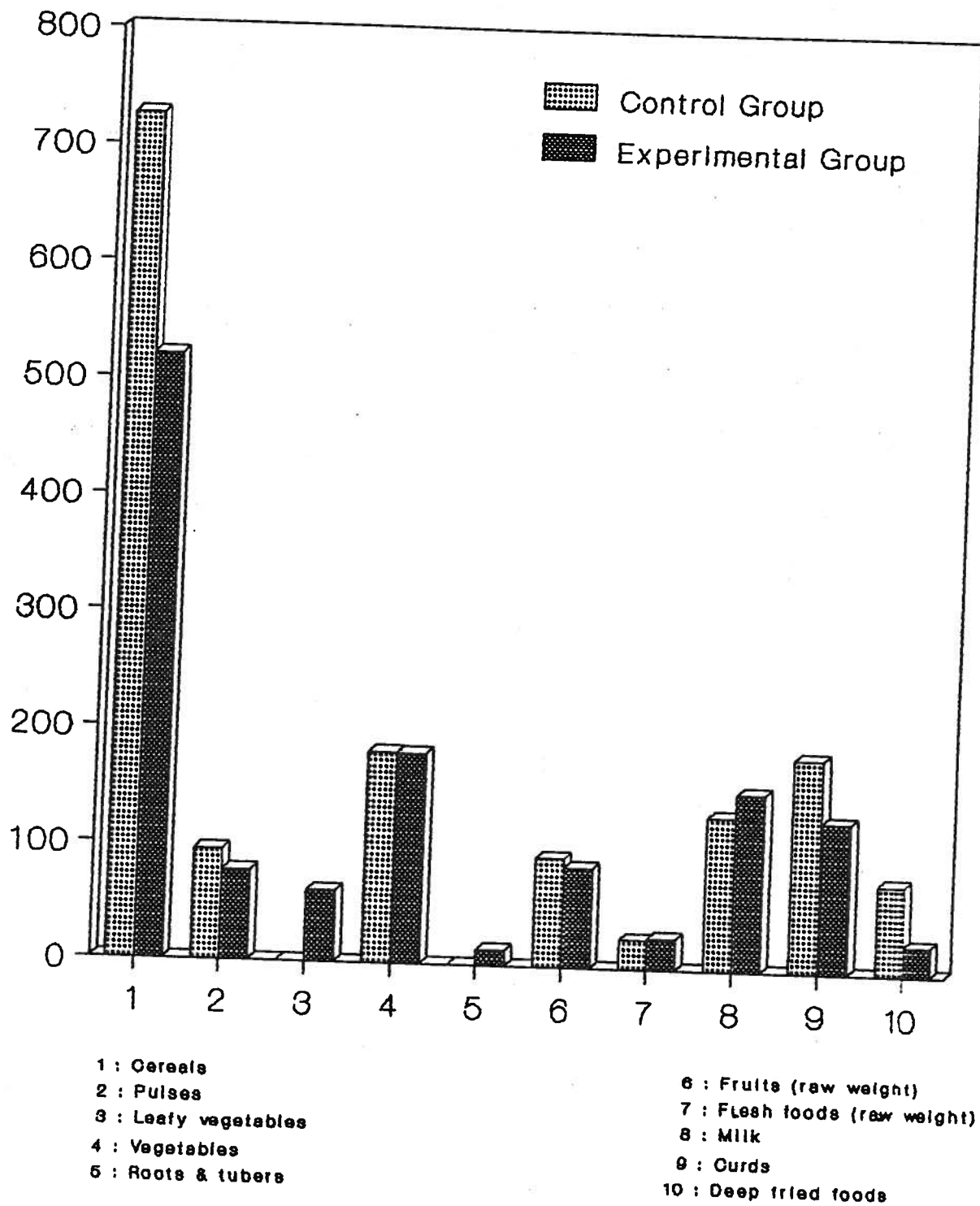


FIG.14: AVERAGE INTAKE OF COOKED FOODS (sub sample)

Table 10: Average intake of Nutrients (Sub sample).

| Nutrients | Control group | Experimental group |
|----------------------------|---------------------|----------------------|
| Protein (g) | 65.03 \pm 33 | 65.99 \pm 13.53 |
| Carbohydrates(g) | 359.6 \pm 106.8 | 312.59 \pm 92.86 |
| Fat (g) | 51.1 \pm 43.5 | 40.07 \pm 13.23 |
| Fibre (g) | 5.86 \pm 1.61 | 7.31 \pm 2.98 |
| Total Energy (Calories) | 2158.84 \pm 950.7 | 1874.86 \pm 544.63 |

Table 11 : Percentage of calories derived from the nutrients (sub sample).

| Nutrients | Control group % | Experimental group % | Recommended values % |
|---------------|-----------------|----------------------|----------------------|
| Protein | 12.05 | 14.07 | 15-20 |
| Carbohydrates | 66 | 66.7 | 55-60 |
| Fat | 21.32 | 19.23 | < 30 |
| Fibre | 5.76 g | 7.28 g | 40 g |

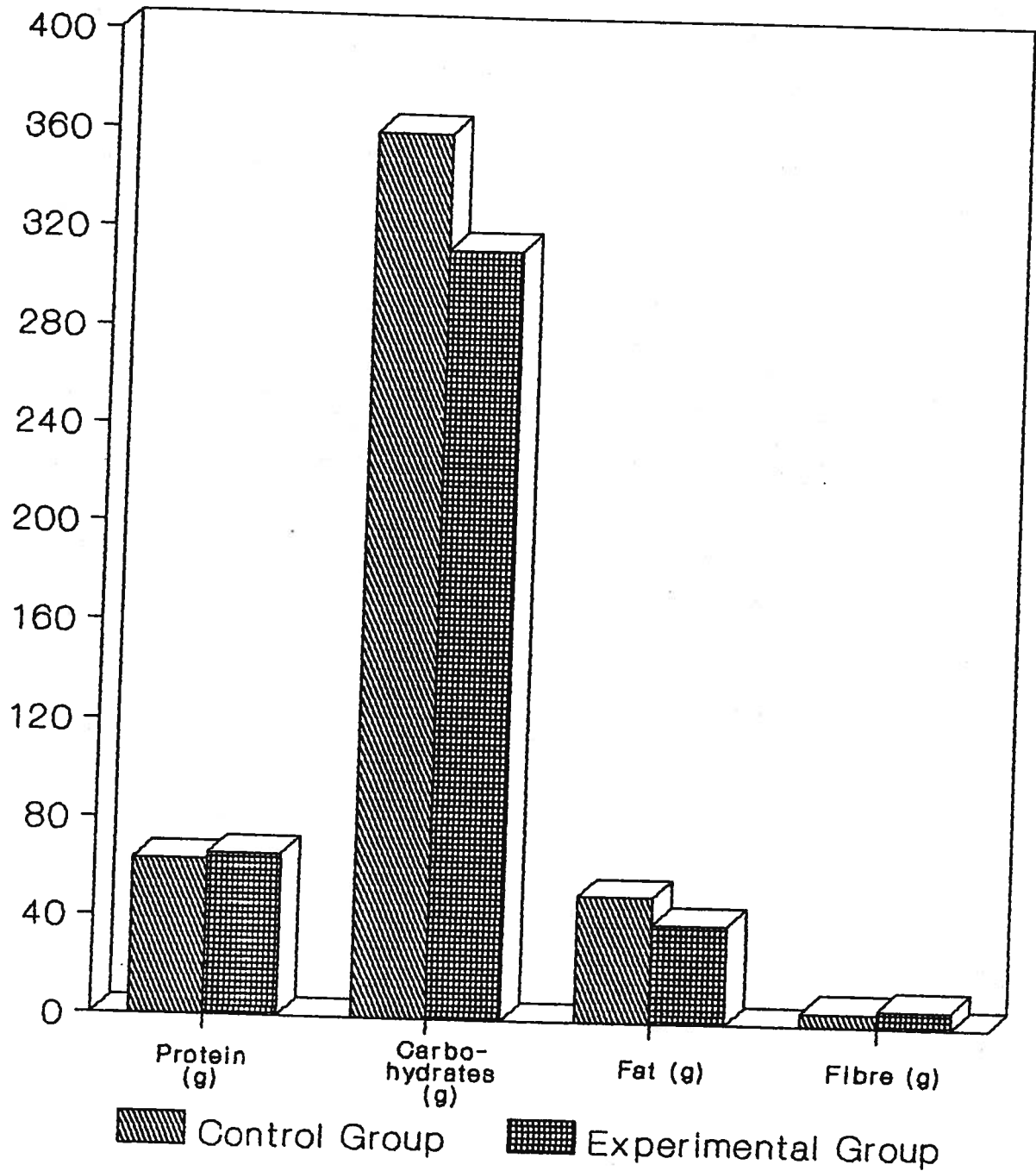


FIG.15: AVERAGE INTAKE OF NUTRIENTS (sub sample)

DISCUSSION

CHAPTER V

DISCUSSION

Present study was designed to evaluate the impact of education intervention in the dietary management of 32 diabetic subjects, attending the out patient clinic in Osmania General Hospital. The education programme was carried out for a period of 6 months with regular counselling sessions with the subjects. Special education material such as booklet were developed. Diet displays and planning of appropriate diets for diabetics was also demonstrated. At the end of the education programme, the impact was evaluated based on the improvements and changes in body weight, blood glucose levels and gain in knowledge. The results of the study are discussed in this chapter.

5.1 GENERAL PROFILE OF THE SUBJECTS

Information of the subjects collected regarding the age, sex, occupation, income level, family history of diabetes and personal habits (Table 2) revealed the mean age of the subjects to be 46.1 ± 10.1 years ranging from 30-65 years. Maximum incidence was seen among the age group of 36-55 years with mean age of occurrence of disease being 43.1 ± 7.94 years. Similar results were evidenced by Ahuja (1991), who reported that in India, maximum incidence of diabetes

was found in third, fourth and fifth decades. Ramachandran et al. (1988) also found that prevalence rate of diabetes was 21 per cent in the age group of over 40 years and 41 per cent in the age group of 55-64 years in south Indian urban population.

Varma et al. (1986) and Rao et al. (1989) from their surveys conducted in North India and South India from Delhi and Eluru found 9.1 and 6.1 per cent prevalence respectively in the age group above 40 years respectively. Rao (1992) revealed that highest number of diabetic patients were in the age group of 40-50 years in Libya.

Present study also showed highest number of subjects (53%) in the age group of 40-50 years, which indicates that persons as they enter into their thirties need to be more cautious and should have regular health checkups, so that they do not suffer from diabetes unnoticed.

According to 1990 census, age profile of population in India reveals that 50 per cent of the population are in the age group of 15-54 years. This brings us to another important point that, not only NIDDM persons are in this age group but IDDM persons also come under the lower limit of same age group. Since this age group is economically, socially and

intellectually most productive, better screening methods need to be employed to prevent or control diabetes.

Out of 32 subjects in the present study, 59 per cent of them are males and rest (41%) are females (Table 2). Even though results indicate greater incidence in males than females, because of the limited number of subjects these results cannot be generalised. Moreover, these results were from a single hospital only. However, sex standardized prevalence of diabetes mellitus showed different results in different studies. Population studies in 1978 by Gupta et al. showed that diabetes was more frequent in men than in women in India and in migrant Indians in other countries. Similar results were reported by Ahuja (1979), Mohan et al. (1985) and Ramachandran et al. (1986). Omar et al. (1984 and 1985) reported female preponderance in Indian diabetic subjects, which was attributed to increased frequency of obesity in females as compared with males. Dowse et al. (1990) in Mauritius found that prevalence of diabetes mellitus was similar in men and women of four ethnic groups which included Hindus and Muslims. Similar pattern was observed by Swai et al. (1990) in Indian muslim population of Tanzania. In Mauritius and Tanzania females were found to have higher values than males.

Similar pattern was observed in the present study which comprised of Hindus and Muslims, 9 per cent of the subjects were Muslim females with BMI of > 3.0 and also have central adiposity.

Ninety per cent of the subjects in the present study were sedentary workers comprising of office executives, housewives and business personnel. Physical inactivity is a risk factor for diabetes mellitus. Taylor et al. (1984) reported an association of NIDDM with sedentary physical activity independent of obesity, same was reinforced by Swai et al. (1990) after controlling for obesity. Role of exercise need to be induced in the NIDDM patients during education programmes, to avoid long term complications and improve metabolic control.

Earlier people used to think that diabetes was more common among the richer class in India. Acceptance of the classification of malnutrition related diabetes by WHO (1985) opened a new chapter in the much talked about diabetes mellitus. Malnutrition in this context is the presence of a history of nutritional deficiency mostly seen in lower income families. 1990 census indicates per capita income as Rs.2000/year in India. In the present study 69 per cent of the subjects belonged to lower income and just above the lower income level. If we could trace back to their earlier

(childhood) food habits and health records, clue to nutritional deficiency could be found. Because the hospital is run by government where health services are provided free of cost, much use of the hospital is by the poor section. This accounts for the more number of lower income subjects in the study, which was quite obvious by only 9 per cent of higher income subjects in the study.

Family history of diabetes was seen in 25 per cent of the subjects while 75 per cent of the subjects did not have any known family history. This is a clear indication that diabetes need not be of genetic origin alone and that environmental factors do have a strong pre-disposition towards it. Different studies have noted some important points regarding genetic susceptibility. Rao and Inbaraj (1979) in their population studies stated that consanguinous marriages (47%) seen in south India as one of the factors pre-disposing genetic susceptibility. According to National Diabetes data group criteria (1985) 50 per cent of offspring have diabetes while 12 per cent have impaired glucose tolerance.

Personal habits like smoking, alcohol and tobacco were present in half of the subjects while rest of the subjects did not have any of these habits and

some had either one or more than one habits at a time. The most prevalent habit was tobacco chewing along with pan and was seen only in Muslim subjects. This was followed by 13 per cent of the subjects who did smoke, drink alcohol and chew tobacco in pan, 3 per cent had only smoking habit and 9 per cent were found to smoke and drink alcohol. The common feature in all of them was tobacco either as a cigarette or in original form (smoke less tobacco). It is a well known fact that nicotine present in tobacco is a vaso constrictor and death rate with vascular complications in diabetics is much higher than their counterparts. Smoking immediately accelerates heart-rate and shoots up blood pressure which imposes extra burden on the heart. Heavy smoking elevates blood cholesterol. The nicotine in tobacco, also increases the tendency of the blood to clot in arteries (Saraph, 1989).

Mela (1989) reported that smoke less tobacco decreased the perceived intensity of salty, sour and bitter stimuli and greater intakes of alcohol. Intake of carbohydrates, fruits, grains and sweet foods were lowered in the subjects taking smokeless tobacco.

Thus, the general profile of the patients presents a group of subjects prone to risk factors like age, sex, physical inactivity, lower income and deteriorating personal habits. All these seem to have

a cumulative effect on the management of disease. A health educator needs to look into these factors and assess the personality to prescribe a tailored programme for each individual.

5.2 DISEASE PROFILE OF THE SUBJECTS

The disease profile of the subjects is presented in Table 3. Duration of the disease after diagnosis ranged from less than 6 months to more than five years and majority of them were seen in both the groups, indicating ever increasing incidence of diabetes adding to the already increased number of diabetics. This pattern was closely followed by less than one year duration after diagnosis.

Diabetes mellitus today is reported to affect atleast 100 million people in the world (Krall, 1989). While in the west epidemiological studies indicate one unknown diabetic for every known case, the situation in developing countries seems to be vastly different in that, for every known diabetic there may be upto 4 others who are undiagnosed, this situation becomes even clear if we look at the doctor population ratio, which ranges from 1:1000 to 1:15000 with an average of 1:4000 (Ahuja, 1991).

Fifty per cent of the subjects were diagnosed as diabetic solely on routine check up i.e. without any

classical symptoms of diabetes. This is probably due to lack of effective screening methods available in the locality, which could have been a preventive measure. A very close watch is needed to identify the persisting health problems which could be a probable symptom of diabetes.

Among the subjects in the study, 47 per cent presented the classical symptoms of diabetes i.e. polyphagia, polyuria, polydipsia and weight loss indicating altered metabolic control. However, by the end of the study period these symptoms subsided indicating improved compliance. Tingling and numbness of the toes and fingers was seen in 19 per cent of the subjects indicating the onset of long term complications involving the peripheral nervous system (Neuropathy). Interestingly this was seen in elderly patients (50-60 years of age) who were diagnosed as diabetic, recently (i.e. from 1 week to 1 year range) which once again indicates the lack of earlier detection facilities and negligence of the people, which could have been a preventive or control measure.

Dietary advice by a qualified dietitian was received by 59 per cent of the subjects while rest of the 41 per cent were not given any dietary advice which also includes 22 per cent of newly diagnosed patients.

All the subjects who received dietary advice were found to be following the prescribed diet. Observation revealed that number of patients coming in contact with dietitian was minimum when compared with doctor and patient. However, time spent by a patient with doctor is very less compared with time spent with dietitian. This may be due to overcrowding of the patients in the Endocrinology out patient ward, which not only deals with diabetic patients but also patients with other endocrine problems. A nutritional prescription not only requires signs and symptoms and biochemical and anthropometric parameters to assess metabolic control but also food habits, type of work, likes and dislikes, present meal pattern, economic and social back ground of the person and the goals to be attained by the patient. Therefore patients spending more time with dietitian is reasonable and is most essential (WHO, 1985). In addition dietitian/nutritionist have to answer numerous doubts posed by patients regarding their food intake unlike medical prescription. Thus a dietitian/nutritionist is a behaviour changing agent (Ferguson, 1978) in the area of a very personal habit - 'Food' and nutrition education should take advantage of this fact. Long time with dietitian keeps the other patients in long waiting by which time some patients would go back home. To avoid this, an ongoing educational programme or posters or other educational

material would be of benefit and stimulate patients to pose sensible questions to dietitian/doctor. It also helps the patients to retain knowledge for longer periods and follow the dietary prescription in correct manner.

In the present study 28 per cent of the subjects were controlled by diet alone which includes 6 per cent who did exercise also. Six per cent of the subjects stopped oral hypoglycemic drugs by the end of the education intervention, other 6 per cent decreased their dosage while other 6 per cent decreased insulin dosage. The common factor of all these patients was that they were all recently diagnosed (less than 1 year) which indicates that after the initial restoration of metabolic control, normoglycemia can be maintained with diet therapy thereby decreasing drugs and also by involving patient in a group education intervention.

5.3 BLOOD SUGAR VALUES OF THE SUBJECTS

In the present study a mean decrease of 76.41 ± 45.74 mg% was observed in FBS values and 135.72 ± 83.03 mg% (Table 5) in PPBS values by the end of the study indicating improvement in the regulation of blood glucose levels. Recently many studies have shown that therapeutic intervention can produce major improvements

in insulin secretion and action and appears, to a great extent, that the abnormalities causing fasting hyperglycemia are reversible, atleast for short periods of time (Savage and Knowler, 1984). Although insulin secretion in response to a glucose challenge was not measured directly in this study, a marked decrease in fasting hyperglycemia serves as an index of improved metabolic control, which was supported by a significant weight reduction in over weight patients. Mazzuca et al. (1986), Bloomgarden et al. (1987), Mani et al. (1989), Pratt et al. (1987), Wood (1989), Mount et al. (1991) and Boulton et al. (1981) have demonstrated similar results with education intervention programmes in the management of diabetes. However, 6 patients in the present study were still having fasting and post prandial hyperglycemia. Eventhough their final blood sugar values were much lesser than the initial blood sugar values and weight reduction upto 2-4 kgs was observed. This could be due to slow rate of compliance and further investigations might be helpful. Six other patients were having only post prandial hyperglycemia indicating that either, food intake prior to blood test was higher in simple sugars or that drug dosage was not appropriate. Counselling revealed that some of these patients had consumed extra food than the usual and

some others did not present with any cause. However, all these PPBS values were near to normal figure.

5.4 CHANGE IN BODY WEIGHT AND BODY MASS INDEX OF THE SUBJECTS

Results of the body weight and body mass index were tabulated in Table 6 and 7. Body weight of the patients ranged from a minimum of 40 kg to maximum of 80 kgs, requiring weight increase at the lower limit and weight decrease at the upper limit. Statistically significant increase/decrease in weight was observed by the end of the study. Similarly body mass index ranged from 1.719 to 3.72, before education and 1.805 to 3.626 after education intervention, even though, decrease at upper limit and increase at lower limit was observed, it was statistically not significant. Improvement in body weight is a good indication of improvement in metabolic control, as was evidenced by decreased blood sugar values. However, the decrease or increase of the body weight was modest (3-4 kgs) and patients remained in the same categories of weight as before education intervention. For example patients requiring 1-2 kgs weight reduction were already in the acceptable weight range, like the patients requiring > 10 kgs weight reduction who were in obese category, a reduction of mere 2-3 kgs did not change their category to which they belong. No change in BMI categorisation is a

clear reflection that even though there was significant change in overall body weight, a substantial weight reduction needs to be aimed in the case of overweight and obese patients, which indicates a long term study.

In the case of near under weight patients a slight increase in weight would suffice, which was evidenced by a statistically significant increase in weight. Patients having IBW, maintained so through out the study period which can be attributed to their awareness of the importance of maintaining desirable body weight through education intervention. Mount et al. (1991), Pratt et al. (1987), Mazzuca et al. (1986), Mani et al. (1989) have demonstrated a decrease in weight after education intervention.

5.5 KNOWLEDGE OF THE SUBJECTS

The knowledge test scores of the patients were given in Table 8. The highly significant scores of the subjects after education intervention reveals the effectiveness of the developed material in increasing the knowledge of the subjects. Patients were able to measure food accurately, differentiate between refined and complex foods, perform urine testing by Benedicts method and were able to tell the steps to be taken during an occurrence of hypoglycemia. However, some patients were not able to tell about OGTT and expressed

the idea that, it is not necessary for them to know and that it was the doctor's duty. The mean score for knowledge obtained after education intervention was 14.28 ± 1.17 indicating almost 100 per cent gain in knowledge of the patients regarding disease management.

Counselling sessions, diet displays, demonstrations and slide shows helped patients to gain knowledge, generated discussions and initiated problem solving. Years of medical research have clearly indicated that human behaviour patterns are not easily altered simply by furnishing information (WHO, 1985). An active interaction between the counsellor and the participant helps to achieve modifications in behaviour, as was evidenced during counselling sessions and diet displays. Bell (1981) strongly recommended continued out patient counselling by nutritionist/dietitian towards better patient compliance, same was observed during the entire study period.

A specific meal plan was designed for each patient during the initial counselling session. Counselling tailored to individual needs was used to increase the patients knowledge of such topics as blood-glucose, fat, fibre, carbohydrates, protein and physical complications, preventive measures etc.

Since OGH is a teaching hospital an atmosphere of learning always prevails and patients showed much interest in learning about their dietary modifications.

Patient's self-monitoring records have helped in counselling, for example an episode of hypoglycemia was traced with the help of food record. Here patient was asked to keep record of what he/she has consumed through out the day and at the end of the day, how he/she felt. Patients were also asked to keep record of any restricted food item that has caught their fancy but did not attempt to eat it, from which patient was motivated to follow their prescribed diet.

Food records also helped in patients feed back which is an important component in patient monitoring. Marlatt's relapse prevention model (Kayman, 1989) suggests that, new behaviour can be lost when an individual encounters a situation that puts him/her at high risk for returning to the old behaviour. This was commonly observed during the present study, which took place during the marriage season. Some of the subjects used to attend marriage functions and come back next day with hyperglycemia, saying that they had eaten sweets in the function and it was hard for them to resist the temptation. Patients were guided to come back to their changed behaviour pattern, even if they

slipped sometimes, as was suggested by Marlatt's relapse prevention model.

The health specialist's impact on a patient is usually transmitted in 3 ways - by direct verbal instruction (counselling), by audio-visual aids (charts and slide shows were used for this purpose), reading materials (a book-let on dietary management of diabetes was supplied to each patient in three languages—Telugu, Urdu and English, of their choice), family and friends. Some evidence suggests that family and friends are critically important to a patient's success and that when patient leaves the counsellors office, he returns to the every day environment that dictates his health pattern and a counsellor is unlikely to have a major or enduring impact on the patient's health unless he or she has made an impact on the environment (Mahoney, 1978). Therefore patient's family and friends participation was encouraged. Booklet on diabetes was a great help to patients for reviewing it in their home environment and to discuss or relate the material to their family or friends.

Similar results were demonstrated by Mount et al. (1991); Wedman (1987); Bloomgarden et al. (1987); Muhlhauser et al. (1983); Prett et al. (1987); Schreiber et al. (1983); Ney et al. (1983); Bell (1981) and Boulton et al. (1981); Garber et al. (1977)

Mani et al. (1989). It was stressed that improvements in knowledge and behaviour pattern led to improved metabolic control.

5.6 FOOD AND NUTRIENT INTAKE OF THE CONTROL AND EXPERIMENTAL GROUPS

The food and nutrient intake of the control and experimental groups were given in the Tables 9 and 10. Observations reveal a similar type of food intake in both the groups. However, pattern of food intake differed largely in both the groups. Foods from all the food groups were consumed by experimental group while control group did not consume any leafy vegetables, roots and tubers. Total vegetable intake was similar in both the groups, eventhough experimental group consumed more variety of vegetables. Control group exhibited large variation in total intake of vegetables, small variation was observed in experimental group. This indicates that experimental group were following same pattern of consumption of vegetables. Flesh foods were used sparingly in both the groups. Milk and milk products consumption was also similar in both groups, while experimental group consumed more milk, control group consumed more curds. Fruit intake was slightly more in control group compared to experimental group, similarly pulse intake was also more in control group.

Experimental group followed the pattern of taking smaller meals at shorter intervals, i.e. upto 3-4 hours gap between each meal while control group was found to be following an erratic pattern of food intake subject to their daily activities.

Contribution of cereals and vegetables to the bulk of total calories indicates higher consumption of complex carbohydrates, contribution of complex carbohydrates as well as fibre was more in the case of experimental group as they were consuming leafy vegetables also. Consumption of deep fried foods was observed to be 2 times more in control group than in experimental group. Consumption of milk and its products was more in quantity than pulses, indicating high biological value protein intake in both the groups. 24 hour recall of the experimental group at the initial visit revealed a higher consumption of fried foods large amounts of rice and very less or minimum amounts of leafy vegetables and fruits. An average of 1800 Kcals diet was prescribed to these patients and same was also displayed during diet displays. Importance of complex carbohydrates, meal timings was stressed during counselling and demonstrations and reduction of refined foods. By the end of the study, patients in the experimental group

were found to be following the prescribed 1800 Kcals diet.

Although many different dietary approaches to the treatment of type 2 diabetes have been reported, the common denominator that made them successful was not clear. For example in a six month out patient study by Hadden et al. (1975) calorie restricted diet with 40 per cent of the calories derived from carbohydrates have resulted in decreased hyperglycemia. While Barnard et al. (1982) reported lowered hyperglycemia with 77 per cent of calories derived from carbohydrates. These observations suggest that reduction in caloric intake is more important in reducing the hyperglycemia.

In the present study, 66 per cent of the calories were derived from carbohydrates in both the experimental and control group.

Weight reduction and maintenance of normoglycemia in experimental group can be attributed to their increased intake of leafy vegetables as a source of fibre, decreased intake of fats and oils, fried foods and taking smaller meals at shorter intervals. Wood (1989) reported maintenance of regular meal timings by diabetic patients after attending education programme.

Thus it was evident that education intervention can lead to a positive change in the dietary management of diabetes leading to a significant decrease of hyperglycemia and over weight of the patients.

5.7 CHANGES IN ATTITUDES OF THE SUBJECTS

Although not formally evaluated, during the education intervention, patients reactions to the programme were observed before, during and after the education intervention. The beliefs, expectations and acceptations of the patients were consolidated.

Before education intervention, some of the patients believed strongly that they should not take 'rice', because it increases sugar level, while taking of chapathi/phulka was believed to decrease their sugar level. Some believed that to control 'diet' or 'to restrict' certain foods means restricting milk, chilli powder, tamarind salt and fruits. Many had the idea that 'dieting' is only scarificing what one enjoys. Some were surprised that eventhough they had never eaten any sweet how could they become diabetic. When doctor/dietitian asked them to restrict certain food item, patients believed that, because of that particular food they might have got diabetes. Many patients under observation displayed a correlation to fate and accepted alien physiology. The point that

nutrition educator not only imparts knowledge to patients, but also corrects acquisition of pseudo-knowledge from different sources with good examples, becomes clear from the above mentioned beliefs of the patients. However, there were some positive points from the patients side which are advantageous to nutrition educator. Patients were very much interested in knowing what they could eat and how much they could eat etc. Many believed that exercise was an important component in diabetes management and that cigarette smoking and alcohol are not good for health.

During the education intervention i.e. during diet displays, slide shows and demonstrations, questions posed by patients generated much discussions. Coming together for a common purpose of learning helped patients to exchange ideas, experiences, problems and frustrations incurred while attempting to adjust to the disease. Diet displays stimulated lot of discussion, interest and all patients liked it, and it induced a sort of commitment to stick to the prescribed diet. All patients liked the inbetween meal snack items and were surprised by the variety, eventhough it lacked sweeter things. Many patients accepted that they break all the rules now and than. One patient actually narrated the recipe of mixing all green leafy vegetables and making curry and its superb taste to

other co-patients. All patients showed eagerness in participating in demonstrations and particular interest in charts of restricted foods, free foods, for your victory over diabetes and how are you? Patients were seen copying normal blood sugar values during the display of charts. Most interesting of all was patients were seen embracing each other, holding hands etc. as they continued to meet for each programme. Participation of family and friends also added importance to what they have been attending all these days watching and doing.

After the education intervention, patients seemed to feel closer to each other and expressed comfort in the knowledge that they were not alone in dealing with diabetes. There was an increased follow up care and public awareness, even though some patients showed trouble in coming solely for the purpose of counselling.

Conversations and discussions between patients-dietitian/nutritionists, patient-doctors, patient-patient, patient-other patients friends/family members in a congenial atmosphere served as a source of motivation. An improved esteem for the hospital in general was observed. Similar findings were reinforced by Small (1978) and Bloomgarden et al. (1987) and Wood (1989).

SUMMARY

CHAPTER VI

SUMMARY

With the increase in the incidence of diabetes during last three decades, the need for education programme for the dietary management has assumed as an essential component in the hospitals.

Present study was focussed on the impact of educational intervention in the dietary management of the diabetes. This study was based on both knowledge based and behavioural based interventions, which are essential to change the patients performance.

Considering the fact that 98 per cent of diabetic cases are non-insulin dependent, 32 diabetics of this category were selected for the study from the out-patient clinic of Endocrinology Department, Osmania General Hospital, Hyderabad. Before the initiation of the education programme, general information such as age, sex, anthropometric measurements, occupation, income, disease history, dietary habits, management practices, was collected from each patient using a schedule developed for this purpose. A sub sample of nine newly diagnosed patients along with 6 controls who did not receive the present education programme were selected to compare food and nutrient intake.

Education programme was carried out for a period of 6 months using the educational material specially developed for the study. The material developed include - (1) book-let on dietary management of diabetes - 'For victory over diabetes' (2) charts - (a) 'For your victory over diabetes' showing areas of control and importance in disease management; (b) 'How are you?' showing the graphic representation of blood sugar values of the hypo, hyper and normoglycemic patients; (c) 'Avoid' showing the restricted foods and (d) 'Free-foods' showing some of the vegetables and fruits which can be consumed liberally by a diabetic patient. Blood glucose levels, body weight and food intake were monitored through out the study period with the help of 'Your management goals' - folder and 'Your meal management' - food intake record. Diet displays, demonstrations of food measurement, urine testing and slide shows were also organised.

The general profile and disease profile of the subjects revealed that majority of them as belonging to lower socio-economic group prove to risk factors like sedentary work, smoking, alcohol, tobacco chewing and irregular meal pattern. Seventy five per cent of the subjects did not present with the family history of diabetes, indicating that environmental factors as causative factor for the onset of the diabetes. Half

of the subjects were diagnosed as diabetics on routine check up without any symptoms which suggests a need for effective screening methods.

The mean age of the subjects was 46 ± 10 years and the mean age at which disease occurred was 43 ± 8 years. Almost half of the subjects including newly diagnosed, presented symptoms of polyphagia, polyuria and polydipsia. Fifty per cent of the subjects were on oral hypoglycemic drugs along with dietary modification before the education intervention.

A mean decrease of 76.4 ± 45.7 of FBS and 135.7 ± 83 of PPBS values was observed by the end of the study. Similarly a mean decrease of 1.9 ± 1.0 kgs of weight in overweight subjects and a mean increase of 1.5 ± 1.0 kgs in underweight subjects was observed. A mean decrease of 0.164 ± 0.26 and a mean increase of 0.063 ± 0.020 was observed in the BMI of overweight and underweight subjects respectively. A mean increase of 6.56 ± 2.45 was observed in the knowledge test score of the subjects.

Significant decrease of FBS and PPBS at 1 per cent level ($P = > 0.001$) was observed. Significant change in body weight (decrease/ increase) at 1 per cent level ($P = > 0.001$) was observed indicating effectiveness of education intervention. A small

change was observed in the BMI of the subjects, indicating the need for long term study to obtain significant change. A highly significant increase in knowledge level at 1 per cent level ($P = > 0.001$) strongly indicates the effectiveness of the intervention in increasing the knowledge level of the patients.

By the end of the study period, 6 per cent totally stopped taking oral hypoglycemic drugs, 12 per cent decreased insulin dosage and number of visits to the hospital with complaints of hyperglycemia or hypoglycemia decreased.

The food and nutrient intake of the sub sample revealed consumption of a variety of vegetables and leafy vegetables by experimental group while controls consumed less variety of vegetables and no leafy vegetables. Consumption of deep fried foods was twice in controls compared to experimental group. Milk and curd intake was similar in both the groups. Similarly flesh foods were used sparingly in both the groups. The per cent contribution of calories from carbohydrates, and fats were similar to that of recommended values in both the groups. Fibre intake was less in controls compared to experimental group, however, both the groups did not meet the recommended 40 gms of fibre intake per day. Calorie contribution

from carbohydrates and fats was more in control group compared to experimental group. Education intervention has led to regular meal timings, increased consumption of leafy vegetables, decreased consumption of deep fried foods and maintenance of the prescribed calorie intake in the experimental group.

The attitudes of the patients has changed after education intervention. There was an increased follow up care and public awareness. Patients seemed to feel closer to each other and expressed comfort in the knowledge that they were not alone in dealing with diabetes. Presence of patients family members, friends during the displays and demonstrations created a congenial atmosphere for learning and served as a source of motivation.

From the findings of the study, it is inferred that the material developed for counselling diabetic patients were effective in improving the patient compliance in the management of the disease. General information revealed a need for effective screening methods for the diagnosis of the disease and an on going intensive educational programmes in the out patient clinics of diabetes.

LITERATURE CITED

LITERATURE CITED

- Abraham A. and Geevarghese P J 1990 Young onset of diabetes in central Kerala - A preliminary report. International Journal of Diabetes in Developing Countries 10: 17.
- Abraira C and Derler J 1988 Large variations of increase in constant CHO diets in type 2 diabetics. American Journal of Medicine 84:193-200.
- Ahuja M M S 1979 Epidemiological studies on diabetes mellitus in India. In Epidemiology of diabetes in developing countries. Ed. Interprint, New Delhi.
- Ahuja M M S 1991 A decade of epidemiology of diabetes. International Journal of Diabetes in Developing countries pp 11.
- Ajgaonkar S S 1986 Comprehensive plan for control of diabetes in the 7th Five year plan of government of India. Sub Committee meeting of Diabetic Association of India. A Preliminary Note 2.
- American Diabetes Association 1979 Special report: Principles of nutrition and dietary recommendation for individuals with diabetes mellitus. Diabetes 28: 1027-1030.
- American Diabetes Association 1987 Nutrition recommendations and principles for individuals with diabetes mellitus. Diabetes care 10:126-132.
- American Heart Association Nutrition Committee Report 1982 Rationale of the Diet Heart Statement of the American Heart Association. Circulation 65: 839A.
- Anderson J W 1977 Effect of Carbohydrate restriction and high carbohydrate diets on men with chemical diabetes. American Journal of Clinical Nutrition 30: 402-408.

- Anderson J W and Ward K 1979 High carbohydrate, high-fibre diets of insulin treated men with diabetes mellitus. American Journal of Clinical Nutrition 32: 2312-2321.
- Anderson J W and Bryant G A 1986 Fibre : Diabetes and obesity. American Journal of Gastroenterology 81: 898-906.
- Anderson J W and Gustaffson N J 1989 Adherence to high carbohydrate and high fibre diets. Diabetes Education 15: 429-434.
- Anderson R A 1986 Chromium metabolism and its role in disease process in man. Clinical Physiology and Bio-chemistry 4: 31-41.
- Anthony L Mc C 1992 Perspectives in diabetes : The impact of diabetes on the Central Nervous System. Diabetes 41: 557.
- Assal J P, Muhlhauser I, Pernet A, Gfeller R, Jorgens V and Berger M 1985 Patient education as the basis of diabetes care in clinical practice. Diabetologia 28: 602.
- Bantle J P, Laine D C, Castle G W, Thomas J W, Hoogwerf B J and Goetz F C 1983 Post prandial glucose and insulin responses to meals containing different carbohydrates in normal and diabetic subjects. New England Journal of Medicine 309:7-12.
- Barnard R J, Lattimore L, Holly R G, Cherny S and Pritikin N 1982 Response of non-insulin dependent diabetic patients to an intensive program of diet and exercise. Diabetes Care 5: 370-374.
- Bell D M 1981 Patient nutrition education follow up in hospital setting. Journal of American Dietetic Association 79: 309.
- Bennion M 1979 Clinical nutrition. Harper and Row Publication.

- Bloomgarden Z, Karmally W, Metzger M, Brothers M, Nechmias C, Bookman J, Faireman D, Ginsberg Fellner F, Ray field E and Brown V 1987 Randomized controlled trial of diabetic education. Improved knowledge without improved metabolic status. Diabetes care 10: 263.
- Boulton W E, Anderson B J, Schwartz N L, Drexler A J 1981 A group approach to the management of diabetes in adolescents and young adults. Diabetes Care 4: 620-623.
- Bray G A 1978 Definition, measurement and classification of the syndromes of obesity. International Journal of Obesity 2:99.
- Brenner B M, Meyer T W and Hosteller T H 1982 Dietary protein intake and the progressive nature of kidney disease. The role of hemodynamically mediated glomerular injury in the pathogenesis of progressive glomerular sclerosis in aging, renal ablation and intrinsic renal disease. New England Journal of Medicine 307: 652-659.
- British Diabetes Association 1982 Dietary recommendations for diabetics for the 1980 - A policy statement by the British Diabetic Association. Human Nutrition : Applied Nutrition 36A: 378-394.
- Cohen M and Zimmet P 1983 Self monitoring of Blood glucose levels in NIDDM. Medical Journal of Australia 8: 317-380.
- Cohen M, Doolds R and Vibertti G 1987 Effect of protein nutrition in IDD at risk of pregnancy. British medical Journal 294: 795-798.
- Campbell L V, Barth R, Gasper J K 1990 Impact of intensive educational approach to dietary change in NIDDM. Diabetes Care 13: 841-847.
- Cooper P L, Wahcoust M L and Simpson R W 1986 Sucrose in the diabetic diet. Recent Advances in Clinical Nutrition 2: 271-283.

- Cruickshank J K, Beevers D G, Asbrone V L, Haynes R A, Corlett J C R and Selby S 1980 Heart attack, stroke, diabetes and hypertension in West Indians, Asians and Whites in Birmingham, England. British Medical Journal 2: 1108.
- Daly PR, Dodson P M and Taylor M P 1986 The effect of a high fibre lowfat, low sodium diet on diabetics with intermittent classification. British Journal of Clinical Practice 40: 313-317.
- Dechamp Rouse C, Valensi P and Assal N 1990 Beliefs in diabetics about the etiology of their disease. Influence of ethnicity. Diabete Metabol 16: 207-212.
- Doar J W H, Wilde CE, Thompson M E and Sewell P F J 1975 Influence of treatment with diet alone on oral glucose tolerance test and plasma sugar and insulin levels in patients with maturity onset diabetes mellitus. Lancet 1: 1263-1266.
- Dowse G K, Garecloo H and Zimmet P 1990 The high prevalence of non-insulin dependent diabetes and impaired glucose tolerance in Indians, Creole and Chinese Mauritious Diabetes 39: 390.
- Etzuiler D M 1976 Pateient participation: The key to succesful diabetes control. Diabetes Forecast Sept.-Oct.
- Everhart J E, Pettitt D J, Peter H, Benett and Knowler W C 1992 Duration of obesity increases the incidence of NIDDM. Diabetes 41: 235-240.
- Ferguson J 1978 Dietitians as behaviour change agents. Journal of Americal Dietetic Association 73: 231.
- Franz M J 1987 Exercise and the management of diabetes mellitus. Journal of the American Dietetic Association 87: 872.

- Friedman G J 1980 Diet in the treatment of diabetes mellitus. In Modern Nutrition in Health and disease 6th ed Edited by Goodhart R S, Shils M E Lea & Feliger Publications Philadelphia.
- Fukuda M, Tahara V, Yamamoto Y, Onishi T, Kumhara Y Tanaka A and Shima K 1989 Effects of very low caloric diet weight reduction on glucose tolerance insulin secretion and insulin resistance in obese non-insulin dependent diabetics. Diabetes Research and Clinical Practise 7: 61-67.
- Gamble D R and Taylor K W 1969 Seasonal incidence of diabetes mellitus. British Medical Journal 3:631.
- Gamble D R, Taylor K W and Cumming H 1973 Coxsackie viruses and diabetes mellitus. British Medical Journal 4: 260.
- Garber A, Christman B, Alogna M and Devidson J 1977 Evaluation of diabetic education programmes. Diabetes 26: 61.
- Ginter C and Vathova V 1983 Vitamin 'C' and diabetes mellitus. Nutrition and Health 2: 3-11.
- Gopalan C, Rama Sastri B V and Balasubramanian S C 1991 Nutritive value of Indian foods. National Institute of Nutrition. Indian Council of Medical Research Publication, Hyderabad.
- Gries F A and Alberti K G M M 1991 Management of non-Insulin -Dependent Diabetes Mellitus in Europe: A Consensus Statement. International Journal of Diabetes in Developing Countries 11: 5.
- Gupta O P, Joshi M H and Dave S K 1978 Prevalence of diabetes in India. Advances in Metabolic Disorders 9:147.
- Hanson P K R 1983 Exercise and diabetes. Primary Care 10: 653-661.

- Hazra D K, Wahal P K, Ram Singh, Gupta M K, Lahiri V L, Elhence B R and Jain N K 1980 Coxsackie virus infection in relation to Asian Diabetics in India. Journal of Diabetes Association of India XX: 157.
- Hadden D R, Montgomery D A D, Skelly R J, Trimble E R, Weaver J A, Wilson E A and Buchanan K D 1975 Maturity onset diabetes mellitus: response to intensive dietary management. British Medical Journal 3: 276-278.
- Hirata Y 1988 Diabetes and alcohol. Asian Medical Journal 31: 564-569.
- Hitman G A and Niven M J 1989 Diabetes - Genes and Diabetes mellitus. British Medical Bulletin 45: 191-205.
- Hoet J P 1954 Carbohydrate metabolism during pregnancy. Diabetes 3:1.
- Hosey G M and Freeman W L 1990 Designing and evaluating diabetes education material for American Indians. Diabetes Education 16: 407-14.
- Jackson W P U 1961 Is pregnancy diabetogenic? Lancet 2: 1369.
- Jenkin D J A, Goff D V and Leeds A R 1976 Unabsorbable carbohydrate from diabetes; decreased post prandial hyperglycemia, Lancet 2: 172.
- Jenkin D J A, Wolener T M S, Jenkins A L, Lee R, Wong G S and Josse R 1983 Glycemic response to wheat products: reduced response to pasta but no effect of fiber. Diabetes Care 6: 155-159.
- Kayman S 1989 Applying theory from social psychology and cognitive behavioural psychology to dietary behaviour change and assessment. Journal of American Dietetic Association 89: 2.

- Kelsey J L, Behall K M and Prather E S 1978 Effect of fiber from fruits and vegetables on metabolic responses of human subjects. I. Bowel transit time, number of defecations, fecal weight, urinary excretions of energy and nitrogen and apparent digestibilities of energy, nitrogen and fat. American Journal of Clinical Nutrition 31: 1149-1153.
- Kendell P A and Jensen G R 1990 Educating patients with diabetes: comparison of nutrient - based and exchange group methods. Journal of American Dietetic Association 90: 238-243.
- Koplan R and Davis V 1986 Evaluating the costs and benefits of out patient diabetes education and nutrition counseling. Diabetes Care 9: 81.
- Korhonen T, Uusitupa M, Aro A, Kumpulaiven T, Slltonen O, Voutilainen E and Pyorala K 1987 Efficacy of dietary instructions in newly diagnosed non-insulin dependent diabetic patients, comparison of two different patient education regimens. Acta Scandinavica 222: 323-331.
- Kosaka K, Kuzuya T, Akanuma Y and Hgura R 1980 Increase in insulin response after treatment in overt maturity onset diabetes independent of the mode of treatment. Diabetologia 18: 23-28.
- Krall L P and Beaser R S 1989 Joslin Diabetes Manual. Lea and Febiger Publications, London.
- Lele R D 1990 Fish oils. Journal of the diabetic Association of India 30:1.
- Lingarde F, Mabmquist J and Belke B 1983 Physical fitness, insulin secretion and glucose tolerance in healthy males and mild type-2 diabetes. Acta Diabetologia Latina 20: 33-40.
- Lorini R and Ciriaco 1990 The influence of dietary education in diabetic children. Diabetes Research; Clinical Practise 9:279-285.

- Lundgren H H, Bengtsson C, Böhme G, Lapidus L and Silostrom L 1989 Adiposity and adipose tissue distribution in relation to incidence of diabetes in women; Results from a prospective population study in Gothenburg, Sweden. International Journal of Obesity 13: 413-423.
- Mahoney M J 1978 Applying behavioural methods to nutritional counselling. Journal of the American Dietetic Association 72: 372.
- Mani V V, Srivastava R, Mani I, Parekh A, Shah V, Mehata N C, Patel H R, Dosi M and Iyer U N 1989 Effect of Diabetes Patient Education in the Management of Diabetes Mellitus. Diabetes Bulletin 9, 4: 200.
- Margaret AP 1987 Hand Book of Diabetes Nutritional Management. As ashen Publications.
- Mazucca S, Moorman N, Wheeler M, Norton J, Fineberg N, Vinicor F, Cohen S and Clark C 1986 The diabetes education study: A controlled trial of the effects of diabetes patient education. Diabetes Care 9: 1.
- Mela D J 1989 Gustatory function and dietary habits in users and non users of smokeless tobacco. American Journal of Clinical Nutrition 2193: 482-489.
- Milchovich K S and Dunn-long B 1991 Diabetes mellitus a practical hand book. Galgotia publications, New Delhi.
- Miller L and Goldstein J 1972 More efficient care of diabetic patients in a country hospital setting. New England Journal of Medicine 286: 1388.
- Mohan V, Ramachandran A and Snehalatha C 1985 High prevalence of maturity onset diabetes of the young (MODY) among Indians. Diabetes Care 8: 371.
- Mohan V 1989 Diabetic dieting without tears. Health Action 2: 32.

- Mount M A, Kendrick O W, Draughon M, Stitt K R, Head D and Mount R 1991 Group Participation as a method to achieve weight loss and blood glucose control. *Journal of Nutrition Education* 23: 25-29.
- Muhlhauser I, Jorgens V, Berger M, Graninger W, Gurtler W, Horuke L, Junz A, Schernthaner G, Scholz V and Vass H 1983 Bicentric evaluation of a treatment program for type I diabetic patients: Improvement of metabolic control and other measures of diabetes care for upto 22 months. *Diabetologia* 25: 470.
- Nagulesparan M, Savage P J, Mott D M, Johnson G C, Unger R H and Bennett PH 1980 Increased insulin resistance in obese, glucose intolerant South Western American Indians: evidence for a defect not explained by obesity. *Journal of Clinical Endocrinology and Metabolism* 51: 739-743.
- National Diabetes Data Group 1979 Classification and diagnosis of diabetes mellitus and other categories of glucose intolerance. *Diabetes* 28:1039-1057.
- National Diabetes Data Group 1985 Diabetes in America, Diabetes data compiled 1984, Dept. of Health and Human Services. NIH Publication.
- National Cholesterol Education Program 1987 Highlights of the report of the expert panel on the detection, evaluation and treatment of high blood cholesterol in Adults, Bethesda, NIH Publications.
- Ney D, Stubblefield N and Fischer C 1983 A tool for assessing compliance with a diet for diabetes. *Journal of the American Dietetic Association* 82: 287-290.
- Nise J E, Kein K S, Huising A J and Willmann L 1989 Effect of sucrose containing snacks on blood glucose control. *Diabetes Care* 12: 423-426.
- Omar M A K and Asmal A C 1984 Patterns of diabetes mellitus in young South African Black and Indian diabetic patients. *Trop. Geogr. Med.* 36: 133.

- Omar M A K, Seedet M A, Dyer R B, Rajput M C, Motala A A and Joubert S M 1985 The prevalence of diabetes mellitus in large group on Indians. South African Medical Journal 67:924.
- Piagessi A, Giampietroo, Picaro L and Miccoli R 1989 Individual teaching as a first-step intervention for the education of diabetic subjects. Acta Diabetol Lat 26: 225-35.
- Power L 1973 New approaches to the old problem of diabetes education. Journal of Nutrition Education 5:230.
- Pratt C, Wilson W, Leklem J and Kingsley L 1987 Peer support and nutrition education for older adults with diabetes. Journal of Nutrition for the Elderly 6:31-43.
- Pushpamma P, Geervani P, Usharani M, Anurag C, Manorama R, Lakshmi Devi N, Krishna Kumari K, Jayashree K and Jyothi E 1981 College of Home Science, Andhra Pradesh Agricultural University Publications, Hyderabad.
- Raghuram T C, Swaran Pasricha and Sharma R D 1991 Diet and Diabetes National Institute of Nutrition. Indian Council of Medical Research Publication, Hyderabad.
- Raheija B S, Phatak R B, Vispute K V and Koppika G D 1989 Dietary habits and prevalence of type 2 (NID) diabetes in Indian subjects. Journal of Diabetes Association of India 29: 3-8.
- Raheija B S 1990 Essential fatty acids, atherosclerosis and diabetes. Journal of Diabetes Association of India 30:20-25.
- Ramachandran A, Gallagher F and Mohan V 1986 Comparative study of the clinical pattern of diabetes from two referral centres for diabetes in the United States of America and India. Journal of Diabetic Association of India 26: 83.

- Ramachandran A, Jali M V, Mohan V, Snehalatha C and Viswanathan M 1988 High prevalence of diabetes in an urban population in South India. British Medical Journal 297:587.
- Ramaiya K L, Kodali V R R and Alberti K G M M 1991 Epidemiology of Diabetes in Asians of the Indian sub-continent. International Journal of Diabetes in developing countries 11:15.
- Rao G M M 1992 Diabetes mellitus in libya A retrospective study. Indian Journal of Medical Sciences 46:174.
- Rao K V R, Hariharan R S and Sessaiah V 1986 Body mass, sex distribution and parity among South Indian NIDDM diabetics. Journal of Diabetes Association of India 26:7.
- Rao P S S and Inbaraj S G 1979 Trends in human reproductive wastage in relation to long-term practice of inbreeding. Annals of Human Genetics 42:401.
- Rao G N 1983 Tests of hypothesis. Statistics for Agricultural Sciences pp 89 Oxford and IBH Publishing Company.
- Rao P V, Ushabala P, Sessaiah V, Ahuja M M S and Mather H M 1989 The Eluru survey: prevalence of known diabetes in a rural Indian population. Diabetes Research in Clinical Practice 7:29.
- Rudnick P A and Taylor K W 1965 Effect of prolonged carbohydrate restriction on serum insulin levels in mild diabetes. British Medical Journal 1: 1225-1288.
- Sadikot S M 1990 Oral hypoglycemic agents - I. Journal of Diabetes Association of India. 30:44.
- Sadikot S M 1991 Oralhypoglycemic agents - II. Journal of Diabetes Association of India 31:2.

- Sadikot S M and Raheija B S 1991a Vitamin E supplementaion and oxident stress in diabetics. Journal of Diabetes Association of India 31: 68.
- Sadikot S M and Raheija B S 1991b Vitamin 'C' supplementation decreases the oxidant stress and free radical damage in diabetics. Journal of Diabetes Association of India 31:68.
- Samal K C, Tripathy B B and Das S 1990 Profile of childhood onset diabetes in Orissa. International Journal of Diabetes in developing countries 10: 27.
- Saraph M 1989 How to prevent and control hypertension. Health Action 2: 6.
- Savage P J and Nagulesparan M 1983 Contrasting response of insulin secretion to different stimuli in glucose intolerance treated by hypocaloric diet. Diabetes 32: 141a.
- Savage P J and Knowler W C 1984 Diet therapy for type 2 diabetes mellitus: Can new approaches improve therapeutic results; Nutrition Abstracts and Reviews, Reviews in Clinical Nutrition 54:2-3.
- Schreiber J H and Amos R J 1983 Hospital diabetes education: Classroom and self- instruction. Journal of the American Dietetic Association 83: 581-583.
- Simpson R W, Mann J I, Eaton J, Moore R A and Cartar R 1979 Improved glucose control in maturity onset diabetes treated with high CHO modified fat diets. British Medical Journal 1: 1753-1756.
- Small D 1978 A patient education program. American Journal of Nursing 78: 889.
- Stanik S and Marcus R 1980 Insulin secretion improves following control of plasma glucose in severly hyperglycemic obese patients. Metabolism 29: 346-350.

- Steven M H, Braxton D M, Hlen P H and Michael P S 1991 Greater influence of central distribution of adipose tissue on incidence of non-insulin - dependent diabetes in women than men. American Journal of Clinical Nutrition 53: 1312-17.
- Swai A B M, Mclarty D G and Chwa L M 1990 Diabetes and impaired glucose tolerance in an Asian Community in Tanzania. Diabetes Research In Clinical Practice 8: 227.
- Tattersall R B 1974 Mild familial diabetes with dominant inheritance. Quarterly Journal of Medicine 43: 339-357.
- Taylor R, Ram P, Zimmet P, Raper L R and Ringrose H 1984 Physical activity and prevalence of diabetes in Melanesian and Indian men in Fiji. Diabetologia 27: 578.
- Varma N P S, Mehta SP, Madhu S V, Mather H M and Keen H 1986 Prevalence of known diabetes mellitus in an urban Indian environment. The Daryaganj diabetes survey. British Medical Journal 293: 423.
- Venkataraman S, Suresh K, Sundaram R S, Hariharan, Madhavan R, Manjula N and Seshaiiah V 1990 Diabetes in the young - A profile. International Journal of Diabetes in Developing Countries 10:17.
- Vinke B, Nagelsmith W F, Vanbuchen F S P and Smid L J 1959 Some statistical investigations in diabetes mellitus. Diabetes 8:100.
- Viswanathan M, Mohan V and Snehalatha C 1985 High prevalence of diabetes among offspring of conjugal type 2 diabetic parents in India. Diabetologia 28: 907.
- Wales J K 1982 Treatment of type 2 (non-insulin dependent) diabetic patients with diet alone. Diabetologia 23: 240-245.

- Wedman B and Kahan R S 1987 Diabetes graphic aids used in counselling improve patient compliance. Journal of the American Dietetic Association 87:1672-1674.
- West K M 1973 Diet therapy of diabetes: an analysis of failure. Annals of Internal Medicine 79: 425-434.
- World Health Organisation study group report on Diabetes mellitus 1985 Geneva 11-16 February.
- West K M 1978 Factors associated with occurrence of diabetes. In diabetes and its vascular complications. Ed. Elsevier Publication, New York.
- Williams D R R, James W P T and Evans I E 1980 Dietary fibre supplementation of a 'normal' breakfast administered to diabetics. Diabetologia 18: 379-383.
- Willians D R R 1984 Nutrition education is appropriate for the long-term management of diabetes in the community. Proceedings of the Nutrition Society U K 43: 227-229.
- Wood E R 1989 Evaluation of a hospital - based education program for patients with diabetes. Journal of the American Dietetic Association 89: 354-358.
- Zifferblatt S M and Wilbur C S 1977 Dietary counselling: Some realistic expectations and guide lines. Journal of the Americal Dietetic Association 70: 591-595.

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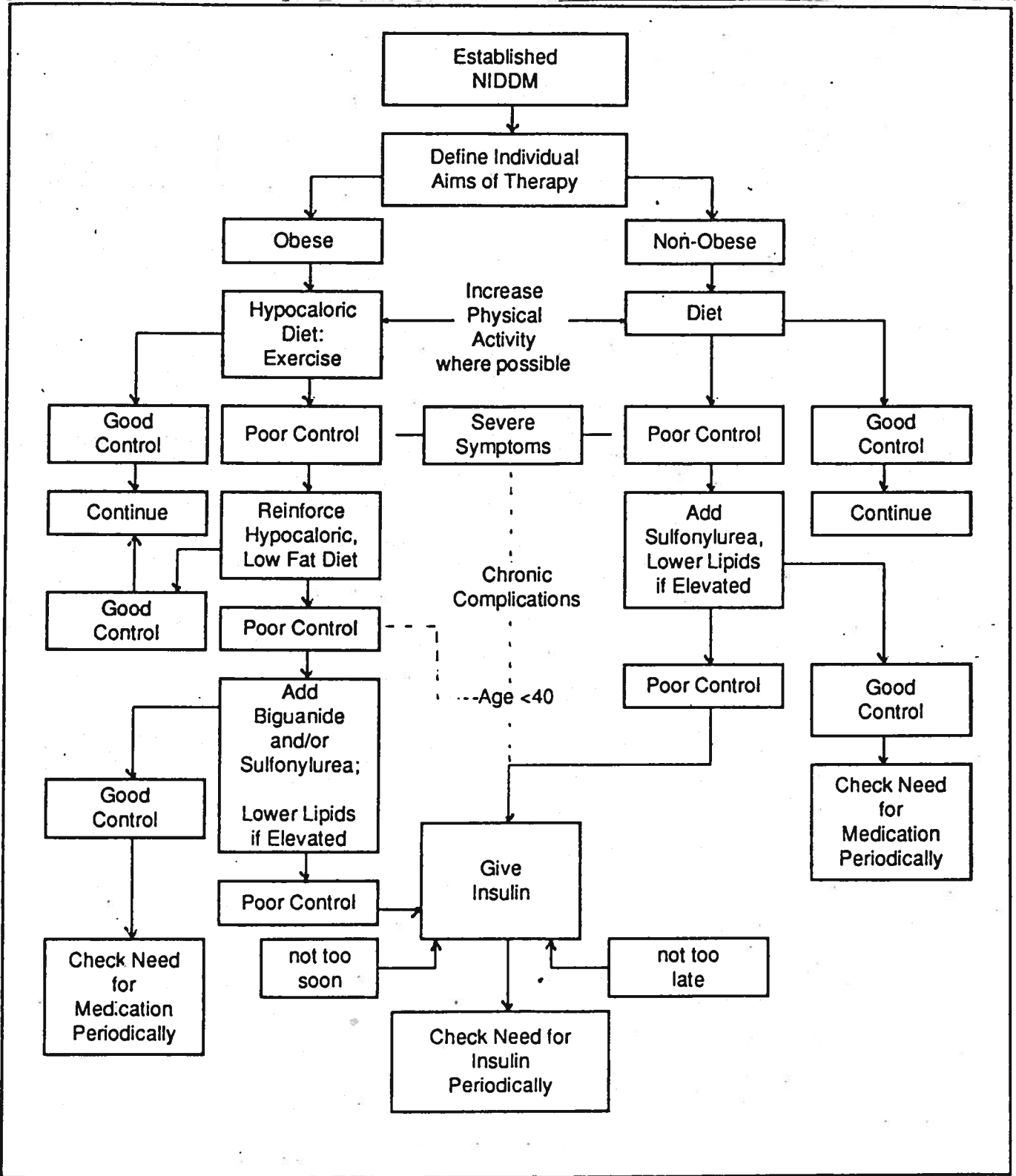
APPENDICES

Appendix - 1

Plan of the Study

- Collection of general information
- Development of knowledge questions & health monitoring folder & book
- Pre-testing the knowledge questions by expert panel and by lay persons
- Pre-testing patients knowledge, blood sugar testing (initial blood sugar), distribution of health monitoring folder and book, dietary prescription, counselling
- Development of charts & book let
- Dietary prescription and diet counselling, blood sugar testing
- Food display - sample diet of 1800 Kcals; restricted foods; inbetween meal snacks; charts display
- Diet counselling and blood sugar testing
- Food display of break fast items of 300 KCals maximum, food measurement demonstration charts display
- Counselling and blood sugar testing
- Slide show and urine testing demonstration distribution of book-let
- Diet counselling and blood sugar testing
- Slide show (repeat)
- Final blood sugar testing
- Post-knowledge test of the patients

A P P E N D I X - 2



SOURCE: Gries FA and Alberti KG MM 1991

Appendix 3

CASE HISTORY OF THE PATIENT

1. Sl.No. _____ Date: _____
 Name : _____ Hospital: _____
 Age : _____ Sex : _____ Occupation: _____
 Income: _____
2. Personal habits
 1. Smoking : Yes/No
 2. Alcohol : Yes/No
 3. Tobacco/Pan : Yes/No
3. History of diabetes :
 - i) Age at which disease occurred: _____
 - ii) Diagnosed: _____
 - iii) Known Diabetics : Yes/No
 If yes, Duration _____
 Drug History : _____

| Name of the drug | Dose | Disage Interval |
|------------------|------|-----------------|
| 1. _____ | | |
| 2. _____ | | |
| 3. _____ | | |
 - iv) Newly diagnosed diabetic : _____
 Symptoms : _____
 Polyphagia:
 Polyuria:
 Polydipsia:
 Tingling and numbness:
 Weight loss: _____
4. Family History : Present/absent
 If present, the relationship of the affected to the diabetic.
5. Associated disorders : _____
 If any, specify :

6. Dietary history :

Has any dietary advice given : Yes/No

Is there any change in dietary intake : Yes/No

7. Anthropometric measurements :

Weight (Kg) :

BMI :

Height (cm) :

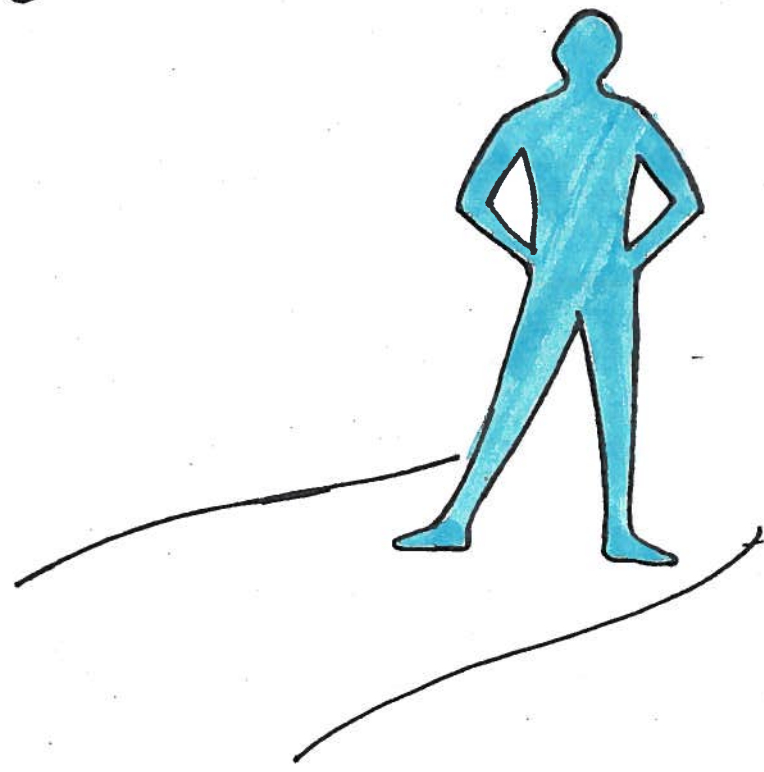
Appendix 4

Knowledge Questions

1. Diabetics have high blood glucose levels. Yes/No
2. Glucose tolerance test is done to confirm Diabetes. Yes/No
3. Insulin is required for transfer of glucose to cells. Yes/No
4. Simple carbohydrates are easily digestible and can be taken by diabetics. Yes/No
5. Fatty foods like deep fried foods, cakes, butter, cream should be avoided by diabetics. Yes/No
6. Fibre helps in decreasing the sudden release of glucose into blood. Yes/No
7. Examples of complex carbohydrates - whole grams and whole gram flours. Yes/No
8. Examples of fibre rich foods - green leafy vegetables, fruits, whole grams and whole gram flours. Yes/No
9. Examples of refined foods - sweets, sugars, ghee, butter, sweetened drinks and chocolates. Yes/No
10. Controlled portion size is important in maintaining energy intake. Yes/No
11. Exercise is as important as food planning in the management of diabetes. Yes/No
12. Diabetes can be kept at bay by maintaining ideal body weight. Yes/No
13. Tobacco and alcohol are harmful to diabetics. Yes/No
14. Low blood sugar levels can occur suddenly, person sweats and may faint. Yes/No
15. Irregular control of diabetes leads to long term complications to eye, heart, kidney and nervous systems. Yes/No

A P P E N D I X - 5a

Your Management Goals



MODE OF TREATMENT

DIET :

EXERCISE :

ORAL HYPOGLYCEMIC DRUGS :

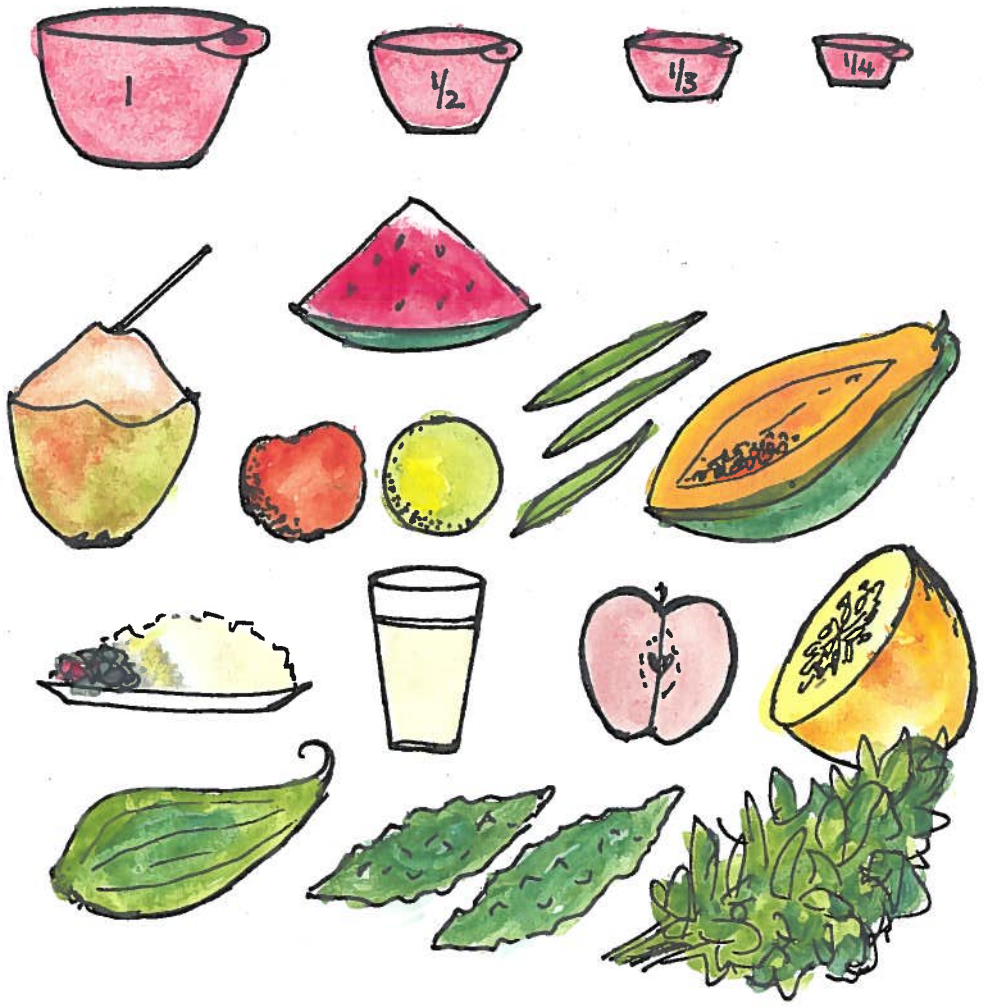
INSULIN :

BLOOD SUGAR LEVELS

NORMAL
-----FBS : 60 - 120 mg %
-----PPBS : 110 - 160 mg %
-----WEIGHT :
-----HEIGHT :
-----IBW :
-----BMI :

A P P E N D I X - 5b

Your meal management



DATE:

| TIME | NAME OF THE FOOD ITEM | APPROXIMATE QUANTITY |
|------|--------------------------|-------------------------|
|------|--------------------------|-------------------------|

HOW ARE YOU TODAY : _____

APR 19 1954
APR 19 1954
DATE

A P P E N D I X - 6

APR 19 1954
APR 19 1954

For-victory over Diabetes

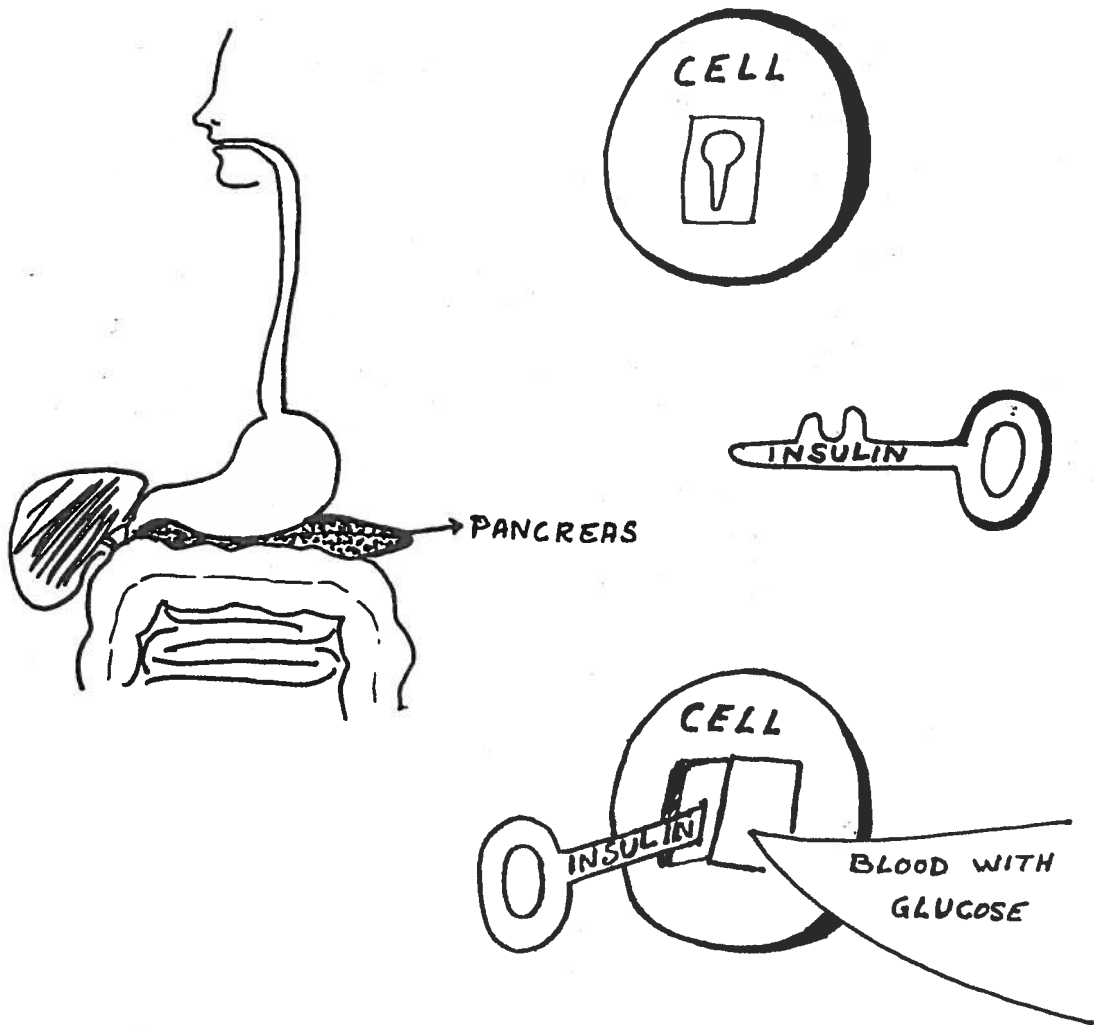
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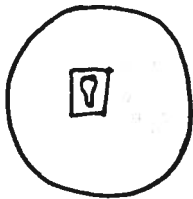
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21

WHAT IS DIABETES?

Food when eaten is digested and much of it is converted into glucose which is the main fuel for the body. Blood carries glucose to the individual cells of the body. Pancreas, an endocrine gland just behind the stomach, produces a hormone called INSULIN, which is essential for glucose to enter into the cell.



We can say that insulin acts as a key to the cells, to allow the entry of glucose. The cell then performs its normal functions. Therefore when sufficient insulin is not produced by the pancreas or even if produced, insulin is not utilised properly as in the case of over-weight persons, digested food in the form of glucose builds up in the blood leading to increased glucose levels in the blood. This condition is known as DIABETES.



NON DIABETIC CELL



DIABETIC CELL
(STARVED STATE)

TYPES OF DIABETESS

1. INSULIN DEPENDENT DIABETES MELLITUS (IDDM)

This type of diabetes usually occurs at a younger age and the person requires insulin injections daily.

2. NON-INSULIN DEPENDENT DIABETES MELLITUS (NIDDM)

This occurs at middle-age and is usually associated with obesity and stress.

3. MAL-NUTRITION RELATED DIABETES MELLITUS (MRDM)

This occurs in persons who present a history of nutritional deficiency.

DIABETES AND YOU

CAUSES

1. HEREDITY
2. OBESITY
3. STRESS
4. MALNUTRITION
5. DISEASES OF THE PANCREAS
Ex: Pancreatitis
6. TOXICITY OF CERTAIN FOODS
Ex: Tapioca (Cassava)

Symptoms to detect diabetes

* EXCESS THIRST

(POLYDIPSIA)



*** EXCESS HUNGER**
(POLYPHAGIA)



*** EXCESSIVE URINATION**
(POLYUREA)



* LOSS OF WEIGHT



* TIREDNESS AND FATIGUE



* SLOW HEALING OF CUTS AND WOUNDS



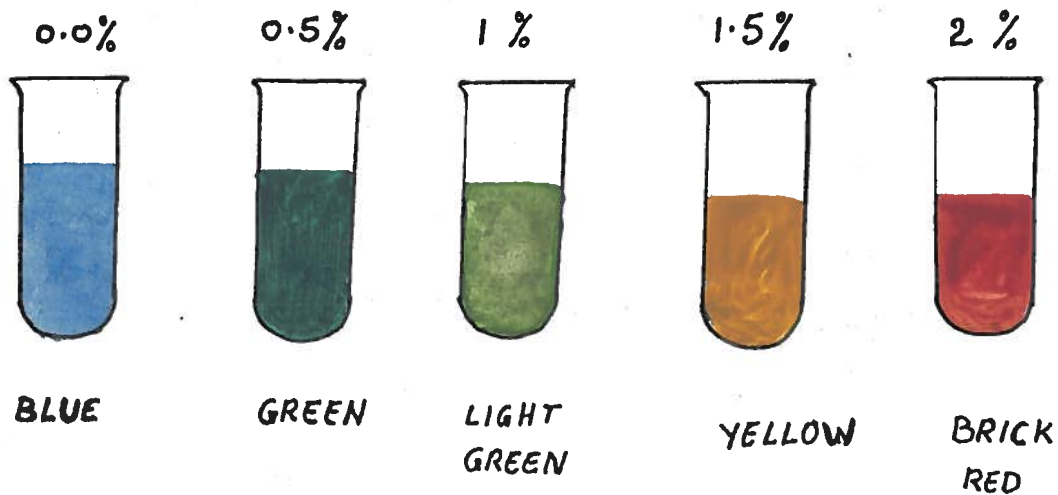
* DIZZINESS AND GIDDINESS



If you have been observing these symptoms for sometime then it is best to consult doctor, who will suggest you to go for clinical confirmation. The tests that are used to detect diabetes are based on urine and blood analysis.

1. URINE TEST

This test is done at the clinical laboratory and can also be done at home by yourself with the help of Benedict's Solution or Strips available in a medical store. Follow the instructions given carefully for accurate results. The change in colour of urine indicates the extent of diabetes. Keep a record of the results along with date and time.



2. BLOOD TEST

This test is done in the laboratory for which you will have to give two samples of blood, one a fasting sample and the other post-lunch sample.

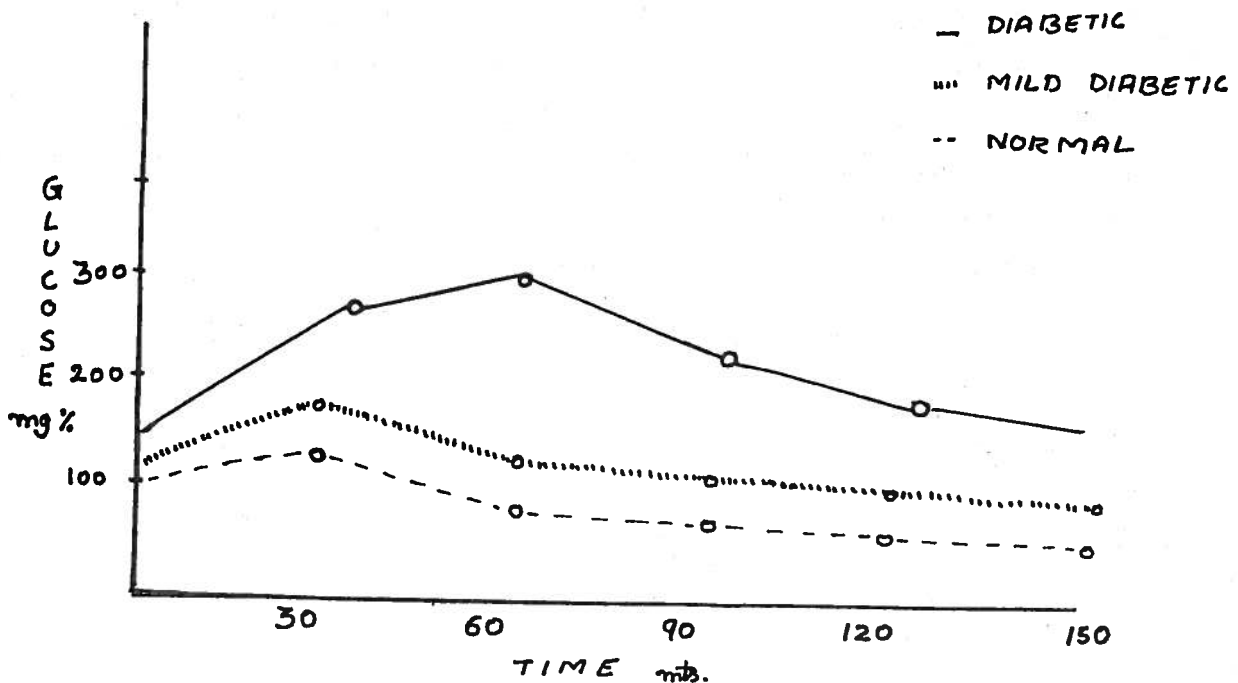
NORMAL VALUES OF BLOOD - SUGAR -

FASTING - 60 to 120 mg. %

POST-LUNCH - 110 to 160 mg. %

3. GLUCOSE TOLERANCE TEST (GTT)

To do a GTT your fasting blood sample will be first taken and then 75 gms of glucose dissolved in 200 ml water (1 glass) will be given to drink. Blood samples will be collected every thirty minutes during the next 150 mts. time period. The glucose content in each sample is analysed and plotted against time.

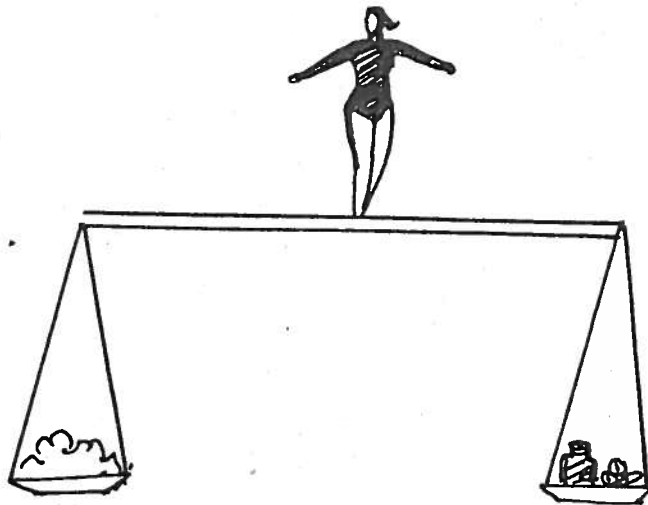


MANAGEMENT OF DIABETES

The three most important aspects in the management of diabetes are -

- * FOOD
- * REGULAR EXERCISE
- * MEDICATION - That is drugs and insulin if necessary

All these are inter-dependent. A fine balance between food, exercise and medication leads to an excellent management of Diabetes.



FOOD

There are no restrictions on food intake as long as your ideal body weight is maintained, by taking required amount of calories. You and your dietician can work out a meal plan according to your height, weight, likes, dislikes and type of work that you usually do and the medicines you are taking.

PRINCIPLES OF GOOD NUTRITION

* What you eat should be balanced with how you spend your energy, then only you can maintain your ideal body weight.

ENERGY BALANCE :



2000 Kcals
INTAKE



2000 Kcals
EXPENDITURE



NORMAL
BODY WEIGHT



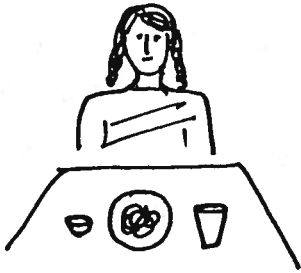
2000 Kcals
INTAKE



1000 Kcals
EXPENDITURE



INCREASE IN
BODY WEIGHT



2000 kcals
INTAKE



3000 kcals
EXPENDITURE



DECREASE IN
BODY WEIGHT

* Do not eat foods which raise your blood glucose levels suddenly (i.e. refined foods need to be avoided).

Ex. Sugars, sweets, syrups, sherbets, chocolates, sweetened fruit drinks, cakes, pastries etc.

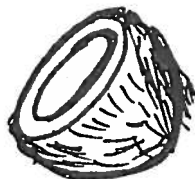
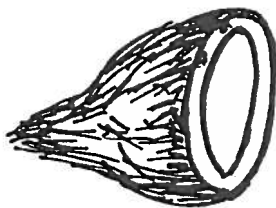
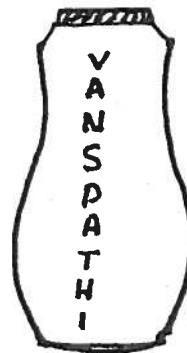
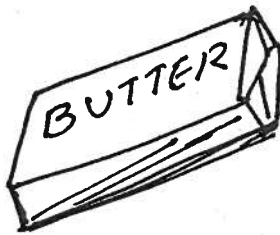
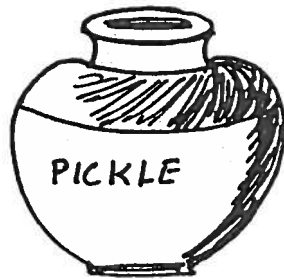


- * Eat more fibre contained foods, because fibre takes time to get digested, thereby causing a slow release of glucose into the blood. It also helps in decreasing the cholesterol content of the blood. Foods rich in fibre are whole cereals, millets and pulses, whole grain flours, fruits, vegetables, green leafy vegetables.

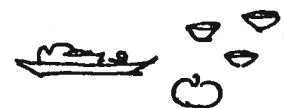
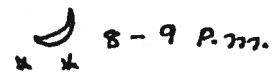
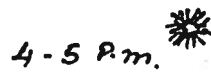
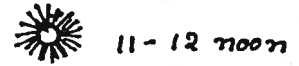
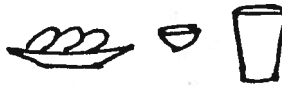
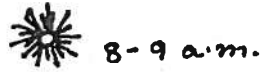
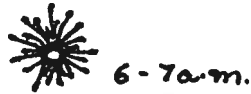


- * Limit consumption of foods which increase the fat content of your blood (pre-disposing factor for hypertension and heart problems).

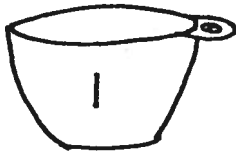
Eg. Ghee, butter, cream, ice-cream, pastries, cakes, pickles, coconut, deep fried foods, fatty tissues of meats, pork.



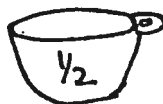
* Take smaller meals more number of times so that your blood glucose level does not fluctuate widely, but maintains a normal range.



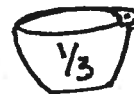
* Measure the food (prescribed by your nutritionist) at least for the first few days, to find out the portion size of foods.



200 ml.



100 ml.



80 ml.



50 ml.



200 ml.

=

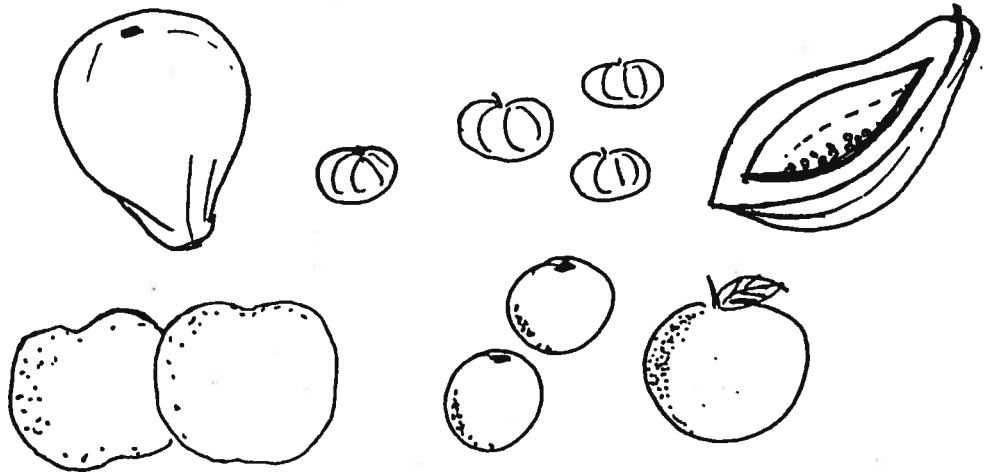


glass

200 ml.

- * Take at least 2 servings of fruit rich in vitamin 'C' every day (Vitamin 'C' helps in healing of cuts and wounds and diabetes is known to cause local vitamin 'C' deficiency).

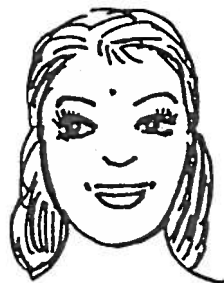
Ex. Guava, amla, orange, lime, mousambi, papaya.



- * Maintain regular meal TIMINGS according to your insulin injections or tablets.

EXERCISE

- * Helps to maintain ideal body weight
- * Helps to decrease fat content of blood
- * Improves circulation to heart and extremities, thereby avoiding complications like gangrene.
- * Helps in making you more vibrant & fit.



COMPLICATIONS

Improper management of diabetes leads to short & long term complications.

SHORT TERM COMPLICATIONS**LOW BLOOD GLUCOSE LEVELS (HYPOGLYCEMIA)**

Occurs within hours.

CAUSES

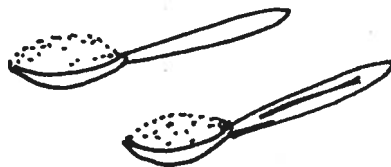
1. Too little food
2. Too-much exercise
3. Too-much insulin

**SYMPTOMS**

Sweating, trembling, restlessness, anger, fainting, confusion and unconsciousness (coma).

WHAT TO DO

Give two spoons of glucose immediately or give sweetend fruit juice.



HIGH BLOOD GLUCOSE LEVELS (HYPERGLYCEMIA)

Occurs within days

CAUSES

- * Too much food
- * Too little exercise
- * Too little insulin

SYMPTOMS

Excess thirst, excess urination, excess hunger, tiredness.

WHAT TO DO

See the doctor immediately. Take lot of fluids (without sugar).

OTHER SERIOUS COMPLICATIONS -

1. EYES



Vision becomes dim, due to damage to retina.

2. HEART



Diabetics are more prone to heart complications (Ex: Coronary heart disease). Therefore foods rich in saturated fats like ghee, butter, fatty tissues of chicken, mutton and pork should be avoided.

3. KIDNEY



More work load on the kidney due to excess urination leads to kidney failure in diabetics.

4. NERVES

Damage to nervous system causes paralysis, loss of sensation, tingling and numbness.

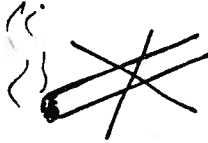
POINTS TO PONDER

- * During illness do not miss your daily insulin intake. Dosage should be taken as suggested by your doctor.
- * Diabetic pregnant women and children need extra nourishment (Discuss with nutritionist).
- * Take good care of hands, feet and folds of the skin.
- * Use Aspartame (Sugar-free) sweetener in tea or other food preparations if you desire.
- * Always carry glucose or a chocolate to use if you feel giddy due to low blood glucose levels.

- * Do not use alcohol as it does not contain food value and causes liver damage.



- * Do not smoke as it constricts veins causing spasms.



- * Do not use tobacco in pan. (upsets stomach as well as causes spasms in veins).
- * Discuss your plans for exercise with your doctor or diabetes educator, before starting it.
- * Always carry your diabetic identity card with you.
- * Your meal plan may need to be changed with time, changes in lifestyle such as work, vacation or travel require adjustments in your meal plan. Visit nutritionist regularly.

FOOD EXCHANGE LIST

To avoid monotony you need to change your foods frequently. The foods you change should approximately give you the same amount of calories. Here are some foods which provide you with the same amount of calories.

| Food | Raw Quantity (gms) | Cooked Quantity in stan- dard cups Vol. | Household measure Cooked Quantity Vol. |
|--|--------------------------|---|--|
| I. CEREALS & PULSES (give 100 cal) | | | |
| Jowar | 30 | - | 1 roti |
| Rice | 30 | 1/2 cup | 1/2 glass |
| Ragi | 30 | - | - |
| Suji | 30 | 1/2 cup | 1/2 glass |
| Wheat flour | 30 | - | 3 small pulkas |
| Channa Dal | 30 | 1/4 cup | 1/2 glass |
| Urad Dal | 30 | 1/4 cup | 1/2 glass |
| Green gram | 30 | 1/4 cup | 1/2 glass |
| Masur Dal | 30 | 1/4 cup | 1/2 glass |
| Rajmah | 30 | 1/4 cup | 1/2 glass |
| II. MILK, MEAT, EGG & FISH (give 100 calories) | | | |
| Milk | 160 ml | 3/4 cup | 3/4 glass |
| Meat | 80 g | - | - |
| Poultry | 100 g | - | - |
| Egg | 185 gms | (6 egg whites) | |
| Fish | 100 g | (Meckerel) | |

Contd..

| Food | Raw Quantity (gms) | Cooked Quantity in stan- dard cups Vol. | Household measure Cooked Quantity Vol. |
|---|--------------------------|---|--|
| III. ROOTS AND TUBERS (provides 50 cal) | | | |
| Carrot | 100 g | 1/2 cup | 1/2 glass |
| Onion | 100 g | 1/4 cup | 1/4 glass |
| Colocasia (Arvi) | 50 g | 1/4 cup | 1/4 glass |
| Potatoe | 50 g | 1/4 cup | 1/4 glass |
| IV. VEGETABLES (provides 50 cal.) | | | |
| Snake groud | 270 g | 1+1/4 cup | 1+1/4 glass |
| Bitter gourd (Karela) | 200 g | 1 cup | 1 glass |
| Bottle gourd | 400 g | 2 cups | 2 glasses |
| Brinjal | 200 g | 1 cup | 1 glass |
| Cauliflower | 160 g | 1 cup | 1 glass |
| Broad beans | 100 g | 1/2 cup | 1/2 glass |
| Drumsticks | 200 g | - | - |
| French Beans | 200 g | 1 cup | 1 glass |
| Cluster beans | 300 g | 1+1/2 cup | 1+1/2 glass |
| Kovai (Donda) | 270 g | 1 cup | 1 glass |
| Ladies finger (Bhendi) | 140 g | 1 cup | 1 glass |
| Cucumber | 385 g | 1+1/2 cup | 1+1/2 glass |

Contd..

| Food | Raw Quantity (gms) | Cooked Quantity in stan- dard cups Vol. | Household measure Cooked Quantity Vol. |
|----------------|--------------------------|---|--|
| Radish (Mooli) | 300 g | 1 cup | 1 glass |
| Tomatoe | 250 g | 1 cup | 1 glass |
| Peas (Mutter) | 50 g | 1/4 cup | 1/4 glass |
| Plantain | 75 g | 1/3 cup | 1/3 glass |

V. FRESH FRUITS
(provides 60 Cals.)

| | | | |
|--------------------------|-----|---|---|
| Amla | 100 | - | - |
| Apple | 100 | - | - |
| Guava | 100 | - | - |
| Watermelon | 375 | - | - |
| Mango | 80 | - | - |
| Orange | 125 | - | - |
| Muskmelon | 375 | - | - |
| Pappaya | 185 | - | - |
| Lemon | 100 | - | - |
| Sweet lime (Mousambi) | 140 | - | - |

VI. GREEN LEAFY VEGETABLES
(provides 50 Cals.)

| | | | |
|-----------------------|-----|---------|-----------|
| Amaranth | 100 | 1/2 cup | 1/2 glass |
| Cabbage | 185 | 1 cup | 1 glass |
| Coriander (Kotmir) | 110 | - | - |

Contd..

| Food | Raw Quantity (g) | Cooked Quantity in stan- dard cups Vol. | Household measure Cooked Quantity Vol. |
|-----------------------------|------------------------|---|--|
| Fenugreek leaves (Methi) | 100 | 1/2 cup | 1/2 glass |
| Gogu | 90 | 1/3 cup | 1/3 glass |
| Mint (Pudina) | 100 | - | - |
| Spinach (Palak) | 200 | 1/2 cup | 1/2 glass |

MISCELLANEOUS

| | | |
|----------------------|---|--|
| Bread slices | - | 2, give 70 calories |
| Coconut water | - | 1 glass or 200 ml gives 50 calories. |
| Salt Biscuits | - | 3, gives 50 calories |
| Sweet Biscuits | - | 2, gives 50 calories |
| Popcorn | - | 15 g (1 table spoon before popping) 50 Cals. |
| Vegetable clear soup | - | 1 glass (200 ml) 15 calories |
| Vegetable thick soup | - | 1 glass (200 ml) 90 calories |
| Tomato cream soup | - | 1 glass (200ml) 110 calories |
| Chicken cream soup | - | 1 glass (200ml) 110 calories |



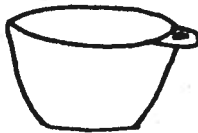
TEASPOON - 5 gm



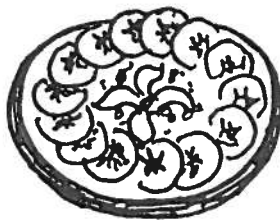
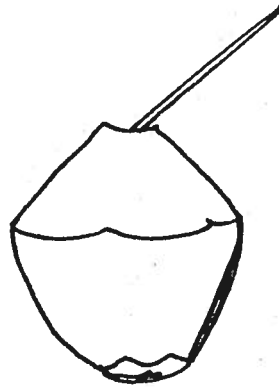
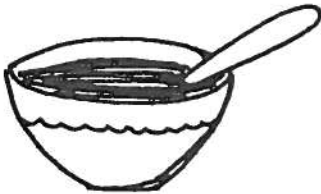
TABLESPOON - 15 gm



GLASS - HOUSE HOLD MEASURE
200 ml



STANDARD MEASURING CUP
200 ml



SAMPLE MENU

1400 CALORIES WEIGHT REDUCING DIET

| | | |
|--------------------------------------|--|---|
| EARLY MORNING : 6 - 7 a.m. | Tea or Coffee | without sugar |
| BREAK-FAST : 8 - 9 a.m. | Idli - 2 (Medium size) | with sambar or chutney |
| | Pulka (flour 25 g) | 2 small size with green leafy vegetable curry |
| | Milk 100 ml | 1/2 glass |
| MID-MORNING : 11 - 12 noon | Butter milk 200 ml. 1 Egg white (boiled) | 1 glass |
| LUNCH : 1 - 2 p.m. | Rice - 50 g | 1 glass cooked rice |
| | Dal - 25 g | 1/4 glass cooked dal |
| | Vegetables - 100 g | 1/2 glass cooked |
| | Green leafy veg. - 100 g. | 1/2 glass cooked |
| | Curds - 50 g. | 1/2 glass |
| SNACKS : 4 - 5 p.m. | One fruit serving or Tea or coffee with 2 Marie biscuits | |
| DINNER : 8 - 9 p.m. | Same as lunch, take 4 small pulkas in place of rice (if you desire) | |
| BEFORE BED : | 100 ml of milk | 1/2 glass |

Oil allowance per day - 30 gms. i.e. 2 table spoons

SAMPLE MENU

1800 CALORIES DIET

| | | |
|--------------------------------------|---|---|
| EARLY MORNING : 6 - 7 a.m. | Tea or Coffee | without sugar |
| BREAK-FAST : 8 - 9 a.m. | Idli - 4 (Medium size) | with sambar or chutney |
| | Pulka - 4 (50 gms. flour) | Small size with vegetable curry |
| | Milk - 200 ml | 1 glass |
| MID-MORNING : 11 - 12 noon | 1 Egg white Butter milk 200 ml vegetable salad | (boiled) 1 glass |
| LUNCH : 1 - 2 p.m. | Rice - 75 gm Dhal - 25 gms Vegetables - 100 g Green leafy vege- tables - 100 g Curds - 100 g | 1 1/2 glass cooked rice 1/4 glass cooked dhal 1/2 glass cooked 1/2 glass cooked 1/2 glass |
| SNACKS : 4 - 5 p.m. | 2 fruit servings Tea or Coffee | or with 4 Marie biscuits |
| DINNER : 8 - 9 p.m. | Same as lunch, take 6 small pulkas in place of rice if you desire | |
| BEFORE BED : | Milk - 100 ml | 1/2 glass |

Oil allowance per day - 30 gms. i.e. 2 table spoons

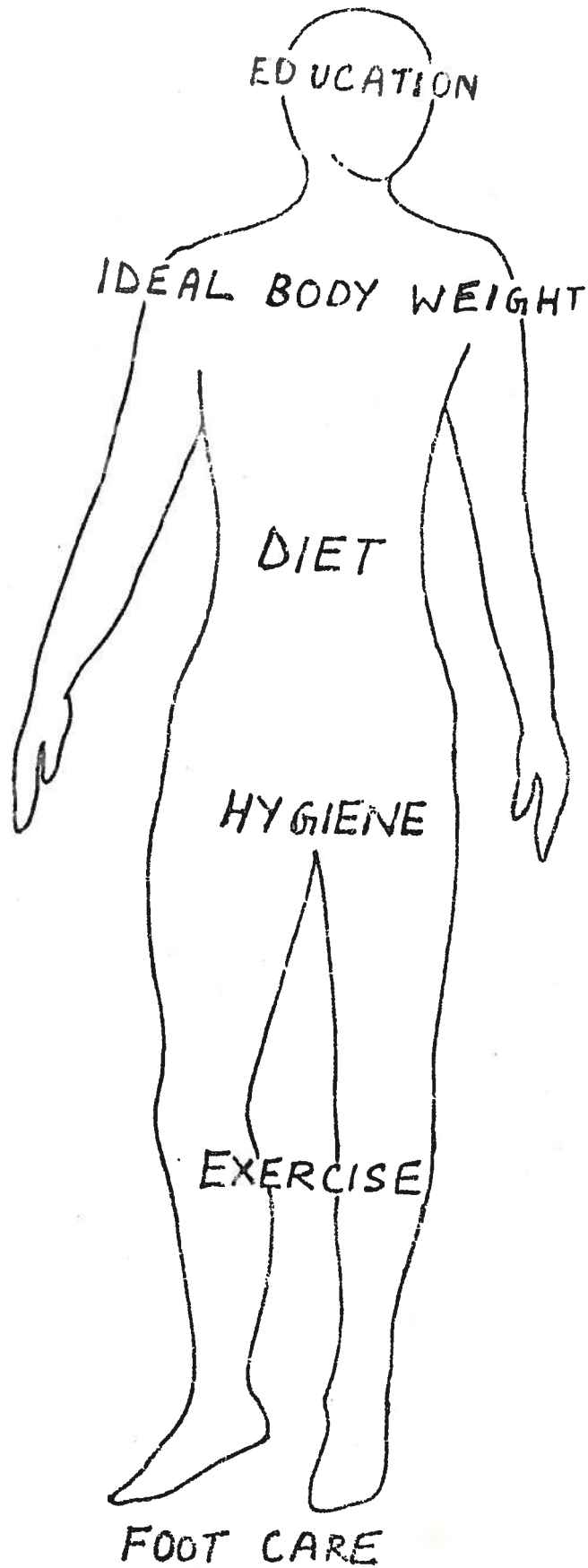
SAMPLE MENU

2200 CALORIES DIET

| | | |
|--------------------------------------|--|------------------------------------|
| EARLY MORNING : 6 - 7 a.m. | Tea or Coffee | without sugar |
| BREAK-FAST : 8 - 9 a.m. | Idli - 6 (Medium size) | with sambar or chutney |
| | Pulka - 6 (75 gms. flour) | Small size with vegetable curry |
| | Milk - 200 ml | 1 glass |
| MID-MORNING : 11 - 12 noon | 1 Egg white Butter milk 200 ml Salad | (boiled) 1 glass |
| LUNCH : 1 - 2 p.m. | Rice - 100 gm | 2 glasses cooked rice |
| | Dhal - 50 gms | 1/2 glass cooked dhal |
| | Vegetables - 100 g | 1/2 glass cooked |
| | Green leafy vege- tables - 100 g | 1/2 glass cooked |
| | Curds - 100 g | 1/2 glass |
| SNACKS : 4 - 5 p.m. | 2 fruit servings Tea or Coffee | or with 4 Marie biscuits |
| DINNER : 8 - 9 p.m. | Same as lunch, in the place of rice take 8 small pulkas if you desire | |

Oil allowance per day - 35 gms. i.e. 2 table spoons +
1 tea spoon

FOR YOUR VICTORY OVER DIABETES



Appendix - 7

Sample of High Carbohydrate high fibre, low fat and optimum protein 1800 kilocalories Diet

| | | |
|---------------------------|---|--|
| Early morning 6-7 A.M. | : | Tea 1 cup |
| Break-fast 8-9 A.M. | : | Idli - 4 in number Sambar - 1 cup Milk - 1 glass |
| Mid-morning 11-12 noon | : | One Egg boiled (without yellow) Butter milk - 1 glass |
| Lunch 1-2 P.M. | : | Rice - 1 1/2 cups Phulka - 2 (small size) Mutton - 1 Katori Cabbage - 1/2 cup Amaranth - 1/2 cup Curds - 1 Katori |
| Snacks 4-5 P.M. | : | Tea 1 cup Sprouted greens - 1/4 cup |
| Dinner 8-9 P.M. | : | Phulka - 4 (small size) Rice - 1/2 cup Beans - 1/2 cup Spinach - 3/4 cup + Dal Salad |
| Before bed 10 P.M. | : | 1 cup hot milk |

Appendix - 8

Restricted foods

Sugar, jaggery, chocolates, cool drinks, cakes, ghee, butter, cream, pickles, jams and deep fried foods.

Appendix 9

Inbetween meal snacks

1. Ragi porridge (15 gms of Ragi powder used)
2. Salad (Kheerai - 100 g; Tomatoe - 50 g; carrot - 50 g)
3. Cucumber salad (cucumber - 100 g; sprouted gram - 10 g; tomatoe - 10 g)
4. Water melon - 290 g
5. Musk melon - 240 g
6. Pappaya - 150 g
7. Apple - 87 g
8. Orange - 100 g
9. Lemon - 87 g
10. Coconut water - 200 ml

Appendix 10

Break fast items

1. Phulka 6 (small size)
greens curry 1 katori
2. Idli 4 in number (medium size)
sambar 1 cup
3. Dosa - 3 in number
roasted Bengal gram dhal chutney - 1/2 katori
4. Pesarattu - 3 in number (medium size)
with cucumber slices

5. Upma - 1 cup
roasted Bengal gram dhal chutney 1/2 katori
6. Pongal - 2 cups
curds - 1/4 cup
7. Missi Roti - 4 numbers (small size)
curds - 1/4 cup
8. Jowar Roti - 2 in number (medium size)
greens curry - 1 katori

Appendix - 11

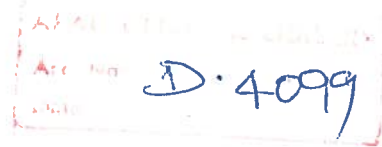
Historical Review of Diabetes Nutritional Management

| Date | Scientist/Source | Recommendations |
|-------------|---|--|
| 1550 BC | Papyrus Ebers | Wheat grains, fresh grits, grapes, honey, berries, sweet beer. |
| 5 AD | Aretaeus the Cappadosian | Milk or cereals and starch with fruits and sweet wines. |
| 6th Century | - | Limit rice, flour and sugar |
| 1675 | Thomas willis | Increased carbohydrates to replace lost sugar, choices limited to milk and barley water boiled with bread, lime water and opium. |
| 1797 | John Rollo | Avoid carbohydrates; break fast was a milk mixture and lime water with bread and butter, lunch was plain blood puddings, mode of blood and suet only, supper was game or old rancid meats and fat; opium. |
| 1857 | Piorry | Agreed with Willis; 125 g of sugar candy and 2 portions of meat, fluid restriction. |
| 1860 | Charles Henry Pile Appolinoire Bouchardat | Strict use of animal food alone, more fat, no milk, to break monotony of the Rollo diet, he added green vegetables, that were boiled and the water discarded, encouraged low calories and even periodic fasting. |
| 1869 | Donkin | Skim milk diet |
| 1880 | Bernhard Naunyn | Pioneered use of measured diets, limited protein and carbohydrates, restricted calories, 24 hour fasts in less severe cases. |

Contd..

| Date | Scientist/Source | Recommendations |
|------|------------------------------|---|
| 1892 | Dmjardin Beaumetz/ Mosse | Diet high in potatoes |
| 1902 | Carl Hanko Von Noorden | Oat meal diet |
| 1912 | Frederick Allen | "Allen starvation treatment" low carbohydrates increased fat, calorie restricted diet. |
| 1921 | - | Insulin identified, diminished interest in diet. |
| 1929 | H. Rawlegeylin | Attempted to 'normalize' the diet did not find a need to increase insulin with drastically increased amounts of carbohydrates (5 times) related effect of carbohydrates to amount of fat that was limited. |
| 1930 | Israely M. Rabin- Oscitch | Limited fat to less than 50 g/d to help lower cardio-vascular- renal disease, patients to be 5 to 10 lbs under weight. |
| 1935 | Sir Harold Himsworth | Increased carbohydrate in Diet |
| 1935 | Walter Kempuer | 90-95% carbohydrate diet |
| 1935 | William F.VonEck | 90% carbohydrate diet |
| 1935 | Ernest & Co-workers | 72% carbohydrate diet |
| 1950 | - | Hypoglyceamic agents. Introdu- ction of exchange diet with pre-calculated meal plans. |

Source : Margaret, 1987 Hand Book of Diabetes, Nutritional
Management



Appendix 12: Hormonal Response to exercise

| Hormone | Effect | Result |
|---------|----------|---|
| Insulin | decrease | facilitates glucose production by liver stimulates glucose utilization by muscle, mobilize FFA (free fatty acids) from adipose tissue). |

Counter-regulatory hormones:

| | | |
|---------------------------|----------|---|
| Glucagon | increase | stimulates glucose production by liver |
| Epinephrine | increase | stimulates glucose production by liver stimulates lipolysis in adipose tissue |
| Norepinephrine | increase | stimulates glycogenolysis in both liver and muscle stimulates lipolysis in adipose tissue |
| Growth hormone & cortisol | increase | over longer periods of exercise: increase gluconeogenesis increase lipolysis Decrease insulin-stimulated glucose uptake in tissue. |

Source: Margaret 1987 Hand Book of Diabetes Nutritional Management.