DIABETES MELLITUS

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CONTINUING EDUCATION

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Course Objectives

(Overall behavioral objectives of the entire course)

At the end of this course, each nurse will be able to:

1. Define diabetes mellitus as a genetically determined disorder of metabolism.
2. Identify the manifestations of insulin deficiency and the inability to tolerate carbohydrates.
3. Identify diabetes mellitus in its completely developed state characterized by fasting hyperglycemia, microangiopathic and arteriosclerotic vascular disease and neuropathy.
4. Identify the various types of lesions that may lead to insulin insufficiency.
5. Discuss and identify insulin as a hormone that regulates the amount of glucose in the blood.
6. Identify and discuss Type 1, Type 2, and Gestational diabetes and their respective goals.
7. Identify the factors that increase the risk of diabetes.
8. Identify the factors in the development of DKA and its treatment.
9. Discuss the pathogenesis of diabetic ketoacidosis.
10. Identify the pathophysiology of hypersosmolar coma and its treatment.
11. Identify and discuss hypoglycemia.
12. Identify the laboratory procedures used in the diagnosis and treatment of diabetes mellitus.
13. Identify the different types of insulin and their use.
14. Understand blood sugar monitoring and blood sugar fluctuations.

15. Identify oral hypoglycemic agents and their use.

16. Identify the diabetic diet as a well-balanced meal plan tailored to the individual needs, tastes, activity level and life-style.

17. Understand the variety of complications associated with diabetes mellitus and their treatment.

CHAPTER I

INTRODUCTION TO DIABETES MELLITUS

THE ROLE OF INSULIN

AND

RISK FACTORS

INTRODUCTION

Diabetes is a condition that affects the way the body uses food. A genetically determined disorder of metabolism, it is manifested by insulin deficiency and the inability to tolerate carbohydrates.

Diabetes in a completely developed state is characterized by:

- Fasting hyperglycemia
- Microangiopathic and arteriosclerotic vascular disease
- Neuropathy

The incidence of diabetes is high, with more than 16 million Americans having some form of the disease. More than 700,000 new cases are diagnosed each year.

The term diabetes covers a wide spectrum of disability ranging from the elderly, a symptomatic individual with glucose intolerance, to the young patient dependent on exogenous insulin.

The manifestation of diabetes mellitus can be divided into two main groups:

- The acute diabetic syndrome characterized by hyperglycemia, ketoacidosis, and if untreated, death.
- The chronic diabetic characterized by diffuse microangiopathy involving vital tissue.

Diabetes has a heterogeneous etiology. Different types of lesions may lead to insulin insufficiency along with environmental factors, causing an alteration in the function and integrity of the beta cells in the pancreas. These factors include pregnancy, diet, obesity, and infective agents such as the mumps virus.

THE ROLE OF INSULIN

Insulin is a hormone produced by the pancreas. The pancreas either produces no insulin, too little insulin, or the body does not respond to the insulin produced. This causes a build-up of glucose in the blood, creating high glucose levels in the body since the body is unable to utilize the glucose for energy. This high concentration of glucose in the blood is called hyperglycemia.

During the normal digestive process, the body converts food into glucose to be used by the body's cells as an energy source.

Foods eaten are made of carbohydrates, proteins, and fats. These foods are broken down to provide fuel for the cells, which are converted into glucose, a simple sugar.

Glucose enters the cells through receptor sites that accept insulin, which facilitates the passage of glucose to the cell. Excess glucose is stored in the liver and muscles in the form of glycogen. When the body is low on fuel and the blood sugar is low, glycogen, stored in the liver, is released to form glucose. The liver is also able to make new sugar, which it gets from protein taken from muscles.

The pancreas is located in the abdomen behind the stomach. It is attached to the small intestine and spleen. Inside the pancreas are groups of cells called Islets of Langerhans. The Isles of Langerhans comprise approximately 1% to 3% of the weight of the pancreas. These islet cells can be classified as A, B, D, PP cells. Each type cell contains a specific hormone with the B cell, or beta cell, being insulin.

Beta cells regulate blood glucose levels constantly and deliver the required amounts of insulin needed to transfer glucose into the cells. This process keeps glucose levels in the normal range of 60-120mg/ml. Glucose has difficulty entering the cells when there is little or no insulin in the body, or when there is an inappropriate response to the insulin produced.

When blood glucose rises above 180mg/ml, or what is termed the kidney threshold, it is removed from the body in the urine. This is called glycosuria. Often, people with long-term diabetes or kidney disease develop a high kidney threshold, which means that glucose does not spill over into the urine until the blood sugar is extremely high.

The regulation of glucose is closely regulated in normal individuals. In healthy people, the secretion of insulin and the amount of glucose are closely coordinated. The amount of glucose released by the liver is directly proportionate to the amount of glucose used by the tissues. Therefore, if glucose concentration decreases, insulin concentration deceases, and if glucose concentrations increase, so does the need for insulin.

In the diabetic, the renal tubules are unable to absorb all of the glucose filtered by the glomeruli. The renal excretion of glucose requires excretion of water and produces an osmotic diuresis. This diuresis is called polyuria or excessive urination. Polyuria can cause dehydration, resulting in dry skin and blurred vision, which is due to fluctuation in the amount of glucose and water in the lenses of the eye during dehydration. Glucose needs water to flow from the body. Loss of water causes an increase in the serum polarity that stimulates the thirst center in the hypothalamus. This results in a condition called polydipsia, or excessive thirst. Symptoms may range from pronounced to nothing more than a dry mouth.

Polyphagia, or excessive hunger, is caused by the body's inability to transfer, via insulin, glucose through the receptors into the cells. Without glucose as fuel, the cells starve. Since the cells cannot produce energy, the diabetic patient feels weak and tired. The glucose needed for fuel is being lost through the urine. Weight loss occurs in people that produce no insulin because fuel does not enter the cells.

When insulin is low, the body breaks down as fuel, and rapid weight loss occurs. As fat cells breakdown, fatty acids are formed. These fatty acids pass through the liver to form ketones. Ketones are excreted in the urine. This is called ketonuria.

FACTORS THAT INCREASE THE RISK OF DIABETES

Heredity:

There is approximately a 5% risk of developing diabetes if your mother, father, or sibling has diabetes. The risk increases to almost 50% of reported cases, if parents or siblings have diabetes and are overweight.

Obesity:

80% of people with Type 2 diabetes are overweight, with symptoms disappearing with weight loss.

Age:

Fewer beta cells produce insulin with age.

Viruses:

Certain viruses may destroy beta cells.

Faulty Immune System:

Multiple factors may cause the immune system to destroy beta cells, such as infection.

Physical Trauma:

Injury or trauma may destroy the ability of the pancreas to produce insulin.

Drugs:

Drugs used for other conditions could cause the development of diabetes.

Stress:

Hormones at times of stress may block the effectiveness of insulin.

Pregnancy:

Hormones produced during pregnancy can block the effectiveness of insulin.

CHAPTER II

DIABETES MELLITUS

TYPE I

DKA

TYPE 2

HHNC

GESTATIONAL and PREGNANCY

HYPOGLYCEMIA

TYPES OF DIABETES

TYPE I (IDDM)

People who have diabetes fall into one of two categories, referred to as Type 1 and 2. Type 1 diabetes, also known as insulin dependent diabetes mellitus, (IDDM), accounts for approximately 10% of diabetes cases. In Type 1, the body does not produce insulin. Type I diabetes is usually diagnosed during childhood or young adulthood, which is why it is referred to as juvenile diabetes. This disease has a sudden onset with symptoms such as polydipsia, polyphagia, polyuria, weight loss, and fatigue. Type I diabetes tends to be unstable and is very sensitive to exogenous insulin and physical activity.

Symptoms are caused by hyperglycemia and a breakdown of body fats. In order for proper metabolism of carbohydrates, fats, and proteins to take place, insulin must be present. If insulin is not present, multiple changes in the products of metabolism will occur.

DIABETIC KETOACIDOSIS
DKA

As insulin deficiency persists, hyperglycemia and glucosuria intensifies as ketonemia develops. Symptoms continue to progress to dehydration, resulting in low blood volume, increased pulse rate, and dry flushed skin. The plasma pH begins to drop as acetone and ketones breakdown. When the plasma level reaches 7.2, the respiratory center is stimulated, and the patient's breathing becomes shallow and rapid (Kussmaul respiration) with a fruity odor. This is the body's way of trying to prevent a further decline in pH. The increased loss of CO2 from the lungs reduces plasma carbonic acid to acceptable levels. Metabolic acidosis occurs when the body's buffer system is unable to maintain normal pH with the assistance of the respiratory compensatory mechanisms. A continued decline in pH with progression of symptoms including stomach pains, vomiting, and decreased level of consciousness, results in diabetic ketoacidotic coma (DKA) and death.

Protein catabolism is a result of insulin deficiency. This causes a release of nitrogen and potassium into the circulation. Late in ketoacidosis, renal function falters due to hypovolemia and dehydration. Urinary loss exceeds that of electrolytes so that plasma sodium and potassium may be increased in spite of the total body deficiency of these ions. With the administration of insulin, the potassium re-enters the cells along with the glucose. Fluid therapy causes expansion of plasma volumes, and a return of normal renal function, which is essential.

Factors in development of DKA

- Failure to take insulin
- Insufficient amount of insulin taken
- Infection
- Nausea/ vomiting with omission of insulin since food isn't eaten
- Resistance to endogenous insulin produced by the body

DKA TREATMENT SUMMARY

NURSING IMPLICATIONS

Dehydration:

The patient in DKA is treated initially with intravenous fluids, progressing to oral fluids as the patient's condition improves. Urinary output, vital signs and specific gravity are monitored closely in order to evaluate the effectiveness of fluid replacement. Normal saline is usually the IV solution of choice. The adult fluid deficit is approximately 6-12 liters.

Insulin:

Patients in DKA require rapid-acting insulin. Regular insulin U-100 is administered IVP, or in severe cases of DKA, an insulin drip is started. Insulin is given until the blood sugar reaches about 250mg/ml. An insulin drip consists of placing 100 units of regular insulin in 500cc's of D5W solution. The drip is then titrated according to the desired dosage per hour.

Electrolytes:

Electrolyte replacement is managed based on laboratory results. As hydration progresses, laboratory values will change as the electrolytes move from compartment to fluid compartment. Potassium replacement becomes more crucial with the administration of insulin. Insulin causes potassium to move into the intracellular compartment at the time acidosis is corrected. Potassium chloride is given IV up to 10 mEq/hour or 100 – 200 mEq/day. Magnesium sulfate is administered up to 2 mEq/kg every four hours. Sodium and phosphate are given as required by laboratory findings.

PATHOGENESIS OF DIABETIC KETOACIDOSIS

INSULIN DEFICIENCY

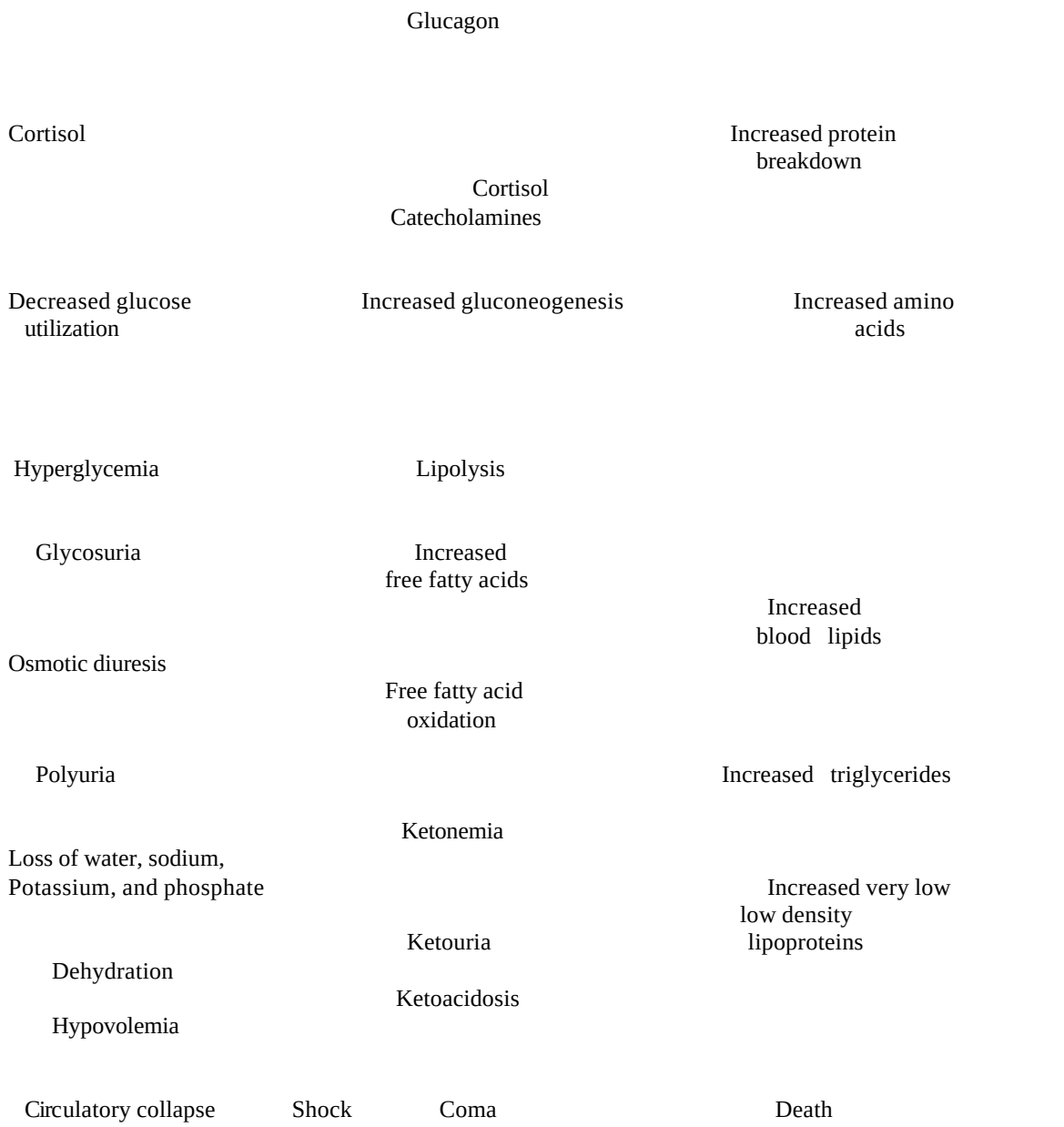

FIG: 1

CLINICAL PICTURE AND TREATMENT
OF THE DKA PATIENT

SIGNS AND SYMPTOMS	LABORATORY FINDINGS	TREATMENT
Insidious onset, Fatigue, muscle weakness	Serum glucose less than 800 mg/ml	I.V. regular insulin
Nocturia Polyuria Polydipsia	Positive serum ketones Serum sodium usually low	Fluid regulation Electrolyte replacement
Polyphagia Weight loss	Serum potassium-elevated, normal or low	
Dehydration	Less than 10 mEq potassium	
Nausea, vomiting, diarrhea, blurred vision	Blood Ph less than 7.32	
Acetone breath Hot, dry flushed skin Sunken eyeball Tachycardia Hypotension Kussmaul respirations Coma Death	Serum osmolarity less than 330 mOsm/l Urine glucose 2+ or greater Urine ketones, large	
TABLE: 1		

TREATING TYPE I DIABETES

A regimen of insulin injections, diet, exercise and monitoring of blood glucose levels is used in treating Type I diabetes. The diabetic must learn to balance insulin, food and exercise.

Successful treatment of diabetes mellitus requires a combined effort by:

- The physician having a complete understanding of the particular problems in each case
- The nurse educator, and how well the patient has been instructed
- The patient's conscientious following of instructions

Diabetics can lead a relatively normal life if they are well informed concerning their disease and how it should be managed.

TREATMENT GOALS FOR TYPE I DIABETES

NURSING IMPLICATIONS

- **Insulin** injections are necessary for Type I, since the body is unable to produce insulin to transport glucose into the cells.
- **Diet** for the diabetic is well balanced, and controls the types and amounts of food eaten.
- **Exercise** improves muscle tone, strength, and the feeling of well-being, while reducing insulin requirements.
- **Monitoring** blood glucose is essential in order to design a diabetic care program and maintain good control.

PERSONAL GOALS FOR TYPE I DIABETES

- Increase self-reliance and self-sufficiency
- Lead an active life that is as close to normal as possible
- Balance diet, exercise, and insulin
- Control blood glucose level, thereby increasing protection of the heart, nerves, blood vessels, eyes, and kidneys
- Maintain body weight within normal range.

TYPE 2 DIABETES (NIDDM)
NON-INSULIN DEPENDENT DIABETES MELLITUS

TYPE 2

Approximately 85% to 90% of all diabetics have Type 2 diabetes, or non-insulin dependent diabetes mellitus, (NIDDM). Type 2 diabetics are usually over 40 and usually overweight. Type 2 diabetes often goes undetected for long periods of time, since symptoms are usually not pronounced. Insulin is produced, but it is not enough, or it does not work properly to transport glucose through the receptor cells. Type 2 diabetics can often be controlled with a carefully planned diet, an exercise program, oral medication, or insulin, used as necessary.

Uncontrolled Type 2 diabetes results in hyperglycemia. Since symptoms have an insidious onset, the patient may not recognize that there is any difficulty. Some of the following symptoms that may be experienced include: polydipsia, polyuria, increased fatigue, blurred vision, slow-healing cuts or sores, dry, itchy skin, numbness and tingling feet.

Left uncontrolled for a long period of time, Type 2 diabetics develop more serious symptoms such as severe hyperglycemia, dehydration, confusion, and shock. This is called "hyperglycemic hyperosmolar non-ketotic coma."

These symptoms are most common in the elderly population and people suffering from illness or infection.

TREATMENT GOALS FOR TYPE 2 DIABETES

NURSING IMPLICATIONS

- **Diet** control helps regulate weight, which is extremely important in Type 2 diabetes. Eating healthy foods and following a diet program if overweight, is essential and should be stressed in diabetic teaching.

- **Exercise** burns calories, which helps control weight. It also stimulates insulin function to allow the body's production of insulin to work more efficiently.

- **Oral hypoglycemic agents** stimulate the beta cells in the pancreas to produce more insulin and help the insulin work more effectively.

- **Monitoring** blood glucose is essential for all diabetics.

PERSONAL GOALS FOR TYPE 2 DIABETES

- Learn about your disease and how it is managed
- Balance diet, exercise and medication
- Maintain appropriate blood sugar levels
- Maintain body weight within normal range

TYPE I VERSUS TYPE 2 DIABETES

	TYPE I (IDDM)	TYPE 2 (NIDDM)
Age of onset	Usually younger than 40	Usually older than 40
Body weight	Thin	Usually overweight
Symptoms	Sudden onset	Insidious onset
Insulin produced	None	Too little, or not effective
Insulin requirements	Exogenous insulin required	May require insulin

Table: 2

WARNING SIGNS OF DIABETES

Type I (IDDM)	Type 2 (NIDDM)
Sudden onset	Insidious onset
Polyurea	Fatigue
Polydipsia	Blurred vision
Polyphagia	Tingling or numbness in hands and feet
20 pound weight loss	Itching
Irritability	Any symptoms of IDDM or hard to heal wounds
Weakness and fatigue	Frequent bladder infections
Nausea, vomiting	

Table: 3

GESTATIONAL DIABETES

Due to hormonal changes during pregnancy, some women experience a rise in blood sugar level during pregnancy. Gestational diabetes can be controlled by diet, with some women needing insulin. Pregnant women are routinely checked for gestational diabetes between the 24^{th} and 28^{th} week of pregnancy. A 50% glucose challenge test is given to the patient. If the level of glucose exceeds 150mg/ml, one hour after administration, the test result is considered positive. These patients are then given an oral glucose tolerance test. If the results of this test are normal, it is recommended that the test be repeated at 32 weeks if the patient is obese, and older than 33 years of age. Patients with a history of gestational diabetes automatically test during their initial pre-partum visit. If the result is negative, the test is repeated at 24 to 28 weeks.

PREGNANCY AND DIABETES

For women with insulin dependent diabetes, pregnancy requires special attention. Hypoglycemia may be a problem during the first trimester due to morning sickness or nausea. Hypoglycemia may also occur because the baby takes sugar from the mother's blood to support its rapid growth. Insulin requirements may double or triple during the second and third trimesters of pregnancy. With a good meal plan, adequate exercise, rest, frequent blood glucose monitoring, and insulin adjustments, staying in control of diabetes can be achieved.

Care during pregnancy

In order to maximize the chances of delivering a healthy, normal baby the mother should:

- See the doctor every 1 to 2 weeks
- Maintain prescribed diet and exercise plan
- Check blood glucose levels 4 to 8 times per day, and record the results
- Inject insulin as prescribed, and make adjustments as necessary
- If control is not possible, hospitalization may be necessary

Pregnant patients with diabetes are at risk for developing polyhydramios or excessive amniotic fluid and toxemia, which is characterized by elevated blood pressure, protein in the urine, and edema of the hands and feet. Retinopathy and proteinuria, which is associated with decreased renal function, progresses while the patient is pregnant, but usually reverses after delivery.

Delivery

Most pregnant women with diabetes or gestational diabetes may complete pregnancy and begin labor naturally. However, to be safe, obstetricians may deliver their patients slightly before the due date by inducing labor or by Cesarean section. These babies are usually cared for in a high risk or intensive care nursery. This ensures a close watch and quick treatment for any problems that may occur.

Diet

In any pregnancy it is essential to eat the proper foods to meet the nutritional needs of the mother and fetus. In a patient with diabetes, it is also important to maintain a proper diet to keep blood sugars as normal as possible.

During the second and third trimesters of pregnancy, the daily need for calories increases by 300 calories. Since the need for protein also increases during pregnancy, it is necessary to increase the protein intake without increasing the fat intake. Increasing low-fat dairy products and making certain that whole grain cereals and bread, along with fruits and vegetables, are part of the daily diet usually increase vitamins and minerals, which are needed in large amounts

The diet plan can be divided into six groups:

- Milk and milk products (4 servings per day)
- Meat, fish, poultry, meat substitutes (5-6 servings per day)
- Bread, cereals, and other starches (5-6 servings per day)
- Fruits (2 servings per day)
- Vegetables (2 servings per day)
- Fats

It is essential to include the specified servings of foods from all groups since each group provides its own combinations of vitamins, minerals and nutrients. Meals plans should contain a variety of foods within each food group. By using all six groups, and the amounts recommended, the fetus is likely to get all of the nutrients needed for its growth and development. Omitting foods from one food group will leave the diet inadequate in other nutrients.

NEONATAL MACROSOMIA

Neonatal macrosomia is a weight gain by the fetus greater than 4.5 kgs. This weight gain is in direct relationship to the control of maternal glucose. This is especially true of patients with gestational diabetes and those with insulin-dependent diabetes. If the maternal blood has an excess amount of glucose, the fetus produces more insulin in an attempt to use the glucose. The extra glucose is converted to fat. The combination of high maternal glucose levels, plus high insulin levels in the fetus, results in large fat deposits that cause an excessively large fetus, over 10 pounds. If after doing a physical examination it is felt that the fetus is macrosomic, an ultrasound is used to measure the size of the fetus. With glycemic control, the incidence of macrosomia is greatly reduced.

PERINATAL MORTALITY

Before exogenous insulin was developed, infant mortality associated with diabetes was 100%. With the careful administration of insulin, the control of glucose, along with close obstetrical monitoring, the perinatal mortality associated with diabetes has been reduced to that associated with normal pregnancy.

CONGENITAL MALFORMATIONS

Congenital malformations in infants whose mothers have diabetes are approximately four to five times the normal rate. These abnormalities are responsible for 50% of the prenatal deaths. The critical period of control for the maternal diabetic must be during the first 3 months of pregnancy. Abnormalities are found throughout all the organ systems.

APPROACHES TO TREATMENT

NURSING IMPLICATIONS

Glucose levels with the pregnant insulin-dependent diabetic should be maintained in the 60 to 100 mg/dl range. The blood glucose level should be monitored before meals and at bedtime. This value is assessed every 6 weeks in the pregnant patient.

Control of blood sugar levels during labor is extremely important. If the maternal blood sugar becomes elevated, the baby's blood sugar will also rise. After delivery, the high insulin levels in the baby can drop the newborns blood sugar level to a hypoglycemic level. This happens because it no longer has the high blood sugar concentrations of the mother.

Sulfonylureas are usually discontinued, and intermediate-acting insulin is usually combined with regular insulin to maintain normoglycemia in the mother.

Insulin requirements during the last half of the pregnancy can double from the prepartum insulin requirement. When labor begins, the mother may be given insulin intravenously throughout labor.

Diet is an extremely important aspect of care with gestational diabetes and diabetes and pregnancy in general. The diet should be tailored to provide adequate nutrition for the mother and the fetus. A proper diet can often keep blood sugar levels within normal range. Specific attention should be paid to the total caloric intake eaten daily. It is also necessary to avoid foods that increase blood sugar levels, and to use the foods that help the body maintain a normal blood sugar, such as fiber provided by vegetables, dried beans, cereals and other starchy foods. These foods decrease the amount of insulin the body needs to keep blood sugars within in normal range.

With adequate teaching and conscientious medical care, the diabetic patient can expect to have a relatively normal pregnancy.

HYPERGLYCEMIC HYPOSMOLAR NONKETOTIC COMA (HHNC)

Hyperglycemic hyperosmolar nonketotic coma (HHNC), is described as severe hyperglycemic and hypertonic dehydration without significant ketoacidosis.

HHNC is responsible for approximately 5% to 15% of all hospital admissions for diabetic coma. This disorder is not restricted to the diabetic patient. Patients with diabetes, central nervous system damage, insipidus gastrointestinal hemorrhage, hypothermia, or drugs such as thiazide diuretics, can develop HHNC. Most patients affected with HHNC have a predisposition to impaired renal function.

Hyperglycemia and hyperosmolarity are present in DKA as well as HHNC. This makes it very difficult to completely separate these diseases. The precipitating factors are the same and usually occur in patients with mild diabetes mellitus not requiring insulin therapy.

Hyperglycemia is extreme, between 900mg and 3000mg/100 ml, while ketonemia is mild or undetected, and acidosis is absent.

Low serum insulin levels cause problems in carbohydrate, fat and protein metabolism. Because of these alterations, blood sugar increases in direct relation to the increased production of glucose by the liver and the deceased use of glucose by the cells. With the lack or total absence of insulin production, glycosuria develops, which causes a fluid and electrolyte imbalance.

The production of protein is stopped along with amino acid uptake. With the breakdown of protein, the liver is supplied with more products for gluconeogenesis. Fat is broken down causing an increase in fatty acids. Even though there is an increase in fatty acids, the serum level of free fatty acids in HHNC is lower than in DKA. This is one way to distinguish the two diseases. Increased glucagon levels and gluconeogenesis is responsible for elevated blood sugar levels, along with impaired renal function. Since the kidneys are unable to excrete the excess glucose, or maintain fluid balance, hyperosmolarity and dehydration occur. Serum osmolarity is increased due to excessive blood sugar while increased sodium concentration occurs because of dehydration.

Patients that progress to a hyperosmolar nonketotic coma have sufficient circulating insulin to retard the massive release of fatty acids that are seen in ketoacidosis, but not enough insulin to stimulate the entry of glucose into peripheral tissues.

PATHOPHYSIOLOGY OF HYPEROSMOLAR COMA

HYPEROSMOLAR COMA

DIMINISHED INSULIN RESERVE

DECREASED GLUCOSE UTILIZATION
(Muscle, Liver)

INCREASED GLUCOSE PRODUCTION
(Liver)

HYPERGLYCEMIA

GLUCOSURIA

OSMOTIC DIURESIS

LOSS OF WATER, SODIUM, POTASSIUM

DEHYDRATION

HEMOCONCENTRATION
HYPOVOLEMIA

PRERENAL AZOTEMIA

HYPERVISCOSITY

ACIDOSIS

SHOCK

THROMBOSIS

LACTIC ACIDOSIS

INCREASED SHOCK

IRREVERSIBLE SHOCK

DEATH

FIG: 2

HHNC TREATMENT SUMMARY

DEHYDRATION

Patients with HHNC are treated with rapid IV fluid replacement (Table 4). Lactated Ringers is the solution of choice. The following plasma expanders are also used:

- Dextran 70 (macrodex), 500-1000 ml of 6 % solution, IV
- Dextran 75 (gentran), 500-100 ml of 6 % solution, IV
- Dextran 40 (LMD), 10-20 ml/kg of 10% solution, IV

INSULIN

Regular insulin U-100 is given IVP until the blood sugar reaches about 250 mg/ml.

ELECTROLYTES

Sodium and phosphate are given as required by laboratory findings. Potassium chloride is given IV at 10 mEq/hr., up to 100-200 mEq/day. Magnesium sulfate is given up to 2 mEq/kg every four hours.

CLINICAL PICTURE AND TREATMENT
HYPERGLYCEMIC HYPEROSMOLAR NONKETOTIC COMA
HHNC

SIGNS AND SYMPTOMS	LABORATORY FINDINDS	TREATMENT
Rapid thready pulse	Blood sugar over 900 mg/ml	Regular insulin
Cool extremities	Ketones moderate, small	Fluid regulation
Normal to low blood pressure	Sodium, normal, elevated or low	Electrolyte replacement
Orthostatic hypotension	Potassium, normal or elevated	
30% in “frank shock”	Blood pH, normal	
Extreme thirst	Free fatty acids less than 1000mEq/L	
Weight loss	Serum osmolarity, greater than 350 mOsm/L	
Nausea and vomiting	Serum bicarbonate greater than 16 mEq/L	
Impaired consciousness		
Visual changes		
Increased or decreased reflexes		
Aphasia		
Seizures		
Hemiparesis		
Muscle weakness, abdominal and leg cramping		
Hypothermia		
Flushed face and sunken eyeballs		

TABLE: 4

NURSING INTERVENTION FOR THE PATIENT WITH DKA OR HHNC

NURSING DIAGNOSIS	NURSING INTERVENTION	PATIENT OUTCOME DATA
Fluid Deficit	Start and maintain an IV line and administer fluids at prescribed rate Assess vital signs Mouth care	Vital signs within normal limits Intake equals output Skin turgor is normal No sign of shock
Acid-base Balance	Administration of insulin Monitor laboratory results Assess respiratory status Check urine sugar and acetone	Normal blood pH Blood sugar within normal limits Normal respiratory status Patient is alert and oriented
Electrolyte Imbalance	Close monitoring of electrolytes Electrolyte replacement as ordered Observe for signs and symptoms of electrolyte imbalance	Electrolytes within normal range
Psycho-social Concerns	Explain all procedures to the patient and patient's family Encourage participation in care by the patient and family	Patient and family actively participate in care Patient is able to verbalize fears and concerns
Education	Help the patient and family understand the disease by providing adequate teaching	Patient is able to verbalize an understanding of his disease and is able to function effectively
TABLE: 5		

HYPOGLYCEMIA

The most frequent cause of hypoglycemia in the diabetic patient is self-administration of insulin. This could be a result of an improper insulin regimen, dietary omission, or excessive physical activity.

In the pre-diabetic, glucose is assimilated at a normal rate. Insulin secretory response is delayed, as if the sensory mechanism were not functioning properly. When the response occurs, it is excessive, causing hypoglycemia approximately 3 to 5 hours after a meal.

Regardless of its cause, the symptoms of hypoglycemia are the same. In mild hypoglycemia, the patient experiences:

- Perspiration
- Tremors
- Blurred vision
- Impaired mental function
- Hunger
- Weakness

Bizarre behavior and resistance to help, follow mental confusion in hypoglycemia. A staggering gait and irrational behavior is sometimes misconstrued as drunkenness. Finally, the patient becomes comatose and seizures may occur. If hypoglycemia remains untreated, permanent brain damage or death results.

Treatment for hypoglycemia is aimed at increasing the blood sugar with a fast acting sugar given by mouth. For more severe reactions, it is given in the form of D50%, 25 GMs, IVP.

The correct diagnosis of coma in the diabetic patient is crucial, since the two common causes of coma in the diabetic patient are treated differently. The main factors that differentiate hyperglycemia from hypoglycemia are listed on the following chart.

In emergency situations, before a differential diagnosis can be made, the patient is given D50% 25 GMS, IVP. If the patient does not respond, it is felt that no harm has occurred in treating the patient as though he were hypoglycemic. Treatment is then continued for hyperglycemia.

HYPERGLYCEMIA VS HYPOGLYCEMIA

	HYPERGLYCEMIA	HYPOGLYCEMIA
ONSET	Hours to days	Minutes
EVENTS	Omission of insulin	Omission of a meal; increased activity
SYMPTOMS	Headache, nausea, abd pain, vomiting, polyphagia, polydipsia, acetone breath	Hunger, perspiration, confusion, stupor, headache tremors, fatigue, nervousness, seizures
PHYSICAL FINDINGS	Kussmaul respirations, dehydration, tachycardia	Tachycardia, normal to fast respirations
URINE	Positive for glucose and ketones (strongly)	Negative for glucose and ketones
SERUM	Glucose 400mg/100 ml or more	Glucose less than 40mg/100 ml, ketones are negative
RESPONSE TO GLUCOSE	None	Dramatic
TREATMENT	Regular insulin, fluids, electrolyte replacement	Fast acting glucose given orally or IV in the form of 50% dextrose
TABLE: 6		

CHAPTER III

LABORATORY TESTS

AND

MONITORING

LABORATORY PROCEDURES

The diagnosis of diabetes mellitus has been primarily dependent upon the results of specific glucose tests, along with a physical examination, presence or absence of symptoms, and a medical history. The two main tests used to measure the presence of blood sugar irregularities are the direct measurement of glucose levels in the blood during an overnight fast and measurement of the body's ability to appropriately handle the excess sugar presented after drinking a high glucose drink.

In June 1997, the American Diabetes Association devised new criteria for the diagnosis of diabetes. The blood glucose level considered to be normal was lowered and new recommendations for when to test for diabetes were put into place.

The new criteria for diagnosis are as follows:

- A fasting plasma glucose (FPG) of 126mg/dl or more after no food for 8 hours
- A plasma glucose of 200 or above taken randomly during the day with no regard to meals, along with classic symptoms of diabetes
- An oral glucose tolerance test (OGTT) of 200mg/dl at 2 hours after ingestion of glucose

The classic symptoms of diabetes include:

- Polydipsia
- Polyuria
- Unexplained weight loss

Along with changing the diagnostic criteria for diabetes, two new categories have been added. Impaired fasting glucose (IFG) is when fasting plasma glucose is found to be at or above 110mg/dl but below 126mg/dl. The second category is when the results of the oral glucose tolerance test (OGTT) are between 140mg/dl in the 2-hour sample. This is called impaired glucose tolerance (IGT). Patients in these two categories need to be monitored closely since they are at a higher risk for developing diabetes as well as hypertension and heart disease.

Criteria for who should be tested have also changed. The following should be tested:

- People 45 years and older should be tested, with follow up tests every 3 years
- anyone 10% over their normal body weight
- close blood relatives of those already having diabetes
- those in high risk ethnic groups (Hispanic, African-American, Native American and Asian)
- women who have had gestational diabetes or have delivered a baby weighing over 10 pounds
- those with hypertension
- those with high cholesterol
- those diagnosed with IPG or IFG

Finally, the terms used to describe the types of diabetes have been changed as noted earlier in this text to Type 1 and Type 2. Type 1 has been divided into Immune Mediated Diabetes, or diabetes due to self-destruction of the beta cells by the body, and Idiopathic diabetes, or diabetes having no known cause.

Type 2 diabetes is the inability of the body to use insulin it produces due to insulin resistance.

FASTING BLOOD GLUCOSE

The normal range of fasting blood glucose is between 70-110mg/dl of whole blood. This test is performed after an overnight fast has been initiated. A value above 140mg/dl on at least two occasions typically means a person has diabetes.

ORAL GLUCOSE TOLERANCE TEST

The test is begun with the patient in a fasting state. The fast is maintained for at least 10 hours but not longer than 16 hours. After obtaining an initial blood glucose level, 75-100 Gms of glucose are given to the patient. The blood glucose is measured at ½ hour, 1 hour, 2 hour, and 3 hour intervals. In a normal individual, after ingestion of glucose, the plasma glucose level rises initially, but returns to baseline within 2 hours. In a diabetic, glucose levels rise higher than normal after drinking the glucose drink and come down to normal much more slowly.

The results of a glucose tolerance test in a healthy individual are influenced by diet, physical activity, and age. For the test to be reliable the patient must be in good health and taking no medications that could affect the blood glucose. It is essential that the patient be placed on a diet containing 250-300 Gms of carbohydrate 3 days prior to testing. Other factors known to affect GTT are infection, liver disease, fever, myocardial infarctions, cerebral vascular accidents, and certain medications such as diuretics. Since any of these factors may cause the oral glucose tolerance test to vary, tests that show mildly elevated glucose levels may be run again to make sure the diagnosis is correct.

Glucose tolerance tests may lead to one of the following diagnoses:

Normal Response

A normal response to the OGTT is when the 2-hour glucose level is less than or equal to 110mg/dl.

Impaired Glucose Tolerance

A patient is said to have impaired glucose tolerance when the 2-hour glucose results from the oral glucose tolerance test are greater than or equa1 to 140 but less than 200 mg/dl.

Diabetes

A patient has diabetes when oral glucose tolerance tests show that the blood glucose level at 2 hours is equal to or more than 200 mg/dl. This is confirmed by a second test.

CORTISONE GLUCOSE TOLERANCE TEST

Cortisone GTT is considered the most sensitive test of carbohydrate tolerance. Cortisone increases glucose production through stimulation of gluconeogenesis and interference with the action of insulin at the cellular level. When blood glucose levels rise under the effect of cortisone in people without diabetes, more insulin is secreted, bringing plasma levels back to normal. In patients with diabetes, or a predisposition to diabetes, there is an inability to secrete the increased amount of insulin, resulting in sustained increased plasma glucose levels. In cortisone GTT, the patient is given 50 to 625 gms of cortisone prior to receiving the glucose load. A GTT is then performed as described previously.

A positive test is determined if blood glucose is:

- Greater than 195 mgs at 1 hour
- Greater than 175 mgs at 1 ½ hours
- Greater than 160 mgs at 2 hours

INTRAVENOUS GLUCOSE TOLERANCE TEST

Occasionally, an intravenous glucose tolerance test is performed. This test is used when intestinal absorption of glucose may interfere with an oral glucose tolerance test. This is sometimes indicated when there is a history of gastrointestinal surgery.

BLOOD SUGAR MONITORING

In order to establish an insulin treatment plan, it is essential that blood sugar levels are checked at various times to determine how each part of the treatment plan is working. The blood sugar results help identify adjustments needed in insulin, food and exercise to achieve better control of the disease process.

Reasons for blood sugar fluctuations are:

- Changes in exercise or activity level
- Delayed meals, or change in amounts eaten
- Adding or omitting snacks
- Infection, illness
- Skipped insulin injection
- Mal-absorption of insulin
- Alcohol consumption

There are two common ways that physicians may assess how well diabetes is being controlled. They are:

- Frequent measurements of blood glucose
- Measurement of Glycohemoglobin

Combined, these tests give a fairly accurate picture of the state of glucose control in a diabetic.

Frequent measurements of Blood Glucose

Blood glucose levels can be measured randomly (RBS) from a sample taken any time, or it can be measured in the fasting state. A fasting blood glucose sample is taken when the patient has not eaten in the past 8 hours and is usually done overnight. In a patient with normal insulin production, blood glucose levels return to normal fasting levels within 3 hours of eating. After a meal, diabetics are not able to return to a normal fasting level within a 3-hour period.

The goal is to keep fasting blood sugars under 140mg/dl or lower (in the 70-120mg/dl range). If possible, blood sugars should be tested four times per day to monitor how well sugars are being controlled. Blood glucose measurements are done before each meal and at bedtime. A 2 a.m. blood sugar may be indicated to assess what the blood sugar is doing overnight. A blood sugar of 65mg/dl or greater is needed to avoid overnight hypoglycemia. It is desirable for the patient to keep a diary of these measurements to aid in maintaining normoglycemia.

Measurements of Glycohemoglobin

Hemoglobin A1c or Glycohemoglobin (or glycosylated hemoglobin) is used to assess blood sugar levels over a 2-3 month period. The level of hemoglobin A1c correlates with the patient's recent overall blood sugar levels. If blood sugar levels have been running high the previous month, the level of hemoglobin A1c will be high. Values in the better ranges are consistent with fewer incidences of diabetic complications in later life. Type 1 diabetics will usually have hemoglobin A1c levels drawn every 3 to 4 months, while Type 2 diabetics will often require measurements less frequently.

Values vary from lab to lab. Below are common values for Hemoglobin A1c.
Hemoglobin A1c
Normal: less than 6.5
Excellent: 6.5-7.5
Good: 7.5-8.5
Fair: 8.5 to 9.5
Poor: Greater than 9.5

Blood Glucose monitoring provides data for the patient and the health care team to:

- Identify trends in glucose control
- Identify factors that may cause high or low glucose values
- Evaluate the impact of food, activity or medications on the disease process
- Identify where changes in the treatment plan are needed
- Decide what the patient needs to do when sick
- Confirm whether or not the feelings the patient is having are the result of a low or high blood glucose, or something not related to the diabetes

Increase the frequency of blood glucose checks:

- During periods of stress, illness, or surgery
- During pregnancy
- When low blood glucose is suspected (patient feels symptomatic)
- When there are changes in the patient's treatment program (medication, diet plan, or activity)
- When taking new medications such as steroids

Keeping a log book

The patient should keep a record of their glucose values in a logbook. The records should be include:

- Glucose levels
- Medications taken, especially any changes.
- Changes in activity, food, illness, stress or insulin reactions
- The logbook should be brought to all appointments with the health care providers.

SELF-MONITORING OF BLOOD GLUCOSE

Self-monitoring blood glucose (SMBG) is used as a guide to diabetes control. SMBG is a direct method of monitoring blood glucose level. SMBG allows the heath care provider and the patient to determine the pattern of blood glucose levels and make necessary changes in diet, exercise or the insulin dose. SMBG measures precisely the effects of changes in diet, exercise, and insulin dosage as they relate to blood glucose levels. The immediate information made available by SMBG helps in avoiding insulin reactions and allows for a rapid response to an elevation or decline in blood sugar, and can be used as a basis for treatment.

BLOOD GLUCOSE LEVELS

Time:	**Excellent**	**Good**	**Fair**	**Poor**
Fasting	60-100	100-140	140-180	over 180
After a meal	110-140	140-180	180-220	over 220

Using Glucose Monitoring as a Tool

The patient should be taught the following in order to use glucose monitoring to their best advantage:

- Know glucose target levels
- Learn how to check glucose levels
- Decide when to check glucose levels
- Identify glucose patterns
- Determine what causes blood glucose changes
- Decide what to do to get blood glucose levels back on target

URINE TESTS

Testing for Ketones in Urine (Type I Diabetes)

When there is not enough insulin present to channel glucose into the cells, the body uses stored fat to make fuel available to the cells. The fat in fat cells is broken down to fatty acids, which pass through the liver and form ketones (acetone). Ketones are exhaled and excreted in urine.

A blood sugar greater than 200 along with ketones in the urine is a warning sign of a low insulin level and requires immediate action.

Ketostix, Chemstrip UK, or Acetest tablets can be used to test for ketones.

Testing for Glucose in Urine

Urine testing is still used despite the invention of SMBG because it is easy, painless, non-invasive, and inexpensive. This is important for people on fixed incomes or for those whose insurance policies do not cover the strips used in SMBG.

The result of the urine test indicates the concentration of glucose in the urine. The higher the result of the urine test, the higher the concentration of glucose in the urine. Test results will vary from ½% to 5% depending on the concentration of glucose.

CHAPTER IV

INSULIN AND

ORAL ANTIDIABETIC AGENTS

INSULIN FACTS

Patients with Type I diabetes produce no insulin and must receive insulin by injection. Type II diabetics receive injections if their disease cannot be controlled by diet, exercise, and oral medication. Insulin cannot be taken orally because it is a protein that would be broken down during digestion.

Insulin is measured in units. The unit is a measure of weight. 24 units equal 1 mg. U-100 insulin means that there are 100 units of insulin in 1cc of solution. U-100 syringes are used for measurement.

Because each patient with diabetes is unique, the development of an insulin therapy schedule is dependent upon several factors including:

- How much insulin each person's pancreas is still making
- How sensitive the patient is to insulin
- Lifestyle and activity level
- Eating habits

Insulin regimes can vary from 1 or 2 injections per day, up to 3 or 4 injections per day. The main goal of insulin therapy is to keep the blood sugar levels as close to normal range as possible.

CHARACTERISTICS OF INSULIN

It is very important for the health care provider and the patient to have a clear understanding of the characteristics of each type of insulin to be taken. When reactions occur, or when blood sugar levels are not well controlled at certain times of the day, knowing the action and characteristics of each insulin taken will help to determine where changes need to be made in the insulin therapy schedule.

Characteristics of insulin:

- **Onset:** the time it takes the injected insulin to reach the blood stream
- **Peak:** the time period in which the insulin is working its hardest to lower the blood sugar
- **Duration:** the length of time is insulin is working in the blood stream

Insulin absorption can be affected by:

- the injection site
- exercise
- the accuracy of dosage measurement
- the depth of injection
- environmental temperatures

In order to maintain consistency in daily insulin absorption and action, sites should be varied within the same anatomical region. The abdominal area provides consistent

absorption of insulin, whereas the arms and legs are often affected by exercise. While repeated injections in the same area may delay absorption, massaging the site of injection may cause increased absorption.

Lipodystrophy is characterized by either hypertrophy or atrophy of the subcutaneous adipose tissue at the injection site. Rotating injection sites will facilitate absorption of insulin and help prevent scarring at injection sites. This condition is seldom seen with the use of human insulin and pure pork insulin.

PREMIXED INSULIN

Premixed insulin works well for patients whose insulin requirements are set and for those who may have difficulty measuring their dosage. Although this type of packaging is convenient, premixed insulin does not allow for flexibility in dosage adjustment.

Premixed insulin is a combination of specific proportions of short acting (R) and intermediate acting (N) insulin in one bottle or in an insulin pen cartridge.

Premixed insulin combinations include:

- 10/90
- 20/80
- 40/60
- 50/50

STORAGE OF INSULIN

Insulin is stable at room temperature for approximately one month. Pen-filled cartridges are stable for 21 days. Insulin stored in the refrigerator is good until the expiration date. Extreme temperatures such as freezing, heat or sunlight, can damage insulin.

Always check insulin for normal appearance. All cloudy insulin should appear uniformly cloudy when mixed by rolling between your hands, not shaken. If floating particles are seen in the insulin solution, or if insulin is adhering to the sides of the bottle, do not use the insulin. Short acting insulin should be clear and colorless.

ALLERGIC REACTIONS AND MILD REACTIONS

A small, hard red area at the injection site with itching is indicative of a mild allergic reaction. This usually goes away within a day or so. If a more generalized allergy to insulin occurs, hives and itching will develop over other parts of the body. This disappears by itself, but, if itching persists, the physician should be notified.

Insulin edema may develop at the start of insulin therapy. This swelling occurs in the legs, ankles, feet, hands or face and will usually go away in a few weeks.
Lipoatrophy is used to describe the "pitted" areas that may form at the injection sites. This is a result of loss of fat in the area due to repeated infections with impure insulin.

TYPES OF INSULIN

Insulin is beef, pork, beef and pork, or human. This refers to the species of the insulin, which can be found on the label of the bottle.

Human insulin is genetically engineered from E. coli bacteria Humulin, or from yeast, Novolin. Human insulin accounts for more than 60% of all insulin used today.

Regular insulin (pork, beef, beef/pork, human) is fast acting and lasts a short time in the body. It is used in emergencies for treatment of ketoacidosis. It is used before meals to control the post meal rise in blood sugar, and to lower blood sugar quickly when an immediate reaction is needed.

Semi-Lente insulin (beef, pork) is short acting, but twice as long as Regular insulin. It can be used to control post-meal rises in blood sugar and may be used with Lente insulin.

NPH insulin (beef, pork, beef/pork, or human) contains added protamine for an intermediate-acting effect. NPH insulin provides a basal amount of insulin. Two injections per day are usually ordered.

Ultra-Lente insulin (beef, human) contains a lot of added zinc to give it the longest acting effect. Humulin Ultra-Lente has a shorter peak and duration then beef Ultra-Lente. Ultra-Lente insulin provides the steadiest basal amount of insulin. One injection a day is used in combination with Regular insulin before meals.

Insulin Lispro has the trade name Humalog. This insulin is man-made and similar to naturally occurring human insulin. The onset is rapid and it has a shorter duration of action than human regular insulin. Humalog should be taken within 15 minutes of eating and is used in combination with longer-acting insulin.

70/30 insulin (human) is a mixture of 70% NPH and 30% Regular insulin. Two injections per day are usually ordered.

GENERAL INSULIN PREPARATIONS

TYPE	ONSET	PEAK	DURATION
Regular	30-60 minutes	2-3 hours	4-6 hours
NPH	2-4 hours	4- 10 hours	14-18 hours
Semi-Lente	1-½ hours	6-8 hours	12-14 hours
Lente	3-4 hours	4-12 hours	16-20 hours
Ultra-Lente	6-10 hours	12-18 hours	20-30 hours
Lispro	minutes	30 minutes	1- 5 hours
70/30	15-30 minutes	2-3 hours, 8-12 hrs.	18-24 hours

TABLE: 7

When mixing two different types of insulin in one syringe, Regular insulin is drawn into the syringe first, then the longer acting insulin.

NOVOLIN PEN INJECTOR

The pen-shaped device holds pre-filled cartridges of NPH, regular, or 30/70 insulin. The pen is convenient and easy to use. The needle is placed on one end and the dose is dialed into the pen and then injected. This device is ideal for people on intensive insulin therapy or for those who are away from home at mealtime.

THE INSULIN PUMP

Pump therapy mimics the action of normal beta cells. This pump delivers both basal rate and bolus doses of insulin. The basal rate is preprogrammed into a continuous delivery of 0.1 unit increments of insulin. Bolus doses are programmed before meals and snacks. Unfortunately, insulin doses are based on blood glucose test results, so the patient must test blood glucose levels four to eight times per day. Blood glucose levels are then interpreted and an appropriate insulin dosage is delivered.

Pump wearers must be highly motivated and able to operate the pump. They must be willing to test their glucose frequently, and be able to identify a developing problem. Patients must also have access to a health care team familiar with pump therapy. This device uses computer chip technology, a syringe reservoir, and battery power to deliver insulin automatically to the body. The syringe is filled with buffered Regular

insulin such as Velosulin or Humulin BR, which is connected to a thin plastic tube called an infusion set. A catheter is connected to the infusion set.

The pump is worn 24 hours a day on a belt, in a packet or on a bra. During swimming or bathing, the syringe is removed from the pump or the tubing may be disconnected from the syringe and capped. It is approximately 2 inches by 3 inches by 5/8 inches.

Advantages:

- greater flexibility of meals, exercise and daily schedule
- improved physical and psychological well-being
- increased control of blood glucose levels
- lower glycosylated hemoglobin

Disadvantages:

- risk of infection
- more frequent hypoglycemia
- constant reminder of diabetes

INSULIN REACTIONS

Hypoglycemic symptoms may come on suddenly, and may progress from mild to severe. Prolonged, severe hypoglycemia can result in damage to the brain or heart.

The normal blood sugar range is from 60-120 mg/dl. During an insulin reaction, the blood sugar can fall below 60 mg/dl, or fall rapidly from a high level to a lower level or fall below the patient's usual level. Insulin reactions are due to:

- injection of too much insulin
- skipping meals, eating too little, or waiting too long between meals
- increasing exercise or work

An insulin reaction is the body's response to low blood sugar. When the brain senses low blood sugar, it signals the release of hormones called catocholamines. Catecholamines produce the effects of pallor, sweating, shaking, a pounding heartbeat, nervousness and irritability. Catecholamines also cause the release of stored glucose in the liver (glycogen), which raises the blood sugar.

Symptoms of Mild Hypoglycemia

- hunger
- cold sweats and a clammy feeling
- dizziness, weakness, shakiness
- pounding heart or increased heart rate

Treatment for these symptoms includes giving juice or sugar.

Symptoms of Serious Hypoglycemia

- nervousness or confusion
- headache
- blurred or double vision
- numbness or tingling of lips or fingers

Treatment includes juice or sugar along with possible additional medical assistance.

Symptoms of Severe Hypoglycemia

- paleness and slurred speech
- bizarre behavior, such as hallucinations
- convulsions

If these symptoms occur, the patient is to be given glucose gel if conscious, or glucagon, IVP if unconscious.

ORAL ANTIDIABETIC AGENTS

Oral hypoglycemic agents have a definite place in the treatment of Type 2 diabetes. It is important to understand that none of the oral hypoglycemic agents are insulin. Hypoglycemic agents cannot replace insulin in conditions such as DKA. Oral hypoglycemic agents are used as a supplement to diet and exercise for controlling diabetes. If diabetes cannot be controlled by medication, diet, and exercise, insulin may be needed.

TYPES OF ORAL ANTIDIABETIC AGENTS

Medication that helps put more insulin into the bloodstream:

Sulfonylurea

The use of sulfonylureas has been the main form of oral monotherapy and combination therapy for Type 2 diabetics for more than 40 years. There are now three generations of these agents. The first-generation oral hypoglycemic agents include Chlorpropamide, Tolbutamide, and Tolazamide. The second-generation oral hypoglycemic agents include Glipizide and Glyburide. These drugs have been in use since 1984. The advantages of using these drugs include reduced side effects, 100-200 times greater potency, and a wide range of treatment options. Glimepiride, the third-generation and the newest addition to this group of drugs, was introduced in 1997. In spite of minor differences in side effects, potency and pharmacokinetic properties, all Sulfonylurea agents have the same mechanism of action.

***Mechanism of action*:**
Sulfonylurea is a sulfa-containing compound, which is used to improve insulin's ability to move glucose into the body's cells. This is accomplished by directly stimulating insulin release from pancreatic beta cells. This action is thought to be achieved through antagonism of Sulfonylurea- specific receptors found on beta cells to facilitate insulin release. Treatment with these agents can only be effective in patients with reserve beta cell functional capacity.

Efficacy in therapy
Sulfonylureas demonstrate an estimated 65% to 75% success rate. Patients most inclined to experience success with these agents are identified as:

- over 40 years of age
- within 5 years of diagnosis
- weighing no more than 10% above their ideal weight
- having a FBG less than 200mg/dl

Failure to respond to therapy as defined by unacceptable hyperglycemia may occur due to:

- alteration of drug metabolism
- advancement of beta cell deficiency and insulin resistance
- changes in body weight
- non-compliance
- aging

Limitations of use

Sulfonylureas can be taken once or twice a day and are long acting. Certain drugs such as non-steroidal anti-inflammatory agents may increase the hypoglycemic action of sulfonylureas. Drugs that are highly protein bound such as salicylates, probenecid, coumarins and monoamine oxidase inhibitors also affect the action of sulfonylureas. Good kidney and liver function is a must if taking sulfonylureas.

Sulfonylureas can cause hypoglycemia that can be prolonged and misdiagnosed. Hypoglycemia can be so severe and frequent that it inhibits the maximal glycemic control possible in some patients. This is seen in patients with impaired renal function and the elderly. Therefore, use of sulfonulureas in older patients must be carefully monitored. Less evidence of hypoglycemia in the elderly has been found with the use of Tolazamide, Tolbutamide, and Glipizide, while Glyburide and Chlorpropamide cause a greater tendency toward hypoglycemia. In patients with increased renal dysfunction, Glipizide and Tolbutamide may be more suitable choices. Glimepiride is totally metabolized by the liver and can be utilized in renally impaired patients. Administration with caution to patients with hepatic conditions is recommended, since sulfonylureas are hepatically metabolized.

Weight gain is a common occurrence with the use of sulfonylureas, on average 10 to 15 pounds. Treatment with sulfonylureas must be carefully considered in patients with cardiovascular disease or for those who are obese. In these patients, weight gain may oppose the benefits of the agents, since obesity exacerbates diabetes.

SULFONYLUREAS

ORAL ANTIDIABETIC AGENTS: FIRST GENERATION

MED	ROUTE	ONSET	PEAK	DURATION
Chlorpropamide (Diabinase)	Po 100 mg 250 mg	1 hour	2-4 hours	24 hours
Tolbutamide (Orinase)	Po 250 mg 500 mg	20 minutes	3-4 hours	Sm. amt. detected after 24 hours ½ life 4.5-6.5 hrs.
Tolazamide (Tolinase)	Po 100mg 250 mg 500mg	20 minutes	4-6 hours	10 hours
Acetohexamide (Dymelor)	Po 250 mg	1 hour		12-24 hours

ADVERSE REACTIONS:

CNS: parethesia, fatigue, dizziness, vertigo, malaise, headache
EENT: tinnitus
GI: nausea, heartburn, and epigastric distress
HEMATOLOGIC: leukopenia, thrombocytopenia, aplastic anemia, and agranulocytosis

TABLE: 8

SULFONYLUREAS

ORAL ANTIDIABETIC AGENTS: SECOND GENERATION

MED	ROUTE	ONSET	PEAK	DURATION
Glyburide (DiaBeta, Micronase, Glynase	Po 1.25 mg 2.5 mg 5 mg micronized 1.5mg, 3 mg, 6 mg	1-4 hours	4 hours	24 hours

ADVERSE REACTIONS:

CNS: dizziness, drowsiness, and headache
EENT: changes in accommodation or blurred vision
GI: nausea, constipation, diarrhea, heartburn, and epigastric fullness
Hematologic: leukopenia, hemolytic anemia, agranulocytosis, thrombocytopenia, and aplastic anemia
Hepatic: abnormal liver function, jaundice
Skin: rash, pruritus
Other: hypoglycemia, arthralgia, myalgia, and angioedema

TABLE: 9

SULFONYLUREAS

ORAL ANTIDIABETIC AGENTS: THIRD GENERATION

MED	ROUTE	ONSET	PEAK	DURATION
Glimepiride (Amaryl)	1mg 2mg 3mg	Unknown	2-3 hours	> 24 hours

ADVERSE REACTIONS:

CNS: dizziness, asthenia, and headache
GI: nausea
Hematologic: leukopenia, hemolytic anemia, agranulocytosis, aplastic anemia, thrombocytopenia
EENT: changes in accommodation
Skin: allergic reaction, urticaria, and erythemia
Hepatic: cholestatic jaundice
Other: hypoglycemia, dilutional hypotenatremia

TABLE: 10

Repaglinide (Prandin)

Mechanism of action:
Prandin was released in April of 1998. This agent is chemically distinct from the sulfonylureas, but its mechanism of action is similar. Prandin controls serum glucose by binding to high-affinity receptors found on the pancreatic beta cell to stimulate insulin release. It does this in response to serum glucose levels. The insulinotropic action of Prandin diminishes with lower glucose concentrations. This drug is rapidly absorbed from the intestines, achieves peak serum levels in approximately one hour and is quickly eliminated.

Efficacy in therapy:
Prandin used in combination with other oral agents has been shown to improve glycemic control. This is especially true when added to Metformin therapy. When monotherapy in patients taking Metformin or Prandin fails, addition of the other agent provides a good therapeutic option.

Limitations in use:
Side effects common to this drug are similar to those of sulfonylureas, including hypoglycemia, URI, headache, rhinitis, nausea, diarrhea, and arthralgia.

Patients on Prandin therapy who miss a meal in a day do not proceed to experience hypoglycemia. It is also important to note that hypoglycemic events are no more prevalent in the elderly than in youths. Prandin is most useful when taken before meals and may be administered up to 30 minutes before each meal. Doses of 0.5 to 4 mgs may be given two to four times a day depending on the meal schedule.

Patients with hepatic insufficiency should be watched carefully since a decrease in metabolism could cause elevated blood levels of Prandin and hypoglycemia. Patients that are debilitated, malnourished, or those with adrenal or pituitary insufficiency should be watched carefully since they are more susceptible to the hypoglycemic effect of glucose-lowering drugs.

Medication that decreases the production of glucose by the liver:

Biguanide
Biguanides increase the ability of tissues to take up glucose and reduce the amount of glucose released by the liver. In 1977, a biguanide phenformin was taken off the market in the United States due to fatal cases of lactic acidosis. In 1995 Metformin, a safer biguanide, was introduced, and has demonstrated adequate glycemic control in regulating Type 2 diabetics with much less incidence of lactic acidosis. This drug offers a therapeutic option for diabetic management which includes management of both glucose and lipid metabolism.

Metformin (Glucophage)

Mechanism of action; Metformin (Glucophage)
Metformin is used in managing hyperglycemia in Type 2 patients who cannot control blood glucose with diet alone. The drug has been found to reduce fasting and postprandial blood glucose levels. It also enhances insulin sensitivity at postreceptor levels and stimulates insulin-mediated glucose disposal, but does not stimulate insulin secretion. By indirectly improving glycemic control, Glucophage may lower triglyceride and cholesterol levels. Administer this drug with meals: once-daily dosage should be given with breakfast and twice daily dosage should be given with breakfast and dinner.

Glucophage, unlike the sulfonylureas, does not cause weight gain. In fact, glucophage may cause weight loss since it does not stimulate insulin secretion, or because the drug can cause anorexia in 20% to 30% of patients. Hypoglycemia is not a factor in the administration of Glucophage since it diverts glucose in the intestinal wall to undergo an anaerobic enzymatic conversion of glucose to the simpler compound lactate. This process is called glycolysis, resulting in energy stored in the muscle in the form of ATP or adenosine triphosphate. Lactate undergoes gluconeogenesis in the liver to regenerate glucose. This increase in lactate production can cause lactic acidosis, although rare (one case per 100,000 patients).

Efficacy in therapy:
Glucophage is administered two or three times daily. It is rapidly absorbed and cleared through the kidneys. The number of patients on the drug responding to therapy decreases over the years due to factors that include:

- Drug noncompliance because of side effects
- Dietary noncompliance
- Declining beta-cell function
- Increase use of drugs including glucocorticoids, thiazides, and beta-blockers

Limitations of use:
Side effects usually occur the first few weeks of therapy and include diarrhea, nausea, and a metallic taste in the mouth. Taking the medication with meals will help reduce side effects.

Glucophage is contraindicated in patients with liver and renal dysfunction or conditions that compromise renal function such as acute myocardial infarction, cardiovascular collapse, septicemia, severe trauma or surgery.

Lactic Acidosis:

Lactic acidosis has a mortality rate greater than 30%. Patients are advised that symptoms include malaise, rapid breathing, shortness of breath, and severe weakness. If laboratory tests indicate lactic acidosis, glucophage should be immediately discontinued and supportive care given. Lactic acidosis can be precipitated by drug accumulation due to renal failure, and is contraindicated in patients who exhibit a serum creatinine greater than 1.4 or 1.5 mg/dl. Lactic acidosis can also be precipitated by lactate overproduction by hypoxic tissues in respiratory and circulatory failure, and impaired lactate removal due to liver damage, for example, which inhibits gluconeogenesis. Glucophage is contraindicated for patients suffering from liver dysfunction as indicated by elevated serum transaminase levels and patients who chronically use alcohol. These contraindications impose considerable restrictions in the use of glucophage for diabetic treatment.

Medication that interferes with the absorption of glucose:

Alpha-glucosidase inhibitors:

Acarbose is a mild anti-hyperglycemic agent. It was approved for use in the United States in 1995 as a means to control postprandial hyperglycemia. Acarbose has an important role in the treatment of glucose levels over 200mg/dl, which correlates directly to an increased risk for developing microvascular complications. Acarbose therapy may delay the onset of retinopathy, nephropathy, and neuropathy. It serves as adjunct therapy in severe cases of DM, but may be used in mild and early Type 2 diabetes.

Mechanisms of action: Acarbose (Precose)
Acarbose works in the intestines, slowing down the digestion of carbohydrates, and lengthening the time it takes for carbohydrates to convert to glucose. Decreasing the rate of carbohydrate degradation and absorption through enzyme inhibition in the small intestines, Acarbose lowers peak glucose levels after meals and controls postprandial glucose levels, thereby facilitating better blood glucose control. The amount of carbohydrates ingested is not altered, so caloric reduction of meals and weight loss is not affected.

Efficacy in therapy
Acarbose offers no hypoglycemic effect if used as the only diabetic agent. Most people find it easier to lose weight with this medication. It can be used alone as the first drug therapy of diabetes, or in combination with any other diabetes pill, and with insulin. It may also help control blood glucose when used with insulin in Type 1 diabetes.

Limitations of use:
Acarbose mainly affects the postprandial blood sugar. Doses of Acarbose are administered to patients prior to meals. Gastrointestinal problems are the most common adverse effects: flatulence, diarrhea, abdominal distention and pain, resulting from fermentation of undigested carbohydrates in the colon, usually subsides within weeks of therapy after the intestines adapt. Adverse effects are dose-related and can be avoided with low initial dosing and slow titration. Initially the 25mg recommended daily dose is taken before the first bite of the largest meal of the day. The dose is then gradually titrated up to as much as 100 mg three times a day over a period of 6 to 8 weeks. Acarbose is contraindicated in patients with active obstructive or inflammatory bowel disorders and ulceration of the colon. The drug is not recommended for people with limited kidney function since Acarbose metabolites can be found in the urine.

Probably the most limiting aspect of this agent is that it is a relatively mild medication that may not be able to maintain adequate glycemic control in patients with more severe hyperglycemia. Using Acarbose in combination with other agents may be more beneficial, especially for controlling postprandial hyperglycemia.

Acarbose itself does not cause hypoglycemia. However, when given with insulin and sulfonylureas, blood sugar levels may drop low enough to cause life-threatening hypoglycemia. Acarbose can increase the hypoglycemic potential of these agents. Since Acarbose prevents the breakdown of sucrose or table sugar, patients should have a source of glucose available to treat symptoms if Acarbose is used in combination with these agents.

Medication that reduces the body's resistance to insulin:

Thiazolindinedione: TZDs

Avandia and Actos belong to a relatively new family of drugs called thiazolidinediones. These drugs make the body more sensitive to insulin or reduce the resistance to insulin. The first agent in this class that was released was troglitazone (Rezulin). This agent was taken off the market shortly after its release because of its tendency to cause liver damage, which led to several fatalities.

Mechanism of action: Rosiglitazone (Avandia) and Pioglitazone Actos)
Avandia and Actos are oral antidiabetic agents that act primarily by improving sensitivity to insulin in muscle and adipose tissue and inhibiting hepatic gluconeogenesis. Actos and

Avandia are potent and highly selective agonists for perocisome proliferator-activated receptor-gamma (PPAR), described as microbodies present in vertebrate animal cells, which are rich in the enzymes peroxidase. In humans PPAR nuclear receptors are found in key target tissues such as adipose tissue, skeletal muscle, and the liver. Activation of PPAR receptors regulates the transcription of insulin responsive genes involved in the control of glucose production, transport, and utilization. PPAR-responsive genes also help in the regulation of fatty acid metabolism.

Efficacy in therapy:
Avandia and Actos are indicated as an adjunct to diet and exercise to improve glycemic control in patients with Type 2 diabetes. Caloric restriction, weight loss, and exercise are essential for the proper treatment of the diabetic patient because they help improve insulin sensitivity. This helps maintain the efficacy of drug therapy. These drugs are indicated for monotherapy and combination therapy with Sulfonylurea, Metformin, or insulin in the case of Actos, when diet and exercise plus a single agent does not produce adequate glycemic control.

Avandia can be given as a single dose of 4 mg to 8 mg per day, or in divided doses. Actos is given once a day at doses ranging from 15 mg to 60 mg per day.

Limitations of use:
It is important to adhere to dietary instructions and to have blood glucose and glycosylated hemoglobin tested regularly. Patients should be informed that blood tests for liver function precede the start of therapy, and continue every two months for the first year, and periodically thereafter.

Immediate medical advice should be sought for unexplained nausea, vomiting, abdominal pain, fatigue, anorexia, or dark urine. Medical advice is also needed during periods of stress such as fever, trauma, infection, or surgery.

The risks of hypoglycemia and conditions that predispose to its development are always present when using combination therapy with insulin or oral antidiabetic agents.

Therapy with Avandia and Actos may result in ovulation in some premenopausal anovulatory women. These patients may be at risk for pregnancy while taking thiazolidinediones. Therefore, adequate contraception in premenopausal women should be recommended.

Adverse reactions reported consist of upper respiratory tract infections, headache, sinusitis, back pain, hyperglycemia, fatigue, and myalgia.

OTHER ORAL ANTIDIABETIC AGENTS

MED	ROUTE	ONSET	PEAK	DURATION
Repaglinide	Po 0.5mg 1 mg 2 mg	Unknown	1 hour	Unknown
Metformin (Glucophage)	Po 500 mg 850 mg	Unknown	Unknown	Unknown
Acarbose (Precose)	Po 50 mg 100 mg	Unknown	1 hour	2-4 hours
Rosiglitazone (Avandia)	Po 4 mg 8 mg	Rapid	2-3 hours	Unknown
Pioglitazone (Actos)	Po 15 mg 30 mg 45 mg	30 minutes	2 hours with food 3-4 hours	Unknown

TABLE: 11

SULFONYLUREA MONOTHERAPY

All oral antidiabetic agents can be used as monotherapy. Sulfonylureas are usually the first choice for Type 2 patients with reserve beta-cell secretory function. Acarbose, Metformin, Repaglinide, and thiazolidinediones may all be effective options for monotherapy. Determination of selection among these agents and the sulfonylureas depends on the evaluation of risks and benefits of each agent as they apply to each patient. It is necessary to be familiar with side effects, contraindications, mechanism of action, and efficacy of each agent in order to select the proper agent. Compliance is an important factor when selecting an appropriate drug regimen and will encourage adherence to drug therapy that will contribute to positive patient outcome.

Diabetic patients that are more than 40 years of age and have a progressive diabetic history, yet are less than 5 years since onset, seem to respond best to the sulfonylureas. Patients with normal to moderate body weight have a good response to these agents. Because obese patients have been associated with insulin resistance, and because weight gain is a side effect associated with sulfonylureas, these agents would not be the optimal choice for overweight patients, since persistent obesity can facilitate disease progression along with its associated complications. In patients who are normal weight or lean, Sulfonylurea may be a better choice, since these patients are associated with greater insulin deficiency.

An indicator of effective pharmacological management would be maintaining the FPG (fasting plasma glucose) at less than 140 mg/dl. Sulfonylurea therapy is started low and gradually titrated up slowly to the target range of FPG levels. It should be remembered that drug efficacy is increased with a proper diet and exercise program.

METFORMIN MONOTHERAPY

Metformin's primary effect is lowering fasting blood sugar without affecting insulin levels. About 80% of Type 2 diabetics have a significant response to metformin monotherapy, but only about a quarter of these patients demonstrate an adequate FPG of 140 mg/dl. Patients that respond positively with improved glycemic control usually maintain the effects for more than 5 years, which is longer than the efficacy demonstrated by Sulfonylurea therapy.

Metformin has been shown to improve serum lipid levels and promote weight loss. In obese patients, initiation of this agent may be preferred to the Sulfonylurea agents since one of their side effects is weight gain.

SULFONYLUREA-METFORMIN COMBINATION THERAPY

There are times when Sulfonylurea therapy improves glycemic control, but is unable to achieve optimum glycemic control, despite maximization of dose. Under these circumstances a second oral agent may be added. Supplementing sulfonylurea with metformin has an additive effect on glycemic control when sulfonylurea by itself is not adequate. Metformin reduces the hepatic glucose output and increases the sensitivity to insulin. Sulfonylurea-metformin combination is one of the most potent oral therapies available to diabetic patients. A trial of this combination therapy is recommended before advancing to insulin therapies.

In approximately half of patients that fail sulfonylurea-monothrapy, the addition of metformin may achieve glycemic control and offer dosing flexibility that may result in the avoidance of adverse drug effects.

ACARBOSE MONOTHERAPY AND COMBINATION

Acarbose is taken with meals to help control postprandial hyperglycemia. In mild conditions it may be used as monotherapy and in combination with another oral agent when either agent has not achieved glycemic control with monotherapy. At present, Acarbose has been approved only for use in combination with sulfonylureas.

DRUG COMBINATIONS

The goal of combination therapy is to maximize glucose-lowering effects by using drugs that lower blood glucose by different mechanisms. The increased number of oral medications available for the treatment of Type 2 diabetes allows for a larger number of combination therapies. The combinations that have been proven to provide the most significant glycemic control are:

- Metformin and Rosiglitazone
- Metformin and Pioglitazone
- Rosiglitazone and Sulfonylurea
- Pioglitazone and Sulfonylurea
- Pioglitazone and insulin

INSULIN AND ORAL THERAPY

Over time, patients on oral regimens, whether oral monotherapy or in combination, can fail to maintain adequate control of hyperglycemia requiring the use of insulin. Many patients become therapeutically unresponsive to oral agents due to progression of beta-cell secretory failure and peripheral insulin resistance, prolonged glucose toxicity, inadequate diet and exercise, non-compliance, and weight gain. This is especially true of Sulfonylurea agents that have a high incidence of secondary failure. Insulin therapy is initiated in Type 2 diabetics when newly diagnosed patients exhibit severe hyperglycemia (>300 mg/dl) and postprandial plasma glucose is >350 mg/dl, progression of the underlying cause results in uncontrolled hyperglycemia, or when primary or secondary failure occurs with oral agents. At this point in therapy, a decision must be made to discontinue the oral agents and begin insulin monotherapy or to begin a combination of oral and insulin therapy.

Most diabetic patients will exhibit a reduction of plasma glucose with an adequate provision of exogenous insulin. Although insulin can provide effective glycemic control, it is usually not the treatment of choice in Type 2 diabetes. Rather, it is the last resort since it requires an invasive route of administration, multiple doses throughout a day, and imposes a significant burden on the patient's life, while causing considerable weight gain. It is also relatively expensive and is associated with a 33% rate of hypoglycemia in patients treated with insulin. In most cases the clinical use of insulin should be minimized when possible. With the introduction of several new oral antidiabetic agents, there are now more therapy options to replace insulin or oral-insulin therapy.

When insulin monotherapy at doses greater than 70 units/day has failed, or when oral combination therapy has failed, insulin-oral combination therapy should be initiated. With the availability of agents that are able to decrease insulin resistance in Type 2 diabetes, attempting to improve insulin resistance by introducing an insulin-sensitizing

agent such as metformin or by using the added stimulatory effect of sulfonylureas combinations may permit the use of lower doses of insulin. This helps in the avoidance of complicated regimens, adverse weight gain, the development of cardiovascular disease, and provides control of serum glucose by insulin, while decreasing hypoglycemic episodes.

Use of insulin combinations has largely been with sulfonylureas, since they have been in existence longer than the other oral agents. Insulin-sulfonylurea therapy has been shown to reduce fasting, postprandial, and glycosylated hemoglobin, while decreasing considerably the adverse reactions associated with higher doses of insulin. A regimen that utilizes evening intermediate-acting insulin, which decreases hepatic glucose production and controls fasting blood glucose, and a daytime sulfonlurea that promotes insulin secretion to cover postprandial requirements throughout the day, seems to work best. This regimen is called BIDS and can be started with a low dose of NPH insulin at bedtime, along with maximal doses of a sulfonylurea agent. Patients must be aware that the dose of insulin should be taken right before bedtime and not given with the evening meal.

CHAPTER V

THE DIABETIC DIET

DIET IN THE TREATMENT OF DIABETES

The Diabetic Diet

In the spring of 1994, the American Diabetic Association issued new guidelines in nutrition for people with diabetes. The guidelines state that people with diabetes may substitute sugar-containing food for other carbohydrates as part of a balanced meal plan. This means that the eating guidelines for people with diabetes can now be more varied.

The beliefs up to 1994 were that people with diabetes should avoid foods that contain "simple" sugars and replace them with "complex" carbohydrates such as those found in potatoes.

The May 1994 issue of Diabetes Care supports the theory that simple sugars are more rapidly digested and absorbed than starches, causing high blood sugar levels. Because of this theory, the emphasis over the past four years has evolved so that many patients are being taught to focus on how many total grams of carbohydrate they can eat throughout the day at meals and snacks, and still keep their blood sugars under control.

This means that the patient who has worked with a dietitian and their diabetic treatment team to figure out how many grams of carbohydrate they may eat throughout the day can decide at any given meal what they will eat. Diabetics that are not on insulin need to focus on keeping the amount of carbohydrate they eat consistent throughout the day. Those on insulin can decide both what and how much to eat at a given meal as long as it does not exceed their daily allotment. The patient needs to learn how to spend their grams of carbohydrates over the course of the day.

Patients are able to use regular home blood sugar monitoring to keep track of the effects of their meals and activity levels on their blood sugars. By working with their health care team to make adjustments in their food intake, exercise program and medication, patients are able to keep their blood sugars as close to normal as possible.

Before the development of exogenous insulin, diabetes was treated strictly by diet. Diet is an essential factor in the overall control of the diabetic program. Developing a personal meal plan to help the patient attain appropriate glucose, cholesterol, and triglyceride levels is a vital goal for the diabetic educator, dietician, and doctor.

A well-developed diet for Type 2 diabetes helps in the achievement and maintenance of correct weight and produces a balance between the foods eaten and the insulin produced by the body. Type 1 diabetics must follow a comprehensive meal plan to ensure a balance between injected insulin and the foods eaten.

The diabetic diet is a well-balanced meal plan designed to meet the individual needs, tastes, and activity level of the individual patient. The better the diet is understood, the more flexibility the patient may enjoy.

Patients with Type1 diabetes should have a diet that has approximately 16 calories per pound of body weight per day. Patients with Type 2 diabetes usually are put on a 1500-1800 calorie diet per day to promote and /or maintain the ideal body weight. This may vary however, depending on the person's age, sex, activity level, current weight and body style. Obese individuals may need more calories at first until they are able to lose weight since it takes more calories to maintain a larger body and 1600 calories may cause too rapid weight loss. Men generally have more muscle mass and may require more calories, since muscle burns more calories per hour than fat. People whose activity level is low will have a decrease in daily caloric needs.

What is a calorie?

A calorie is a unit of heat used to define the energy-producing content of a specific food. Calculation of a patient's daily caloric intake depends on many factors including age, height, weight, activity level, metabolism, growth needs and life style. If the body takes in more calories than is needed to produce energy, the excess calories are stored as body fat. Losing weight is achieved by consuming fewer calories than the body needs for the usual activity level and by increasing exercise.

Exchange System

The exchange system provides a quick way to estimate energy, carbohydrates, protein and fat content in any food or meal. The ADA has devised an exchange food group system that classifies food into six exchange lists. These exchange lists include starch, meat, vegetables, fruit, milk, and fat. Food from each exchange is defined so that one serving of each food contains approximately the same amount of carbohydrate, protein, fat and energy or calories. Serving sizes vary for different choices on each exchange list.

Many vegetables are considered "free" foods, including celery, lettuce, spinach, cabbage, cucumbers and zucchini. Free foods are foods and drink that have less than 20 calories per serving and less than 6 grams of carbohydrate per serving. When eaten in moderation, 1-2 servings per meal, free foods do not need to count in calculations. If one consumes 8 servings of a free food (160 calories), it is equivalent to one English muffin.

Free foods have high water content. When they are cooked they lose the water and become more concentrated. Water loss occurs as heat breaks down the membranes of the cells that compose the food product. As the membranes break, the content of the cell, including water, leaves, forming a more concentrated food item. Therefore, cooked free vegetables have smaller portion sizes.

The following chart shows the amounts of carbohydrates, proteins, fat and calories in one serving from each exchange list.

EXCHANGE LISTS

	Carbohydrate (grams)	**Protein (grams)**	**Fat (grams)**	**Calories**
Starch/Bread	15	3	Trace	80
Meat				
lean		7	3	55
medium fat		7	5	75
high fat		7	8	100
Vegetable	5	2		25
Fruit	15			60
Milk				
skim	12	8	Trace	90
low fat	12	8	5	120
whole	12	8	8	150
Fat			5	45

TABLE 12:

SAMPLE 1500-CALORIE ADA DIET

BREAKFAST	1 Fruit 2 Starch/Bread 1 Fat 1 Milk * Free Foods
LUNCH	1 Meat 2 Starch/Bread 1 Vegetable 1 Fruit 1 Fat *Free Foods
AFTERNOON SNACK	1 FRUIT
DINNER	2 Meat 2 Starch/Bread 1 Vegetable 1 Fruit 2 Fat * Free Food
EVENING SNACK	1 Starch/Bread 1 Milk 1 Fruit

TABLE 13:

SAMPLE OF EXCHANGE LISTS

STARCH / BREAD LIST

The general rule when selecting a starch food is:

- ½ cup of cereal, grain, or pasta is one serving
- 1 ounce of a bread product is one serving

CEREALS/GRAINS/PASTA

Bran cereals, flaked	½ cup
Cooked cereals	½ cup
Grapenuts	3 tbsp
Pasta, cooked	½ cup
Rice (white or brown), cooked	½ cup
Unsweetened cereals	¾ cup
Table 14:	

DRIED BEANS/PEAS/LENTILS

Lentils, cooked	1/3 cup
Baked beans	¼ cup
Beans and peas, cooked (kidney, white, split)	1/3 cup
Table 14:	

STARCHY VEGETABLES

Corn	½ cup
Corn on cob, 6”	1
Lima beans	½ cup
Potato, baked 1 small (3 oz)	1 small
Potato, mashed	½ cup
Squash, winter (acorn, butternut)	¾ cup
Table 14:	

BREAD

Bagel	½ (1 oz.)
Croutons, low fat	1 cup
English muffins	½
Pita, 6”	½
Raisin bread, unfrosted	1 slice (1oz.)
White bread	1 slice (1 oz.)
Whole wheat bread	1 slice (1 oz.)
Table 14:	

MEAT LISTS

Each item is considered one exchange, which is usually 1 ounce of meat containing 3 to 8 grams of fat and from 55 to 100 calories.

LEAN AND MEDIUM-FAT MEAT AND MEAT SUBSTITUTES

Beef, most beef products: ground beef, roast (rib, chuck, rump), steaks (cubed, Porterhouse, T-bone), meat loaf, (medium fat) tenderloin, chipped beef, (lean fat)	1 oz.
Pork, most pork products: chops loin roast, cutlets, Canadian bacon, lean ham	1 oz.
Poultry, chicken, turkey, Cornish hen (without skin = lean) chicken with skin, duck or goose (well-drained of fat) ground turkey (medium fat)	1 oz.
Lamb: most lamb products: chops, leg, roast (medium fat)	1 oz.
LEAN FISH Fish, all fresh and frozen fish Crab, lobster, scallops, shrimp, clams (fresh or canned in water) Oysters Tuna (canned in water) MEDIUM FISH Tuna (canned in oil and drained), salmon (canned)	 1 oz 2 oz. 6 medium ¼ cup ¼ cup
LEAN CHEESE Cheese, any cottage cheese Grated parmesan Diet cheeses (less that 55 calories per oz. MEDIUM CHEESE Skim or part –skim mild cheeses: Ricotta Mozzarella Diet cheeses (56-80 calories per oz.)	 ½ cup 2 tbsp 1 oz. ¼ cup 1 oz. 1 oz.
Table 15:	

VEGETABLE LIST

In general, the serving size is ½ cup for cooked vegetables and vegetable juices or 1 cup for raw vegetables. One exchange is considered one serving size and contains about 25 calories and 2-3 grams of dietary fiber.

VEGETABLES

Artichoke Asparagus Beans (green, wax, Italian) Bean sprouts Beets Broccoli Cabbage Greens (collard, mustard, turnip) Mushrooms (cooked)	Onions Peppers Sauerkraut Spinach Summer squash (crookneck) Tomato (large) Water chestnuts Zucchini
Table 16:	

FRUIT

Each fruit is considered one exchange and contains about 60 calories. Fresh, frozen, and dried fruits contain about 2 grams of fiber per serving.

FRUIT

Apple (raw, 2" diameter)	1 apple
Applesauce (unsweetened)	½ cup
Banana (9" long)	½ banana
Blueberries	¾ cup
Cantaloupe	1/3 melon
Cherries (large, raw)	12 cherries
Cherries (canned)	½ cup
Grapefruit (medium)	½ grapefruit
Grapes (small)	15 grapes
Orange (2.5" diameter)	1 orange
Peach (2.75" diameter	1 peach or ¾ cup
Pear	2 halves or ½ cup
Strawberries (raw, whole)	1 ¼ cup
Tangerine (2.5" diameter)	2 tangerines
Watermelon (cubes)	1 ¼ cup
Table 17:	

FRUIT JUICE

Apple juice/cider	½ cup
Cranberry juice cocktail	1/3 cup
Grapefruit juice	½ cup
Grape juice	1/3 cup
Orange juice	½ cup
Pineapple juice	½ cup
Prune juice	1/3 cup
Table 17:	

MILK LIST

Each item of milk is one exchange and contains trace amounts to 8 grams of fat and from 90 to 150 calories.

SKIM AND LOW-FAT MILK

Skim milk, ½%, 1%, low-fat buttermilk	1 cup
Evaporated skim milk	½ cup
Dry non-fat milk	1/3 cup
Plain non-fat yogurt	8 oz.
Table 18:	

LOW-FAT MILK

2% Milk	1 cup
Plain low-fat yogurt	8 oz.
Table 18:	

WHOLE MILK

Whole milk	1 cup
Evaporated whole milk	½ cup
Whole plain yogurt	8 oz.
Table18:	

FAT LIST

Each item in the fat exchange is considered 1 exchange and contains about 45 calories. All fats are high in calories and must be measured carefully.

UNSATURATED FATES

Avocado	1/8 medium
Margarine	1 tsp.
Mayonnaise	1 tsp.
Mayonnaise, reduced calorie	1 tbsp.
Nuts and seeds:	
Almonds (dry roasted)	6 whole
Cashews (dry roasted)	1 tbsp.
Pecans	2 whole
Peanuts	20 small or 10 large
Walnuts	1 tbsp.
Pumpkin seeds	1 tsp.
Sunflower seeds	2 tbsp.
Oil (corn, safflower, soybean, sunflower, olive, peanut)	1 tsp.
Olives	10 small or 5 large
Salad dressing, mayonnaise-type	2 tsp.
Salad dressing, mayonnaise-type, reduced-calorie	1 tbsp.
	1 tbsp.
Salad dressing (all varieties)	1 tbsp.
Salad dressing, reduced-calorie	2 tbsp.
Table 19:	

SATURATED FATS

Butter	1 tsp.
Bacon	1 slice
Coconut (shredded)	2 tbsp.
Coffee whitener, liquid	2 tbsp.
Coffee whitener, powder	2 tbsp.
Cream	2 tbsp.
Cream, sour	2 tbsp.
Cream (heavy, whipping)	1 tbsp.
Cream cheese	1 tbsp.
Salt pork	¼ oz.
Table 19:	

UNDERSTANDING FOOD GROUPS

Although foods contain many nutrients, the three basic food groups are fats, proteins and carbohydrates. It is important to have all three-food groups in a diet to have good nutrition.

Carbohydrates

It is important to count carbohydrates since carbohydrates intake leads directly to increased blood glucose levels. Although foods containing carbohydrates have the most impact on blood sugars, the calories from all foods will affect blood sugar.

Most of the carbohydrates we consume belong to three main groups: starch, fruit and milk. Vegetables are also source of some carbohydrates, while proteins and fats have very little. Sugars may be naturally present as in fruits, or may be added. When digested, carbohydrates provide fuel for energy.

To make counting carbohydrates easy, one serving of starch, fruit, or milk contains 15 grams of carbohydrate or one carbohydrate serving, while three servings of vegetable contain 15 grams of carbohydrates.

The following is an example of carbohydrates calculated for an 1800-calorie diet. Each gram of carbohydrate provides 4 calories. A diabetic on an 1800-calorie diet should get 50% of these calories from carbohydrate. A total of 900 calories, or 225 Gms of carbohydrate (at 4 calories per gram) should be consumed over an entire day. At 15 grams per exchange, this would equal 15 exchanges of carbohydrates per day.

Proteins

Protein foods are meat, poultry, fish, eggs, cheese, dried beans and legumes. When digested, these foods are used to build and repair the body, but may also be used as fuel. According to the American Diabetic Association, approximately 20 % of the total number of calories should come from protein.

Fats

Fats are found in butter, margarine, cooking oils, cream, nuts and many other foods. When digested, fats are stored as fat cells and later used as fuel for energy. No more than 30% of calories should come from fat.

A pound of fat is equal to 3,500 calories. In order to lose one pound a week, caloric intake must be decreased by 500 calories a day times seven days to total 3,500 calories.

Types of Fats

- Triglycerides are neutral fats synthesized from carbohydrates found in the blood that may increase the risk of heart disease.

- Saturated fat is one that raises total blood cholesterol. It is found in hydrogenated vegetable fats, coconut and palm oils, meat fat, whole milk, butter, ice cream and fatty cheese.

- Polyunsaturated fat is a vegetable fat that lowers total blood cholesterol. It is liquid at room temperature found in cottonseed, soybean, sunflower and safflower oils.

- Monounsaturated fat is a type of unsaturated fat that lowers blood cholesterol. It is found in olive oil and peanut oil and is liquid at room temperature. Monounsaturated fatty acid is the most common of all food and tissue acids.

- Low-density lipoprotein (LDL) is a harmful type of cholesterol that deposits on artery walls and increases the risk of heart disease.

- High–density lipoprotein (HDL) is a type of cholesterol that may protect against heart disease.

- Cholesterol is a fatty substance found in animal foods such as meat, poultry, egg yolks, and high fat dairy products. Cholesterol levels should be less than 200 mg./dl.

Fiber

Another important dietary consideration in a healthy diet is the amount of fiber consumed. Fiber, also known as roughage, is the part of the plant food that the body cannot digest. Increased amounts of fiber lower the concentration of plasma glucose. Glycemic control may be improved by slowing the absorption of carbohydrates, thereby reducing carbohydrate induced elevations of the blood sugar.

Alcohol

Alcohol should be used only with meals or snacks and in moderation. Alcohol can cause problems with control of diabetes. It can lower blood sugar levels by blocking the release of glycogen or stored glucose, leading to a severe insulin reaction.

Oral diabetes medications when combined alcohol can cause dizziness, flushing, and nausea.

Alcohol should be avoided especially if the disease is not under control, or if weight reduction is an issue. Alcohol has the calories without the vitamins, minerals and other

nutrients that are essential for maintaining good health. Alcohol is equal to 7 calories per gram and stimulates the appetite. The body digests alcohol as a fat; therefore one fat exchange should be removed from the diet for every 45 calories in an alcoholic beverage.

POINTS TO REMEMBER

Eat a variety of foods.

By varying the foods eaten, there is a better chance of getting all the vitamins and minerals needed for a healthy diet. This means eating from all food groups every day and choosing different foods from each group from day to day.

For the best blood sugar control, patients need to keep the amounts eaten from the bread/starch, the fruit group, and the milk group about the same from day to day. These are the high carbohydrate foods that have the most effect on blood sugar. They are high in nutrition and important to eat, but keeping amounts approximately equal from day to day will make diabetes control easier.

Maintain a healthy weight.

Staying at a healthy weight is important for all people with diabetes. Extra body fat makes it harder for people with Type 2 diabetes to make and use their own insulin. For these people, losing some extra pounds can be a powerful tool for diabetes control, especially in the first few years after their diabetes is diagnosed. People with Type 1 diabetes may have trouble keeping their weight high enough if their blood sugars are too high.

The right nutrition plan includes:

- Eating the right foods
- Eating the right amount of foods
- Balancing food intake with exercise and diabetes medicine

Doing theses things will help protect the patient's health, control the diabetes, and assist in maintaining healthy weight.

Choose a diet low in fat, saturated fat, and cholesterol.

Pure fats like oil, margarine, and butter are to be used sparingly. They give very few vitamins and minerals. For best health, they should be only a small part of nutrition. Fats that are solid at room temperature indicate high fat saturation, which affects the cardio-vascular system.

- Foods that are naturally low in fat can be made into high fat foods by the way they are cooked or served. (For example, potatoes are fat-free but French fries are high fat.)
- Many choices from the milk and meat sections (like whole milk, cheese, and spare ribs) are naturally high in animal fat.
- Choose low-fat protein foods like chicken, turkey, and lean meat instead of high-fat ones, like cold cuts, sausage, lamb patties, ground pork, or spareribs.
- Non-stick cooking spray should be used instead of butter, oil, or shortening to "grease" a pan before cooking.
- Limit high–fat add-ons like butter, margarine, oil, shortening, salad dressing, and gravy.
- Eat less fried food. Baked, broiled, steamed, grilled or poached foods are better.
- Choose 1% low fat and non-fat dairy products.
- Eat higher-fat cuts of red meat no more than three times a week.
- Loin or sirloin cuts tend to be leaner than other cuts. Cut all visible fat from meat and poultry before cooking. Keep serving sizes of meat small (about the size of the palm of the hand).
- Season food with low-fat flavorings and with spices. Try lemon or lime juice, flavored vinegars, low-calorie salad dressings, low-fat yogurt, or a small amount of wine instead of butter, margarine, sour cream, or other high-fat choices.
- In order to estimate how much fat to eat each day, divide present or goal weight by three and this yields the recommended number of grams per day. This will be about 25%-30% of the total energy needs of the average individual.

Choose a diet with plenty of vegetables, fruits, and grain products.

By choosing the high-carbohydrate whole grain foods, the patient can increase the intake of important vitamins and minerals as well as fiber. Certain fiber may help lower blood fats, promote regular bowel movements, and slow the absorption of sugars after a meal. To get the most fiber in your diet:

- Eat fresh fruits and vegetables
- Eat the skins and peelings, if edible.
- Choose whole grain breads and cereals.
- Eat at least one meatless main dish that includes dried beans, peas, or lentils each week.

Use sugar in moderation.

High sugar foods, like desserts and regular soft drinks give very little nutritional value. For best health, these foods should be only a small part of the overall food intake.

- Small amounts of high-sugar foods can be part of a healthy diet, even for people with diabetes. Eating sugary foods should be considered carefully if weight or blood sugars are out of control. When they are eaten, they should be a part of the dietary plan.
- Artificial non-caloric sweeteners in diet soft drinks, recipes, prepared puddings, gelatins and drink mixes provide diabetics with a sweet taste, but are less likely to contribute to weight or blood pressure problems.

Use salt and sodium in moderation.

Most Americans eat more salt than they really need. For some people, extra salt adds to their risk for high blood pressure. High blood pressure is more common in people with diabetes. Uncontrolled high blood pressure greatly increases the risk for many health problems related to diabetes. The following are some suggestions to help control salt intake.

- Use little or no salt at the table or in cooking.
- Avoid foods canned, boxed, or frozen with extra salt.
- Use herbs, spices, and salt-free seasonings mixes instead of salt.
- Use black, white, or red pepper to season food.
- Remember to choose food "close to nature." Less processed foods have less salt.

MEAL PLAN CHART

It is often helpful to use a chart like this one to help make a meal plan that is nutritious and good for taking care of the diabetic patient.

Food Groups	Breakfast	Snack	Lunch	Snack	Dinner	Snack
Starch						
Fruit						
Vegetable						
Milk						
Protein						
Fat						
Carbs						

TABLE 20:

CHAPTER VI

DIABETIC FOOT AND WOUND

CONDITIONS

AND

TREATMENT

FOOT AND WOUND PROBLEMS

Foot infections are a major cause of hospitalizations in the diabetic population. Approximately 20 % of all admissions are the result of foot problems, which can become limb, and life- threatening.

It is important to remember that once a problem develops, appropriate diagnosis and treatment is essential if the foot is to be returned to full function. Without appropriate treatment, chronic problems can be created, which can eventually lead to amputation.

PERIPHERAL VASCULAR DISEASE

In the lower extremity, diabetes affects the vascular and nervous systems. The large arteries in the lower leg can become calcified or clogged while the capillaries become thickened, decreasing the ability for oxygen and nutrients to pass into the surrounding tissue and skin, therefore causing injuries to heal poorly. Poor circulation can also lead to swelling and dryness of the foot. With decreased blood flow, necrosis can develop, which interferes with the body's ability to fight infection. This inability can develop into a rapidly spreading cellulitis or take the form of localized pockets under the skin or abscesses. Although infection is usually not the direct cause of an ulceration or wound, it does have great importance in the outcome. If neglected or improperly treated, ulcers may become infected due to prolonged exposure to the external environment or to excessive weight bearing. While these conditions are associated with long standing problems, small scrapes or cracks in the skin may lead to severe infections when inadequate blood flow is present.

Since infection is the number one predisposing factor in lower extremity amputation, it is essential that the patient and health care team take an aggressive approach in the prevention and management of infections. In the diabetic patient, foot infection is the result of several factors, which include neuropathy, vascular disease, and decreased resistance to infection. If trauma is added to the equation, the diabetic foot is at risk for ulceration and infection. Not all foot infections are the result of bacterial invasions, but these sequences of events are the most common leading to lower extremity sepsis.

Immunopathy and defects in leukocyte function are associated with diabetic's susceptibility to infection. Leukocyte phagocytosis, with impaired intracellular killing of bacteria, is significantly reduced in patients with poorly controlled diabetes.

Diabetic foot infections are polymicrobial in nature. These wounds contain anaerobic bacteria along with a wide spectrum of aerobic organisms. Three to five different organisms can usually be cultured from moderate to severe infections. The most common organisms are the aerobic gram-positive cocci, including staphylococcus aureus, coagulase negative staphylococci, and group B streptococci. Aerobic gram-negative pathogens frequently cultured are proteus.

Anaerobic organisms can be found in 80% of patients with moderate or severe infections. Gram-negative rods especially bacteroids, are extremely pathogenic and can cause septicemia in advanced diabetic foot infections.

NEUROPATHY

Over 50% of diabetics can be affected by a condition called neuropathy. High glucose levels can damage nerves resulting in abnormal or decreased sensation. A sign of neuropathy in the early stages includes a burning pain on the bottom of the feet. As neuropathy progresses, numbness starting at the toes and progressing to the foot, the inability to feel pain or hot or cold temperatures develops. This protective mechanism can be decreased to the extent that minor cuts, scrapes, blisters, and abnormal spots may develop, leading to severe infection.

In the absence of pain, a pressure spot can develop into a thick callus. With the breakdown of the underlying skin regions, mal-perforant ulcers develop. Such wounds should be treated as an emergency and medical attention should be obtained within 24 hours once an ulcer or bleeding within the skin occurs.

Charocot arthropathies are micro-fractures that are a result from loss of sensation. The foot becomes red, swollen and deformed with minimal or no pain. If not treated the foot can become deformed, making it difficult to walk or wear shoes.

Footcare and Diabetes
Nursing Implications

The diabetic patient should be continuously aware of any potential problems or existing problems concerning the feet. Proper foot care is critical since diabetics are prone to foot problems. Simple daily foot care and diabetes management can prevent these problems, and should be made a part of the daily routine:

- **Adequate management of diabetes**

  It is important to develop a life style that consists of healthy choices to maintain glucose levels as close to normal as possible.

- **Check feet daily**

  Feet should be checked daily for red spots, bruises, cuts, blisters, and dryness or cracks. This should include checking between and under the toes and feeling for tenderness, hot spots or other signs of infection.

- **Wash feet daily**
  Feet should be washed in warm water with mild soap. Before bathing, the water should be tested for temperature with the hand or elbow. The feet should be dried well, especially between the toes. Talcum powder may be used to keep the skin dry between the toes.

- **Keep the skin soft and smooth**
  Lanolin-based creams can be applied to dry skin, but not between the toes due to risk of infection.

- **Shoes and socks**
  Shoes and socks should be worn at all times. Socks, stockings or nylons should be seamless and worn with shoes at all times to prevent the formation of blisters or sores. Check shoes daily for foreign objects and to make sure the lining is smooth. Shoes should fit well and protect the feet.

- **Trim toenails as needed**
  Trim toenails with clippers after they are washed and dried thoroughly. Toenails are trimmed straight across and smoothed with an emery board. If the nails are yellow or thick a podiatrist should be consulted.

- **Protect feet from hot and cold**
  Insulated boots should be worn to keep feet warm on cold days. During warm days sunscreen can be used on the tops of the feet to protect them from the sun.

- **Exercise feet**
  Exercising regularly can help blood flow and increase flexibility. Walking is the best overall conditioner for the feet. Walking not only improves circulation, but also promotes good general health.

THINGS TO AVOID

- Do not walk barefoot, even indoors. This can expose the skin to cuts and foreign bodies. Walking on hot sand can result in serious burns.

- Do not use heating pads or hot water bottles on feet. This may cause burns due to decrease sensation.

- Do not wear shoes, socks or stockings that are tight or worn out. This could cause pressure sores or decrease circulation.

- Do not soak feet unless specifically ordered. Soaking can dry out the skin and cause the skin to crack.

- Do not trim calluses, corns or abnormally thick nails unless advised by a podiatrist.

- Do not cross legs for long periods of time.

- Do not smoke. Smoking decreases blood flow to the feet.

- Do not rely on the absence of pain to determine if treatment is necessary.

- Do not wear sandals or other open footwear.

ASSESSMENT OF THE DIABETIC FOOT WOUND

NURSING IMPLICATIONS

Location

Wounds on the medial aspect of the foot are caused by constant low pressure, as in wearing tight shoes. Wounds on the plantar aspect of the foot are caused by repetitive moderate pressure.

Description of wounds

When describing the wound, terms such as granular, fibrotic, or necrotic are used. The size, presence or absence of drainage can be described as serous or purulent. Further description of odor and color should be included. Wounds with purulence and /or two of the following signs should be considered infected:

- Erythema
- Lymphangitis
- Cellulitis
- Loss of function
- Edema
- Pain
- Fluctuant underneath the skin

Signs of Peripheral Vascular Disease (PVD)

- Non-palpable pedal pulses
- Pallor on elevation of limbs
- Dusky color or appearance of toes
- Lack of hair on digits and dorsum of foot
- Atrophic shiny skin
- Pain at rest alleviated with dangling the foot or walking

CHAPTER VII

COMPLICATIONS

OF

DIABETES MELLITUS

NEUROPATHY

RETINOPATHY

NEPHROPATHY

BLOOD VESSEL COMPLICATIONS

NEUROPATHY

Neuropathy or nerve damage as a result of chronically high blood sugars can be one of the most debilitating and frustrating complications of diabetes. Available treatments are not always successful, resulting in chronic pain, discomfort and disability.

By keeping blood sugars as closely controlled as possible, exercising regularly, and maintaining weight control, patients are able to find some relief from their neuropathies. Neuropathy is more likely to affect people with long-standing diabetes who have poor glycemic control.

Symptoms of neuropathies can be varied, although pain or numbness in the legs or feet may be the most common complaint. Neuropathy can cause many different complaints depending on whether nerves in the legs, GI tract, or elsewhere are affected.

Symptoms related to neuropathy

- Inability to adequately empty the bladder of its contents, resulting in frequent infections
- Nausea, vomiting, abdominal fullness or bloating, diarrhea or constipation
- Low blood pressure upon standing that causes fainting or dizziness
- Inability to lift the foot or new deformities of the foot or foot ulcers
- Trouble achieving or maintaining and erection

Diabetic neuropathy is a group of nerve disorders affecting the peripheral nerves, which includes the motor, sensory and autonomic nerves.

TYPES OF NEUROPATHY (body parts affected)

Motor neuropathy affects the nerve fibers that carry signals regulating muscles, allowing motions such as walking and fine finger movements. Loss of motor fibers causes muscle weakness.

- Diabetic amyotrophy can be symmetric or asymmetric and is centered in the pelvic girdle and thigh muscles. A progressive atrophy of muscle tissue, the weakening and wasting of muscles is accompanied by aching or stabbing pain.

Sensory neuropathy or peripheral neuropathy affects the nerves that carry information to the brain about sensations from different parts of the body. Loss of sensory fibers ultimately causes visual changes, including an inability to interpret shapes, movement, texture, and pain caused by sharp objects and heat and cold.

- Distal neuropathy is the most frequently diagnosed type of neuropathy. This form of sensory neuropathy affects the hands or feet. It can be asymmetric, but is usually symmetric. Symptoms include numbness and prickling or tingling sensations. The feet can be so tender that walking on a rough surface can cause pain.

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- Femoral neuropathy can be symmetric or asymmetric and is centered in the thigh muscles.

Autonomic neuropathy affects the nerves that control involuntary activities of the body such as the action of the stomach, intestines, bladder, heart and blood pressure. Autonomic neuropathy may lead to impotence in men, bladder neuropathy, diarrhea or a bloated stomach.

- Gastroparesis affects the stomach, preventing it from emptying normally. The resulting symptoms are ulcer-like which includes vomiting, bloating and poor absorption of food resulting in malnutrition and hypoglycemic episodes.

- Diabetic diarrhea is the result of an erratic functioning of the small intestine. This can cause unformed stools to be passed. If the sphincter muscles are involved, stool can pass without warning, resulting in fecal incontinence. If the large intestines are involved, and stool remains in the large intestines too long and constipation will result.

- Bladder neuropathy occurs when the bladder nerves no longer respond normally to pressure as the bladder fills with urine. The bladder is unable to empty entirely leading to urinary tract infections. Symptoms include cloudy urine, low back pain, fever and painful urination.

- Postural hypotension is an autonomic neuropathy that results in low blood pressure when standing. The pulse does not rise to compensate for the change in blood pressure resulting in dizziness or fainting.

- Impotence is caused by autonomic and or sensory neuropathy that leads to blood vessel disease and the inability to have or maintain an erection .

Neuropathic arthropathy

- Charcot's joint or neurological arthropathy occurs when the bones in the feet fracture and feet become misaligned. The foot becomes deformed as a result of the lack of nerve stimulation. This causes the muscles to lose the ability to support the foot properly. People who have neuropathy in their feet and have lost sensation are at a greater risk of developing Charcot's. Symptoms include swelling, redness, heat, strong pulse and insensitivity to the foot. Early treatment can stop bone destruction and aid healing.

- Unilateral foot drop occurs when the foot cannot be picked up because of nerve damage in the leg either by blood vessel disease or compression.

Cranial neuropathy affects the twelve pairs of nerves that are connected to the brain and control sight, eye movement, hearing and taste. Usually cranial neuropathy affects the nerves that control the eye muscles. It starts with pain on one side of the face near the affected eye. With time the eye muscle becomes paralyzed, resulting in double vision. Symptoms usually get better in 2-3 months.

Compression mononeuropathy occurs when a single nerve is damaged, by blood vessel disease that restricts blood flow to a part of the nerve, or when nerves must pass through a tight tunnel or near a lump of bone. Carpal tunnel syndrome is probably the most common form of compression mononeuropathy known. This occurs when the median nerve of the forearm is compressed at the wrist. Symptoms include swelling, numbness, or prickling in the fingers with or without pain.

Thoracic or lumbar radiculopathy occurs most often in people with Type 2 diabetes. It affects the torso as a band around the chest or abdominal wall on one or both sides. People with this neuropathy get better with time.

CAUSES OF NEUROPATHY

There are many theories as to why neuropathy occurs in people with diabetes. Generally, diabetic neuropathy is thought to be a result of chronic nerve damage caused by high blood sugars. One theory states that a covering of cells, called Schwann cells, surrounds nerves. Excess sugar circulating throughout the body interacts with an enzyme in the Schwann cells called aldose reductose. Aldose reductose transforms the sugar into sorbitol. This draws water into the Schwann cells, causing them to swell. The swelling pinches the nerves, causing damage and at times pain. If this process is not stopped and reversed, the Schwann cells and nerves they surround may die.

DIAGNOSING NEUROPATHY

One way of diagnosing neuropathy is through symptoms being exhibited. A physician may detect early signs of neuropathy by observing decreased responsiveness to knee or ankle jerk tests or by observing orthostatic changes in blood pressure.

There are specific tests used in diagnosing neuropathy. Electromyography is a test that measures the response of muscles to electrical impulses. Nerve conduction studies that study the flow of electrical currents through the nerve inserts a needle into the muscle to measure the electrical charges. This test can show whether a nerve fiber is breaking down or healing.

To diagnose Charcot's joint, the doctor may take an x-ray of the joint or possibly perform a bone scan.

TREATMENT OF NEUROPATHY

There are no cures or treatment for nerves damaged by neuropathy. Although there are a variety of treatments that are helpful, there is no way to heal or replace nerves that have been damaged.

The most important things that people with neuropathy should do is:

- Keep their blood sugar levels as close to normal as possible
- Reach and keep an ideal weight
- Follow a regular exercise program

By keeping blood sugars closer to normal, the damage high blood sugars can cause to nerves is limited. Exercise will have the added advantage of keeping muscles that have been weakened by decreasing nerve activity to remain strong and toned.

No one therapy works best for everyone. Treatment should be tailored to the location of the pain and the type of pain.

A major goal of treatment for neuropathy is relieving pain. Acetaminophen, aspirin and ibuprofen are usually used before narcotics. Pain medications are best used throughout the day before the pain becomes severe. Narcotics can relieve pain, but they are used only as a last resort. Use of narcotics for a long period of time can lead to addiction.

Creams that contain capsecum, an extract of hot peppers that includes cayenne and tabasco peppers, are rubbed on the skin in the painful. These creams block pain signals although they do not work for everyone.

Anti-depressants are used to relieve the pain associated with neuropathy. Tricyclic drugs including Elavil, Norpramin or Imipramine are among the drugs prescribed to block neuropathy pain. Patients take these medications at night, as they help with depression, anxiety, and insomnia associated with neuropathy. These anti-depressants decrease the patient's awareness of pain, which is usually more severe at night. Anti-depressants can take several weeks to become effective. Uncomfortable side effects can include dry mouth, constipation, and nausea.

There are other types of drugs that sometimes help. Anti-convulsants such as Dilantin, Tegretol, and Neurontin are used along with drugs such as Mexitil, normally used to treat irregular heart rhythm. Many of these drugs can have unpleasant side effects such as dizziness or confusion when taken in large doses, especially by the elderly.

Gastroparesis

Reglan or Propulsid are drugs used for gastroparesis in which the stomach is not emptying properly. These drugs help the stomach push food along and get it through the rest of the digestive process. Carafate is used in conjunction with these drugs to help control extra acid that may be sitting in the stomach.

Bladder Neuropathy

Urecholine treats bladder neuropathy that results in the bladder never completely emptying. Because patients with this problem are more susceptible to developing urinary tract infections, antibiotic therapy may be prescribed to try and keep the bacterial count in the bladder and urinary tract at a manageable level. Patients should be encouraged to urinate every 3-4 hours when they are awake even if they don't feel the need.

Erectile Dysfunction

Erectile dysfunction as a result of neuropathy or blood vessel damage can be treated using certain drugs that are inserted into the end of the penis or injected to cause an erection before intercourse. Vacuum devices that enable an erection to be achieved or surgically implanted prosthesis are options to be explored. Viagra is another option that can be considered by patients with erectile dysfunction. Diabetics however, are more susceptible to heart and vessel disease than non-diabetics, and use of Viagra by individuals with underlying cardiovascular disease should be reviewed thoroughly. In

addition to its known side effects, the use of Viagra with nitroglycerin tablets has been associated with several reported deaths.

Postural Hypotension

Certain blood pressure-raising medications may be prescribed for patients with postural hypotension or they may benefit from support stockings to prevent pooling of blood in the legs. If the diabetic patient also has high blood pressure, the process of balancing blood pressure-lowering medication with medication that will keep the blood pressure from dropping while reclining or sitting can be very difficult and may require several adjustments.

Charcot's

Treatment for Charcot's joint is geared toward preventing further damage. The joint should be immobilized and weight bearing should be avoided while the joint is healing. The foot is usually put into a cast or a special brace for a period of weeks. This preserves the joint function and limits damage. As the foot heals, special shoes are worn. If the joints have healed into a deformed shape, surgery may be necessary to restore the joint to a more normal shape foot.

DIABETIC RETINOPATHY

Diabetic retinopathy is the leading cause of blindness and visual impairment in the diabetic population. Compared to non-diabetics, people with diabetes are four times more likely to become blind. 12% of new blindness diagnosed each year and 8% of the legally blind in the United States are attributed to diabetes as the underlying cause. New diagnosis of Type 2 diabetics' show approximately 21% has some degree of eye damage. The main diabetes-related eye disease is retinopathy. Retinopathy is defined as impairment of the retina due to deterioration of the capillaries. According to the American Diabetic Association, after 15 years of diabetes, 2% of people become blind while 10% develop severe visual handicap. 97% of people on insulin and 80 % of Type 2 diabetics show evidence of retinopathy.

Retina

The retina is a delicate layer of nerve tissue at the back of the eye. Light enters the eye and is focused by the lens through a clear gel-like fluid called the vitreous. This nerve-rich, light-sensing area in the back of the eye is crucial for sight, functioning by changing the image into electrical impulses that are transmitted to the brain via the optic nerve.

The retina is composed of two parts. The macula is the middle portion closest to the optic nerve and is responsible for central vision and color vision. The peripheral or outer region is responsible for side vision and night vision.

TYPES OF RETINOPATHY

Background Retinopathy

Background retinopathy or nonproliferative diabetic neuropathy is an early stage of retinopathy that usually involves no apparent damage. In this type of retinopathy, blood vessels within the retina develop tiny bulges or microaneurysms, which leak fluid and hemorrhage. This causes swelling and forms deposits or exudate. If the macula, the part of the retina where central vision occurs, becomes swollen, distorted vision is the result. Mild background retinopathy is normally not treated. This type affects approximately 90-95% of long- term diabetics to some degree. This is generally perceived as a warning sign and can progress to the more severe form proliferative retinopathy.

Proliferative Retinopathy

Proliferative retinopathy is a more advanced and severe from of retinopathy. In this form of retinopathy, the retinal vessels close, causing growth of abnormal blood vessels over the retina and into the vitreous. These abnormal vessels block light from reaching the retina. Connective tissue growing along with the abnormal vessels may grow into the vitreous and contract causing the retina to detach. This is called traction retinal detachment. The patient may notice a dark shadow and vision is poor. The corrective

procedure for this condition is surgery to release the traction, remove scar tissue, and re-attach the retina. Surgery must be performed as soon as possible to preserve vision.

If the retinopathy affects the peripheral retina, the patient may notice no signs and symptoms aside from difficulty with night vision. Patients will generally notice more difficulty if the macula is affected.

Prevention of Retinopathy

To minimize the risk of diabetic retinopathy it is essential to:

- Adequately control blood sugar levels
- Maintain a normal blood pressure
- Schedule an ophthalmology examine at least once a year

Treatment of Retinopathy

Laser treatment of leaking blood vessels for macular edema reduces the risk of future loss by 50%. Laser treatment to the peripheral retina or pan-retinal photocoagulation is used for proliferative diabetic retinopathy. Brief intense bursts of laser light can repair leaking blood vessels, destroy those that cannot be fixed, and prevent formation of new ones. Laser surgery does not reverse retinopathy. Non-proliferative diabetic retinopathy may reduce further loss of vision and delay the onset of proliferative retinopathy.

A vitrectomy is used when massive bleeding into the vitreous has occurred. In this procedure, the bloody vitreous is removed and replaced with clear, sterile fluids, which restores vision.

Fluorescein angiography is ordered if after an eye examination diabetic retinopathy is suspected. This is a series of pictures taken as iodine-based dye travels through the retinal vessels. In conjunction, an ultrasound may be used to detect retinal detachment.

CATARACTS

Cataracts are clouding of the normally clear lens. A cataract develops over years and causes progressive blurred vision. Causes of cataracts include aging, eye injuries, disease, heredity and birth defects. Senile cataracts are common eye problems among the elderly. Poor diabetic control can hasten the formation of senile cataracts. Metabolic cataracts are sometimes found in younger people with diabetes. Treatment for senile and metabolic cataracts is surgical removal of the lens. Vision is restored after surgery by using eyeglasses, contact lenses, or intraocular lens implants.

GLAUCOMA

The neovascularization of proliferative retinopathy can cause blood vessels to grow into the iris, leading to glaucoma. Glaucoma is a group of diseases characterized by damage to the nerve due to increased pressure in the eye. The vision loss from glaucoma is permanent. The best protection against glaucoma is prevention and early detection.

Early detection and treatment of diabetic eye disease is essential to prevent blindness. Early diabetic eye disease initially may have no symptoms. An initial eye examination with an ophthalmologist, keeping regularly scheduled appointments, along with proper management of diabetes can help prevent the devastating complication of blindness.

DIABETES AND NEPHROPATHY

Diabetic nephropathy is a complication of long-term diabetes that affects nearly one million Americans. This secondary complication of diabetes is characterized by progressive loss of kidney function, which ultimately requires dialysis and frequently leads to end-stage renal disease requiring kidney transplant.

The kidneys filter waste products from the blood through capillaries. As the blood passes through the capillaries, the kidneys draw waste out and produce urine. In people with nephropathy, capillaries become blocked and leaky. Waste stays in the blood and protein, which should remain in the blood, leaks into the urine.

One-third of people with Type1 diabetes develop kidney complications within 15 years of diagnosis. 15% of people with Type 2 diabetes develop nephropathy within 15 years of diagnosis. The first sign of kidney complications is microalbuminuria, or small amounts of protein in the urine.

Microalbuminuria generally appears around ten years after diabetes has been diagnosed. Over a period of time, the protein content of the urine increases. When the protein content reaches 30mg/dl, which is generally after 15 to 20 years as a diabetic, the diagnosis of nephropathy is made. As kidney function decreases over the years, kidney function declines and the protein content of the urine continues to increase. Microalbuminuria becomes proteinuria. After 25 to 30 years of diabetes, the kidneys no longer function, resulting in end-stage renal disease. (ESRD)

People with diabetes are twenty times more likely to develop ESRD than non-diabetics. ESRD is fatal unless a kidney transplant is performed or dialysis is started. About 15% of kidney transplant recipients die within two years, and about 35% of diabetics on dialysis die within two years.

Diabetics should have their urine checked regularly for protein along with watching for symptoms of nephropathy including fatigue, insomnia, weakness, vomiting, and body swelling due to fluid build-up.

RISK FACTORS FOR NEPHROPATHY

Heredity: In families where some family members develop kidney complications, other diabetics in the family are at higher risk for nephropathy.

High blood pressure: Hypertension stresses the kidney capillaries, increasing risk of capillary leakage.

High protein intake: The more protein eaten, the more protein passes into the urine. Most people consume far more protein than necessary to maintain good health.

PREVENTION AND TREATMENT OF NEPHROPATHY

Reduce blood pressure: Blood pressure medications slow the progression of nephropathy along with a low-salt diet. It is also advisable to reduce protein consumption and to keep the blood sugar level under control.

Treatment choices for ESRD

There are new and better treatments for ESRD that replace the function of healthy kidneys. There will be changes in the patient's life no matter which treatment is chosen. The health care team plus the patient will discuss the various options, and together choose the best treatment.

Hemodialysis

Hemodialysis cleans and filters the blood. It cleans the body of metabolic wastes and extra fluid. This procedure is done approximately 3 to 4 times a week. Each treatment can last anywhere from 4 to 6 hours at a time.

Complications of Hemodialysis

Due to rapid changes in the body's chemical and fluid balance, side effects may include muscle cramps, along with nausea, dizziness, and weakness associated with hypotension. By following a proper diet and taking medications as directed, many of the side effects can be avoided.

Diet

A proper diet can help reduce the wastes that build up in the blood. The following points should be considered when choosing foods:

- Eat balanced amounts of foods with limited amounts of foods high in animal protein such as meat and chicken.
- Potassium is a mineral found in salt substitutes, vegetables, milk, chocolate, some fruits and nuts. It is essential to eat the proper amount of potassium, since it has a direct effect on the functioning of the heart.
- The intake of fluids should be limited. Since the kidneys are not working properly, a build up of fluids can cause not only edema of the tissues, but can cause high blood pressure and heart problems.
- Salt and salty foods should be avoided since they can cause the body to retain fluid.

- Foods such as milk, cheese, dried beans and soft drinks should be limited due to their phosphorus content. Too much phosphorus in the blood causes calcium to be pulled out of the bones. This in turn can lead to problems with bones.

Peritoneal Dialysis

Peritoneal dialysis is a procedure that replaces the work of the kidneys. This procedure removes wastes, extra water, and chemicals from the body. The peritoneal membrane of the abdomen is used to filter the blood.

Dialysate, a cleansing solution, is introduced through a tube into the abdomen. Fluid, wastes, and chemicals pass from tiny blood vessels in the peritoneal membrane into the dialysate. The dialysate is drained from the abdomen after several hours, taking with it the wastes from the blood.

There are three different types of peritoneal dialysis.

- Continuous Ambulatory Peritoneal Dialysis **(CAPD)**
  The most common type of peritoneal dialysis is CAPD. With CAPD the blood is being clean continuously. The dialysate passes from a plastic bag through a tube into the abdomen. The dialysate stays in the abdomen for four to six hours via a sealed catheter. The solution is drained back into the bag bringing with it the waste products. The abdomen is refilled with fresh solution through the same catheter and the process begins again. The process of draining the dialysate and replacing fresh solution takes approximately 30 to 40 minutes. The solution is changed four times a day. While the solution is in the body, the plastic bag may be folded and hidden under the clothes, around the waist or in a pocket.

- Continuous Cyclic Peritoneal Dialysis **(CCPD)**
  CCPD is similar to CAPD except that a machine is connected to the catheter. The machine allows the abdomen to be filled and drained with dialysate automatically. The procedure takes 10-12 hours and is done at night to minimize inconvenience to the patient.

- Intermittent Peritoneal Dialysis **(IPD)**
  The same machine is used for IPU as in CCPD. Whereas CAPD and CCPD are done at home, IPD is usually performed in the hospital. IPD treatments are done several times a week for a total of 36 to 42 hours per week. Sessions may last as long as 24 hours.

Complications of Peritoneal Dialysis

Peritonitis, or infection of the peritoneum, occurs if the opening where the catheter enters the body becomes infected. It can also occur if the catheter bag is not connected or disconnected correctly.

Peritonitis can be a very serious problem with peritoneal dialysis. It may cause fever and abdominal pain. Signs include reddening or swelling around the catheter, discharge around the catheter and cloudy dialysate.

Diet

The diet for peritoneal dialysis is slightly different than the diet for hemodialysis.

- The patient may be able to have more salt and fluids
- More protein may be eaten
- There are usually different potassium restrictions
- The number of calories eaten is reduced. The limitation is due to the sugar in the dialysate, which can cause weight gain

Kidney Transplant

Kidneys used in transplantation may come from different sources. A kidney received from a family member is called a living-related donor. A kidney received from a person who has recently died is called a cadaver donor. If a spouse or a close friend donates a kidney, it is called a living-unrelated donor.

In order for the transplant to be successful, the donor's tissue and blood must match the recipient's tissue and blood. This will help prevent the body's immune system from rejecting the new kidney.

The new kidney is placed between the upper thigh and abdomen. The artery and vein of the new kidney is connected to the recipient's artery and vein. Blood flows through the new kidney and functions like the old kidney when it was healthy. The old kidneys are left in place unless they are causing infection or high blood pressure. The new kidneys may start working immediately or it may take up to a few weeks. The transplant surgery takes approximately 3 to 6 hours, with the usual hospital stay of 10 to 14 days.

Complications

The most severe complication is the possibility of rejection. The chance of the body accepting the new kidney depends on age, race, and overall medical condition.

75 to 80 percent of transplants from cadaver donors are working one year after surgery. Transplants from living relatives usually work better than the cadaver donor, since the living relative donors form a closer match.

Immunosuppressant drugs are given to help prevent rejection. These drugs must be taken every day for the rest of the patient's life. However, even with these drugs there is the possibility that the kidney will be rejected. Treatment with these drugs can cause side effects. The most serious side effect is the weakening of the immune system. Other side effects include weight gain, facial hair, and a fuller face along with cataracts and hip disease. There are also a small number of patients that may develop liver or kidney damage when these drugs are used over an extended period of time.

Diet

The diet of a transplant patient is less restrictive than a dialysis patient. The diet will probably change, as the medication, blood values, weight and blood pressure would dictate:

- Medication may increase the appetite and cause weight gain; therefore, counting calories may be necessary.
- Limiting salty foods may be necessary since the medication may cause retention of sodium, resulting in high blood pressure.
- Eating less protein is sometimes necessary to prevent a build-up of waste in the bloodstream.

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BLOOD VESSEL COMPLICATIONS

Macrovascular Disease

Macrovascular disease refers to changes in the medium to large size blood vessels. The blood vessel walls thicken and become hard and arteriosclerotic. Atherosclerosis also becomes a problem, eventually blocking flow.

Peripheral vascular disease refers to diseased blood vessels that supply the legs and feet. When blood flow is partially interrupted, cramps, weakness, or claudication may result. If the artery is completely blocked, severe pain accompanied by cold and discoloration will occur in the legs. Treatment includes replacing the artery surgically or performing an angioplasty.

Coronary artery disease refers to diseased arteries of the heart. When blood flow is decreased, angina may occur. Complete blockage of an artery results in myocardial infarction. Symptoms include chest pressure, cramping, a heavy feeling in the chest, shortness of breath, and extreme fatigue. Suggested treatments include coronary bypass surgery and angioplasty.

Cerebral vascular disease refers to partial or complete blockage of arteries in the brain. Arterial blockage may result in temporary reductions of blood supply to a part of the brain or transient ischemic attacks. When a complete blockage occurs or a blood vessel breaks, a cerebral vascular accident occurs. Symptoms include lightheadedness, dizziness, confusion, aphasia, and inappropriate behavior.

Minimizing Risk of Macrovascular Disease

In order to minimize the risk of macrovascular disease followed these guidelines as closely as possible:

- Maintain blood sugar control
- Maintain normal blood pressure
- Maintain weight control
- Reduce fats and cholesterol in diet
- Exercise in moderation
- Do not smoke

SUMMARY OF COMPLICATIONS

The principal concern for people with diabetes has always been to avoid both acute and chronic complications. It is important to understand the risks of complications the disease can cause. With this knowledge, the patient is better equipped to make decisions regarding health and maintaining good health.

Acute Complications of Poorly Controlled Diabetes			
Complications	**Cause**	**Early signs**	**Prevention**
Diabetic Ketoacidosis (DKA)	Insulin deficit causing severe metabolic alterations	Weight loss Increased urination Increased thirst Vomiting Rapid breathing	Insulin must be given
Hyperosmolar Hyperglycemic Non ketotic Coma (HHNK)	Excessive blood glucose concentration	Increased urination Increased thirst Fatigue Lethargy	Maintaining blood Glucose within lower range
Hypoglycemia	Blood glucose drops significantly below healthy range and can not recover naturally because of diabetes medication	Lightheaded Dizzy Shaky Hungry Weak, Tired	Carbohydrate food intake is balanced with medication and activity

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Chronic Complications of Diabetes		
Systems Effected	**Disease**	**Health Concern**
Eyes	Retinopathy Glaucoma Cataracts	Blindness
Blood Vessels	Coronary artery disease Cerebral vascular disease Peripheral vascular disease Hypertension	Heart attack Stroke Poor circulation in feet and legs Heart attack, stroke, kidney damage
Kidneys	Renal insufficiency Kidney failure	Insufficient blood filtering Loss of ability to filter blood
Nerves	Neuropathies Autonomic neuropathy	Chronic pain Poor nerve signaling to organ systems
Skin, Muscle, Bone	Advanced infections Cellulitis Gangrene	Amputation

Table: 22

CHAPTER VIII

The Family and Diabetes

Children and Diabetes Management

THE CHILD WITH DIABETES

As part of the health care team, it is partially up to the caregiver to help the family and the child accept his or her diabetes with a minimum of stress. The American Diabetes Association and the Juvenile Diabetes Foundation can be of great help. Other parents who have faced the same problem and learned to cope with it are more than willing to share ideas and advice. It is important to help the family learn to protect without dominating, to supervise while encouraging self-care. It is important to work with the child and the parents for the best control, but to help them understand that "ideal" control is not always possible.

The child's self image and self-esteem are threatened by diabetes. Encourage the parents to be understanding and supportive. No child can be expected to assume complete responsibility for diabetes control at a very early age. Ultimately, responsibility for eating properly, injecting insulin, testing blood sugar and planning exercise will be the child's responsibility. Avoiding unnecessary anxiety about "cheating" and guilt feelings is essential in order to help the child develop the maturity, independence, self control, and self esteem needed as the child learns self-care.

The parent's role changes as the child grows.

The role of the parent changes as the child grows. Every child is different but there are some things that remain the same. A child with diabetes is a child first, and a child with diabetes second. Accept, love, teach, discipline just as you would a child without diabetes as a factor. Remember that by overprotecting or overindulging, the child might develop feelings that reinforce feelings of inadequacy and over-anxiousness.

Birth through 7 years

The parent has full control of all responsibilities during early childhood. It is important for the parent to involve the child at an early age. In order to start the process of developing self-reliance, offer the child some choice in his/her care such as picking out the spot to inject or selecting which finger to get a drop of blood. Parent approval is always important, be sure to describe results as high, low or normal, and not in terms of good or bad.

7 years through 12 years

During this time of the child's life, the child can assume more responsibility for his/her own care. The child can take over blood glucose testing and insulin injections some of the time. By the age of 12, most children can manage their own injections. It is important for the parent to remind the child of his newly assumed responsibilities. Once again by encouraging the child to participate in his/her own care the child learns to become independent and self-reliant.

12 years through 17 years

By the time the child reaches adolescence, it is essential that the child be permitted to participate in treatment decisions. Even with a child that is educated about their disease process, adolescents may act as if they did not have diabetes. This includes ignoring their diet and falsifying blood sugar tests. Depression is common at this time and contacting a professional counselor might be helpful. It is important that the child understand that people with diabetes live full lives.

17 years through 20 years

At this age the child will begin to mature in attitude and responsibility. By setting realistic goals, and by educating the child about their disease process all through childhood, the child by this time has progressed from dependence to independence and self-reliance.

Physical Education and Sports

It is essential to remember that exercise is an important factor in diabetes control. With some planning, a child with diabetes may enjoy all the benefits of physical education and sports. If the child has gym class before lunch, increase the morning snack of carbohydrate and protein. If the child has a late gym class or participates in after-school sports, increase the afternoon snack. Make sure the child understands to carry fast acting sugar at all times. It is also important for the child's gym teacher or coach to know how to be of assistance if the child needs help in case of a reaction.

Parties

Diabetes is no reason for missing out or not being able to enjoy social occasions with friends as long as a few simple rules are followed. If there is a social occasion pending, plan ahead by saving some food exchanges from dinner. Bring diet soda or make sure to drink only diet soda at the party. Choose foods carefully and replace calories used for dancing. Through trial and error and with the help of blood testing, the teen will learn how many calories to replace, and always carry fast acting sugar.

School

School personnel should be informed that a student has diabetes. Teachers, coaches, school nurse and other school personnel need to understand what having diabetes means and how they may assist the child if a reaction occurs. By giving the school personnel a concise overview of diabetes, this will help school personnel cope with the child's special needs. It is advisable to prepare a written plan, which should include the following information:

- When to check blood sugar and take insulin
- Meal and snack time
- Preferred snack and party foods
- Usual symptoms of high and low blood sugar
- Preferred treatment of high and low blood sugar
- Phone numbers for parents and other emergency contacts
- Treatment plans should be reviewed and updated with school personnel as needed during the school if change occurs, and then every new school year

CHAPTER IX

COPING WITH DIABETES

NURSING IMPLICATIONS

Dealing with diabetes

The newly diagnosed diabetic patient is probably left feeling confused and overwhelmed. They may have difficulty concentrating on learning all the things they have to follow in order to develop a healthy diabetic care plan.

Good diabetes care requires a daily effort to follow a diet, stay active, and take medicine as indicated. By taking one day at a time and learning all they can about their disease, the patient may begin to feel less frightened and more in control of their lives.

Some people have difficulty believing that they have diabetes despite all the evidence that is presented. Other people understand that they have diabetes, but still do not follow their care plans. The goal at this point is to accept the diagnosis so that learning may begin and eventually achieve independence and good health by following a care plan designed specifically for the patient.

The newly diagnosed diabetic may feel angry. Angry feelings are normal and a healthy part of adjusting to their disease, as long as the patient does not allow it to interfere with achieving the goals necessary to developing a good health plan.

Depression can be very debilitating for people with diabetes. Symptoms of depression include feelings of helplessness, loneliness, lack of self-esteem, fatigue, irritability, and changes in sleep patterns or eating habits. If any of these symptoms are identified, refer the patient counselors with experience in helping people with diabetes.

It is normal to grieve over the loss of the healthy self when first diagnosed with diabetes or when complications occur. Time and support from family, health-care providers and friends can help the patient resolve grief.

One of the most difficult things for the patient to come to terms with is the fact that even if the patient follows his/her care plan to the letter, it still might not be possible to achieve perfect control.

Treatment Plan and Regular Assessments

With the help of the health-care team, people with diabetes can live long, healthy lives. It is very important for people with diabetes to understand how to stay healthy, follow a proper diet, exercise, and be aware of changes in their bodies.

Points to Remember

- Good diabetes care is a daily responsibility.
- Local diabetes organizations offer programs so people with diabetes can share experiences and support.
- Good health care prescribed for people with diabetes, can also benefit anyone who wishes to stay healthy.
- Medical guidance is available from a variety of sources such as diabetes groups, local medical societies and hospitals, diabetes clinics, and the diabetic health care team.

Treatment Plan

It is essential that the health care team include the following in the patient's treatment plan:

- Each patient needs personalized advice on proper eating, which includes types of food, amounts, and timing of meals and snacks.
- Exercise is critical in achieving and maintaining diabetic control. Helping the patient understand the appropriate exercise level and how to incorporate exercise in their daily lives can help them control diabetes.
- Each patient needs to know the dose and timing of oral medication or insulin and how to take the medication.
- The patient needs to know the appropriate values for blood glucose, blood pressure, and weight.
- The patient needs to have a working understanding of the complications associated with diabetes.

Regular Treatment Assessments

At each regular visit, the patient should be assessed for the following:

- Encourage the patient to talk about any problems and questions they might have.
- Changes in diet, medication, monitoring, and exercise should be evaluated at each visit.
- All self-monitoring results and treatments should be reviewed and re-evaluated.
- A physical exam should be performed and include an evaluation of weight, blood pressure, eyes and vision, kidney function, feet and skin care.
- Diabetes education should be a continuous process.

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