Honor's Project
for ID 499

JUVENILE DIABETES

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PREFACE

This paper was written to provide the nurse and other members of the health team with the information necessary for the total care of a child with Juvenile Diabetes. The topic is introduced with specifics about the disease Diabetes, to provide the reader with a background which is essential in understanding the treatment and nursing care of this patient. The care of the diabetic child is broken down into three main sections: the general treatment, the specific nursing care and observations, and the education of the patient. The most important segment of the patient's treatment is in educating the patient to care for himself. Section IV includes a checklist with the major areas that must be covered, followed by a detailed outline of the methods used to present the topic to the patient. The case study included at the end of Section III serves to illustrate the course of Diabetes in a hospitalized child and exemplify the topics covered throughout the paper, particularly illustrating the need for organized care.

I wish to express my appreciation to the two faculty advisors who aided me in the preparation of this paper, Miss Wantz, instructor in Maternal Child Nursing, and Mrs. Holmes, instructor in Medical Surgical Nursing, Ball State University. I want to also thank the nursing staff on Pediatrics at Ball Memorial Hospital for their help and co-operation.

Jennifer L. Harley
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JUVENILE DIABETES

Section I: The Disease

Definition

Diabetes mellitus may be defined as a disturbance in carbohydrate metabolism resulting from insulin deficiency. There is a tendency for abnormal elevation of blood sugar levels (hyperglycemia) leading to glycosuria causing a failure of nutrition and the regulation of water and acid-base balance.\(^1\) Diabetes mellitus which has its onset before the age of 15 years is termed Juvenile Diabetes.\(^2\) Diabetes having its onset in adulthood is called Mature Onset Diabetes. A comparison of these two forms of Diabetes can be found in Table 1.

Incidence

The children affected by Juvenile Diabetes comprise five percent of the total diabetic population.\(^3\) In comparison to the entire population 40 children per 100,000 have Diabetes; and there are approximately 10,000 new cases each year in the United States.\(^4\) Some literature states


Table 1: Comparison of Childhood and Adult Forms of Diabetes

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Child</th>
<th>Adult</th>
</tr>
</thead>
<tbody>
<tr>
<td>Onset</td>
<td>Rapid, obvious</td>
<td>Slow, incidious</td>
</tr>
<tr>
<td>Obesity</td>
<td>Plays no role</td>
<td>A predisposing factor</td>
</tr>
<tr>
<td>Dietary Treatment</td>
<td>Not adequate alone (used in combination with insulin)</td>
<td>Single treatment for 1/3 of cases</td>
</tr>
<tr>
<td>Use of oral hypoglycemic agents</td>
<td>Contraindicted</td>
<td>Useful in 1/3 of cases</td>
</tr>
<tr>
<td>Need for insulin</td>
<td>In all cases</td>
<td>Present in 1/3 of cases</td>
</tr>
<tr>
<td>Hypoglycemia and Ketoacidosis</td>
<td>Commonly seen</td>
<td>Uncommon</td>
</tr>
<tr>
<td>Systematic degenerative vascular changes</td>
<td>Only after adolescence</td>
<td>May be present at diagnosis</td>
</tr>
</tbody>
</table>

that there is little difference in incidence between the sexes. Dr. Monteleone found, in his studies of diabetic patients admitted to Cardinal Glennon Memorial Hospital for Children, that the "incidence of Diabetes was consistently higher in girls of all ages until adolescence, the most significant difference is in the age group of 4-7 years." Diabetes is the eighth cause of death in children.

Etiology

The basic lesion of this chronic disease remains

1Weil, p.345.
2Traisman, p.1.
4Traisman, p.1.
unknown. It is believed that the fundamental genetic abnormality is present from the time of conception, even though the time of onset of the disease is greatly variable.\textsuperscript{1} This genetic abnormality is believed to be transmitted through Mendelian recessive genes with unknown secondary factors allowing their expression.\textsuperscript{2} Some authors believe that the disease is transmitted in a single recessive gene while others believe there are multiple genes involved. There have also been those who reit that Juvenile Diabetes was a dominate trait and Maturity Onset Diabetes was a recessive trait.\textsuperscript{3}

Six facts in support of the hereditary transmission of diabetes have been proposed. First of all, there is a 50 percent incidence of diabetes in similar twin mates. Secondly, the incidence of diabetes in close blood relatives of patients is seven times that of the control population. Families studied have demonstrated Mendelian ratios of the recessive type in the incidence of the disease. In families tested with glucose tolerance tests this same ratio pattern emerged. There is a high occurrence of diabetes in like sexed siblings. Finally, there is a tendency toward a lowering of the age of onset in each successive generation.\textsuperscript{4}

\textsuperscript{1}Weil, p. 343.
\textsuperscript{2}McQuarrie, p.1.
\textsuperscript{3}Traisman, p. 3.
\textsuperscript{4}McQuarrie, p.2.
Pathophysiology

The result of this genetic determination is the disease Diabetes Mellitus, characterized by an insufficient amount of insulin production. The two main hormones controlling carbohydrate metabolism are insulin and glucagon, produced by the pancreas. Insulin plays an important role in carbohydrate, fat, and protein metabolism. The following functions are performed by insulin: 1) Stimulates the active transport of glucose into the cells of muscles and adipose tissue (the exact mechanism is unknown); 2) regulates the rate at which glucose is burned by the cells; 3) aids in the conversion of fats to fatty acids, and inhibits the breakdown of fatty acids; 4) promotes the conversion of glucose to glycogen and inhibits the reverse; and 5) stimulates the synthesis of protein. All of these actions work to lower serum glucose levels. On the other hand, glucagon promotes a rise in blood sugar.¹

Carbohydrate metabolism, which provides the body with the energy to function, has two phases, a destructive phase (catabolism) and a constructive phase (anabolism). In catabolism the carbohydrate molecule is broken down into smaller components and energy is released. There are three forms of carbohydrate catabolism; they are: 1) Glycolysis—breakdown of sugars to simpler compounds; 2) The Krebs Cycle—a series of steps resulting in the complete breakdown

of the glucose molecule; and 3) Glycogenolysis - the conversion of glycogen to glucose.\(^1\) Carbohydrate anabolism consists of the conversion of carbohydrates, fats, and proteins into glycogen and stored in the liver. The two processes involved with anabolism are glycogenesis, conversion of sugar to glycogen, and glyconeogenesis, the changing of fats or proteins to glycogen.\(^2\) Figure 1 illustrates normal carbohydrate metabolism.

Insulin is released in response to glucose blood levels to transport glucose into cells where it is metabolized and into the liver for storage. Glucose is the product of digestion of carbohydrates, when insulin is lacking from the body (Diabetes Mellitus) glucose is not utilized for energy production. The body instead breaks down fats and proteins for fuel.

The development of this disease has been divided into four stages. These stages are seen mainly in Maturity Onset Diabetes, but they may also apply to Juvenile Diabetes if one keeps in mind that the stage may appear abruptly and be of relatively shorter length. The first stage is termed prediabetes. This stage includes the time from birth to the first abnormalities in carbohydrate metabolism. In this time period biological abnormalities such as increased insulin-like activity has been found in some cases, and early changes in vascular bed takes place. The second stage is

\(^1\) Luckmann, p. 1312.

\(^2\) Ibid, p. 1313.
Figure 1: Pathways of Normal Carbohydrate Metabolism

**DIGESTIVE TRACT**
- Starch, Simple sugar, Fiber → Feces
- Galactose, fructose, glucose

**LIVER**
- Liver Glucogen → Hexose (phosphorylated) → Glucose
- Glucose to glucose
- Pyruvic Acid → Lactic Acid + NH₂
- CO₂ + H₂O (Krebs Cycle) → Amino Acid (aline) → Fatty Acid

**BLOOD**
- Lactic Acid
- Glucose 160-180 mg% → Urine

**MUSCLE**
- Muscle → Phosphorylated Glucose
- CO₂ + H₂O → Lactic Acid
- Pyruvic Acid → Krebs Cycle

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called subclinical Diabetes. In this stage the carbohydrate metabolism is abnormal in children during illness or other stressful situations. The child is otherwise asymptomatic and the disease is noticed accidentally if a urinalysis is taken during an acute illness or following surgery, displaying glucose in the urine. Latent or chemical Diabetes is the third stage of this disease. Serum glucose levels are elevated after meals with corresponding glycosuria. This stage has been found to last only a few days to several months in children. The final stage is Overt Diabetes Mellitus, which usually appears abruptly in children. The transition from latent to overt is often precipitated by stress such as infection. In fact the most readily recognized peaks of Diabetes are at six and twelve years; corresponding to the beginning of school and adolescence. Table compares the physical signs seen in the stages of Diabetes.

In the overt stage of Diabetes, whatever the direct cause, insulin levels are insufficient to meet bodily needs. The metabolic disturbances which follow are summarized in the three general alterations: 1) reduced amount of glucose entering the cells; 2) this leading to unavailability of carbohydrates as a fuel; and 3) utilization of alternate substances, namely fatty acids and amino acids, for fuel.

1Weil, p. 345.
2Ibid., p. 345-346.
3Ibid., p. 344.
Table 2: Comparison of Stages of Diabetes

<table>
<thead>
<tr>
<th>Stage</th>
<th>FBS</th>
<th>GTT</th>
<th>Cortisone GTT</th>
<th>Delayed and/or decreased insulin response - glucose</th>
<th>Clinical features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-diabetes</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>+</td>
<td>Asymptomatic</td>
</tr>
<tr>
<td>Sub-clinical</td>
<td>N</td>
<td>Abnormal</td>
<td>Abnormal ++</td>
<td></td>
<td>Asymptomatic</td>
</tr>
<tr>
<td></td>
<td>N</td>
<td>Abnormal</td>
<td>Abnormal ++</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chemical</td>
<td>N or Increased</td>
<td>Abnormal</td>
<td>NN</td>
<td>+++</td>
<td>Asymptomatic</td>
</tr>
<tr>
<td>Overt</td>
<td>Increased</td>
<td>NN</td>
<td>NN</td>
<td>+++</td>
<td>Classic symptoms</td>
</tr>
</tbody>
</table>

(N=normal, NN=not necessary)

Tissue that is sensitive to insulin (skeletal muscle, heart, adipose tissue, fibroblasts) are deprived of the hormone in Diabetes. The transport of glucose from the blood into the interior of their cells is slowed markedly. The liver, at this time, also increases its rate of glucose release - by glycogenolysis. Thus, the glucose accumulates in the blood, and the amount of glucose rises. Since there is not enough insulin in the blood, this glucose derived from a meal or an administration of glucose cannot be disposed of rapidly. The tolerance for glucose is diminished. Since the glomerulus of the kidney is freely permeable to glucose, the increased serum glucose level causes an increase in concentration in the glomerular filtrate. When the amount


of filtered glucose exceeds the capacity of the tubules to reabsorb it, the tubular maximum for glucose, glucose will show up in the urine. This glucose in the urine causes an osmotic diuresis.¹

The increases in filtered solute limits the capacity of the tubules to reabsorb water and sodium salts. This leads to dehydration and electrolyte loss.² Other electrolytes lost, besides sodium, are potassium, phosphorous, and magnesium. This comes about by the increase in serum levels due to cell breakdown, which is the body's compensation for lack of glucose for fuel, and their excretion via the kidney. Serum levels of potassium are generally normal or elevated in spite of the profound deficits. This is caused by the large amounts liberated into the blood from cellular breakdown at a rate faster than the kidneys can excrete and later by reduced renal function as a consequence of hypovolemia, hypotension, and renal vasoconstriction.³

In the absence of carbohydrates the first substrate utilized is fat. There is an extreme depression of fatty acid formation with the slowed rate of sugar metabolism. This is due to: 1) a lack of building stones (acetate); 2) a lowered supply of reduced coenzymes; 3) because glycerophosphate, normally derived from sugar breakdown, is lacking, fatty acids are not resynthesized into neutral fats.

¹Traisman, p.11.
²Ibid., p.12.
³Ibid.,
All of these events cause a net liberation of stored fat as free fatty acid. These are used in the body for energy production. A large amount of such fatty acids reach the liver. Metabolism in the liver ends in ketone production.

The long chained fatty acid is gradually broken down to the stage of aceto-acetyl CoA and/or Acetyl CoA. Condensation occurs between these molecules and B-hydroxy - B-methylglutaryl CoA. When this molecule splits aceto-acetic acid and acetyl CoA are formed. The liver has only a limited capacity to metabolize fatty acids (ketones). Therefore, aceto-acetic acid and its reduction product, B-hydroxybutyric acid, are released to the blood stream. Normally ketone bodies can be used by the extrahepatic tissue, mainly muscle, for energy. However, their production exceeds the ability of utilization and they accumulate in the blood.

Ketone bodies are strong organic acids. The buffer system of the body, primarily the bicarbonate-carbonic acid system, neutralizes the acid by displacing sodium from the bicarbonate. There is a progressive decrease in plasma bicarbonate concentration. As the buffer system becomes depleted, acidosis (ketoacidosis) results. To compensate for the falling pH the respiratory center is stimulated, breathing is increased, and CO₂ (acid) is blown off. This gives the characteristic rapid and deep (Kussmaul) breathing

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2. Ibid., p. 161.
3. Ibid.
seen in diabetic patients. This rapid breathing rate increases the amount of pulmonary water loss three or four fold.¹

When the body's compensating mechanisms fail, acidosis increases and the activity of the higher brain centers is depressed, leading to coma. Vascular shock results from fluid loss and a reduction in blood volume along with acidosis affecting the contractility of small blood vessels. Diabetic coma and death are the end result of untreated insulin deficiency. Before the introduction of insulin, the end result of all Juvenile Diabetes was death.² Figure 2 illustrates the fatty acid metabolism in Diabetes.

Proteins are also utilized for fuel in the absence of carbohydrate metabolism. Proteins are broken down faster than they can be resynthesized. Therefore, amino acids are released from muscle, brought to the liver, and there changed into urea. A negative nitrogen balance results from this synthesis. The carbon from amino acids forms glucose and some are transferred to ketones.³ These factors combine to produce a slower rate of growth and cachexia frequently seen in children with Diabetes.⁴ Figure 3 diagrams the pathophysiology of Diabetes.

¹Traisman, p. 13.
²Netter, p.160.
³Ibid.
⁴Weil, p.344.
Figure 2: A Diagram of Fat Metabolism in the Diabetic

CIRCULATION

Glucose

Glucose can not enter muscle or fat cells without insulin

ADIPOSE TISSUE

Fatty Acid

Albumin

Fatty Acid

Large amounts of fatty acids are taken up by the liver

LIVER

Acetyl CoA → Aceto-Acetyl CoA → Fatty Acid

↓

B-Hydroxy-B-Methyl Glutaryl CoA

↑

Aceto Acetic Acid

↓

Acetyl CoA

↓

B-Hydroxybutyric Acid

KIDNEY

Ketones

Lost in urine

LUNG

Hyperpnea

BRAIN

Coma

Ketones recombine with base of plasma

Acidosis

Netter, p.160.
Figure 3: Diagram of Pathophysiology of Diabetes.¹

**WEIGHT LOSS**

- **MUSCLE**
  - Protein
  - Amino Acid

- **FAT**
  - Triglycerides
  - Fatty Acids

**GLUCOSE**

- Glucose cannot freely enter muscle or fat cells without insulin
- ACCUMULATED IN BLOOD

- Amino acids
- LIVER
- Fatty acids

- Glucose
  - exceeds renal threshold
  - water loss
  - KIDNEY

- Glycosuria
- Ketoacidosis
- Polyuria
- Mineral Loss
- Nitrogen Loss

- Urea--increased output
- Ketones

- binds with sodium
- Na
- Ketones
- Ketoacidosis
  - BRAIN
  - Coma

---
¹Netter, p.160.
Cardinal Signs and Symptoms

These pathologic developments give rise to the four cardinal symptoms of Diabetes. Polyuria, or frequent urination, is due to the osmotic pull of the glucose in the urine preventing the reabsorption of water. Polydipsia (extreme thirst) is a compensation for the dehydration caused by polyuria. Weight loss is caused by the body's breaking down of fat and protein for energy. Finally, polyphagia (excessive hunger) is the body's compensatory mechanism for the state of starvation due to fat and protein breakdown.¹

Diagnosis

The suspicion of Diabetes is usually brought to the attention of a physician by one or more of the following situations: 1) a family history of Diabetes Mellitus (especially in close relatives, e.g. parents); 2) glycosuria; 3) symptoms suggesting diabetes without glycosuria; 4) a history of polydipsia, polyuria, recent weight loss, and enuresis, and subsequently glycosuria and hypoglycemia; 5) ketoacidosis and coma.² When Diabetes is suspected the physician will perform a complete history and physical examination to rule out the possibility of coexisting or complicating diseases. The family history that is taken must include the incidence of Diabetes in the family, the age of onset, whether it was associated with obesity, and

¹Luckmann, p. 1315.
²Graef, p. 397.
whether they were treated with insulin. The diagnosis is confirmed with tests that show glucose and acetone in the urine, elevated fasting blood sugar, an elevated postprandial blood sugar, and a glucose tolerance test with prolonged high levels of blood glucose.

Fasting blood sugar determination is useful in diagnosis of well advanced Diabetes. The fasting blood sugar levels may reach as high as 500 mg/100 ml and the patient may have surprisingly few others objective or subjective abnormalities. The normal range for fasting blood sugar is 65-100 mg/100 ml. If the fasting plasma glucose is over 120 mg/100 ml further examination is necessary.

A second test, the postprandial blood glucose test, gives more information than the fasting blood sugar levels. Blood samples are taken either one or two hours after the patient has eaten a meal containing about 100 gm of carbohydrate. Results of 160 mg/100 ml one hour after eating is considered diagnostic for Diabetes. Values of 120 mg/100 ml is suspicious for Diabetes and should be investigated.

The glucose tolerance test is the most sensitive test used. This test is not necessary in patients with a


4 Krupp, p. 722.

5 Widmann, pp. 421-422.
fasting blood sugar level of 140 mg/100 ml or higher, or a postprandial level of 180 mg/100 ml. For three days prior to the test the patient must have a high carbohydrate diet (minimum of 150 Gms.). After fasting for at least eight hours, a glucose load is given, the amount being determined by the patient's weight. Samples of blood and urine are taken in the fasting state and at one, two, and three hours after the glucose load is taken. The results of urine testing provides information about renal threshold, valuable in planning later therapy. Glucose levels considered diagnostic for Diabetes are as follows: 160 or 170 mg/100 ml whole blood (185-195 mg/100 ml serum) after one hour; 120 mg/100 ml whole blood (140 mg/100 ml serum) after two hours. One may also add the serum glucose levels after one, two, or three hours and Diabetes is diagnosed if the total exceeds 600 mg.\textsuperscript{1}

\textsuperscript{1}Widmann, p.422.
Section II: The Treatment

General Objectives

There are five main objectives in the management of Diabetes Mellitus. First, treatment is geared to maintain or establish optimum physical health, with emphasis on normal growth and development. Secondly, the control of metabolic alterations, namely hypoglycemia, ketoacidosis, abnormal serum lipid concentrations, and excessive glycosuria are avoided. Thirdly, the patient and his parents must be educated so that they have a complete understanding of the disease and their role in management. Fourth, there must be adequate and immediate control of ketoacidosis and coma. And finally, treatment is designed to prevent or delay the onset of complications. This section will deal with the three main approaches to treatment, insulin therapy, diet, and exercise.

Insulin

Juvenile Diabetes Mellitus requires a daily injection of insulin if the disease is to be managed effectively. Insulin is secreted by the beta cells in the islets of Langerhans, and is necessary in the regulation of the uptake, storage, and release of glucose, amino acids, and fatty

---

1Widmann, p.422.
2Graef, pp.397-398.
3Traisman, p.45.
There are numerous kinds of insulin classified into three groups, long-acting, intermediate-acting, and short-acting. Table 3 lists the kinds of insulin and general information about each type. NPH insulin is often used as the intermediate acting insulin; Lente may be used if a little longer acting insulin is required. Mainly, preadolescents are given a single dose of NPH or Lente before breakfast, which provides adequate control. Some patients require regular insulin in order to obtain control. Adol-

Table 3: Table of Insulins

<table>
<thead>
<tr>
<th>Insulin</th>
<th>Mixture compatible with:</th>
<th>Subcutaneous Injections</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Onset</td>
<td>Maximum Action</td>
</tr>
<tr>
<td>Rapid acting-</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Regular</td>
<td>All other</td>
<td>1/2-1 hr.</td>
<td>3-4 hr.</td>
</tr>
<tr>
<td>Semilente</td>
<td>Other Lentes</td>
<td>1/2-3/4 hr.</td>
<td>5-7 hr.</td>
</tr>
<tr>
<td>Intermediate acting-</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NPH</td>
<td>Regular</td>
<td>1/2-1 hr.</td>
<td>7-11 hr.</td>
</tr>
<tr>
<td>Lente</td>
<td>Regular, Lente</td>
<td>1-1 1/2 hr.</td>
<td>7-11 hr.</td>
</tr>
<tr>
<td>Globin</td>
<td>Regular</td>
<td>1-2 hr.</td>
<td>8-10 hr.</td>
</tr>
<tr>
<td>Long acting-</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Protamine Zinc</td>
<td>Regular</td>
<td>6-8 hr.</td>
<td>10-18 hr.</td>
</tr>
<tr>
<td>Ultra Lente</td>
<td>Regular, Lentes</td>
<td>5-8 hr.</td>
<td>22-26 hr.</td>
</tr>
</tbody>
</table>

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2 Traisman, p. 46.
escent patients are usually best controlled with a combination of NPH and regular insulin. There are some children, who require a large amount of insulin, who are better regulated if they take three-fourths of their required dose in the morning before breakfast, and one-fourth is given after supper. Regular insulin is used whenever there is an increase in the need for insulin. Long-acting insulin is not recommended for use in Juvenile Diabetes because hypoglycemia reactions often occur during sleep and therefore may be prolonged and severe.¹

The amount of insulin given the patient is based on the phase of Diabetes. Initially, if the newly diagnosed patient is suffering from moderate to severe ketoacidosis, regular insulin (rapid acting) is given for recovery from this state. John Graef M.D. and Thomas Cone M.D., in their book Manual for Pediatric Therapeutics give the following guidelines for determining insulin doses: The initial stat dose is roughly approximated at 1-2 units per kg of body weight. Then they recommend one unit/kg subcutaneously every one to two hours until the blood sugar falls below 300mg/100ml, along with diminishing ketouria and acidosis. Then the patient is put on a sliding scale where regular insulin is given every four hours, the amount depending on the urinary acetone and glucose levels. An example of this sliding scale given by Graef and Cone for children over four years of age is as follows: 2-4 units of insulin are given for each (+) for 3+ and 4+ glycosuria (results of Clinitest)

¹Graef, p. 399.
in the absence of acetone; 4-5 units of insulin for each (+) for 3+ and 4+ urinary sugar if acetone is present.1

The requirements for insulin in the stabilization period for the child with Diabetes are high. For about two months following this time, the requirement is low, then it gradually increases. Following this period of fluctuation insulin requirements may stabilize; it will, however, gradually rise with the growth of the child until the growth is complete. The body will require more insulin in periods of stress, such as infection, trauma, burns, surgery, and emotional upset. Insulin needs decrease with physical activity.2

All these variations in insulin requirements must be taken into account by the physician when determining the insulin dosage for his patient.

Diet

The main objectives for dietary treatment of the Juvenile Diabetic is to meet the basic nutritional requirements so that the individual may lead a normal life. The diet is planned specifically for the individual patient taking into account his weight, age, and activity level. The major restriction is on concentrated sources of carbohydrates.3

There are three major diets used in the treatment of Juvenile Diabetes: the weighed diet, the exchange diet, and the free diet.

1 Graef, p. 401.


The weighed diet, if prescribed by the physician, is a restricted diet where the amount of food eaten is limited in grams of protein, carbohydrate, and fat. Parents of the diabetic child must weigh the food on a gram scale, and the child is kept on a strict schedule. This diet is not used often due to the fact that it is almost impossible to use with an active growing child. He is always hungry and frequently cheats on his diet, sneaking food to satisfy his hunger.¹

Most clinicians now advise following an exchange diet. In this diet, foods are arranged in groups of exchanges. The foods in each exchange list have about the same sugar content allowing for variations of the patients diet and facilitating meal planning.² There are seven exchange lists; they are: 1) food allowed as desired; 2) vegetable exchange; 3) fruit exchange; 4) bread exchange; 5) meat exchange; 6) fat exchange; and 7) milk exchange. The physician determines the calorie requirements for the child and prescribes the number of choices to be made from each exchange. Any food in the same exchange list can be substituted for any other food on the same list. Snacks between meals are also prescribed and prepared from the list.³ Table 4 is an illustration of a typical meal plan.

¹Mash, p. 630.
²Lippincott, pp. 642-643.
³Ibid.
Table 4: Typical Meal Plan.¹

Meal Plan for: Carbohydrate; Protein; Fat; Calories;

Your Food for the Day

<table>
<thead>
<tr>
<th>Amount</th>
<th>Kind</th>
<th>Choose From</th>
</tr>
</thead>
<tbody>
<tr>
<td>2 cups</td>
<td>Milk</td>
<td>List 1</td>
</tr>
<tr>
<td>Any Amount</td>
<td>Vegetable Exchange A</td>
<td>List 2A</td>
</tr>
<tr>
<td>1</td>
<td>Vegetable Exchange B</td>
<td>List 2B</td>
</tr>
<tr>
<td>4</td>
<td>Fruit Exchanges</td>
<td>List 3</td>
</tr>
<tr>
<td>3</td>
<td>Bread Exchanges</td>
<td>List 4</td>
</tr>
<tr>
<td>2</td>
<td>Meat Exchanges</td>
<td>List 5</td>
</tr>
<tr>
<td>3</td>
<td>Fat Exchanges</td>
<td>List 6</td>
</tr>
</tbody>
</table>

Divide this food as follows:

Breakfast:
1 Fruit Exchange
1 Bread Exchange
1 Fat exchange

Lunch or Supper:
1 Bread Exchange
3 Meat Exchanges
1/2 cup skim Milk

Mid Afternoon: 1 Fruit Exchange

Dinner or Main Meal:
1/2 Bread Exchange
4 Meat Exchanges
1 Vegetable A Exchange

Bedtime Meal: 1/2 cup skim Milk
1/2 Bread Exchange

The third type of diet used in the treatment of Diabetes is the free diet. When this diet is prescribed the child is allowed to eat a normal diet with the only restrictions on concentrated carbohydrates (ice cream, cake, cookies, etc.). This diet requires the child to learn how much he can eat and what he can eat; thus he controls his own diet, and at the same time he can eat when he is hungry.² Also, insulin doses may need to be adjusted each day using an insulin coverage plan.

¹Luckmann, p. 1324.
²Mash, p. 630.
based on activities and clinitest and acetest results. The goal of this diet is to avoid ketouria but allow some glycosuria.  

The free diet has the advantage that psychological problems and negativistic attitudes toward food are not as frequent when the child is on this diet. There is a demoralizing effect for an active child to be accused of cheating on his diet when his body is craving for more food. The child is hungry, but to satisfy his hunger, he is going against medical restrictions and the expectations of his physician and parents. This can have a long term effect on the child's self esteem.

Those who advocate the restricted diets believe that the excess sugar in the blood stream and spillage into the urine is harmful to the patient and leads to the development of complications. On the other hand, those who believe in the free diet feel that excess sugar is not harmful and with fewer restrictions there is less psychological stress put on the child.

In the book *Current Medical Diagnosis and Treatment*, the authors cite studies which showed no evidence that an increased amount of carbohydrates in the diet caused a deterioration of control, especially when the carbohydrates were in the form of bread, potatoes, or rice instead of simple sugars, as long as the total calories are limited. Vaisrub,  

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1 Hamilton, p. 322.  
2 Mash, p. 630.  
4 Hamilton, p. 322.  
5 Krupp, p. 723.
in his article "Dietary Prudence", reviewed studies on this subject and concluded that levels of serum cholesterol and triglycerides were unaffected by the change in ratio of carbohydrates in the diet.¹

The choice of the type of diet used in the management of Juvenile Diabetes is still dependent upon the physician. The exchange diet is widely used today; however, new evidence concerning the free diet may lead to a swing in that direction.

Exercise

Diabetes Mellitus does not restrict the child from participating in normal activity for his age. On the contrary, the child is encouraged to engage in physical activity.² Exercise promotes the metabolism and utilization of carbohydrates, thus lowering the insulin requirements of the body and enhancing the effects of insulin. Exercise should be regulated and approximately equal each day.³ Moderate exercise in a routine manner coupled with a balance of the size and frequency of meals can stabilize the insulin dosage in diabetics who slip out of control easily. Strenuous exercise, however, could cause a hypoglycemic reaction in an unprepared patient. Diabetics must be taught to prepare for strenuous exercise by decreasing their dose of insulin or taking supplemental carbohydrate.⁴

²Traisman, p. 74.
³Lippincott, p. 644.
⁴Krupp, p. 727.
Section III:
Nursing Care, Observations, and Complications

The nurse providing continuous care for a child with Diabetes, while the child is hospitalized, is responsible for observing the child's reactions to treatment and education of the child and his parents about the disease. Education will be discussed in Section IV. This section will deal with nursing observations and care.

Urine Testing

The urine is a major indicator of the diabetics condition. Both glucose and ketone bodies appear in the urine when they are at high levels in the blood. Therefore, results of urine tests are a major indicator of the patient's response to treatment.

The normal blood sugar level is 90-110mg/100 ml of blood. In a diabetic individual, however, insulin is not present in sufficient quantity, and glucose accumulates in the blood and eventually spills over into the urine. The normal renal threshold for glucose is 160-180 mg/100 ml of blood.\(^1\) Ketone bodies, normally metabolized by the body, also build up in the blood due to their rapid production, and these ketones are filtered out by the kidneys. Determinations of the amount of glucose and ketones in the

\(^1\)McFarltane, p. 1361.
urine gives indications of the diabetics condition.

The most often used test for glucose is the Benedict's Test. In this test sugar reduces blue alkaline cupric sulfate to red cuprous sulfate. This is, therefore, measuring the reducing activity of the substance being tested (urine) not glucose specifically. Other substances in the urine which may reduce copper sulfate are fructose, lactose, creatinine, uric acid, salicylates, and nomogentistic acid. Also drugs or contaminants, such as ascorbic acid or some antibiotics, may cause a positive reaction.¹

The Clinitest is a modification of Benedict's Test providing a quick, easy, and semiquantitative estimation of the amount of glucose in the urine. To show a trace reaction (green) it is necessary to have .25mg glucose/100ml urine, and the colors produced by oxidation progress to orange. The brick red color indicates a glucose concentration of 2 percent or more. A modification of the test allows the estimation of glucose levels up to 5 percent.²

Other tests employed to test urine glucose involve a strip and a chemical glucose oxidase which reacts with glucose producing gluconic acid and hydrogen peroxide. The hydrogen peroxide then induces a color change in a color indicator such as orthotoluidine. The color change indicates the concentration of glucose. Hydrogen peroxide or bleach in the urine will give a false positive reaction

¹Widmann, p. 233.
²Krupp, p. 721.
with this test. Ascorbic acid will delay or even abolish color development. However, other reducing agents have no effect on this test.1

To test for ketones in the urine reagent strips with nitroprusside and glycine are used. These chemicals react with ketones to produce a color change over the range from 5-10 mg/100 ml (trace), 20-30 mg/100 ml (moderate), and 60 mg/100 ml or above (large or severe). Symptoms of ketosis occur at levels of 50 mg/100 ml or above, and levels of 20-30 mg/100 ml signal moderately serious metabolic imbalance.2

It is the nurses job to monitor the results of the urine test results and record these results since they are indicative of the child's condition and effectiveness of treatment. Urine tests are to be done four times a day: in the morning, before breakfast, before lunch, late afternoon, and before going to bed. These test results are also used by the physician to determine the correct insulin dosage for his patient, and they are warnings of diabetic acidosis and hypoglycemia.

**Hypoglycemia**

When the child is started on insulin therapy there exists the possibility of a hypoglycemic reaction. It is the nurses responsibility to observe for signs and symptoms of this condition and help prevent its development.

1Widmann, p. 234.
2Ibid.
Hypoglycemia, also known as insulin shock or hyperinsulinism, is a result of too much insulin in the bloodstream. The condition is characterized by a rapid onset, hypoglycemia, irritability, mood swings, hunger, sensation of weakness or dizziness, sweating, nervousness, tremors, pallor, and rapid pulse. Younger children may have temper tantrums and the older child may complain of hunger, double vision, headache, epigastric pain, and tremors. If these symptoms are not picked up early and treated properly the patient may become semiconscious, then progress to convulsions, coma, and death.\(^1\) Frequent hypoglycemic reactions, particularly when convulsions also occur, can damage the central nervous system of the child.\(^2\) The condition is caused by an overdose of insulin, insufficient food intake, or violent exercise without taking an extra amount of carbohydrates.\(^3\) Table 5 lists the stages of hypoglycemic reactions with the related signs and symptoms.

If a mild hypoglycemic reaction occurs near a mealtime it can best be treated by giving the child the amount of milk he would receive on his meal tray, providing he is conscious and not vomiting. If the patient has a reaction within twenty minutes of his meal he should be served his tray.\(^4\) If hypoglycemia occurs between meals the patient

\(^1\) Mash, p. 631.
\(^2\) McFarlane, p. 1361.
\(^3\) Mash, p. 631.
\(^4\) Ibid.
Table 5: Stages of Hypoglycemic Reaction and Associated Signs and Symptoms.

<table>
<thead>
<tr>
<th>TYPE</th>
<th>PATHOPHYSIOLOGIC CHANGES</th>
<th>CLINICAL MANIFESTATIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild</td>
<td>Diminished glucose for cerebral function.</td>
<td>Behavior changes, inattention, in school (daydreaming), subtle changes in character noted by the family, hyperactivity</td>
</tr>
<tr>
<td></td>
<td>Acute increased systemic epinephrine</td>
<td></td>
</tr>
<tr>
<td>Moderate</td>
<td>Decreased cerebral arteriovenous differences of both glucose and oxygen</td>
<td>Headache, mental confusion, and visual disturbance</td>
</tr>
<tr>
<td>Severe</td>
<td>Hypoxia and markedly decreased glucose for cerebral function</td>
<td>Convulsions, coma</td>
</tr>
</tbody>
</table>

should be given some form of glucose orally (orange juice, candy, lump of sugar, or corn syrup), if the patient is conscious. If the patient is not conscious or unable to take anything by mouth, give glucagon subcutaneously or intramuscularly. Glucagon causes glycogenolysis in the liver which releases glucose into the blood stream. Then give this patient orange juice or ginger ale as soon as he regains consciousness.

If the patient is unconscious for a period of time, he should be given 50% glucose solution I.V. to restore the normal blood glucose level quickly. This is followed by a 5-10% glucose in water solution. Mannitol may be administered to combat cerebral edema if present. After a rapidly absorbed carbohydrate is given a feeding of protein and fat

1 Graef, p. 402.

2 Lippincott, p. 647.
should follow.¹

The nurse is responsible for prevention and early
detection of hypoglycemic reactions, and in teaching the
child and his family to do the same. The child must re-
ceive his meal tray and between meal snacks at the designated
time. Delaying in serving a meal or snack could precipitate
a hypoglycemic attack. The dosage of insulin to be given
should be checked with the physician's orders and the amount
drawn up in the syringe checked by another registered nurse
or qualified individual before the injection is given. The
insulin injection should be given at the same time each day
and promptly recorded. The nurse must also be aware of the
urine test results as these are indicators of the patient's
condition. Also she must observe the patient for signs
and symptoms of hypoglycemic reaction. All observations
made by the nurse should be entered on the patient's chart
so that any cycles or trends in the patient's maintenance
can be discerned.

Ketoacidosis

This is also referred to as diabetic coma. It is
caused by a lack of insulin which results in the disturbance
of carbohydrate, fat and protein metabolism, dehydration
and electrolyte imbalance. There is also an increase in the
number of ketone bodies in the blood due to the rapid break-
down of fats. This reaction can be caused by a failure to
take insulin, not enough insulin, or resistance to insulin.

¹Lippincott, p. 647.
Other causes include ingesting more than prescribed in the diet, infections, vomiting, diarrhea, and physical stress.¹

Early manifestations of this condition include the following: 1) Polyuria which is a result of the large amounts of glucose, ketones, and protein being lost in the urine creating an osmotic pull on water resulting in diuresis; 2) Thirst is a result of the cellular dehydration produced by polyuria; 3) Nausea results from electrolyte imbalance due to glycosuria and ketouria, and vomiting the cause being unknown; 4) Dry mucus membranes, cracked lips, and hot flushed skin is the result of severe dehydration and acidosis; 5) Abdominal pain is possibly associated with dehydration or sodium deficiency.²

If the condition is left untreated additional symptoms will develop. These symptoms are: 1) Kussmaul respirations is the result of the lungs trying to blow off the overload of acetone and excess carbon dioxide which the body buffer system can no longer neutralize; 2) Acetone (sweet) odor of the breath is due to the excess acetone being excreted via the lungs; 3) Hypotension and shock are results of profound dehydration which eventually lead to hypovolemic shock and circulatory collapse; 4) Oliguria or anuria is a dreaded complication arising from severe dehydration and shock, the decreased amount of blood volume lessens the blood flow to the kidney resulting in renal shutdown; 5) Coma or stupor is precipitated by electrolyte imbalance,

¹Lippincott, p. 649.
²Luckmann, p. 1336.
profound shock, and rapidly lowering pH.¹ When the patient is in acidosis his serum glucose is high while the CO₂ combining power is low. Plasma ketones are also present. The urine displays marked glycosuria and ketouria.²

Treatment for this condition is aimed at correcting acidosis, overcoming water and electrolyte losses, and giving the necessary amount of insulin. Normal saline solution is started intravenously, and regular insulin is given both subcutaneously and intravenously as ordered. Blood samples are taken to determine blood chemistry with tests repeated frequently to guide continued therapy. The rate of fluid infusion is decreased and sodium bicarbonate and other electrolytes may be added. Glucose may also be added to the intravenous solution before the blood sugar drops to a low level producing hypoglycemia.³

As in hypoglycemia, the nurse is responsible for observing the child's behavior and general appearance and urine test results for signs and symptoms of ketoacidosis. She is also responsible for the correct administration of prescribed insulin, and educating the patient about this condition and his role in its prevention.

Other Complications

Due to the fact that the child with Diabetes is less able to defend and repair itself, good general hygiene is

¹Luckmann, p. 1336.
²Lippincott, p. 649.
³Mash, pp. 630-631.
very important. While in the hospital the child should be protected from other patients with infectious diseases and from injury. The techniques for administration of insulin must be meticulous. The nurse must see that the child is bathed regularly, has good oral hygiene, and observe for any signs and symptoms of infection or injury.¹

Complications are uncommon in the child with Diabetes except when the child has been inadequately treated or has the disease for a long period of time. The following conditions may possibly appear in the diabetic child as a result of Diabetes: 1) lack of development; 2) stunted growth; 3) amenorrhea; 4) lack of development of secondary sex characteristics. Other physical conditions that may develop after years of having the disease are cataracts, gangrene, and arteriosclerosis. Infections are common, including skin infections, respiratory tract and urinary tract infections. Dental caries are also seen frequently. The course and prognosis of the disease depends upon the correct treatment and the maintenance of good control.²

¹Hamilton, pp. 324-325.
²Marlow, p. 660.
Case Study

The following is a case study of a Juvenile Diabetic admitted to Ball Memorial Hospital, Muncie, Indiana. The review of this patient's hospital stay will serve as an example of the information previously discussed.

Debbie is a twelve year old white female admitted to the hospital with a blood sugar of 850mg%. Debbie was seen by her physician several days prior to her admission with the chief complaints of fatigability and polyuria. She had no complaints of polydipsia of polyphasia. Her urinalysis showed a questionable positive, one Clinitest negative and a stick positive 1+. A two hour post prandial blood sugar was taken and was 850mg% with this data the physician put Debbie in the hospital.

Debbie has no history of serious illnesses or trauma. Her parents are in good health and there is no family history of congenital or hereditary diseases including Diabetes.

During her hospital stay she was taught about her disease and treatment. The nursing staff was responsible for the majority of the patient's education concerning her care. In this case the patient was taught how to test her urine, and draw up and inject her insulin. A dietitian spoke to Debbie and her mother about regulations ordered by the doctor and the exchange method of diet planning. The author of this paper discussed with the patient Diabetes, what it is, its causes, etc.; what the urine testing was for; insulin: what it is and how it worked, sites for injection; regulations
of the diet; signs and symptoms of both hypo- and hyperglycemia; and special skin care. A responsible family member was not taught to give Debbie insulin, before she was discharged, because it was overlooked by the staff.

The following Table 6 outlines Debbie's hospital stay: the care ordered by her physician, her lab results and Clinitest results, and her education that took place by the nursing staff.
Table 6: Outline of the Hospital Experience of Juvenile Diabetic

<table>
<thead>
<tr>
<th>Hosp. Doctors</th>
<th>Lab Reports</th>
<th>CT</th>
<th>Education</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Day</strong></td>
<td><strong>Orders</strong></td>
<td><strong>Reports</strong></td>
<td><strong>Education</strong></td>
</tr>
<tr>
<td>1</td>
<td>Stat blood sugar 1500 cal. ADA diet CT qid with coverage +1-5 +2-10 +3-15 +4-20 Daily weight Routine labs Chest X-ray</td>
<td>Urinalysis Sp gr-34 pH-6 clear yellow Hemoglobin-neg. Ketone-possible Biliruben-neg. RBC-0 WBC-2-3 Epithelial-occ Casts-0 Slight mucus Blood glucose-672</td>
<td>7:00p-4+ 11:00p-4+</td>
</tr>
<tr>
<td>2</td>
<td>Lente insulin 12 units qam Electrolyte osmolarity Up ad lib</td>
<td>Blood WBC-10.3 RBC-4.93 Hgb-14.1 Hct-42.7 Serum osmolarity-286 Cl-105 Na-139 K-3.0</td>
<td>7:00a-4+ 11:00a-0 4:00p-0 8:00p-4+</td>
</tr>
<tr>
<td>3</td>
<td>Increase Lente insulin to 16u qam FBS in am</td>
<td></td>
<td>7:00a-4+ 11:30a-0 4:00p-0 8:00p-0</td>
</tr>
<tr>
<td>4</td>
<td>FBS 80mg Reduce insulin to 10u qam Increase diet to 1800 cal 3hr pprd BS afternoon Give 10u Lente 6u Semilente qam</td>
<td>7a FBS 80mg% 3p pprd 186mg%</td>
<td>11:00a-4+ 4:00p-0 8:00p-3+</td>
</tr>
<tr>
<td>5</td>
<td>4+ 11a CT with 3+ 8p Lente to 16u qam, Semilente 6u qam DC coverage</td>
<td></td>
<td>7:00a-0 11:00a-4+/N 4:00p-3+ 8:00p-0</td>
</tr>
</tbody>
</table>