Chapter 1: Diabetes: A Comprehensive Overview

5 Contact Hours

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Learning objectives

Upon completion of this course the learner should be able to:

- Identify the different types of diabetes and prevalence in the United States.
- Understand recommended screening guidelines, diagnostics tests, and the common signs and symptoms of the disease.
- Identify the nonmodifiable and modifiable risk factors for developing diabetes.
- Discuss the pathophysiology of diabetes as well as the psychological impact of a diagnosis for the patient and family.
- Compare and contrast the various treatment modalities of diabetes, (nutrition/diet, oral medications, insulin, pumps and surgical options).
- Identify and understands the role of the nurse caring for a patient along with the cultural conside3rations related to that care.
- Discuss the future of diabetes, including stem cell research

Introduction

Diabetes mellitus is a common, chronic, complex disease characterized by an insufficiency of, or the body's failure to properly use, insulin [78]. Proper management of diabetes requires a collaborative approach with the patient and family to make lifelong behavioral and lifestyle changes. The nurse has an enormous role in facilitating the treatment regimen and empowering patients and families to achieve maximum states of wellness. Regardless of a nurse's specialty, each of us has been responsible for the care of a patient living with diabetes. Although nurses may have a basic understanding of diabetes, it is important for them to understand the complexity of the disease and feel comfortable in their knowledge of it to ensure that they give patients the right information to control their diabetes and to avoid serious complications.

Diabetes is the world's most prevalent metabolic disease and it is the leading cause of adult blindness, renal failure, gangrene and the necessity for limb amputations [58,63]. Since 2007, diabetes has reached epidemic proportions worldwide, making it the seventh-leading cause of death in the United States [58]. Growing numbers of people are dealing with acute and chronic complications, disability and death due to the diabetic disease process.

Prevalence of diabetes in the United States and worldwide

In 2011, the National Diabetes Information Clearinghouse (NDIC) and the American Diabetes Association (ADA) provided the following prevalence statistics regarding diabetes [19,58]:

Diabetes in the United States

There are about 25.8 million children and adults (8.3 percent of the total United States population) living with diabetes. This included 18.8 million people diagnosed and 7 million who were not diagnosed. Additionally, there are 79 million people diagnosed as prediabetic. In 2011 the National Diabetes Fact sheet used both fasting glucose and A1C levels to obtain estimates for undiagnosed diabetes and prediabetes since these are the most commonly used tests in clinical practice.

- Only a fraction of the total number of cases are found under the age of 20:
  - 215,000 or 0.26 percent of all people in this age group have diabetes.
  - About one in every 400 children and adolescents has type 1 diabetes.
- The majority of diabetes is found in patients over the age of 20:
  - **Age 20 or older**: 25.6 million (11.3 percent) of all people in this age group have diabetes.
  - **Age 65 or older**: 10.9 million (26.9 percent) of all people in this age group have diabetes.
  - **Men**: 13 million (11.8 percent) of all men age 20 or older have diabetes.
  - **Women**: 12.6 million (10.8 percent) of all women age 20 or older have diabetes.
  - **Non-Hispanic whites**: 15.7 million (10.2 percent) of all non-Hispanic whites aged 20 years or older have diabetes.
  - **Non-Hispanic blacks**: 4.9 million (18.7 percent) of all non-Hispanic blacks aged 20 years or older have diabetes.

Prediabetes in the United States

Pre-diabetes is a condition characterized by blood glucose or A1C levels that are higher than normal but not high enough to be called diabetes. Persons who have pre-diabetes are at increased risk for developing type 2 diabetes, heart disease, and

stroke. Research shows that pre-diabetics who lose weight and increase their physical activity can actually prevent or delay the development of type 2 diabetes and may even, in some cases, return their blood glucose levels to normal [58].

- In 2005-2008 (based on fasting glucose or A1C levels) 35 percent of adults in the United States ages 20 years or older had pre-diabetes. Fifty-percent of these individuals were 65 years of age or older. "Applying this percentage to the entire U.S. population in 2010 yields an estimated 79 million Americans ages 20 years or older with pre-diabetes." [58].
- Based on fasting glucose or A1C levels, and after adjusting for population age differences, the percentage of American adults ages 20 years or older with pre-diabetes in 2005-2008 was similar for non-Hispanic whites (35 percent); non-Hispanic blacks (35 percent) ; and Mexican Americans (36 percent) [58].

Gestational diabetes

Gestational diabetes has been reported in 2 percent to 10 percent of pregnancies in the United States. Immediately following their pregnancies, 5 percent to 10 percent of women with gestational diabetes are found to have diabetes (usually type 2).Those women who had gestational diabetes have a 35 percent to 60 percent chance of developing diabetes in the next 10-20 years [58].

Diabetes and death in the United States

According to the Diabetes Research Institute and the Centers for Disease Control and Prevention (CDC), diabetes reduces life expectancy by one-third [37]. Based on the most recent available statistics, diabetes was the seventh leading cause of death based on U.S. death certificates in 2007. This conclusion is based on the 71,382 death certificates in 2007 in which diabetes was the underlying cause of death. Diabetes was a contributing cause of death in an additional 160,022 death certificates. In general, the risk for death among people with diabetes is about two times that of people of similar age but without diabetes [58].

Cost of diabetes in the United States

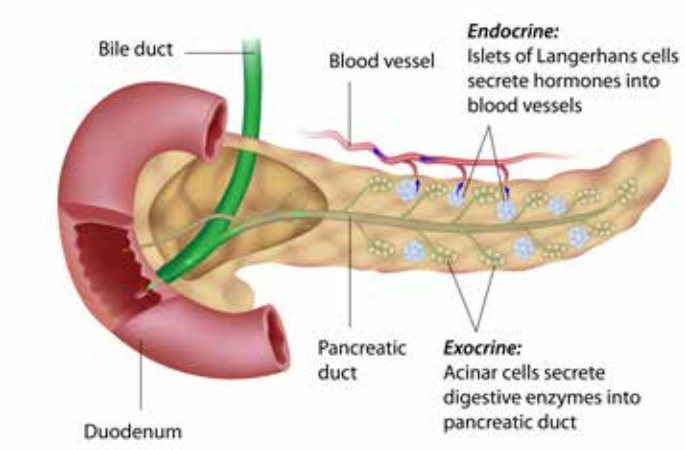
Diabetes is a major public health concern and debt to the U.S. population. In 2007, the most recent year for which statistics are available, the total (direct and indirect) cost in the U.S. was 174 billion. However, according to the Diabetes Research Institute, diabetes costs the American people an estimated $218 billion each year [37,58]. This includes:

- **Direct medical costs:** $116 billion in 2007; up from 91.8 billion in 2002 [19]. After adjusting for population age and sex differences, average medical expenditures among people with diagnosed diabetes were 2.3 times higher than what expenditures would be in the absence of diabetes.
- **Indirect costs:** $58 billion; up from 39.8 billion in 2002 [19]. Indirect costs compromise the amount spent on disability, work loss and premature mortality.

Even though the statistics are astonishing in the U.S., it is more profound globally. According to the World Health Organization (WHO), more than 220 million people worldwide have diabetes. [76] The WHO (2011) released the following data as well [76]:

- More than 80 percent of deaths due to diabetes occur in low-and middle-income countries.
- WHO predicts that diabetes deaths will double between 2005 and 2030.
- Healthy diet, regular physical activity, maintaining a normal body eight and avoiding the use of tobacco products can prevent or delay the onset of type 2 diabetes.

Understanding the role of the pancreas and diabetes

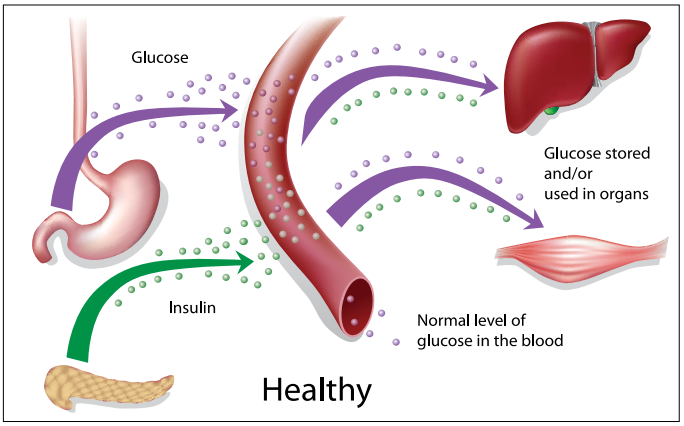


The pancreas is a banana-shaped organ that lies behind the stomach, with the head and neck extending into the curve of the duodenum and the tail lying against the spleen. The pancreas has endocrine and exocrine capabilities [19,79]:

- **Endocrine function** involves the islets of Langerhans, microscopic structures that are responsible for two major hormones, glucagon (secreted by the alpha cells) and insulin (secreted by the beta cells) that have an enormous effect on diabetes.
  - **Glucagon** is a hormone that increases blood glucose levels when the blood sugar is low.
  - **Insulin** is a hormone that stimulates growth and promotes the movement, storage and metabolism of carbohydrates, protein and fat. Insulin plays a role in lowering the blood glucose levels by allowing glucose to move across the cell membranes into many tissues.
- **Exocrine function** is responsible for the digestive enzymes excreted to facilitate the eating process. Although the exocrine function of the pancreas plays an enormous role in excreting various digestive enzymes every day, it does not have any relation or effect to diabetes.

Normal physiology in a non-diabetic individual

When an individual eats or drinks something, the body responds by raising the blood sugar to provide energy and nutrients to the cells and organs. Carbohydrate foods provide most of the glucose absorbed and used by the body; proteins and fats provide smaller amounts [75]. According to the ADA, a patient without diabetes will maintain a fasting blood glucose level between 70-99 milligrams/per deciliter (mg/dl) and less than 140 mg/dl postprandial (two hours after eating) [2]. Our bodies cannot differentiate the type of glucose ingested, but after eating any meal or drinking, the blood sugar normally rises, often to between 120 and 130 mg/dl, but generally not above 140 mg/dl [52]. Once the blood sugar rises, the pancreas will respond by releasing insulin to help control the metabolism of the carbohydrates ingested.



Insulin secreted via the pancreas "opens the doors" of the cells throughout the body by binding to insulin receptors on the cell membranes. Once the doors open, insulin travels immediately to the liver via the bloodstream. The pancreas secretes approximately 40 to 50 units of insulin daily into the liver in a two-step manner [19]:

- Low levels during fasting (basal insulin secretion).
- Increased levels 10 minutes after eating (prandial). Insulin will continue to be released periodically if hyperglycemia persists.

Once the insulin is in the liver, it promotes the production and storage of glycogen (long chains of glucose) [19]. Glycogen is required to ensure that a normal, therapeutic blood glucose level is maintained throughout the day. Therefore, if an individual ingests a large amount of food, the excess glucose will be converted into glycogen and stored within the liver and muscles.

During a hypoglycemic event (blood glucose 70 mg/dl) brought on by an individual choosing to skip a meal or choosing to ingest a smaller portion, the body responds by mobilizing glucose into the bloodstream and cells from the stored glycogen to raise the blood glucose levels [19]. At the same time, another major hormone, glucagon, allows glucose to be released from glycogen as needed from the storage sites (predominantly within the liver and muscles) whenever the blood glucose levels are low [19]. The glucagon will then mobilize the glucose from the storage sites to increase the concentrations of glucose in the bloodstream. It is imperative that the pancreas and hormones respond appropriately to prevent complications and a lack of glucose and nutrients from getting to organs and tissues.

Pathophysiology in a diabetic patient

In the diabetic patient, the glucose is unable to "unlock" the cell and enter into the body's cells, allowing it to stay in the bloodstream and induce a hyperglycemic state that denies the cells their source of energy [75]. Without therapeutic levels of glucose, the body cannot function adequately because glucose is the main fuel for the central nervous system (CNS). The brain cannot store glucose; therefore it requires a continuous supply. The body attempts to compensate for the insufficiency, however. When the cells are unable to absorb the glucose, they rely on fat and protein for energy. Fat (in the form of triglycerides) and proteins should only be utilized as "reserves" for fuel and not used as energy under normal conditions [19]. If the body continues to use fat and protein for the source of energy, the cells break down, inducing a form of emaciation, muscular atrophy and weakness [63,79].

Diabetes also causes insulin to either not be produced (Type 1 diabetes) or production to be decreased (Type 2 diabetes). In either instance, without an adequate supply of insulin, glucose is unable to properly move in the bloodstream to the tissues and organs, further exacerbating the cells that are starving for energy and nutrients for survival [2]. Over time, the patient will exhibit various symptoms due to the buildup of glucose in the bloodstream and lack of insulin production (See Signs and symptoms of diabetes).

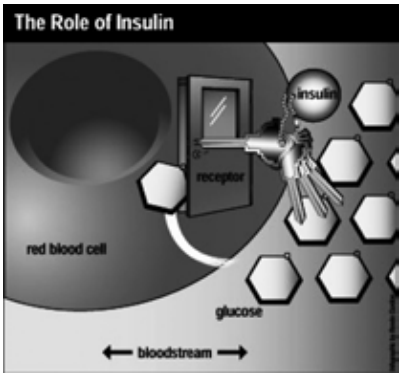


Diagram 1 below (Role of Insulin)

Diagram 1: Carbohydrates that we eat make our blood glucose rise. To utilize the carbohydrates and lower the blood sugar, insulin opens the doors of the body's cells to glucose circulating in the blood. The glucose enters the cells and is used as the cells' fuel for energy. Insulin binds to a spot on the cell surface called a receptor. Likened to a lock and key, insulin is the key that opens up the lock (receptor) so that glucose can pass through the door into the cell. Using this analogy in type 1 diabetes, the keys have been stolen because no insulin is produced (the pancreas does not make the insulin). With type 2 diabetes, the door will not open fully even with the right key, which is known as insulin resistance. Source: [52].

Type 1 diabetes

According to the ADA and the CDC, type 1 diabetes is classified in one of the following categories [5, 31, 48]:

Type 1 diabetes was previously referred to as insulin-dependent diabetes mellitus (IDDM), juvenile-onset diabetes, ketosis prone diabetes, brittle diabetes and idiopathic diabetes. Type 1 diabetes is a multifactorial disease caused by an autoimmune destruction of insulin-producing pancreatic beta cells. It typically develops when the body's immune system destroys the pancreatic beta cells and insulin is no longer produced in response to the individual's ingestion of food. It is speculated that type 1 diabetes is caused by a family history, women who have had gestational diabetes, the pancreas attacking itself following certain viral infections or after the administration of certain drugs (autoimmune response) [75]. Other risk factors include the following:

- **Genetics** plays an enormous role in predisposing individuals to develop diabetes. In Type 1, family members are at risk of developing diabetes throughout their life. Researchers have identified genetic markers that determine immune responses, specifically DR3 and DR4 antigens on chromosome number six of the human leukocyte antigen (HLA) system, amongst 95 percent of people diagnosed with type 1 diabetes [25, 53]. The inherited antibodies can be detected in the blood years before the development of any clinical symptoms [25]. In the general population without any genetic predisposition, an individual has a one in 400 to one in 1,000 risk; however, a child of a diabetic patient has a one in 20 to one in 50 risk [51]. Interestingly, the offspring of a mother with type 1 diabetes has a 3 percent risk that increases to 6 percent if the father is affected [53]. The risk increases tremendously among identical twins, up to 25 to 50 percent [53].
- **Age**. Type 1 diabetes can occur at any age; however it is more common in children and young adults, typically under 40 years of age. Type 1 diabetes may account for 5 to 10 percent of all diagnosed cases of diabetes [53].

Type 2 diabetes

Type 2 diabetes was previously referred to as noninsulin-dependent diabetes mellitus (NIDDM) or adult-onset diabetes. Type 2 diabetes begins as insulin resistance, a disorder in which the cells do not utilize the insulin produced by the pancreas properly. As the need for insulin rises, the pancreas gradually loses its ability to produce insulin. In type 2, the body either does not make enough insulin (insulin deficiency), something interferes with the action of the insulin that is made by the pancreas (insulin resistance) or there is an increase in the hepatic glucose output [21]. Type 2 diabetes is the most common form nurses will care for in their career.

Type 2 diabetes may account for about 90 to 95 percent of all diagnosed cases of diabetes. Type 2 diabetes is associated with older age, obesity, family history of diabetes, history of gestational diabetes, impaired glucose metabolism, physical inactivity and race/ethnicity – African Americans, Hispanic/Latino Americans, American Indians and some Asian Americans and Native Hawaiians or Other Pacific Islanders are at particularly high risk for type 2 diabetes [31]. The prevalence of type 2 diabetes among African Americans is 1.6 times higher than that of the total U.S. population [50]. It is speculated that African Americans are at a higher risk because of correlating obesity and hypertension rates, especially among African American women [50].

Type 2 diabetes is increasingly being diagnosed in children and adolescents due to genetics, a significant incline in childhood obesity and decreased activity [25, 75].

The patient with type 2 diabetes typically presents as an obese patient with a family history of diabetes and a recent stressor, such as a death of family member, illness or loss of a job [75].

Although the origin is unknown, research has demonstrated that type 2 diabetes is influenced by genetics along with the combination of environmental factors. Type 2 diabetes is very complex and not all that well defined because there have been numerous susceptibility genes identified. The pathogenesis of type 2 diabetes revolves around genes that either influence viability or cellular responses to insulin or beta cell function or both [48].

- **Genetics**. Genetics plays an enormous role in the development of diabetes (90 percent). However, in contrast to type 1 diabetes, there is no identified HLA link.The maturity-onset diabetes of the young (MODY) is thought to be an autosomal dominant as it effects 50 percent of first-degree relatives. At this time, there are at least six types of MODY, and it is considered to be a subset of type 2 diabetes. Each specific type of MODY is caused by a specific mutation in the enzyme that is involved in the beta cell function and insulin action. For example, MODY3 develops after a mutation occurs in the hepatocyte nuclear factor alpha-1, and MODY2 can be attributed to a defect in the glucose-sensing ability (glucokinase mutation). Since only 2 to 5 percent of cases of diabetes are monogenic, they are classified as MODY [48].

  The offspring of people with type 2 diabetes have a 15 percent chance of developing type 2 diabetes and a 30 percent risk of developing glucose intolerance (inability to metabolize carbohydrates normally).
- **Race/ethnicity**. Type 2 diabetes is prevalent in half of all black and Hispanic children and in over two-thirds of American Indian children [46].
- **Environment**. Environmental triggers can be exacerbated by exposure to a viral infection (mumps, rubella or coxsackievirus B4) and chemical toxins (smoked and cured meats) [53].
- **Modifiable risks**. There are certain risks that patients have the power to potentially change and control in their life, such as:
  - **Obesity**. Obesity is defined as being at least 20 percent over the recommended body weight for an individual's

height and weight or having a body mass index (BMI) of at least 27 kilograms per meter squared (kg/m2) [53]. When an individual is obese, it hinders the ability of glucose to enter the liver, adipose tissue and skeletal muscle [48]. Intra-abdominal obesity is the single most important risk factor in determining who is at risk of developing type 2 diabetes. Research has demonstrated that severe obesity creates 10 times the risk of developing type 2 diabetes. In addition, having an excessive caloric intake contributes to obesity and predisposes an individual to type 2 diabetes [48].

The fat accumulation in nonadipose tissue (ectopic fat) is very common in insulin resistance and type 2 diabetes. The accumulation of lipids in the islets can be attributed to the impairment of insulin secretions, and insulin resistance has been attributed to excess fat in the muscle. Nonalcoholic steatohepatitis is the infiltration of fat within the liver and may result in cirrhosis and hepatic failure. Very little is understood about the pathogenesis of ectopic fat, but overnutrition is clearly the main culprit. Pancreatic fibrosis, which occurs in 33 to 66 percent of type 2 diabetics, can also contribute to the loss of beta cell function.

- **Physical inactivity**. The Diabetes Prevention Program, DPP) a prevention study of persons at significant risk for diabetes, found that losing weight and increasing physical activity reduced the development of type 2 diabetes by 58 percent during a 3-year period. The reduction was even greater (78 percent) in adults 60 years of age and older [31].
- **Hypertension** (greater than 130/85 in adults), High density lipidproteins (HDL) cholesterol less than 35 mg/dl and/or a triglyceride level greater than 250 mg/dl. [78].

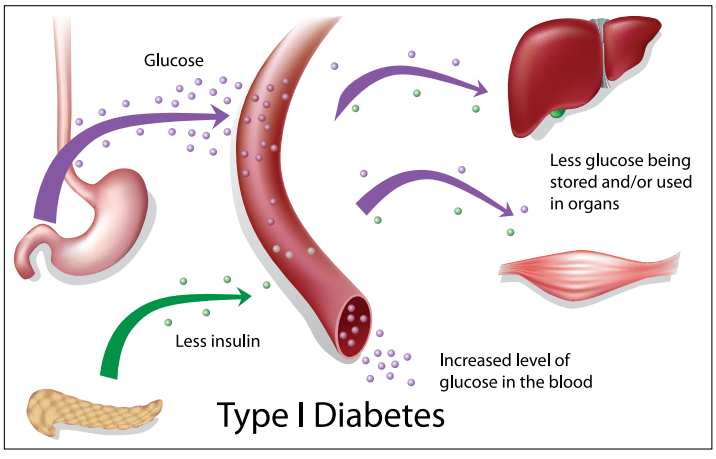
- **Insensitivity to insulin**. Interestingly, patients living with or at risk for diabetes typically have a cellular resistance factor 60 to 80 percent of the time. Resistance to insulin also increases with obesity and a condition called metabolic syndrome. An integral part in the pathogenesis of type 2 diabetes and metabolic syndrome is the decrease in beta cell responsiveness to the plasma glucose levels as well as abnormal glucagon secretions. (See the subsequent section for further explanation).

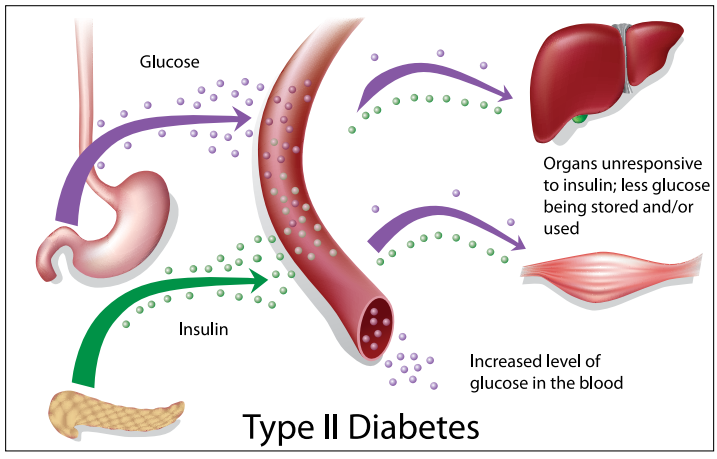
Pre-diabetes and insulin resistance (metabolic syndrome)

Type 2 diabetes does not just occur overnight; often a patient will have one of the following conditions before being diagnosed with diabetes [29, 57]:

1. **Prediabetes** is a condition in which a patient will have a higher-than-normal blood glucose level but not high enough to be classified as diabetes. People with prediabetes have an increased risk of developing type 2 diabetes, heart disease and a stroke. Prediabetes is a condition that needs to be assessed and monitored by health care professionals because of the seriousness and the risk of developing complicating diseases. As noted, 79 million Americans ages 20 years or older are estimated to have pre-diabetes." [58].
   People with prediabetes have impaired fasting glucose (IFG) and/or impaired glucose tolerance (IGT) [19, 29, 52]:
   - IFG is a condition in which the fasting blood sugar level is 100 to 125 milligrams per deciliter (mg/dL) after an overnight fast. This level is higher than normal but not high enough to be classified as diabetes.
   - IGT is a condition in which the blood sugar level is 140 to 199 mg/dL after a two-hour oral glucose tolerance test (OGTT). This level is higher than normal but not high enough to be classified as diabetes.
   - An A1C of 5.7 percent to 6.4 percent indicates an increased risk of type 2 diabetes and is diagnostic for prediabetes [80].

   Various research studies and the Diabetes Prevention Program (DPP) have demonstrated that most people with prediabetes develop type 2 diabetes within 10 years unless they make the following lifestyle changes [29, 57]:
   - **Decrease weight.** Lose 5 to 7 percent of their body weight, approximately 10 to 15 pounds for someone who weighs 200 pounds. Losing just 5 to 7 percent of body weight prevents or delays diabetes by nearly 60 percent. The body mass index (BMI) chart is a measurement of body weight relative to the individual's height. Adults aged 20 or older can use the BMI table





below to find out whether they are normal weight, overweight, obese or extremely obese.
It is important to note that the BMI table has certain limitations as it may overestimate body fat in athletes and others who have a muscular build and underestimate body fat in older adults and others who have lost muscle. BMI for children and teens must be determined based on age and sex in addition to height and weight. Information about BMI in children, teens and adults, including a BMI calculator, is available from the Centers for Disease Control and Prevention (CDC) at www.cdc.gov/nccdphp/dnpa/bmi.

- **Diet changes.** Change their diet by decreasing the total calories and cutting out the total fat intake to reduce their risk of complications (such as heart disease).
- **Physical activity.** Increase their level of physical activity, such as walking 30 minutes a day five days a week. Physical activity helps muscle cells use blood glucose for energy by making the cells more sensitive to insulin.

In the DPP study, people aged 60 or older who made lifestyle changes lowered their chances of developing diabetes by 70 percent. Many participants in the lifestyle intervention group returned to normal blood glucose levels and lowered their risk for developing heart disease and other problems associated with diabetes. The DPP also showed that the diabetes drug Metformin reduced the risk of developing diabetes by 31 percent (See the section Medication Modalities for the Type 2 Diabetic for further discussion about Metformin.)

It's important for health care professionals to urge their patients to be physically active, make wise food choices and reach and maintain a healthy weight.

2. **Insulin resistance** (commonly referred to as metabolic X syndrome) is a condition in which the body produces insulin but does not use it properly. Over the past decade, there has been a lot of hype correlating diabetes, metabolic syndrome and cardiovascular disease. However, it should be noted that since the late 1960s, research has demonstrated a significant association among diabetes, hypertension, obesity and hyperlipidemia [1]. Then, in the early 1990s, researchers discovered that the same chronic cluster of disorders was caused by insulin resistance and concluded that "insulin resistance syndrome" (syndrome X and metabolic syndrome) was the appropriate name for this condition. At the same time, researchers discovered from the Framingham Offspring Study that a clustering of risk factors, including hyperinsulinemia, dyslipidemia, hypertension and glucose intolerance (rather than hyperinsulinemia alone), characterized the underlying features of the insulin resistance syndrome [1].

   Individuals who are insulin resistant are unable to respond appropriately to insulin, and as a result, their bodies need more insulin to help glucose enter cells. The pancreas tries to keep up with this increased demand for insulin by producing more, but eventually the pancreas can't keep up with the body's needs. So excess glucose builds up in the bloodstream, setting the stage for diabetes. Many people with insulin resistance have high levels of both glucose and insulin circulating in their blood at the same time.

   Insulin resistance increases the chance of developing type 2 diabetes and heart disease.

   A diagnosis of prediabetes or insulin resistance does not mean that diabetes and/or heart disease will definitely develop over the next few years. However, it should be looked upon as a wake-up call to make lifestyle changes to prevent or delay diabetes. According to the CDC (2008) [29]:

   - Progression to diabetes among those with prediabetes is not inevitable. Studies have shown that people with prediabetes who lose weight and increase their physical activity can prevent or delay diabetes and return their blood glucose levels to normal.
   - The DPP, a large prevention study of people at high risk for diabetes, showed that lifestyle intervention reduced developing diabetes by 58 percent during a three-year period. The reduction was even greater, 71 percent, among adults age 60 or older.
   - Interventions to prevent or delay type 2 diabetes in individuals with prediabetes can be feasible and cost-effective. Research has found that lifestyle interventions are more cost-effective than medications.

   Typically, patients with insulin resistance (metabolic X) and/or prediabetes do not exhibit any diabetic symptoms, and they may have one or both conditions for several years without noticing anything. People with a severe form of insulin resistance (metabolic X) may have a condition called acanthosis nigricans – dark patches of skin, usually on the back of the neck, elbows, knees, knuckles, and the armpits – an early sign of prediabetes. Nurses and health care providers need to be attuned to the major risk factors (similar to the development of diabetes). According to the NDIC (2008) and the ADA, the risk factors for prediabetes, insulin resistance (Metabolic X) and diabetes are typically the same [57].

   Research has demonstrated that 25 percent of the general nonobese, nondiabetic populations have insulin resistance at a magnitude similar to type 2 diabetes [53]. It occurs more frequently in men and Mexican Americans between the ages of 20 to 70. While metabolic syndrome may have a basis from a genetic standpoint, other influential environmental factors may be involved, including lack of exercise, excess nutrients and obesity [48]. Metabolic syndrome has a group of clinical traits that, when combined together, increase the risk for developing cardiovascular disease significantly. According to the American Heart Association (AHA) and the National Heart, Lung and Blood Institute, metabolic syndrome is diagnosed when a minimum of three of the following criteria are met [48]:

   - Elevated waist circumference (abdominal obesity). Increased abdominal adiposity (waist greater than 40 inches in men and greater than 35 inches for woman). The excess fat in the intra-abdominal area is a huge component of the metabolic syndrome. The majority of experts concur that the combination of obesity, obesity-related cytokines called adipokines, excess nutrients and inflammatory cytokines are the main contributors to beta cell death and insulin resistance in type 2 diabetes. Regardless of which event occurred, the mechanisms

that are responsible for insulin receptor binding or postreceptor can be reversed by weight loss [48].
    - Elevated triglycerides (TG) greater than 150 mg/dl.
    - Reduced HDL cholesterol (less than 40 mg/dl in men and less than 50 mg/dl for women).
    - Fasting blood glucose (Hyperglycemia) greater than 100 mg/dl).
    - Increased blood pressures (130/85 mm Hg or greater).

Research has also correlated other risk factors, such as physical inactivity, aging, hormonal imbalance and genetic predispositions [75].

Insulin resistance and prediabetes are diagnosed by one of the following laboratory tests [57]:

- **Fasting glucose test.** This test measures blood glucose in people who have not eaten anything for at least eight hours. This test is most reliable when done in the morning. Fasting glucose levels of 100 to 125 mg/dL are above normal but not high enough to be called diabetes, so that level is called prediabetes or IFG. People with IFG often have had insulin resistance for some time and are much more likely to develop diabetes than people with normal blood glucose levels.
- **Glucose tolerance test.** This test measures blood glucose after people fast for at least eight hours and two hours after they drink a sweet liquid provided by a doctor or laboratory. A blood glucose level between 140 and 199 mg/dL means glucose tolerance is not normal but is not high enough for a diagnosis of diabetes. This form of prediabetes is called IGT and, like IFG, it points toward a history of insulin resistance and a risk for developing diabetes.

For over a decade, the WHO, AHA and the National Cholesterol Education Program (NCEP) Adult Treatment Panel (ATP) III have recognized and appreciated the significant correlation of metabolic syndrome, diabetes and cardiovascular disease [1,20]. Throughout their research they have seen a significant prevalence of metabolic syndrome with worsening glucose tolerance from 26 percent in patients with normal fasting glucose rising to 86 percent in those with diabetes. Metabolic syndrome is a very common disorder in the U.S. population for people over the age of 50. In contrast, diabetes without metabolic syndrome is uncommon in the over-50 population (only 13 percent of diabetic patients do not meet criteria for metabolic syndrome) [1].

In contrast, the ADA and the European Association for the Study of Diabetes state there is no solid evidence that any of the metabolic syndrome health factors contribute more together than they do individually. In other words, the whole is not greater than the sum of its parts. The authors recommend that doctors should [15]:

- Aggressively treat the individual health factors that lead to heart disease (diabetes, insulin resistance, unhealthy triglyceride and cholesterol levels, high blood pressure, and signs of kidney disease).
- Continue to evaluate patients for other risks that can lead to heart disease.
- Avoid labeling patients with the term “metabolic syndrome.”
- Avoid prescribing a treatment for the “syndrome” until new, solid evidence is found.

However, many health care providers have recognized the significant correlation of metabolic syndrome diabetes and cardiovascular disease. The American Academy of Family Physicians (AAFP) (2004) believes metabolic syndrome will overtake cigarette smoking as the number 1 risk for developing cardiovascular disease [18]. It is imperative to monitor and diagnose this condition as it significantly correlates to an increased risk of the patient developing type 2 diabetes and heart disease [75].

According to the NDIC (2008), people whose test results indicate they have prediabetes should be educated about changing their lifestyle behaviors, and their blood glucose levels be rechecked in one to two years. At the time of diagnoses or recognizing a patient with the risk factors for developing diabetes, clinical research trials have demonstrated that the patient and family should be educated on the following [57]:

- The first therapy should always be an intensive lifestyle modification program because weight loss and physical activity are much more effective than any medication at reducing the risk for developing diabetes.
- At this time, several drugs have been shown to reduce diabetes risk to varying degrees, but at this time there is no drug approved by the U.S. Food and Drug Administration (FDA) to treat insulin resistance, prediabetes or to prevent type 2 diabetes. However, the ADA recommends the initiation of Metformin (See Medication Modalities for Type 2 Diabetics).

Gestational diabetes

Gestational diabetes (GDM) is a form of glucose intolerance that is diagnosed in some women during the later part of their pregnancy. The risk increases especially if the pregnant woman has coinciding risk factors for type 2 diabetes [75]. During pregnancy, the extra metabolic demands required to support the pregnancy and potential other co-morbidities or risk factors may cause the onset of diabetes. Throughout the pregnancy, the woman’s hormones are providing growth for the baby, but these same hormones also block the action of the mother’s insulin in her own body, potentially leading to insulin resistance.

In a normal pregnancy, there are complex alterations in the maternal glucose metabolism, insulin production and metabolic homeostasis. It is imperative that these complex alterations occur to ensure adequate nutrition for the mother and developing fetus [77]. Glucose is the primary fuel used by the fetus through the process of carrier-mediated diffusion, implying it is directly proportional to the maternal levels. At 10 weeks gestation, the fetus secretes its own insulin at adequate levels to balance the glucose ingested by the mom. Therefore, as the maternal glucose levels rise, the fetal glucose levels are increased, resulting in increased fetal insulin secretion. Each of the metabolic changes is elaborated upon as follows [77]:

- **During the first trimester** (through week 12 gestation), the pregnant woman's metabolic status is influenced by the rising levels of estrogen and progesterone. The hormones stimulate the beta cells in the pancreas to increase insulin production, promoting increased peripheral utilization of glucose and decreased blood glucose with fasting levels being reduced by approximately 10 percent. There is an increase in tissue glycogen stores and a decrease in the hepatic glucose production, which further increases the risk of hypoglycemia occurring during the first trimester.
- **During the second and third trimesters,** the pregnancy exerts a diabetogenic effect on the maternal metabolic status, thus increasing the risk of developing diabetes. Since there are significant hormonal changes occurring throughout this period, there is a decreased tolerance to glucose, increased insulin resistance, decreased hepatic glycogen stores and increased hepatic production of glucose. Maternal insulin requirements gradually increase from 18 to 24 weeks gestation to about 36 weeks. At 36 weeks, insulin requirements usually level off until labor begins.
- **At birth** (typically between 38 and 40 weeks gestation), the expulsion of the placenta prompts an abrupt decrease in levels of circulating placental hormones, cortisol and insulinase. Therefore, the maternal tissues quickly regain their prepregnancy sensitivity to insulin and she does not receive insulin unless the blood glucose level is greater than 200 mg/dl.
    - If the new mom is not breastfeeding, the prepregnancy insulin-carbohydrate balance returns in about seven to 10 days.
    - If the new mom is breastfeeding, lactation uses maternal glucose, thus the breastfeeding mom's insulin requirements will remain low as long as she is nursing.

There are several risk factors that predispose women without a prior diabetic history to develop GDM, such as [27, 48]:

1. Family history of diabetes.
2. Delivering a baby greater than 4,000 g (macrosomia).
3. Belonging to one of the high-risk ethnic groups (similar to the type 2 diabetic).
4. Older than 25 years of age.
5. Body Mass Index (BMI) greater than 25kg/m2.
6. PCOS.
7. Prior history of GDM and/or a history of complications of obstetrical associated with gestational diabetes (stillbirth).

Other/rare forms of diabetes

Latent autoimmune diabetes in adults (LADA), also referred to as 1.5 diabetes or double diabetes, is diagnosed in individuals over the age of 30. The individuals demonstrate signs and symptoms of both type 1 and type 2 diabetes. Initially, the individual with LADA will still produce their own insulin, similar to a type 2 diabetic, but within a few years must take insulin to control blood glucose levels. In LADA, as in type 1 diabetes, the beta cells of the pancreas stop making insulin because the body's immune system attacks and destroys them [55].

Diabetes related to genetic conditions, such as maturity-onset diabetes of youth (MODY) or mutated genes. These rare genetic forms of diabetes are elaborated as follows per the current medical diagnosis and treatment (CMDT) 2007 guidelines [53]:

- **MODY** is a rare monogenic disorder characterized by noninsulin diabetes with an autosomal dominant inheritance in a person younger than 25 years of age. Typically, the patient presents with hyperglycemia related to impaired glucose, induced secretion of insulin and is nonobese.
- **Diabetes due to mutant insulins** is a rare form of nonobese type 2 diabetes with an autosomal dominant genetic disposition in which the individual has only one normal insulin gene. The diabetes is mild and does not appear until middle age.
- **Diabetes due to two mutant insulin receptors genes**. Over 40 percent of all diabetic patients have a defect in one of their insulin receptor genes; however it is rare for it to occur in two. If an individual has two mutant insulin receptor genes, it will be noted in infancy. The newborn will have a leprechaun-like phenotype and unfortunately, rarely survives.
- **Diabetes associated with a mutation of mitochondrial deoxyribonucleic acid (DNA)** impairs the transfer of leucine or lysine into mitochondrial proteins. This rare form is transmitted only by the mother because sperm do not contain mitochondria. Typically the diabetes is mild and the patient responds to oral hypoglycemic agents. Interestingly, two-thirds of the patients with this form of diabetes have a hearing loss, and others in smaller amount (15 percent) may have coinciding Myopathy (muscular weakness), Encephalopathy (degenerative brain injury), Lactic Acidosis and Stroke-like episodes (MELAS).
- **Wolfram's syndrome** is an autosomal recessive neurodegenerative disorder that presents in childhood. Wolfram's syndrome consists of Diabetes Insipidus (DI), Diabetes Mellitus (DM), Optic Atrophy and Deafness (DIDMOAD). At this time there is no treatment for DIDMOAD, and the patient typically lives to about 30.

Other rare forms of diabetes may result from one of these [31, 55]:

- Surgery.
- Drugs (steroid hormones, Dilantin, thiazide diuretics and thyroid hormones as they may impair the normal action of insulin).
- Malnutrition.
- Infections.
- Other illnesses (pancreatitis or cystic fibrosis).

All of the rare forms of diabetes, including the mutated genes and genetic predispositions, account for 1 to 5 percent of all diagnosed cases of diabetes [31]. Due to the rare cases, little data is available online and/or in textbooks elaborating upon the details and mechanisms.

Signs and symptoms of diabetes

Many times, patients may be unaware of their potential risk or living unknowingly with diabetes. The initial signs and symptoms of diabetes may be very subtle, and a patient may assume it is related to another reason. The classic signs and symptoms of diabetes in general are the "three polys"; polyuria (excessive urine output), polydipsia (increase thirst), and polyphagia (extreme hunger) [63]. Other clinical signs noted during a clinical exam, revealed in blood and urine tests, include: hyperglycemia, glycosuria (glucose in the urine) and ketonuria (ketones in the urine) [49, 63,78]:

- **Polyuria** occurs from increased glucose circulating in the blood, resulting in hyperglycemia. The hyperglycemia causes serum hyperosmolality, drawing water from the intracellular spaces into the general circulation. All of the extra fluid increases blood volume, leading to an increase in flow to the kidneys. The buildup of glucose, especially in the renal tubules, acts as an osmotic diuretic, thus increasing urine output.
  - **Polyuria** can be measured in the urine when the blood glucose level exceeds the renal threshold for glucose, usually about 180 mg/dl. The condition is called glucosuria.
- **Polydipsia** occurs due to a decrease in the intracellular spaces. Water is pulled out into the general bloodstream and then compounded with an increased urinary output leading to dehydration and the patient having an urge to drink continuously.

  The glucose is unable to enter the cell without insulin, therefore the energy level declines (fatigue), but it will stimulate the patient to have the urge to eat more (polyphagia). It is important to note that although the patient is increasing food intake, he or she will typically lose weight (maybe even become emaciated) because as the body loses water, it will break down proteins and fats in attempt to replenish the energy source.

Other potential signs and symptoms of diabetes are [8,45,48, 53,78]:

- Dehydration leading to **hemoconcentration** (increased blood concentration), **hypovolemia** (decreased blood volume), hyperviscosity (thick concentrated blood), **hypoperfusion** (decreased circulation) and **hypoxia** (poor tissue oxygenation).
- **Unusual weight loss** related to the breakdown of protein and fats and depletion of water, glycogen and triglycerides due to the lack of insulin. Therefore, reduced muscle mass occurs as the amino acids are diverted to form glucose and ketone bodies [8, 10].
- **Increased fatigue** due to a lack of energy from inappropriate absorption of glucose in the cells.
- **Irritability** due to fluctuations and/or changes in the blood glucose levels.
- **Blurred vision** typically occurs with polydipsia as it often develops when the lens are exposed to hyperosmolar fluids.
- **Postural hypotension** results from a lower plasma volume.
- **Paresthesias** (numbness and tingling of the lower extremities; "feeling the limbs are asleep") may or may not be present at the time of diagnosis as a result of a temporary dysfunction of peripheral sensory nerves. Paresthesias typically resolves once insulin is replaced and the glycemic levels are restored to a homeostasis level.

In addition, health care professionals should always contemplate a potential diagnosis of diabetes in women who have delivered large babies (macrosomia; greater than 9 pounds, or 4.1 kilograms), history of polyhydramnios (excess amount of amniotic fluid in the sac; occurs in 1 to 2 percent of all pregnancies)or pre-eclampsia or unexplained fetal loss, even if she did not develop GDM [53].

Type 1 diabetes affects the metabolism of fat, protein and carbohydrates so glucose accumulates in the blood and leaks into the urine when the glucose exceeds the kidney's ability to excrete it appropriately. Type 1 diabetes is correlated to the destruction of beta cells. Unfortunately, the patient typically does not exhibit any signs or symptoms until 80 to 90 percent of them are destroyed and insulin falls to critically low levels [63]. The major initial clinical manifestations noted in type 1 diabetes include the "three polys" [48].

Type 2 diabetes is typically more nonspecific because the patient will often complain of polyuria and polydipsia along with often being overweight, hyperlipidemic (high lipid levels) and high blood pressure. However, children and adolescents may not present with only symptoms of polydipsia or polyuria and acanthosis nigricans [25]. Children often present with a preceding minor illness, such as a flulike episode prior to being diagnosed with diabetes [46]. In addition, there have been several cases of children and adolescents who were undiagnosed with type 2 diabetes who had reported to the emergency room or their primary care provider in a hyperglycemic hyperosmolar state (HHS) that was confused with a diabetic ketoacidosis (DKA) and unfortunately had a high mortality rate due to the lack of recognition and proper treatment [25]. (See the section, Hyperglycemia and Diabetes for further explanation).

The type of obesity fat seen in type 2 diabetic patients is predominately distributed on the upper segments of the body (especially in the abdomen, chest, neck and face) and less often on the appendages [53]. However, nurses should never think that type 2 diabetes occurs in only obese patients. Some who suffer it can be emaciated due to the breakdown of fat and protein. Another aspect in type 2 diabetes is the onset is usually slow and insidious, making the diagnosis difficult [48, 53]. Since the symptoms may be very subtle and/or intertwined with other co-morbidities, it is important to ensure the primary care provider recognizes other manifestations such as [8]:

- **Recurrent infections**, which are common due to the proliferation of increasing glucose circulating in the blood stream, impairing the blood supply and thus hindering the healing process.
- **Acanthosis nigricans**, a hyperpigmentation and thickening of the skin with velvety irregularities apparent in skin folds of the neck, axillae, elbows, knees, groin and abdomen [25].
- **Genital pruritus**, common especially in women due to circulating glucose and glycosuria (glucose excreted in the kidneys) which both promote the growth of fungus, such as Candida.
- **Visual changes** that occur from the water balance in the eye fluctuating due to increased glucose levels.

Type 2 diabetes has a unique manifestation, **HHS**, characterized by a plasma osmolarity of 340 mOsm/L (greater than the normal range of 280-300 mOsm/L), elevated blood glucose (greater than 600mg/dl and may be as high as 1,000 to 2,000 mg/dl) and an altered level of consciousness [53]. HHS is a serious, life-threatening complication of type 2 diabetes. (See Hyperglycemia and Diabetes for further explanation)

Gestational diabetes may or may not have any present symptoms. Many women remain asymptomatic throughout their pregnancy if they have no previous history of diabetes prior to conceiving. However, if a woman is at risk or may be developing symptoms, she may test positive with polyuria and glycosuria during her routine prenatal care appointments and she may complain of any of the following symptoms [22]:

- Polydipsia.
- Frequent urination.
- Fatigue.
- Nausea.
- Frequent infections of bladder, vagina and skin.
- Blurred vision.

Screening and diagnosing diabetes

There are over 7 million people in the U.S. living unknowingly with diabetes. Health care professionals must learn to recognize the symptoms and then educate patients and the community to ensure people recognize the signs and symptoms to prevent long-term complications. Some patients may present to their health care provider with the classic symptoms of the "three polys" (polyuria, polydipsia and polyphagia). Others may be diagnosed as they present for another concern or complaint. Unfortunately, there are some patients who skip through the radar because they avoid seeing doctors or they do not have access to adequate health care.

Health care professionals typically complete a routine urinalysis on each of their patients because it is relatively inexpensive. All patients over the age of 3 will have a urinalysis completed before seeing their health care provider. In a routine urinalysis, the health care provider will be able to screen for the following presence of glucose, but the test is not used for diagnostic measures [34, 53]:

- **Glycosuria** (sensitive to picking up less than 0.1 percent of glucose in urine) occurs when the renal threshold for glucose is exceeded (180 mg/dl or greater) due to osmotic diuresis [25].
- **Ketonuria,** with any amount found a concern as it may imply the possibility of diabetic ketoacidosis (DKA). Ketonuria occurs due to an abnormal breakdown of fatty acids (the backup source of energy) that may accumulate in the blood and urine when insulin is not available [49].

If glucose and/or ketones are found on the urinalysis, then the health care provider will initiate blood testing to confirm a diagnosis of prediabetes (insulin resistance/metabolic syndrome) or diabetes. However, nurses should realize there are different recommendations circulating among health care professionals to screen for diabetes, so the practice of the primary care provider may be different.

Children
Since 2007, the ADA and the American Academy of Pediatrics (AAP) have recommended a fasting blood glucose (FBG) at the age of 10 or the onset of puberty, and every two years if overweight (BMI greater than 85th percentile for age and sex), plus two additional risk factors (family history of diabetes in a first- or second-degree relative; high risk race/ethnicity; signs of or conditions associated with insulin resistance (acanthosis nigricans, hypertension, dyslipidemia, or PCOS) [32, 45].

Adults
Since 2003, the U.S. Preventive Services Task Force (USPSTF) has recommended that adults with high blood pressure or high cholesterol be screened for type 2 diabetes (insulin-resistant diabetes) as part of an integrated approach to reduce their risk of cardiovascular disease, but concluded that further research is needed to determine whether widespread screening of the general population would improve health outcomes [62].

The National Guideline Clearinghouse (2009) and Current Practice Guidelines (2008) recommend that between the ages of 18 and 39, screening may be indicated in patients with risk factors for diabetes, previously identified impaired FBG or OGTT, history of GDM, hypertension, HDL-C less than 35 mg/dL and/or triglyceride greater than 250 mg/dL, PCOS, or history of vascular disease. Beginning at the age of 45, a fasting plasma glucose is recommended every three years on all patients, especially if the patient has a body mass index greater than 25 [45, 59].

The ADA (2007) guidelines are partially congruent with the previous recommendations that screening should begin with a FBG or OGTT every three years, beginning at the age of 45, especially if BMI greater than 25. In addition, screening should be initiated earlier and more frequently in overweight patients with diabetes risks factors (family history, high risk ethnic group, history of impaired glucose testing, GDM, mother with an infant birth weight greater than 9 pounds, comorbid conditions hypertension (greater than 140/90), dyslipidemia HDL less than 35mg/dl or Triglycerides (TG) greater than 250 mg/dl), overweight (BMI greater than 25), PCOS or acanthosis nigricans, history of vascular disease and/or habitually physically inactive [45,81].

Adults with comorbid hpertension or dyslipidemia (metabolic syndrome)
The AAFP (2007) and USPSTF (2003) recommend screening for type 2 diabetes in patients with hypertension and/or dyslipidemia due to the potential correlating metabolic syndrome and risk for developing type 2 diabetes. However the specific testing and frequency is not mentioned or suggested. There is strong evidence that in hypertensive patients, the health care provider needs to be more aggressive in controlling the blood pressure when diabetes is coinciding [45].

According to the Journal of Family Practice (2009), research reiterates the information provided by the AAFP and USPSTF

that although there are no specific guidelines for patients with hypertension (HTN) or dyslipidemia, practitioners should still educate their patients at risk. In addition, unless the patient meets another guideline or recommendation, then all patients should be screened beginning at 45 per the ADA [60].

Pregnant women

The USPSTF (2003) and AAFP (2007) have found insufficient evidence that routine screening for gestational diabetes substantially improves the health of mothers or their babies, although it is implemented in care among pregnant women between 24 and 28 weeks gestation [45,62].

According to the ADA (2007), all pregnant women should be screened for the potential risk of developing GDM at their first prenatal visit [45]:

- A woman with a high risk of developing GDM (obesity with a BMI greater than 27, family history, personal history of GDM, glycosuria, previous delivery of a large for gestational age infant, or PCOS), is urged to get an OGTT completed as soon as possible. If an OGTT is not completed with the initial testing, then the woman should be tested between 24 and 28 weeks gestation.
- A woman with an average risk of developing GDM is recommended to test between 24 and 28 weeks gestation.
- For a woman with a low risk of developing GDM (less than 25 years old, [not of Hispanic, African, Native American, South or East Asia or Pacific Islander ancestry], with weight normal before pregnancy, no history of abnormal glucose tolerance, no previous history of poor obstetric outcome and no known diabetes in a first-degree relative) is no glucose testing is recommended.

Most health care providers also will order a routine electrolyte panel at least annually, regardless of any symptoms or family history due to the prevalence of diabetes in our country. However, according to the Quality Adjusted Life Year (QALY) (2004), failure of health care providers to adhere to the recommended guidelines is not cost-effective for our health care system and budget [45].

Before assessing fasting blood glucose (FBG) or OGTT, the patient should be free from any acute illnesses. If so, then the nurse should instruct the patient three days before testing to continue a regular diet with at least 150 to 200 grams (g) of carbohydrates daily and then no caloric intake at least eight hours before the test [35]. If the patient is having a serum fasting blood glucose, the blood will be taken upon arrival at the laboratory. However, if the patient has been ordered an OGTT, the patient will be instructed to drink 75 to 100 g of glucose over five minutes. OGTT may be ordered for one or two hours in nonpregnant adults [35].

The ADA and AAFP (2007) and the USPSTF (2003) comply with the following acceptable diagnostic measurements for children and non-pregnant adults [35, 45, 46, 53]:

- **Diabetes** is diagnosed by:
  1. A fasting blood glucose level greater than 126 mg/dl on two or more occasions (a normal FBG is less than 100 mg/dl).
  2. A random plasma glucose concentration greater than 200mg/dl taken at any time, regardless of the last meal
  3. Two-hour plasma oral glucose tolerance test (OGTT) greater than 200 mg/dl (two hours after ingesting 75g of a glucose load). A normal FBG level for nondiabetics is 70-110 mg/dl).A normal OGTT is less than 140mg/dl. There are certain medications that may skew the OGTT, such as diuretics, contraceptives, glucocorticoids, niacin and phenytoin [10].
- **Prediabetes** (High risk for developing diabetes). Normoglycemia is defined as a plasma glucose level less than 100 mg/dl in the FBG and less than 140 mg/dl in the two hour OGTT.
  1. Impaired glucose tolerance (IGT) is diagnosed in a patient without any prior history of diabetes by:
     - FBG greater than 126mg/dl and a plasma glucose 140-200mg/dl
     - 2 hour- OGTT 140 mg/dl, but less than 200mg/dl
  2. Impaired fasting glucose (IFG) is diagnosed in a patient without any prior history of diabetes by:
     - FBG 100-125 mg/dl and a plasma glucose less than 140mg/dl.

It can be a little tricky to find a normal level because the levels may be different if a patient is pre-diabetic, undiagnosed or living with diabetes. The ADA and the American College of Endocrinology (ACE) have provided the following 2008 ranges to ensure compliance by patients and health care professionals; see Table 1 [3, 13].

Table 1: Diagnostic Results for Pre-diabetes and Diabetes

	Pre-diabetes per the ADA & ACE	**Living with Diabetes guidelines per the ADA and ACE**
Normal fasting blood glucose level	Less than 100 mg/dl	70–130 mg/dl
Postprandial (2 hours after eating)	Less than 140 mg/dl	Less than 180 mg/dl
Diagnostic for diabetes: • Fasting plasma glucose (FPG) • 2 hours Postprandial (after eating) oral glucose tolerance test (OGTT)	• Greater than 126 mg/dl • Greater than 200 mg/dl	• Greater than 126 mg/dl • Greater than 200 mg/dl

*Table 1 devised based upon the ADA & ACE literature review.

In a pregnant woman, the ADA and AAFP (2007) and USPSTF (2003) recommend the following diagnostic measurement for GDM [45, 67]:

- FBG greater than 126 mg/dl or a casual plasma glucose greater than 200mg/dl and precludes the need for an OGTT challenge. However, most providers caring for pregnant woman will order an OGTT between 24-28 weeks gestation. Diagnosis is confirmed if:
  - Initial screening for gestational diabetes is accomplished by performing a 50-g, one-hour OGTT at 24 to 28

weeks of gestation. Normal should be less than 130 to 140 mg/dl; both are accepted. If the patient does not pass the one-hour test (results greater than 140 mg/dl), the woman will have to complete a 75 or 100-g, three-hour OGTT. The patient will be instructed to remain without food or drinks for eight to 12 hours before the exam. During the exam, she will be tested upon arrival to obtain a fasting level. Then she will drink the 75- or 100-g glucose challenge within five minutes, and then be tested every hour for three hours and three-hours after she completes the glucose challenge.
    - Diagnosis of GDM is confirmed if the woman's level is above normal on any two of the following parameters [11]:
        - Fasting 95 mg/dl.
        - 1-hour 180 mg/dl.
        - 2-hour 155 mg/dl.
        - 3-hour 140 mg/dl.

Once diabetes is diagnosed, then the patient can anticipate having the following routine tests completed to monitor the management and appropriate care [36, 53]:

- **Chemistry profile** to assess the electrolyte panel, especially the blood sugar and kidney function.
- **FBG** will be ordered if the patient is experiencing any signs or symptoms of hypoglycemia or hyperglycemia because it is quick and cheaper than the chemistry profile. In addition, it may be ordered to assess for complications, such as DKA or HHS.
- **Glycosylated hemoglobin (c) (HbA1c)** is a laboratory test that should be monitored every six months if the patient is meeting specific treatment goals; otherwise it will be completed every three months. The HbA1c will be ordered approximately every three months to monitor the average glucose level over the previous three months; however it is not used as a diagnostic measurement for diabetes. Anytime the glucose level is elevated or erratic, glucose attaches to the hemoglobin molecule then remains there for the life of the hemoglobin, typically 120 days in a healthy person. The normal level should be below 7 percent, typically 4 to 6 percent in healthy individuals [34]. According to the CDC (2008), for every 1 percent reduction in results of A1C blood tests (e.g., from 8.0 to 7 percent), the risk of developing eye, kidney and nerve disease is reduced by 40 percent [30]. The ADA recommends all diabetics keep their HbA1C less than 7 percent [7].
- **Urinalysis** to assess for the presence of glucose, ketones and proteins [34]:
    - The presence of glucose in the urine indicates hyperglycemia (greater than 180 mg/dl).
    - The presence of ketones indicates that carbohydrates in the body are diminished and fats are broken down. If ketones are present in the urine, ketonuria is diagnosed and is an indicator of DKA. However, if DKA is speculated and the urine ketones are negative, rule out renal insufficiency as it may skew the data. If the kidneys are not functioning appropriately, the kidneys may not be able to filter the ketones, leading to a false negative.
    - The presence of protein in the urine (microalbuminuria). Normally, less than 150 mg of protein is excreted in the urine within a 24-hour period. If microalbuminuria is noted, it is an early indicator of nephropathy in diabetic patients. It is important to assess for the presence of microalbumin to screen all diabetic patients for the future development of renal failure. The primary provider may refer the patient to urologists or order a urine culture because urinary tract infections may cause excretion of proteins in the urine. Other causes of microalbuminuria include heart failure, hypertension and heavy exercise; therefore a positive result is confirmed with three additional tests over the next few months.
- **Serum cholesterol and triglyceride levels**. The lipids are ordered to assess for atherosclerosis and cardiac impairments every three to six months.
- **Thyroid function tests** will be ordered every three to six months to assess for the risk of coinciding thyroid disease that typically occurs in diabetic patients due to the autoimmune effect.

General treatment modalities for all diabetic patients

The key to being successful in the treatment of diabetes and metabolic syndrome (prediabetes) is collaborating with the patient and family by providing education with each visit to ensure the patient is empowered to control the diabetes, rather than the diabetes controlling the patient. Regardless of the type of diabetes, all patients need to make lifestyle changes and monitor their diabetes as recommended per the ADA and European Association for the study of diabetes [45]:

- **Weight loss.** One of the major, first-line treatment modalities is to encourage the patient to lose weight by choosing healthy foods and exercising. Losing weight will decrease the complications related to the heart and mortality risk factors correlated to obesity (BMI greater than 30 and increased abdominal fat).
- **Nutrition therapy.** Nutrition is one of the key factors in order to maintain euglycemia, but it may be impeded or customized based upon the culture, lifestyle and financial issues of the patient and family. It is important to convey that in order to maintain glycemic control, the patient should learn to count carbohydrates or follow the conventional meal plan (three meals a day and three snacks). The ADA does not provide a specific meal plan for every diabetic patient; rather it is important for the patient to be familiar with his or her own nutritional goals and laboratory values to assess whether he or she is within the guidelines. In addition to the education provided by the nurse and physicians, the patient and family should be encouraged to discuss their nutrition with a registered dietician (RD) to help customize their own nutritional needs and goals.

  Although there are no specific recommendations for diabetic patients, there are certain parameters that should be adhered to on a daily basis, as well as the importance of understanding the relationship between foods and insulin [25, 47].

Typically, the ADA encourages most patients to be familiar with the following food groups:

- **Carbohydrates** since they are the body's main and preferred energy source. The ADA recommends that most patients limit their total carbohydrates to 40 to 60 percent of their total calorie intake; however it may be customized by the patient's average blood glucose levels [49. 74]. Carbohydrates are composed of starches (breads, cereals, rice, and pasta), fruit, milk/yogurt, and sugars/sweets (gum, sodas, fruit drinks and pastries) [74].
- **Fiber** is also considered a carbohydrate, but the body does not digest it. Therefore, it cannot raise the blood glucose level. Fiber is an important nutrient that promotes health by improving carbohydrate metabolism and lowering the patient's cholesterol levels [19]. Main sources are whole grains, legumes, fruits and vegetables. All diabetics should be encouraged to increase the fiber in their diet to control their blood glucose as well as adhering to the recommended carbohydrates per meal [44]. The ADA recommends that individuals should be ingesting 20 to 35 grams of fiber a day from a variety of sources [74].
  - The nurse should educate patients to increase fluids and to gradually add high fiber in their diets to reduce abdominal cramping, loose stools and flatulence [19]. In addition, increasing too much high fiber at one time can increase the risk of hypoglycemia.
- **Proteins** help the body function appropriately by maintaining homeostasis and helping in the repair of tissues. Proteins are very complex, since there are complete proteins and nonproteins. A complete protein is a food that contains nine essential amino acids (found in meat, poultry, seafood, dairy products, eggs and soy) [74]. Incomplete proteins are missing one or more essential amino acids (beans, peas, nuts, seeds and vegetables) [74]. Protein is very important to patients with diabetes. The ADA recommends protein intake be approximately 15 to 20 percent of total calorie intake for people with normal kidney function [49, 74]. For patients with microalbuminuria (increased protein in the urine), protein should be reduced to 10 percent of their total calories a day to slow the progression of kidney failure [49].
  - For type 1 diabetics, protein has little effect on glucose levels if the patient is taking enough insulin. However, if the patient ingests large amounts of protein, it can increase the blood glucose level, thus increasing the insulin needed [74]. The newer school of thought per the ADA and research is to have the individual patient monitor his or her protein intake and blood glucose levels to assess whether any changes need to be addressed with their provider.
  - If the patient is a type 2 diabetic, protein stimulates the production of insulin; therefore a small rise in insulin does lower the blood glucose levels [74].
- **Fat** is required in small doses to provide essential fatty acids, carry the fat soluble vitamins (A, D, E and K), maintain healthy skin and produce components needed for some hormones [74]. Similar to proteins, fat typically has little effect on the total blood glucose levels. However, it can affect the blood lipid levels, thus increasing the risk of heart disease. Therefore, the patient should avoid any fat high in trans-fatty acids [49].
- **DASH diet**. The DASH diet may be encouraged and/or collaborated with the diabetic diet if the patient has a coinciding cardiac issue, such as HTN and/or dyslipidemia. The DASH diet emphasizes fruits, vegetables, low-fat dairy foods, whole grains, poultry, fish and nuts, while reducing saturated fats, red meat, sweets and sugar-containing beverages. Reducing sodium intake can further reduce blood pressure or prevent the increase in blood pressure that may accompany aging [20].

There are various approaches to acclimate patients and their families to the importance of maintaining a healthy balance of nutrition and controlling their diabetes. Since 1995, the ADA and the American Dietetic Association have adopted the United States Department of Agriculture (USDA) Food Guide Pyramid [74]. It is a great diagram and tool to teach about food groups and appropriate portion sizes. In addition, the ADA recommends different tools to plan the most appropriate meals for patients and families. People should understand that they can change their plans as they gain better control of their diabetes. Patients should always be referred to a registered dietician to help them customize their meals.

1. **Carbohydrate counting** is a practice that has been utilized for many years. In order to count the carbohydrates, the patient needs to know their allotted carbohydrates for the day based upon the individual food intake, lifestyle, diabetes medications and physical activity [40]. The RD will be the best health care professional to help the patient and family determine this daily allotment. Carbohydrate counting focuses on the total grams of carbohydrates, regardless of the source [49].

   The ADA has determined the following advantages and disadvantages for carbohydrate counting [47, 49]:

   - **Advantages:**
     - The patient may perceive it to be easier to focus only on the carbohydrates ingested on a daily basis.
     - Typically the patient is able to achieve a stable blood glucose control if their carbohydrate intake is consistent every day.
     - Patients on insulin or an insulin pump can match the carbohydrates ingested to the amount of insulin needed. For example, an initial formula of one unit of rapid acting insulin is administered for each 15 g of carbohydrates. Patients become proficient at reading their labels or weighing each item to ensure they calculate the appropriate insulin dosage required for that particular snack or meal.
   - **Disadvantages:**
     - Although the patient may enjoy focusing on just one food group, it may also be a disadvantage if the patient loses focus on the other nutrient value of the food. For example, if a patient eats bacon or sausage for breakfast, the carbohydrates will be counted based upon the total number and type ingested. However, the patient may ignore the fat

content, and too much fat exacerbates the risk of heart disease, cancer and weight gain.

2. **Fat-gram counting** is a practice that has been around for almost 30 years that helps the patient learn about eating a low fat diet to reduce the risk of cancer. Fat gram counting is helpful for type 2 diabetics who are overweight to help reduce their total weight.

   The ADA has determined the following advantages and disadvantages for fat-gram counting [47]:
   - **Advantages:**
     - Provides flexibility and control over the food choices ingested. Typically, the patient who counts fat grams will choose healthier foods such as low fat fruits, vegetables, grains and low-fat dairy products.
   - **Disadvantages:**
     - Blood glucose levels may be inconsistent because the patient is only focusing on the fat ingested.
3. **Food exchange system** is a unique way to group foods with similar nutritional values into lists to help the diabetic patient eat consistent amounts of nutrients [47]. It is probably the most popular since it was initiated back in the 1950s [74].The ADA and the American Dietetic Association have actually published handy books, such as Exchange Lists for Meal Planning on three broad groups (the carbohydrates, meat and meat substitutes, and the fat group). In addition, there are books published every year that discusses the total number of nutrients based upon the patient's food choices at restaurants and their ethnic/ cultural considerations.

   The ADA has determined the following advantages and disadvantages for food exchanges [47]:
   - **Advantages:**
     - The patient has more knowledge of various nutrient groups and the correlation to their glucose level.
     - Typically it results in more consistency in the patient's blood glucose levels.
   - **Disadvantages:**
     - It requires the diligent patience of the patient to truly grasp the concept of "exchanging" foods.
4. **Calorie counting** is a tool that has been encouraged for many years to lose, gain or maintain an individual's weight. Similar to the carbohydrate counting, the RD will be more apt to customize it to the patient based upon their weight, height and activity level.

   The ADA has determined the following advantages and disadvantages for calorie counting [47]:
   - **Advantages:**
     - Allows the patient to expand the choices of foods as long as he or she abides by the total calorie goal a day.
   - **Disadvantages:**
     - May be time-consuming to calculate the calorie content of each food ingested.

Nursing considerations and education for the diabetic patient in regards to nutrition [52]:

- Assess the blood glucose level for the type 1 diabetic and type 2 diabetic (on insulin) within one to two hours after meals to determine whether the insulin/ carbohydrate regimen is adequate to emulate a "functioning" pancreas.
- If the patient is within an average weight (typical type 1 diabetic), encourage the patient to avoid gaining weight. Hyperinsulinemia (chronic high blood glucose levels) can occur with intensive treatment schedules and may result in weight gain.
- If the patient is overweight and/or obese (typical type 2 diabetic), reiterate to the patient the importance of monitoring and reducing their total intake of saturated fat, cholesterol and sodium levels, especially if they have coinciding conditions and co-morbidities. Research has demonstrated that reducing the total calories by 250 to 500 a day and increasing physical activity improves diabetes and weight control. In addition, reducing 10 percent of the body weight can significantly reduce the Hba1c levels.

5. **Exercise**. Exercising has phenomenal benefits on the metabolism of carbohydrates and insulin sensitivity [49]. According to the AAP (2004) and the AHA, all diabetic patients should be encouraged to exercise as it will improve physical fitness, emotional well-being, weight control, decrease cholesterol and triglyceride levels, and improve work capacity and decrease cardiac complications [5, 20]. Regular physical activity reduces very low density lipoprotein (VLDL) levels, raises HDL cholesterol, and in some people, lowers the LDL levels. It also can lower blood pressure, reduce insulin resistance and improve the function of the heart [70]. It should be important for the health care provider to find a level of activity that the patient can accomplish over the long term [18]. The AAFP (2004) recommends a combination of resistance and aerobic exercise, but any activity is better than none, and patients who have been sedentary need to start with walking and gradually increase duration and intensity [19, 20].
   - Initially, the patient can be instructed to use low-weight dumbbells, elastic exercise bands or even heavy food containers that can provide the needed weight for resistance training.
   - Instruct the patient to stretch for five to 10 minutes prior to performing any exercise, then have a five- to 10-minute cool-down period afterwards to reduce the risk of dysrhythmias.
   - Gradually work up to aerobic exercise for 40 to 60 minutes. Aerobic exercise includes walking briskly, running, jogging, stationary or regular bicycling, swimming, dancing, rowing and cross-country skiing as they each improve cardiac output. If the patient is a type 1 diabetic, the patient should limit the exercise time to 20 to 40 minutes four to seven days a week.

   Nursing considerations and exercise education for the diabetic patient [19]:
   - Assess the blood glucose level before exercise.
     - If the patient is hyperglycemic (greater than 250 mg/dl), check the urine for ketones. If the

patient has positive ketones, the patient should be instructed not to exercise because exercise would cause the patient to become hyperglycemic.
    - Type 1 diabetics should only perform vigorous exercise if their blood glucose levels are 80 to 250 mg/dl and no ketones are present.
    - Although the patient is at risk of becoming hyperglycemic, the patient can also become hypoglycemic. Therefore, encourage the patient to keep a snack on standby. Type 1 diabetics are unable to make the shift in hormones because the inadequate insulin supply doesn't allow proper flow of glucose to the cells. Hypoglycemia can occur during exercise and continue for up to 24 hours afterward, so patients might require an additional carbohydrate.
- Avoid exercising within one hour of insulin administration or at the peak of the insulin. Exercise can increase the absorption of insulin from the injection site, increasing the blood glucose levels.
- Avoid exercising in extreme cold or heat.
- Assess for the following complications related to exercise:
    - If the patient has peripheral neuropathy, observe and limit the risk of foot and joint injuries. (See below, ADA recommendations for all diabetic patients to avoid injury).
    - If the patient has retinopathy, educate the patient to avoid the Valsalva maneuver and activities that increase the blood pressure because heavy lifting, rapid head motion or jarring activities can cause hemorrhage or retinal detachments.
    - If the patient has nephropathy, exercise may increase proteinuria (microalbuminuria).
- It is important to note that the ADA recommends that all diabetic patients adhere to the following when they exercise to avoid injury and complications [10]:
    - Utilize proper fitting footwear.
    - Never walk barefoot.
    - Inspect the feet daily and after exercising.
    - Avoid exercise in extreme heat or cold.
    - Avoid exercise during periods of poor glucose control, avoid smoking to ensure adequate circulation, and any diabetic patient over 35 should have an exercise-stress electrocardiogram prior to any exercise routine.

6. **Stress management** (illness, surgery, corticosteroid therapy). Any time a diabetic patient is ill, the blood glucose levels will increase, even though the intake has diminished [49]. The nurse should educate all patients on the following [25,49]:
    - Assess the blood glucose levels at least four times a day while ill. As with patient exercise, assess the urine ketones if the glucose level is greater than 250 mg/dl.
    - Encourage the patient to continue taking the usual insulin dose or an oral hypoglycemic agent.
    - Encourage the patient to drink extra fluids, sipping 9 to 12 ounces of fluid each hour.
    - Encourage the patient to substitute easily digested liquids or soft foods if solids are not being tolerated.
    - Encourage the patient to notify their health care provider if they are unable to eat for more than 24 hours or if vomiting and diarrhea last more than six hours.
7. **Monitoring blood glucose levels**. Self-monitoring of the daily blood glucose level (SMBG) is important for the diabetic patient, and devices to do so are available to purchase. It is important that patients and their families are adequately trained on their specific machine to ensure accurate readings. It is also important to note that self-monitoring is vital for the patient to understand their average blood glucose readings, but a health care professional caring for the patient will not change the treatment plan based upon the patient's home readings because there are multiple variables that may skew the data. The accuracy of self-monitoring results depends upon the patient adhering to the manufacturer's directions and the following variables [68]:
    - **Quality of the meter, test strips and patient's training**. The patient should always read and follow the directions from the manufacturer. Failure to comply may result in inaccurate results. In addition, if the patient has any sensory deficits or is unable to comprehend the directions, the nurse should encourage the patient to bring in their machine to ensure proper understanding and demonstration.
    - **Patient's hematocrit level**. If the patient has a higher-than-normal hematocrit value, the patient will usually test lower on their SMBG than patients with normal hematocrit. In addition, if a patient has lower hematocrit values, their SMBG will test higher.
    - **Other substances in the body** may interfere with the testing results, such as uric acid (a natural substance in the body that can be more concentrated in some people with diabetes), glutathione (an "anti-oxidant" also called "GSH"), and ascorbic acid (vitamin C).
    - **Altitude, temperature and humidity**. Altitude, room temperature and humidity can cause unpredictable effects on glucose results. Therefore the patient should be referred to read the manufacturer's directions if the climate changes or they travel to another part of the country.

    The ADA recommends that all diabetics maintain their daily blood glucose levels as follows [7]:
    1. Pre-prandial 70 to 130 mg/dl.
    2. Postprandial less than 180 mg/dl.
8. **Collaborating with their health-care provider and endocrinologists.** Typically, the patient's diabetes will be ultimately be managed by their primary health care provider, and then followed up by an endocrinologist or a cardiologist if other co-morbidities are present or as needed. Patients can expect their primary care provider or endocrinologist, whichever one is responsible for the treatment modalities, to see them every three months to assess their laboratory data (FBG, A1C, lipids). Other referrals include ophthalmologists and podiatrists.
The patient should be seen annually unless their ophthalmologist or podiatrist informs them otherwise. It is important that any referrals or education are documented

appropriately to avoid any potential complications to the patient and legal consequences for the health care provider.

In addition to the patient taking their diabetic agents (insulin and/or oral meds), the patient may be prescribed any or all of the following medications [24]:

- Annual **flu vaccination** to provide coverage two weeks to a month after injection for up to six months, although the flu vaccination should be avoided by anyone with an allergy to eggs or a current respiratory infection. The patient should be instructed to return for the flu vaccination once the respiratory infection has subsided.
- **Pneumonia vaccination** can be administered anytime during the year. According to the ADA, for most people, one shot will provide protection for life and typically provides effectiveness 60 percent of the time against the deadliest pneumonia pneumococcus and meningitis. However, people under 65 who have chronic illnesses or weakened immune systems should discuss with their doctor a potential repeat vaccination every five to 10 years [9].
- An **Ace inhibitor (ACEI),** such as Lisinopril to help prevent the conversion of angiotension two, especially in the larger blood vessels, which causes vasoconstriction, may be prescribed. ACEI are ideal in diabetic patients to enhance vasodilatation, especially to the kidneys. The JNC 7 and the ADA recommend that the majority of patients with diabetes require two or more antihypertensive agents from different classes. Research has demonstrated that combining agents with two different mechanisms of action can result in an additive blood pressure lowering effect and may permit for lower doses of each agent to be used, possibly decreasing the potential for dose-related side effects. Furthermore, the National Kidney Foundation recommends that patients with chronic kidney disease (including albuminuria and/or nephropathy), should be treated with an ACEI and/or Angiotensin receptor blockers (ARB) (prevent the conversion in the smaller arterioles) in combination with a diuretic.
- **Baby aspirin** up to 325mg every day.
- Due to the coinciding hyperlipidemia/dyslipidemia, the third report of the expert panel on detection, evaluation and treatment of high blood cholesterol in adults (Adult Treatment Panel III) (ATP) and the national cholesterol education program (NCEP) recommend that the lipids should be controlled. The LDL cholesterol goal of therapy for most persons with diabetes should be less than 100 mg/dL. When LDL cholesterol levels are in the range of 100-129 mg/dL at baseline or on treatment, several therapeutic options are available [70]:
  - Increasing intensity of LDL-lowering therapy.
  - Adding a drug to modify atherogenic dyslipidemia (fibrate or nicotinic acid).
  - Intensifying control of other risk factors including hyperglycemia.

  If triglyceride levels are greater than 200 mg/dL, non-HDL cholesterol becomes a secondary target of cholesterol-lowering therapy.

  See Table 2- Drugs affecting Lipoprotein Metabolism [69]

  According to the AHA (2007), children (boys over 10 and girls after menarche) should also be placed on drug therapy if after a six- to 12-month trial of fat and cholesterol restricted dietary management [45]:
  1. LDL is greater than 190 mg/dl.
  2. LDL is greater than 160 mg/dl and there are a positive family history of premature cardiac history and two other risk factors.

  The goal is to maintain the LDL less than 110 mg/dl to 130 mg/dl with a statin (HMG CoA reductase inhibitor).

Medication modalities for the type 1 diabetic

Patients living with Type 1 Diabetes are reliant on insulin for the rest of their lives. Failure to administer exogenous or endogenous insulin injections will result in death. Other patients who may be required to receive insulin include[53]:

- People with type 2 diabetes who are unable to control their glucose levels with oral antidiabetic drugs and/or diet.
- People with type 2 diabetes, who at the time of diagnosis are unable to achieve glycemic control, especially if blood glucose values are greater than 250 or 300 (depending on the health care provider), AIC greater than 10 or the patient initially presented in a DKA state [25].
- People with diabetes who are experiencing physical stress (infection, surgery or corticosteroid therapy) and are unable to control their blood glucose levels between 110 and 180 mg/dl per the ADA.
- People with type 2 diabetics who are already taking two oral agents but are unable to maintain glycemic control.
- Pregnant women, regardless of whether they have a previous history of any form of diabetes or GDM.
- People with DKA or HHS.
- People who are receiving high-calorie tube feedings or parenteral nutrition.

Insulin is derived from animals (pork pancreas) or synthesized in the laboratory from either an alteration of pork insulin or recombinant DNA technology, using strains of Escherichia coli (E.coli) to form biosynthetic human insulin. Insulin is an endogenous hormone, secreted by the beta cells of the pancreas to lower the blood glucose levels by stimulating glucose passage across the cell membranes and uptake into the cells [25]. In addition, insulin also promotes the conversion of glucose to glycogen and inhibits hepatic glucose production from glycogen [25]. It should be noted that there are different types of insulin and it should be prescribed on an individual basis.

Table 2: Drugs Affecting Lipoprotein Metabolism [69]:

Drug Class	Agents and Daily Doses	Lipid/Lipoprotein Effects	Side Effects	Contraindications
HMG CoA reductase inhibitors (statins)	Lovastatin (20-80 mg), Pravastatin (20-40 mg), Simvastatin (20-80 mg), Fluvastatin (20-80 mg), Atorvastatin (10-80 mg), Cerivastatin (0.4-0.8 mg)	LDL-C ↓18-55% HDL-C ↑5-15% TG ↓-30%	Myopathy Increased liver enzymes	Absolute: -Active or chronic liver disease Relative: -Concomitant use of certain drugs*
Bile acid Sequestrants	Cholestyramine (4-16 g) Colestipol (5-20 g) Colesevelam (2.6-3.8 g)	LDL-C ↓15-30% HDL-C ↑3-5% TG No change or increase	Gastrointestinal distress Constipation Decreased absorption of other drugs	Absolute: -dysbeta-lipoproteinemia -TG greater than 400 mg/dL Relative: -TG greater than 200 mg/dL
Nicotinic acid	Immediate release (crystalline) nicotinic acid (1.5-3 gm), extended release nicotinic acid (Niaspan ®) (1-2 g), sustained release nicotinic acid (1-2 g)	LDL-C ↓5-25% HDL-C ↑15-35% TG ↓20-50%	Flushing Hyperglycemia Hyperuricemia (or gout) Upper GI distress Hepatotoxicity	Absolute: -Chronic liver disease -Severe gout Relative: - Diabetes -Hyperuricemia -Peptic ulcer disease
Fibric acids	Gemfibrozil (600 mg BID) Fenofibrate (200 mg) Clofibrate (1000 mg BID)	LDL-C ↓5-20% (may be increased in patients with high TG) HDL-C ↑10-20% TG ↓20-50%	Dyspepsia Gallstones Myopathy	Absolute: -Severe renal disease -Severe hepatic disease

Insulins are available in rapid-acting, short-acting, intermediate-acting and long acting preparations. (See Table 3- Insulin Preparation).

Once an individual has been diagnosed with type 1 diabetes, it is important to discuss the potential options of administering insulin since it is a lifelong commitment. Insulin therapy can be administered in one of the following manners:

1. Subcutaneous shot; dispensed in 100 units per ml (u/ml). For infants and toddlers, a pharmacist may prepare a diluted insulin preparation to ensure a smaller dosage [25].

Table 3- Insulin Preparation [25]:

Preparation	Name	Onset (hours)	Peak (hours)	Duration (hours)
Rapid acting or ultra-shorting acting	Lispro (Humalog)	Less than 15 minutes (typically 5 to 10 minutes)	30 to 60 minutes	3-4
Short acting	Regular Regular Humulin (R) Regular lletin II Velosulin	0.5-1.0	2-3	4-6
Intermediate acting	Lente Humulin (L) Lente lletin II NPH Humulin (N) NPH lletin II NPH	2	6-8	12-16
Long acting	Ultralente (U) Lantus	2 No onset	16-20 No peak	24+ 24
Combinations	Humulin 50/50 Humulin 70/30 Novolin 70/30	0.5 0.5 0.5	3 4-8 4-8	22-24 24 24

- Single daily injection is typically compromised of intermediate-acting insulin or a combined short and intermediate.
- Two-dose protocol injections are combined of short and intermediate-acting insulin injected twice a day.
  - Two-thirds of the dose (intermediate acting and regular insulin in a two-to-one ratio) is administered before breakfast; one-third is administered before the evening meal (intermediate acting and regular insulin in a one-to-one ratio).
- Three-dose protocol.
  - Combination of short and intermediate-acting before breakfast, short-acting insulin administered before evening meal, and intermediate-acting insulin administered at bedtime to lower fasting and after-breakfast blood glucose levels. In addition, it typically prevents a night-time hypoglycemic event.
- Four-dose protocol.
  - Short acting insulin 30 minutes before meals to provide the greatest amount of insulin to be present when needed.
  - Basal insulin is provided twice a day with an intermediate-acting or once-a-day injection of long-acting insulin.

If the patient is required to mix insulins, it is important for the nurse to convey the importance of compatibility to avoid changing the "expected" peak times. Mixing short- and intermediate-acting insulins produce normal blood glucose levels.

2. Alternative methods to administering insulin [19]:
   - **Basal-bolus therapy**, in which it is administered once a day using glargine (a pre-filled lightweight syringe), then a bolus of rapid-acting insulin is administered with each meal and snack.
     - Avoid if the patient has any visual or neurological impairments.
   - **Continuous subcutaneous insulin infusions** (CSII) "pump therapy" is becoming popular among children and teenagers.
     - **Advantages of the CSII:**
       1. CSII are more effective in controlling blood glucose levels than a multiple injection schedule.
       2. Externally worn pump that contains a syringe and reservoir with rapid or short-acting insulin and is connected to the patient via an infusion system.
     - **Disadvantages of the CSII:**
       1. Site infections occur if the infusion site is not cleaned or the needle is not changed every three days.
       2. Hypoglycemia may result if the patient is receiving rapid-acting insulin and has a normal blood glucose level.
       3. Higher risk of developing ketoacidosis if the patient does not adhere to the CSII recommendations due to infection, noncompliance or infusion obstruction.
   - **Implanted insulin pumps** are implanted in the peritoneal cavity where insulin is absorbed by local blood vessels, similar to natural insulin release. The pump provides a reservoir re-filled with 400 units of insulin every one to two months. Complications that may arise include catheter blockage, pump failure and subcutaneous inflammation in the subcutaneous pockets.

Nursing considerations for the patient taking insulin [75,78]:

- Educate the patient on the purpose of their insulin (to emulate their nonfunctioning pancreas) and emphasize that it is required to sustain life.
- Educate the patient about the various types of insulin and the insulin prescribed by their physician. If the patient is prescribed more than one type, reinforce that changing insulins may affect their blood glucose and should be done under only the supervision and guidance of their prescribing provider.
- Educate the patient about the technique and sites.
  - **Technique**. The patient should be instructed to inject the insulin in the subcutaneous tissue at a 90-degree angle. In contrast, if it is a thin patient, inject at a 45-degree angle and avoid intramuscular (IM) since it has a faster absorption. It is not necessary to aspirate the blood.
  - **Sites**. The patient should always rotate the sites to prevent day-to-day changes in absorption and to prevent the development of lipohypertrophy (increased fat deposits in the skin) or lipoatrophy (loss of fatty tissue). Absorption is faster in the abdomen, followed by the deltoid, thigh and buttocks.
- Educate the patient about insulin storage, disposal and syringes. According to the ADA, the following guidelines are recommended [14]:
  - **Storage**. Although manufacturers recommend storing your insulin in the refrigerator, injecting cold insulin can sometimes make the injection more painful. To counter the reaction, many providers suggest storing the bottle of insulin you are using at room temperature; it will last one month. Always put the date and time that the insulin was opened, and check the expiration date to avoid administering insulin that has already expired.
  - **Syringe re-usage**. Ideally, it is better to always use new, clean insulin syringes and needles. However, the ADA says that many people safely reuse their insulin syringes. But it should be avoided if the patient is ill, has any open wounds on the hands or has poor resistance to infection. If the patient will re-use the needle due to financial constraints, instruct the patient to always keep the needle and syringe clean by recapping the needle when it is not in use. Syringe makers will not guarantee the sterility of syringes that are reused. The patient should never use another person's syringe or allow another person to use their personal syringe.
  - **Syringe disposal**. Always dispose of the needles appropriately and safely to avoid anybody else picking up a used needle. The patient should place the needle or entire syringe in an opaque (not clear), heavy-duty plastic bottle with a screw cap or a plastic or metal box that closes firmly.
- In the elderly patient or one with a sensory impairment, it is important to ensure the patient is safe while administering their insulin.

In 2005, the FDA approved, Symlin, an injectable medicine to control blood sugar for adults with type 1 and type 2 diabetes.

Symlin is to be used in addition to insulin therapy in patients who cannot achieve adequate control of their blood sugars on intensive insulin therapy alone [42]. Since the approval in 2005, Symlin is to be used only in combination with insulin to help lower blood sugar during the three hours after meals. Symlin has a medication guide (FDA-approved patient labeling) and a risk minimization action plan (RiskMAP) due to three areas of concern.

- First, the principle risk associated with Symlin therapy is hypoglycemia, and this risk is greatest in patients with type 1 diabetes and in patients with gastroparesis (motility problems of the stomach, a long-term complication of diabetes).
- Second, the potential for medication errors, specifically mixing of Symlin with insulin in the same syringe, which can alter the activity of the insulin, is addressed in the medication guide and in physician labeling.
- Finally, the potential for off-label use in patients where the benefit/risk profile has not been characterized or demonstrated is also a concern and will be monitored by the sponsor.

Symlin should not be used if patients cannot tell when their blood sugar is low, have gastroparesis (slow stomach emptying), or are allergic to pramlintide acetate, metacresol, D-mannitol, acetic acid or sodium acetate. Side effects associated with Symlin include but are not limited to nausea, vomiting, abdominal pain, headache, fatigue and dizziness. Symlin has not been evaluated in the pediatric population [42].

Medication modalities for the Type 2 diabetic

The major goal for treating type 2 diabetic patients is controlling the blood glucose level and HbA1c levels, decreasing weight, increasing exercise, normalizing lipid profiles and blood pressure and preventing complications [25].The treatment laboratory goal for type 2 diabetes is to maintain an HbA1c less than 7 percent and a fasting and pre-prandial blood glucose of 70-130 mg/dl [45]. According to Clinical Diabetes (2002), blood glucose control has been shown to decrease the risk of macrovascular and microvascular complications of type 2 diabetic patients. At this time, it is unknown whether the blood glucose control decreases the risk of cardiovascular mortality, however, the United Kingdom Prospective Diabetes Study (UKPDS) suggests that good glycemic control probably does decrease cardiovascular risk in patients with Type 2 Diabetes [28].

Typically, the initial treatment for type 2 diabetes begins with education on changing the diet and exercising to lower the blood glucose level. In addition, many health care providers typically initiate a single oral diabetic, such as Metformin, as it helps lower the blood glucose and decrease weight. Children are even started on Metformin once they are weaned off insulin if they were initially placed on it due to a hyperglycemic state or DKA [25].

Metformin improves the sensitivity of target cells to insulin, slows the gastrointestinal absorption of glucose and reduces the hepatic and renal glucose production. The dosage can be gradually increased to improve metabolic control. It should only be prescribed under the following conditions:

- Normal liver and kidney function, and no ketosis present.

If Metformin is not well tolerated and/or the patient does not achieve euglycemia and another medication needs to be added, sulfonylurea or meglitinides may be used. However they are not approved for children in the U.S. due to liver toxicity [25].

If the HbA1C is equal or greater than 7 percent, the ADA and European Association (2007) recommends adding either basal insulin (the most effective) or a sulfonylurea (least expensive) [45]. It is important to note that the treatment should be customized to the patient's risk for hypoglycemia, the very young or older age, end-stage renal disease, advanced cardiovascular or cerebrovascular disease and the life expectancy [45].

At this time, there are several distinct classes of antidiabetic (hypoglycemic agents) available, each displaying unique pharmacologic properties designed to correct one or more of the metabolic abnormalities. The patient is typically started at the lowest dose, and then increased every one to two weeks until the patient reaches an acceptable blood glucose level [19]. These classes are the dipeptidyl peptidase IV inhibors (DPP-4), sulfonylureas, meglitinides, biguanides, thiazolidinediones and alpha-glucosidase inhibitors [10,68,82,83,84]:

- **DPP-4** inhibitor sitagliptin was approved by the FDA in 2006. These drugs prolong action of incretin hormones. Sitagliptin can use used as a monotherapy or in combination with metformin or a glitazone. In 2009 the DPP-4 inhibitor saxagliptin was approved and linagliptin in May, 2011. As of this writing, another DPP-4 inhibitor, vildagliptin, is currently under FDA review. DPP-4 inhibitors do not cause the weight gain that other classes (e.g. sulfonylureas) may instigate.
- **Sulfonylureas (SU)** are insulin secretagogue's, meaning they force the pancreas to increase insulin production. Therefore SU's are reserved only for patients with some remaining pancreatic beta cell function. The anticipated reductions while taking a sulfonylurea is a 0.8 to 2.0 reduction in the HbA1c and 60 to 70 points lower on the FBG. It is important that the nurse monitors the renal function and speaks with the doctor before administering as there is a crossover for patients with sulfa allergies. In addition, the nurse should educate the patient that the drugs are potentially photosensitizing and hypoglycemia may occur. Hypoglycemia is more likely to occur with Diabinese and Novo-Propamide, due to their longer duration of action. In addition, underweight older adult patients with cardiovascular, liver or kidney impairments are susceptible.
  - **First generation**: acetohexamide (Dymelor), chlorpropamide (Diabinese, Novo-Propamide), tolazamide (Tolinase), tolbutamide (Orinase).
  - **Second generation**: glyburide (Micronase/DiaBeta), gipizide (Glucotrol), glimepiride (Amaryl). The second generations are used more frequently than the first generation medications because of a higher risk of hypoglycemia with the first-generation medications.

- **Biguanides** are insulin sensitizers as they reduce hepatic glucose output. The anticipated reductions while taking a Biguanide is a 1.5 to 2.0 reduction in the HbA1c and lowering the FBG by 50 to 70 points. It is important to monitor the creatinine level and to avoid if the creatinine level rises above 1.4. The nurse needs to educate the patient to avoid taking the medication and to inform their physcian prior to any radiocontrast use and/or surgery on the day of the procedure and for 48 hours post-recovery. The major risks that may occur if the patient continues the medication is dehydration, impaired renal function and hypovolemia because everything is going through the kidneys, leading to a condition called lactic acidosis. In addition, the nurse should be conscious about signs and symptoms of those conditions, even if the patient is not scheduled for a procedure with radiocontrast and/or surgery. Hypoglycemia may occur if the patient is taking a coinciding SU and/or insulin.
    - Metformin (Glucophage) is one of the most common, first-line medications prescribed for type 2 diabetics, metabolic syndrome and/or PCOS because it has amazing results in reducing weight and inducing ovulation. Patients should be educated that diarrhea is a common complaint once the medication is initiated, but it typically resolves. Regardless, the patient should inform their prescribing health care provider to avoid dehydration.
- **Meglitinides** are similar to the SU agents due to their typical short-acting insulin secretagogues. The anticipated reductions while taking a meglitinide is a 1 to 1.5 percent in the HbA1c. The goal of meglitinides is a reduction in the postprandial glucose level as it helps with the absorption of carbohydrates while eating. Therefore, the patient should be instructed to take it one to 30 minutes before eating. During the meal, the medication provides a quick insulin burst approximately 20 minutes after swallowing the pill. Meglitinides should be strongly encouraged if the patient has difficulty managing postprandial blood glucose levels. The main concern is to use with caution in patients with renal or hepatic impairments. Again, similar to SU agents, the major side effect is hypoglycemia, especially with Starlix. Therefore, if the patient skips a meal, he or she should not take a scheduled dose of Starlix to avoid hypoglycemia.
    - Repaglinide (Prandin), nateglinide (Starlix).
- **Thiazolidinediones (TZD)** are insulin sensitizers as they work by promoting glucose utilization in the muscles and tissues. The anticipated reduction while taking a thiazolidinediones is 1 to 2 percent in the AIC. TZD's are potent drugs on the liver; therefore the nurse should monitor the alanine aminotransferase (ALT) before administering the dose as there is a rare risk of hepatic toxicity. In addition, there is a risk of edema, especially if the patient is on a SU or insulin. Therefore, do not initiate it if there are any signs and symptoms of heart failure. If the patient has any cardiovascular risks (as many diabetics do), TZDs should not be used with nitrates as it may exacerbate the risk of developing edema or heart failure. TZDs are not an ideal medication to administer to a female patient taking an oral contraceptive (OC) as it decreases the effectiveness. Another unique feature is the onset of action is delayed; it requires up to 12 weeks of use before attaining the maximum therapeutic level.
    - Pioglitazone (Actos), rosiglitazone (Avandia)

      In November 2007, the FDA added a box warning for heart-related risks, especially heart attacks in patients taking Avandia. The FDA recommends that patients with type 2 diabetes who have underlying heart disease or who are at high risk of heart attack should talk with their health care provider about the revised warning as they evaluate treatment options. FDA advises health care providers to closely monitor patients who take Avandia for cardiovascular risks. In August 2007, the FDA warned that Avandia may also worsen heart failure in some patients as well [41].
- **Alpha-glucosidase inhibitors** delay the intestinal carbohydrate absorption by reducing postprandial digestion of starches via enzyme action inhibitions, and it helps the dumping effect of carbohydrates. Therefore, the risk of developing hyperglycemia after meals is reduced since the intestinal absorption and digestion of carbohydrates is reduced. The anticipated reduction in the HbA1c is 0.3 to 0.9 percent. Hypoglycemia is rare unless administered with a SU or insulin.
    - Acarbose (Precose), miglitol (Glycet)

Before the primary care provider chooses an appropriate oral agent, the patient's history, age, blood sugar levels, HbA1c levels and costs are considered to ensure the patient will be able to comply with the medication regimen. If the primary care provider is considering the cost alone, SUs are the least expensive, taken once a day and have few side effects [19].

Byetta is an injectable drug that was approved by the FDA in 2005 as adjunctive therapy to improve blood sugar control in patients with type 2 diabetes who have not achieved adequate control on Metformin and/or a SU. However, since 2007, Byetta has been on the FDA list and has received black box warnings. Starting in 2007, FDA reviewed 30 postmarketing reports of acute pancreatitis in patients taking Byetta. At that time, the FDA encouraged health care professionals to educate patients taking Byetta to seek prompt medical care if they experience unexplained persistent severe abdominal pain, which may or may not be accompanied by vomiting. If pancreatitis is suspected, Byetta should be discontinued. If pancreatitis is confirmed, Byetta should not be restarted unless an alternative etiology is identified. In October 2007, the FDA received reports of six cases of hemorrhagic or necrotizing pancreatitis in patients taking Byetta [43].

Treatment of gestational diabetes

Any woman with diabetes contemplating a pregnancy should ideally discuss it with her health care provider before conception. The woman with type 1 diabetes will continue on her insulin, while the type 2 diabetic may discontinue her oral antidiabetic

agents or begin taking insulin throughout the pregnancy [77]. A pregnant woman with diabetes requires prompt, adequate treatment to normalize her maternal blood glucose levels and to avoid complications for the infant. The fetal risks are the same for women with all forms of diabetes (type 1, type 2 and GDM); however maternal risks are greater in women with type 1 diabetes [77]. This is because Type 1 creates erratic blood sugar control because of the absolute lack of insulin production and because women are more prone to have vascular, retinal or renal complications. Therefore, to prevent morbidity of the women and fetus, very aggressive treatment is necessary [48].

Although maternal and fetal morbidity and mortality rates have significantly decreased over the years, the risks of developing complications still exist. Research has demonstrated the most common complications that occur [27, 77]:

1. **Maternal**
    - **Poor glycemic control** around the time of conception and in the early weeks of pregnancy may be associated with an increased incidence of early pregnancy loss in women with a history of diabetes.
    - **Pre-eclampsia or eclampsia** is exacerbated (four times) in women with diabetes.
    - **Hypertensive disorders**, such as pre-clampsia or eclampsia, occur more frequently in women with a prior history of diabetes, especially if she has coinciding renal dysfunction.
    - **Hydramnios (polyhydramnios)** (increased amniotic fluid, greater than 2000 milliliters) (ml) occurs more frequently in diabetic patients, causing an overdistention of the uterus. The overdistention of the uterus leads to additional risks, such as increases in the compression of the maternal abdominal blood vessels (vena cava and aorta), leading to hypotension while in the supine position; premature rupture of membranes (PROM); preterm labor; and postpartum hemorrhage.
    - **Infections** are exacerbated due to disorders of carbohydrate metabolism that alters the body's normal resistance to infection. Although infections are prevalent with any form of diabetes, it is increased due to the pregnancy. It is important for nurses to educate the pregnant woman about the risks of infections and other problems that lead to further complications:
        - **During the pregnancy**, urinary tract infections (UTI) increase the risk of pre-term labor.
        - **After the pregnancy**, postpartum infections.
    - **Ketoacidosis** typically occurs more frequently in the second and third trimesters when the diabetogenic effect is the greatest. If the pregnant woman has coinciding risks, such as stress or infection, the risk for DKA is exacerbated.
2. **Infant/neonatal**
    - **Congenital anomalies** (occurs in 6 to 10 percent of deliveries) of infants; typically cardiac defects are the most common.
    - **Macrosomia** (large infant). Although the pancreas is working overtime to produce more insulin, it is not lowering the blood glucose levels in the woman's body, and all of the extra blood glucose is being transported through the blood brain barrier into the placenta for the baby. The baby is unable to metabolize or excrete the extra blood glucose; thus the mom and nurses caring for the baby can expect the baby will continue to get larger (macrosomia). Macrosomia infants tend to have disproportionate increases in the shoulder and trunk, leading to another consequence: shoulder dystocia. Poor glycemic control in the later portion of the pregnancy, especially in women with a history of coinciding vascular disease, increases the risk of macrosomia. Macrosomia occurs in 25-40 percent of diabetic pregnancies. In addition, at birth, the nurse can expect the following:
        - Baby will develop hypoglycemia because its little body is used to the extra glucose.
        - Difficulty breathing due to the increase weight.
    - Other problems that cause neonatal morbidity includes:
        - Spontaneous abortion (two times the risk) in diabetic women.
        - Macrosomia.
        - Hypoglycemia.
        - Respiratory distress syndrome (RDS).
        - Polycythemia (increased hematocrit level).
        - Hyperbilirubinemia (jaundice).

To prevent the potential risks and complications, the nurse needs to educate the woman and her partner about her diabetes (including the disease process, prevention, treatment and possible complications). During the first and second trimesters of pregnancy, the diabetic woman should see her provider every one to two weeks, rather than monthly as for a nondiabetic woman. It is important to establish and convey the importance of collaborating together to recognize early signs of any potential problems to avoid complications. The overall goal is to achieve and maintain euglycemia for a pregnant diabetic woman in the range of 60 to 120 mg/dl [77]. In order to maintain euglycemia, the diabetic woman needs to comply with the combination therapy of checking the blood glucose levels, diet, insulin and exercise throughout her pregnancy [77]:

- **Blood glucose measurements** are completed frequently to assess compliance of the medical regimen throughout the day, such as before breakfast, lunch and dinner. Typically in a woman with true gestational diabetes, fasting blood glucose levels will be normal, while the postprandial blood glucose levels are elevated [27]. The rationale behind this concept is related to the metabolism of large carbohydrate boluses rather than carbohydrate intolerances at the baseline levels [27].
- **Diet** is individualized to the patient based on the blood (not urine) to allow for increased fetal and metabolic requirements.
    - Energy needs are based upon 30-35 calories per kilogram of the ideal body weight with an average of 2,200 calories (first trimester) to 2,500 calories (second and third trimesters).
    - Carbohydrates, protein and fat are important to balance with approximately:
        - 50 to 60 percent of the total calories being carbohydrates (minimum of 250 g per day). Limit simple carbohydrates and encourage complex carbohydrates that are high in fiber to regulate the blood glucose level by releasing more glucose. It may be easier for the pregnant woman to count carbohydrates at meals, educating her to ingest 30 to

45 grams of carbohydrates at breakfast, 45 to 60 grams at lunch and dinner, then 15 grams for snacks [27].
    - 12 to 20 percent should be protein.
    - 20 to 30 percent from fat, with less than 10 percent from saturated fats.
- **Exercise** regimens should be individualized to the patient with the exact protocol per the physician. However, most encourage walking for 15 to 30 minutes four to six times a week about 30 to 40 minutes after eating to enhance the postprandial blood sugar levels [27].
    - A woman with vasculopathy should be encouraged to do only mild exercise to prevent the risk of injury to the placenta.
- **Medications:** Since 2002, the American College of Gynecology (ACOG) and the AAFP has recommended that women with GDM be treated initially with an adequate, nutritious diet designed to achieve normal glycemic levels and to avoid ketoacidosis [71]. There are various perspectives about treating a woman with GDM with insulin or oral agents. However, in January 2009, a meta-analysis was published in Obstetrics and Gynecology based on research conducted over the previous years that did not demonstrate any significant differences in maternal glycemic control, infant birth weight, neonatal hypoglycemia or congenital malformations [73].
    1. Insulin is imperative to maintain euglycemia and proper metabolism of glucose. In addition, if the previous medical regimen in the type 1 and type 2 diabetic changes, the patient requires adequate education to avoid confusion and potential frustration for the pregnant woman. The following insulins are recommended [77]:
        - The woman will be started on short-acting insulin (Humalog/Lispro or Novolog) in combination with intermediate-acting insulin (NPH) in the morning to cover breakfast and lunch; then short-acting insulin at dinner (Humalog/Lispro or Novolog) [27]. Humalog is ideal for GDM patients to better control the postprandial blood sugar levels with less risk of hypoglycemia developing [27].
        - Several trials have demonstrated a reduced risk of fetal macrosomia if the mother is treated with insulin during the pregnancy. Although insulin treatment is commonly prescribed in GDM, only 9 to 40 percent of treated mothers benefit. Treatment aims to achieve glucose levels of 130 mg per dL one hour postprandial [27].
    2. Oral hypoglycemic agents may be administered if the glucose levels are lower. In one study, Glyburide provided outcomes comparable to those achieved with insulin in patients with GDM who had failed to achieve adequate glycemic control with diet alone [71].

After pregnancy the woman should continue to adhere to eating healthy, exercising and controlling her weight because research has demonstrated that 5 to 10 percent of women with gestational diabetes are found to have type 2 diabetes. In addition, women who have had gestational diabetes have a 20 to 50 percent chance of developing diabetes in the next five to 10 years [48, 75].

Surgical treatment options for diabetic patients

Many nurses may not be aware of the surgical treatment modalities that are available for the diabetic patient as there is little discussion of surgical modalities in recent, published nursing textbooks. However there are various sites available online on recent research studies conducted over the past few years that are bringing hope and promise for the future for patients with diabetes.

According to the Science Daily (March 2008), growing evidence shows that surgery may effectively cure Type 2 diabetes based upon research conducted recently [65]. Since 2005, a study published in Diabetes Care (2005), compared laparoscopic adjustable gastric banding (LAGB) and conventional diet (no-LAGB) in the prevention and remission of type 2 diabetes, hypertension and obesity over a four-year period [18,61]. In the study, there were 122 patients, and 73 chose to have the LAGB surgery. The results were as follows [18, 61]:

- In the primary intervention study, body weight, A1C, and systolic and diastolic blood pressure significantly decreased in the LAGB but not in the no-LAGB group.
- In the secondary intervention study, body weight, A1C, and systolic and diastolic blood pressure significantly decreased in the LAGB group but not in the no-LAGB group. Remission occurred in 45 percent of the LAGB patients and 4 percent of the no-LAGB patients. In addition, remission of arterial hypertension occurred in 51 percent of LAGB patients and 4 percent of no-LAGB patients.

Since 2005, the ADA also has information in regards to a research study published in the Journal of Clinical Endocrinology and Metabolism (2005), focusing on the satiety after achieving the LAGB surgery for 23 patients. The results were as follows [39]:

- Of 23 LAGB patients who attempted the protocol, 17 completed two breakfast tests. Five patients were excluded for failing to consume the meal adequately, three due to regurgitation of food and two due to delayed consumption. These patients were presumably too restricted by their bands to allow passage of the test meal.
- All of the patients became increasingly hungry preprandially, experienced maximal satiation immediately after the meal, and experienced decreasing satiety thereafter.

In 2008, the Journal of American Medical Association (JAMA) conducted a two-year research study on patients recently diagnosed as a type 2 diabetic with a BMI of 30 to 40. The patients were randomly assigned to receive conventional medical/behavioral therapy (medical therapy and a focus on weight loss through lifestyle modification) or LAGB, plus conventional medical/behavioral therapy. The results were amazing [33]:

- Complete remission of diabetes was achieved in 73 percent of the type 2 diabetics who underwent the LAGB due to more weight loss (20.7 verses 1.7 in the medical/behavioral group). The percentage of weight loss generally required for

diabetes resolution was 10 percent, which was achieved in 86 percent of the surgical patients but in only 1 patient in the medical group.
- Complete remission of diabetes was only 13 percent of those in the medical/behavioral therapy group.
- No serious surgical complications were reported.

Although the research studies exemplify promising news for the future of diabetes, especially for the morbidly obese and/or patients with co-existing hypertension, as the ADA indicates, there are limitations to the studies. The majority of the research conducted is based on a limited number of patients, and further research needs to be conducted [18].

According to the American Society for Bariatric Surgery, LAGB is a relatively safe procedure that has been around for almost 30 years. Over the past three decades, the procedure has been enhanced [23]:
- Currently, the LAGB consists of an inflatable balloon that is connected to a silicone band. The surgeon controls the amount and degree of inflation or deflation of the balloon, which can always be adjusted as needed. Inflation of the balloon functionally tightens the band and thereby increases weight loss, while deflation of the balloon loosens the band and reduces weight loss. These bands can be inserted laparoscopically, thereby reducing the complications and discomfort of an open procedure.
- LAGB is a procedure that induces weight loss solely by the patient restricting the amount of food. Typically, 28 to 65 percent of patients lose excessive weight within two years, and 54 percent at five years. However, in order for the surgery to be effective, the patient needs to be compliant with the strict diet and frequent follow-up appointments for band adjustments. Another unique feature is LAGB is a reversible procedure that does not carry the risks of nutritional and mineral deficiencies of other bariatric procedures.

According to the American Society for Bariatric Surgery, remission of diabetes with LAGB is seen in 64-66 percent of patients at one year. At this time, long-term results comparing LAGB with the traditional gastric bypass surgery (surgical incision in the abdomen to make the stomach smaller by creating a small pouch at the top of the stomach using surgical staples or a plastic band) are not yet available. The sleeve gastrectomy is another potential surgical procedure, similar to the LAGB and gastric bypass surgery. During this procedure, the surgeon creates a small, sleeve-shaped stomach that is larger than the stomach pouch created during the Roux-en-Y bypass (traditional gastric bypass surgery) and is about the size of a banana. Sleeve gastrectomy is typically considered as a treatment option for bariatric surgery patients with a BMI of 60 or higher [26].

There are other surgical procedures and transplants that have been implemented to potentially to improve the quality of life for the diabetic patient.

Whole pancreas transplantation can be performed in one of the following manners [49]:
- Transplant of the pancreas alone (PTA).
- Transplant of the pancreas and kidney (PAK).
- Simultaneous pancreas and kidney transplant (SPK), ideal for diabetic patients with uremia.

According to the ADA, whole pancreas transplantation is ideal for type 1 diabetic patients with the HLA genetic composition, because it "tricks" the body into accepting the donor organ recipient. Patients with a transplanted organ must take immunosuppressive drugs in order to prevent the immune system from fighting the new organ, and the side effects of these drugs may be worse than the problems caused by diabetes; the operation itself is serious. According to the ADA, one to two people in 10 die within a year of getting a pancreas transplant [17]. On a positive note, if the transplantation works and the body accepts the organ, the patient no longer has diabetes and is unlikely to get it again. Therefore, the patient does not require insulin shots and frequent blood glucose testing. The ADA has suggested that euglycemia levels may prevent further complications or any current co-morbidities from worsening, although many more studies are needed.

Unfortunately, there are not enough cadaver pancreases to go around because not enough people sign up to be organ donors, and each pancreas must meet strict guidelines. When a whole cadaver pancreas is not available, a person can receive a portion of a pancreas from a living relative.

Anytime a patient with diabetes is receiving a kidney transplant from a living relative, it is usually beneficial to perform a partial pancreas transplant at the same time. Since the transplanted kidney will become damaged by diabetes over time, transplanting a partial pancreas from the same donor will help control blood glucose levels and protect the new kidney from further damage. Transplant success seems higher when patients and donors are matched for HLA types, and a pancreas transplanted along with a kidney is less likely to fail than a pancreas transplanted alone.

The ADA website mentions a recent study conducted by JAMA (2003) indicating patients with functioning kidneys who therefore decline the PAK option have survival rates that are worse than those of patients who manage their diabetes with conventional therapy (insulin, diet, etc.). Therefore, the decision to have a pancreas-only transplant should be very carefully considered by both the patient and physician. Because of the lower survival rates seen with pancreas-only transplants and because a pancreas transplanted along with a kidney is less likely to fail than a pancreas transplanted alone, pancreas transplants are nearly always done only in people with type 1 diabetes who are getting or already have a transplanted kidney. Remember that pancreas transplants work only for people with type 1 diabetes. The major problem in people with type 2 diabetes is not a failing pancreas, but the body's inability to respond to insulin in the right way.

It is important to realize there are many options for the patient, but there also are precise risk factors and history to consider before pursuing any options. The nurse should encourage the patient to discuss the most feasible with his or her primary care provider and consulting surgeon.

All diabetic patients are at risk of developing acute complications related to their diabetes. However, each realistic, potential acute complication is treatable and preventable with appropriate education and knowledge.

Typically, patients will experience hyperglycemia with any of the following occurrences [75]:

- Caloric intake exceeds their daily allowance (1500 to 2000 calories/day).
- Missing a dose of insulin and/or oral antidiabetic agents.
- Stress and illness causes the release of hormones, such as epinephrine, cortisol, growth hormones and glucagon. The diabetic patient is unable to compensate for the fluctuation and changes in the various hormones being released.

The symptoms of hyperglycemia include the "three polys," blurred vision headache, lethargy, abdominal pain, ketonuria (if type 1 diabetic) and/or a coma. It is imperative that nurses educate their patients about hyperglycemia to ensure the patient is aware of how to prevent, recognize and treat the problem. However, anytime a patient has hyperglycemia, it is always important to consider two other potential life-threatening conditions, DKA, typically found in type 1 diabetics, and hyperglycemic-hyperosmolar nonketotic syndrome (HHNS), typically found in type 2 diabetics. Although hyperglycemia, DKA and HHNS all have hyperglycemia in common, always remember the following:

- Hyperglycemia can occur without DKA or HHNS.
- DKA will have a blood glucose greater than 300 mg/dl and positive urine ketones.
- HHNS will have higher blood glucose, typically greater than 600 mg/dl, and no urine ketones.

DKA will be elaborated upon with hyperglycemia since the treatment modalities are similar, and then HHNS will be discussed.

1. **Diabetic Ketoacidosis (DKA)** is a complication of hyperglycemia that develops when there is an absolute deficiency of insulin and an increase in the insulin counterregulatory hormones specific to patients with type 1 diabetes during physical or emotional stress despite continued insulin therapy [51]. DKA is a common and potentially life-threatening condition that occurs primarily in children (20 to 40 percent) [25]. Research has demonstrated that the most common causes of DKA include incorrect or missed insulin doses, inaccurate way of administering the insulin, illness, trauma or surgery [25].

   DKA (insulin deficiency) is accompanied by an increase in hormones, such as epinephrine, norepinephrine, cortisol, growth hormones and glucagon) that are released when there is not enough glucose delivered into the cells [25]. DKA occurs due to the muscle cells breaking down protein into amino acids that are converted to glucose by the liver, leading to hyperglycemia. The increase in adipose tissue releases fatty acids that are transformed by the liver into ketone bodies [49, 51, 75].

   The onset of DKA is typically sudden, and the patient will initially exhibit signs of dehydration (polyuria, polydipsia) that will exacerbate the hyperosmolality process by producing symptoms of anorexia, nausea and vomiting (metabolic acidosis) [25, 53]. The patient will also have positive ketones excreted in the urine. The increased amount of ketones circulating will cause the patient to blow off a "fruity" smell from the mouth. Other characteristic signs that typically occur later include continues dehydration (including warm/dry skin with poor turgor, soft eyeballs, dry mucous membranes, oliguria, malaise, rapid but weak pulse and hypotension), abdominal pain, tachycardia, flushed ears and cheeks, Kussmaul respirations and altered level of consciousness [49, 51]. In addition, children typically exhibit complaints of abdominal or chest pain, nausea and vomiting due to the metabolic acidosis [25]. A patient left untreated will go into a coma, the vascular system will collapse and the patient will go into renal failure, especially when the blood glucose increases between 300 and 800 mg/dl [40].

   Although there are a vast array of symptoms that may be exhibited by the patient, it should be noted that the initial symptoms may be a diabetic coma as the other signs may have been masked or pacified by the patient. The patient's level of consciousness varies based upon the degree of the hyperosmolality [53]:

   - If insulin deficiency develops slowly and the patient is able to maintain an adequate intake, the patient will remain alert and the physical symptoms may be minimal.
   - In contrast, if the patient is vomiting in response to the ketoacidosis process, the body will begin to compensate to the dehydration, further exacerbating the serum osmolality, less than 320-330 milli-osmole per kilogram (mOsm/kg). Essentially, the body is depleted and unable to maintain homeostasis, inducing the patient to be in a stupor or coma.

   The initial diagnostic testing of hyperglycemia begins with the following laboratory findings, especially if DKA is speculated [40, 49, 51]:

   - Blood glucose level greater than 250 mg/dl.
   - Serum laboratory values:
     - Sodium (may be hypo, normal or hypernatremia).
     - Potassium (initially hyper with acidosis, but hypokalemia with dehydration).
     - Phosphate (low).
     - Bicarbonate (low, typically less than 15 mEq/L.
     - Osmolarity (variable).
     - Elevated BUN and creatinine due to the dehydration.
     - Serum ketones (positive).
   - Plasma pH less than 7.35.
   - Presence of urine ketones and glucose.

   The treatment of hyperglycemia or DKA includes all of the following, pending the severity of symptoms. If the patient is at home, the nurse should instruct the patient to understand the risks and then recognize the potential signs and symptoms of hyperglycemia. If the patient feels different or recognizes any of the signs and symptoms, they should check their blood glucose level. If the level is greater than 300 mg/dl, they should check the urine for ketones and increase their fluid intake [75]. The patient should contact

the health care professional who is monitoring their diabetes if their blood glucose is greater than 200 mg/dl for two days or if they are ill or vomiting.

- If the patient is in the hospital, the nurse should always assess the airway, level of consciousness, hydration status, electrolytes (if available) and blood glucose levels depending on the severity of the hyperglycemia and the patient's condition [49]. The nurse should adhere to the hospital's protocol in regards to verifying a high blood glucose level with a serum laboratory test. Additional actions of the nurse may include the following, depending upon the hospital protocol, patient's status and the physician's orders [49]:
  1. Assess the patient's vital signs (blood pressure, heart rate and respirations every 15 minutes).
  2. Record intake and output, temperature and mental status every one hour.
  3. Assess the patient's fluid status. The primary goal is to restore volume and maintain perfusion to the patient's heart, brain and kidneys. Typically the physician will order one liter of an isotonic saline solution over 30 to 60 minutes. Another goal in replacing fluid therapy is to replace the total body fluid loss by administering a slow, 0.45 percent of normal saline; then when the patient's blood glucose levels reaches 250 mg/dl, add 5 percent of dextrose to the 0.45 percent of normal saline to prevent hypoglycemia when the serum osmolarity declines rapidly.
  4. In order to lower the serum glucose, the nurse may administer an intramuscular (IM) or intravenous (IV) bolus of regular insulin. Typically, it is administered in an initial IV bolus of 0.1 units/kilogram, followed by an IV drip of 0.1 units/kilogram/hour. Most physicians will prescribe a continuous infusion because of the four-minute half life of IV insulin.
  5. If the patient has any significant changes in potassium and symptoms (fatigue, malaise, confusion, muscle weakness, shallow respirations, hypotension and weak pulse), the patient may have an electrocardiogram (ECG). In hypokalemia, the nurse can anticipate seeing ST-segment depression, flat or inverted T waves and increased U waves on the ECG [51]. Prior to administering an IV bolus potassium, make sure the patient has voided at least 30 ml/hr to prevent developing hyperkalemia.

It is important to assess and monitor for hypokalemia anytime a patient is hyperglycemic because it is a significant cause of death in the treatment of DKA [49].

2. **Hyperosmolar hyperglycemic nonketotic syndrome (HHNK), also known as hyperosmolar hyperglycemic state (HHNS)**, occurs in type 2 diabetics, especially in the older adult who may not even be aware that he or she has diabetes [49]. HHNS is a significantly dangerous, life-threatening complication with a high risk of mortality due to severe dehydration from prolonged hyperglycemia. Older adults are more prone to mortality, as high as 40 to 70 percent [52]. The development of HHNS rather than DKA is related to residual insulin secretion. In HHNS, the patient is able to secrete insulin to prevent the serum and urine ketones, but not enough to prevent hyperglycemia [49]. HHS is precipitated by one of the following conditions, although infection is the most prevalent cause [51]:
   - **Therapeutic agents**.
     - Glucocorticoids.
     - Diuretics.
     - Beta-adrenergic blocking agents.
     - Chlorpromazine.
     - Diazoxide.
   - **Acute illnesses**.
     - Infection.
     - Gangrene.
     - Urinary tract infection.
     - Burns.
     - Gastrointestinal bleeding.
     - Myocardial infarction.
     - Pancreatitis.
     - Stroke.
   - **Therapeutic procedures**.
     - Peritoneal dialysis.
     - Hemodialysis.
     - Hyperosmolar alimentation (oral or parenteral).
     - Surgery.
   - **Chronic illnesses**.
     - Renal disease.
     - Cardiac disease, including congestive heart failure (CHF).
     - Hypertension.
     - Previous stroke.
     - Alcoholism.

The patient with HHS may present with subtle, insidious symptoms. However, after the nurse collects the history, it will be noted there has been a decreased consumption of fluids with polyuria, polydipsia and weakness. If the patient presents with the initial symptoms, the patient may not be lethargic, confused or in a coma state as it presents with a serum osmolality greater than 310 mOsm/kg [40, 51].

The patient with HHNS will be diagnosed by exhibiting the following findings [40]:

- Severe hyperglycemia (typically greater than 600 mg/dl) in the absence of serum and urine ketones. Severe hyperglycemia occurs due to the coinciding severe hyperglycemia and the glucose is not filtered into the urine [49].
- Severe hyperosmolality (310 mOsm/L).
- Dehydration (the patient may lose up to 15 to 25 percent of his or her body fluid) [49].
- Hypokalemia and/or hyponatremia.
- Altered levels of consciousness.

The nurses' role in treating HHS includes recognizing the signs, symptoms and diagnostic findings, replacing fluids and restoring normal blood glucose levels within 36 to 72 hours, correcting insulin and electrolyte imbalances, assessing urine output and vital signs [40, 49]:

- The first treatment is providing adequate fluid replacement to increase the fluid volume. If the patient is in shock or has severe hypotension, administer a hypotonic intravenous fluid (0.45 percent normal saline). However, if the patient

has hypovolemia, then an isotonic solution (0.9 percent normal saline) is required. The patient will typically receive four to six liters of fluid over eight to 10 hours. Ideally, the nurse should expect to see a slow but steady improvement in the central nervous system function.

- The nurse should assess the patient hourly for signs of cerebral edema, abrupt changes in the mental status, abnormal neurological signs and coma. If the symptoms continue, it indicates that the patient is not getting the correct volume of fluid replacement or a rapid reduction in plasma osmolarity.
- Administer IV insulin at 10 units/hour to reduce the blood glucose levels. Once the patient's blood glucose maintains 250mg/dl, the physician should be notified to change the intravenous fluids to 5 percent Dextrose and 0.45 percent or 0.9 percent normal saline solution.
- Hyponatremia and hypokalemia replacements as needed.
- Ensure urine output is 50 mg/hour or more.

Hypoglycemia and diabetes

Another common, acute complication of diabetes is hypoglycemia (low blood glucose, less than 70 mg/dl), which occurs when there is not enough glucose available in relation to the circulating insulin [75]. Normal insulin secretion decreases when the blood glucose levels drop to approximately 83 mg/dl and the "counterregulatory" hormones (glucagon and epinephrine) are activated at about 68 mg/dl [49]. It is important to educate the patient to prevent hypoglycemia as it may cause neurological damage because the brain starves for glucose [75]. Hypoglycemia can be very dangerous for a type 1 diabetic because [49]:

- After one to five years of diagnosis, the regulation of circulating insulin dissipates because the patient is administering an injection, rather than the pancreas supplying the insulin as needed.
- Another problem is with long-standing hypoglycemia, the patient no longer has warning signs of the impending hypoglycemia. Unfortunately, this occurs in about 25 percent of all patients, and about 50 percent of patients who have had type 1diabetes for 30 years or more.

Hypoglycemia typically occurs if the patient undereats (skip a meal), administered too much insulin/oral antidiabetic agents and/or exacerbated during exercise. If a nurse is caring for a pregnant woman, it is important to note that she is more likely to develop hypoglycemia because her ideal glucose control is lower (60 to 120 mg/dl) [77].

The most common symptoms of hypoglycemia include hunger, sweating, tremor, blurred vision, headache, irritability, confusion, seizures and coma [49, 77]. If the patient should experience any of the symptoms (which typically occur around 50 mg/dl) or a family member finds the patient in a stupor or coma, confirm the blood glucose level with a SMBG (if at home). If a nurse is caring for the patient in the hospital, he/she should check the accu-check machine and then notify the hospital laboratory to confirm. However, if the nurse is working in the hospital, he/she should not sit around and wait for the laboratory department, and should treat the patient to prevent further lowering of the glucose level.

If the patient is at home or in the hospital setting with a mild case (patient remains alert, hungry, irritable, shaky, weak, headache and a blood glucose less than 60 mg/dl) of hypoglycemia, then treat the patient with a 10 to 15 gram carbohydrate snack, such as [49, 77]:

- Glucose tablets or gel.
- 4 ounces of orange juice.
- 6 ounces of regular soda.
- Miniature box of raisins.
- Six to eight Life Savers.
- Three graham crackers.

If the patient is at home or in the hospital with a moderate hypoglycemia event (cold, clammy skin, pale, rapid pulse, shallow respirations, marked change in mood, drowsiness and a blood glucose less than 40 mg/dl), then [49]:

- Treat symptoms with 15 to 30 grams of rapidly absorbed carbohydrates.
- Ingest additional foods, such as a low-fat milk or cheese, after 10 to 15 minutes.

If the patient is at home with a severe case of hypoglycemia (unable to swallow, unconscious, convulsions or blood glucose less than 20 mg/dl) [49]:

- The family member should administer 1 mg of Glucagon as an intramuscular (IM) or subcutaneous (SQ) injection.
- Administer a second dose in 10 minutes if the patient remains unconscious.
- Notify the physician immediately.

If the patient is in the hospital with a severe case of hypoglycemia, the nurse should [49]:

- Administer Glucagon IM or SQ and 50 percent of dextrose intravenous (IV). The nurse should always be cautious when administering Glucagon, as it may cause aspiration, thus inducing a vomit.
- Once the patient awakens, give a simple sugar, then a small snack or meal.

The blood glucose level should be rechecked every 15 minutes; avoid overtreating as it may cause hyperglycemia and rebound hypoglycemia [75, 77]. The nurse's role in treating DKA includes recognizing the signs and symptoms, collaborating with the treating provider to correct the dehydration process, normalize the electrolytes and correct the acidosis. Throughout the treatment, the nurse will monitor the patient's blood glucose levels, amounts of insulin being administered, urine volume, vital signs and serum chemistries. Once the DKA has been corrected, the nurse needs to educate the patient and family explicitly about the importance of insulin and providing guidelines for "sick" days to prevent future occurrences.

Long-term complications of diabetes

Failure to properly identify or control any form of diabetes will increase the risk of developing severe, multisystem complications. Over time, uncontrolled hyperglycemia will lead to the following complications [53]:

- **Cardiovascular disease** is the leading cause of death from type 2 diabetes [28]. Heart disease occurs due to changes in the macrovascular (large blood vessels) and microvascular (small blood vessels) to compensate for the increased flow leading to atherosclerosis, abnormal platelets, red blood cells, clotting factors and changes in the arterial walls [28, 49, 51]. Each of the cardiovascular compensatory mechanisms is exacerbated if hypertension, hyperlipidemia, smoking and/or obesity are coinciding with the diabetes. The macrovascular complications include the following [49]:
  - **Coronary artery disease** (CAD) is the most common cause of death among diabetic patients; accounting for 40 to 60 percent of deaths. CAD is also a major risk factor in the development of a myocardial infarction (MI), especially in type 2 diabetes.
    - Most diabetic patients will die from a massive MI due to their extensive CAD, cardiomyopathy and abnormal blood clotting factors [49].
    - Heart failure occurs in approximately 50 percent of all patients after an MI [49].
  - **Hypertension** (greater than 130/80 if diabetic) affects 20 to 60 percent of all people with diabetes, leading to microvascular complications, such as retinopathy and nephropathy. From 2003 to 2004, 75 percent of adults with self-reported diabetes had blood pressure greater than or equal to 130/80 millimeters of mercury (mm Hg) or used prescription medications for hypertension [58]. The overall goal is to maintain the blood pressure at less than 130/80, according to the seventh report of the JNC 7 [45].
  - **Hyperlipidemia** (abnormal blood lipids). The overall goal is to maintain the LDL less than 100mg/dl (may be less than 70mg/dl), TG less than 150mg/dl and HDL greater than 40mg/dl. Therefore the ADA recommends that the patient should have at least an annual lipid profile, then every two years if the patient has a low risk. Interestingly, although the patient is typically prescribed a statin (s) to maintain a lower lipid profile, the ADA does not recommend routine monitoring of liver and muscle enzymes in asymptomatic patients, unless the baseline enzymes were abnormal or the patient is taking drugs that interact with statins [45].
- **Cerebrovascular disease**
  - Stroke is increased two to six times among older adults with type 2 diabetes because of the risk factors and elevated blood glucose levels. A patient who maintains a high HgbA1c due to elevated blood glucose levels at the time of the stroke could have a greater brain injury and higher mortality [49].
  - Peripheral vascular disease of the lower extremities.

According to the National Diabetes Information Clearinghouse (NDIC) (2008) [58]:

- In 2004, heart disease was noted on 68 percent of diabetes-related death certificates among people aged 65 years or older.
- In 2004, stroke was noted on 16 percent of diabetes-related death certificates among people aged 65 years or older.
- Adults with diabetes have heart disease death rates about two to four times higher than adults without diabetes.

The microvascular complications include retinopathy, nephropathy and neuropathy.

- **Diabetic retinopathy** is the leading cause of new-onset blindness among adults' ages 20 to 74 years. Diabetic retinopathy occurs due to the alterations in the blood flow to the eyes, eventually leading to retinal ischemia. Interestingly, almost all patients with type 1 diabetes and 60 percent of patients with type 2 diabetes will have some degree of retinopathy. Diabetic retinopathy causes 12,000 to 24,000 new cases of blindness each year [58]. Retinopathy has been linked to fasting blood glucose levels above 129 mg/dl [49]. The overall goal is to prevent vision loss; therefore the diabetic patient should be encouraged to have dilated eye exams or 7-field 30-degree fundus photography by an ophthalmologist annually. In addition, it is important to maintain the blood glucose levels and blood pressure [45]. Other eye complications related to diabetes include [49]:
  - Retinal hemorrhages, detachments and venous bleeding due to microaneurysms (small capillary wall dilations formed in the retinal vessels, leading to poor circulation).
  - Macular degeneration.
  - Double vision.
  - Open angle glaucoma.
- **Diabetic nephropathy** is the leading cause of renal failure requiring dialysis or a transplant in the U.S. The condition occurs in about 20 to 40 percent of all diabetic patients. Diabetes is the leading cause of kidney failure, accounting for 44 percent of new cases in 2005. In 2005, 46,739 people with diabetes began treatment for end-stage kidney disease in the United States and Puerto Rico. In 2005, a total of 178,689 people with end-stage kidney disease due to diabetes were living on chronic dialysis or with a kidney transplant in the United States and Puerto Rico [58]. The exact pathologic origin is unknown, but thickening of the basement of the glomeruli eventually impairs renal function. The first indication of nephropathy is the presence of albumin (microalbuminuria) in the urine. The overall goal is to prevent renal failure. Therefore the blood glucose and blood pressure should be controlled, the serum creatinine and microalbuminuria should be screened annually, there should be an annual GFR calculation and the patient should be instructed to limit the protein intake to 0.8 g/kg in patients with any degree of chronic kidney disease (CKD) [45].
  - If the patient tests positive for microalbuminuria, the ADA recommends the patient be started on the following medications [45]:
    - Type 1 diabetic should be on an ACE inhibitor.
    - Type 2 diabetes with hypertension should be on an ACE or ARB.

In addition, patients should maintain their blood pressure at less than 130/80 and limit protein to 10 percent of their dietary intake. The patient should be referred to a urologist.

- **Peripheral neuropathies** (somatic neuropathies) initially appear in the toes and feet, then progress upward into the fingers and hands. The diabetic patient will complain of numbness or tingling and pain that are described as "aching," "burning" or "shooting," and feelings of coldness in the feet. About 60 to 70 percent of people with diabetes have mild to severe forms of nervous system damage. The results of such damage include impaired sensation or pain in the feet or hands, slowed digestion of food in the stomach, carpal tunnel syndrome, erectile dysfunction or other nerve problems. Almost 30 percent of people over 40 with diabetes have impaired sensation in the feet, and at least one area that lacks feeling.
    - Foot injuries are one of the most common complications of diabetes leading to hospitalizations related to sensory neuropathies, ischemia and infections [49].Diabetes is the leading cause of amputations worldwide, approximately 60 percent. In 2004, about 71,000 nontraumatic lower-limb amputations were performed in people with diabetes [58]. The five-year mortality rate after a leg or foot amputation ranges from 39 to 68 percent [49].
        - Claw toe is a deformity that causes the toes to hyperextend, resulting in ulceration [49]. Health care professionals, therefore, should visually inspect patients' feet at every visit, and patients should be instructed to inspect their own feet daily with a mirror and have an annual foot exam by a podiatrist.

Other complications include any of the following [45]:

- **Visceral neuropathies** (autonomic neuropathies) that involves various entities:
    - Sweating dysfunction with an absence of sweating (anhydrosis) of the hands and feet.
    - Abnormal papillary function (constricted pupils that dilate slowly in the dark).
    - Cardiovascular dysfunction with a fixed cardiac rate that does not change with exercise, postural hypotension.
    - Gastrointestinal dysfunction, including upper gastric motility (gastroparesis) that results in constipation, dysphagia, anorexia, heartburn, nausea, vomiting and altered blood glucose control.
    - Genitourinary dysfunction, resulting in changes in bladder (inability to empty completely or a sensation of fullness) and sexual function (decreased libido, failure to ejaculate or no vaginal lubrication).
- **Periodontal disease**. Periodontal, or gum, disease is more common in people with diabetes. Among young adults, those with diabetes have about twice the risk of those without diabetes. Persons with poorly controlled diabetes (A1C greater than 9 percent) were nearly three times more likely to have severe periodontitis than those without diabetes. Almost one-third of people with diabetes have severe periodontal disease with loss of attachment of the gums to the teeth measuring 5 millimeters (mm) or more [58].
- **Male erectile dysfunction (ED)** occurs at an earlier age in men with diabetes (10 to 15 years earlier). Half of all diabetic men experience ED. ED is related to poor glucose control, obesity, hypertension, heavy smoking and other chronic micro and macrovascular conditions [49].
- **Increased susceptibility to infections.**

There are a vast array of complications that can occur in diabetic patients, so nurses must properly assess their patients' overall condition, risk factors, sensory deficits, access to health care and willingness to comply with the medical regimen to prevent injury or an early death.

Diabetes and cultural considerations

Nurses in a diverse America may be challenged whenever they encounter patients who have different beliefs, values and cultures from their own. It is important to be sensitive and respectful to the patient and family and come to know them as an individuals. In order to be most effective and respectful toward each patient that we care for, it is imperative for nurses to be culturally competent (learning, understanding and respecting the values and beliefs of others) [44].

Upon admission to a hospital facility, the nurse is required to ask the patient if they have any religious or cultural considerations that may affect their health care. It is important to truly ask the patient and respect their response. In addition, nurses should convey the patient's concerns to the appropriate disciplinarians and other nurses responsible for the care of the patient. If nurses are working in an outpatient facility, there are no set for health care professionals to ask their patients about their religious or cultural considerations. However, if a nurse works in an outpatient facility, it should be considered and asked of all patients.

The purpose of this section is to familiarize nurses on how to consider patient's cultural and religious beliefs. If you are caring for a patient, learn about their personal concerns to ensure you customize their care toward things the patient will be most apt to abide by to reduce their risk of developing diabetes and/or complications of diabetes. In addition, always inquire with the individual patient because he or she may not adhere to all of the recommendations of their religious or culture. Although there is a vast array of knowledge to be learned about various cultures, some of the major cultural and religious considerations are listed as follows [44]:

- **Diet**
    - Islam prohibits followers from eating from sunrise to sunset during the month of Ramadan.
    - Judaism requires individuals to fast from sundown to sundown during Yom Kippur and the Day of Atonement.
    - Orthodox Jews are forbidden to eat port, shellfish, and nonkosher red meat and poultry.
    - Hindus and Seventh Day Adventists are forbidden to eat meat.
    - Philippinos enjoy eating rice with every meal and may feel deprived without it.

- Japanese prefer small amounts of beef or chicken mixed with vegetables and rice or noodles.

Mexican Americans, African Americans and Asian Indians have a high prevalence of developing diabetes in their lives, so it is important to understand the typical food choices that may be a factor contributing to their risk:

- **Mexican Americans** enjoy eating lots of salt and fats in their cooking, which may be a contributing factor for their high development of type 2 diabetes. Therefore, the nurse should consider diabetes treatment and prevention programs for Mexican-Americans to include the following [50]:
  - Foods found in their traditional diet. Traditionally, Mexican-Americans tend to eat diets that are low-fat and high-fiber, which is certainly beneficial.
  - Assess the level of acculturation to American eating practices.
  - Ascertain the integration of folk medicine in consideration of their foods, hot and cold in regards to healing.
- **African Americans** enjoy eating "soul food," such as food that is breaded and fried, especially in the summer months [50]. Their food choices are based on the following:
  - Health beliefs passed down each generation: "the soul food."
  - Socioeconomic statuses along with education level.
  - Physical and financial limitation also plays an important role in terms of planning a meal and nutrition education for African-American patients.

  A research study conducted by a diabetes clinic in Atlanta, Ga, the Grady Memorial Hospital, concluded that unfamiliar food options was the main reason African Americans did not adhere to low fat, low cholesterol diet recommendations. Therefore, it is critical that patients fully understand and realize the significance of their new diet; otherwise they may very well not follow it, which could lead to serious consequences, such as diabetes, obesity and/or hypertension [50].
- **Asian Indians** living in the U.S. are composed of a very diverse culture that will vary from each family based upon their religion. Although there is a vast array of religions practiced amongst Asian Indians, the most prevalent is Hinduism.

  A survey among 73 Asian Indians adults residing in New York City and Washington, D.C., implemented per Diabetes Care (2004) and the ADA, demonstrated the following [50]:
  - Acculturation of this population in the U.S. has led to more frequent selection of American or other ethnic foods for main meals and replacement of traditional sweets with cookies, doughnuts and other Western pastries.
  - Length of stay in the U.S. had an effect on the choice of fats used in cooking; those who had lived here more than five years appeared to have decreased their consumption of butter and ghee (clarified butter) and used margarine as an alternative. These individuals continued to consume rice, chappati (flat bread), yogurt, dhal (a spiced lentil dish), and curried vegetables. Asian Indians also reported an increase in intake of whole grain breads, fish, poultry, meat, potato chips, cakes, cheese, fruit, and alcoholic and nonalcoholic beverages (other than water) after immigration to the U.S.

  Therefore, the Americanized Asian Indians changed their diet radically as they went from a low-fat, high fiber diet to a higher-fat animal protein, low fiber, and high levels of saturated fat. One reason for this dramatic change is the increased tendency for Asian Indians to consume convenience foods at fast food restaurants. In addition, there are other factors to consider such as stress and sedentary lifestyles that increase their risk for developing chronic diseases, increasing their risk of developing diabetes, obesity and/or hypertension.

- **Pregnancy**
  It is important to note that many Asian and Hispanic cultures practice a system of hot and cold body balance. During pregnancy, the woman is considered to be in a "hot" condition and she will avoid "hot" foods such as protein. Therefore, it is important for the nurse to inquire during pregnancy for a woman who believes in a hot and cold practice if they are eating protein-rich foods. In addition, many Hispanic women avoid iron supplements and pre-natal vitamins because they are considered "hot."

Nurses caring for patients should always consider nutrition-related cultural variations by asking the patients about their specific food habits. Collaborating with the individual patient and family will provide a wealth of information for the nurse to provide appropriate suggestions for the patient to modify their traditional eating patterns to prevent and treat type 2 diabetes [50]. In addition, always discuss and refer the patient and family to a RD to ensure individual diets can be customized.

Paying for Diabetes

In April 2007, the ADA recognized that there were millions of American children and adults living with diabetes without insurance or limited access to obtaining the supplies, medications and education necessary to successfully manage the disease and prevent diabetes-related complications [6]. Currently, in 2009, the problem is compounded and expected to exacerbate due to the economic hardships encountered by many Americans. The nurse needs to be empathetic, respectful and conscious about the initial costs of the SMBG machines, monthly costs of the supplies (lancets, alcohol wipes, syringes), medications (oral and/or insulin), choosing healthy nutritious foods and any other expenses that may incur. Nurses should never assume that a patient and/or family is "noncompliant" in their health care regimen, but instead take the time to assess the patient and to find out the reasoning.

Throughout the U.S., each state has recognized the major effects diabetes plays and the impact on patients and society. As of January 2009, 46 states and the District of Columbia require mandated health insurance coverage for diabetes treatment. At this time, the laws in Mississippi, Missouri and Washington require only that insurers offer coverage, but not necessarily include the coverage in all active policies. The majority of the states require coverage for both direct treatment and for diabetes equipment and supplies that are often used by the patient at home. The four states that do not have a mandate or insurance requirement are Alabama, Idaho, North Dakota and Ohio [54]. Eleven percent of diagnosed diabetics under the age of 65 were uninsured and most likely to be from low income backgrounds [54]. The other 6 percent of diabetic patients are unaccounted, for according to the National Conference of State Legislatures.

However to date, private health insurance companies are not congruent in the costs and coverage, as it varies in each state. The majority of patients diagnosed with diabetes are either covered by a private insurance company (39 percent) or Medicare (44 percent). Medicare is the leader in providing the most coverage for diabetic patients.

Even with health insurance, Medicare and/or Medicaid, patients may not receive all of their required treatment modalities, such glucose monitors, test strips, lancets, emergency kits and medications. Therefore, it is imperative for physicians, nurses and educators to assess the community resources and programs within their community to find other potential coverage options. For example, Wal-Mart provides $4 prescriptions on various medications; on Wal-Mart's main web site (2009), the following oral anti-diabetic agents are offered in a 30-day supply for $4 [72]:

- Chlorpropamide 100mg tablets.
- Glimepiride 1mg, 2mg and 4mg tablets.
- Glimepiride 2 and 4mg tablets.
- Glipizide 5mg and 10mg tablets.
- Glipizide 10mg tab.
- Glyburide 2.5mg, 5 mg and micronized 3mg and 6mg tablets.
- Metformin 500mg, 850mg and 1000mg tablets.
- Metformin 500mg extended release tablet.

However, what happens to people who have coverage but then lose their job? The cost of Consolidated Omnibus Budget Reconciliation Act (COBRA) insurance is very pricey, especially for a family or individual who recently lost a job. The NDIC and the ADA recognized in 2004 that a person with diabetes spends an average of $13,243 a year on health care expenses [56]. Therefore, if a patient is not eligible for Medicare, Medicaid, or private health insurance, there are other potential programs available according, to the NDIC, that the nurse can recommend for the patient and family dealing with diabetes (2004) [56]:

- **State Children's Health Insurance Program**, supported by the U.S. Department of Health and Human Services, established the State Children's Health Insurance Program (SCHIP) to help children without health insurance. SCHIP provides health coverage for children whose families earn too much to qualify for Medicaid but too little to afford private health insurance.
- **Health care services**. The Bureau of Primary Health Care, a service of the Health Resources and Services Administration, offers health care for people regardless of their insurance status or ability to pay. Encourage the patient to find a local health center by calling 1-800-400-2742 or visit the bureau's website at *www.bphc.hrsa.gov* on the Internet.
- **Hospital care** is provided to patients who are uninsured and require hospital care. In 1946, Congress passed the Hospital Survey and Construction Act, which was sponsored by Sens. Lister Hill and Harold Burton and is now known as the Hill-Burton Act. Although the program originally provided hospitals with Federal grants for modernization, today it provides free or reduced-charge medical services to low-income people. The program is administered by the Department of Health and Human Services. For more information, call 1-800-638-0742 or visit *www.hrsa.gov/hillburton/default.htm* on the Internet.
- **Food and nutrition.** Food, nutrition education and access to health care services are also available through the U.S. Department of Agriculture's WIC (Women, Infants, and Children) program. Pregnant women who meet residential, financial need and nutrition risk criteria are eligible for assistance. GDM is considered a medically based nutrition risk and would qualify a woman for assistance through the WIC program if she meets the financial need requirements and has lived in a particular state the required amount of time. WIC Phone: 703-305-2746, Internet: *www.fns.usda.gov/wic*.
- **Local resources** that may be available, include:
  - **Lions Clubs** help with vision care.
  - **Rotary Clubs** help with humanitarian and educational assistance.
  - **Elks Clubs** provides charitable activities for youth and veterans.
  - **Shriners** offer need-based treatment for children at Shriners hospitals throughout the U.S.
- **Dialysis and transplantation**. In 1972, Congress passed legislation making people of any age with permanent kidney failure eligible for Medicare. To qualify, a patient must need regular dialysis or have had a kidney transplant, and must have worked under Social Security, the Railroad Retirement Board or as a government employee (or be the child or spouse of someone who has), or must already be receiving Social Security or Railroad Retirement benefits. Every American needing dialysis for chronic kidney failure is eligible for dialysis assistance. For more information, call the Centers for Medicare & Medicaid Services at 1-800-MEDICARE (633-4227) to request the booklet Medicare Coverage of Kidney Dialysis and Kidney Transplant Services. This booklet is also available on the Internet at *www.medicare.gov* under "Publications."
  - For information on financing an organ transplant, contact the following organization: United Network for Organ Sharing (UNOS) by calling: 1-888-894-6361 or visit the Internet at: *www.unos.org*.
- **Prosthetic care**. If a patient has had an amputation and is paying for compounding rehabilitation expenses, one of the following organizations provides financial assistance or information about locating financial resources for people who need prosthetic care:
  - Amputee Coalition of America
    Phone: 1-88-AMP-KNOW (267-5669)
    Internet: *www.amputee-coalition.org*

- Easter Seals
  Phone: 1-800-221-6827
  Internet: *www.easterseals.com*

- Prosthetics for Diabetics Foundation
  Phone: 770-267-0019
  Internet: *www.expage.com/page/pfdfoundation*

The future for diabetics

Researchers, scientists and physicians have been diligently attempting to control and cure diabetes for decades. One of the most intriguing developments was presented in April 2009 in Japan with the Diabetes Research Institute. For the first time, scientists discussed a hopeful development on how transplanted insulin-secreting cells called "islets" function when they are inside a living organism, or in vivo. In the past, researchers could only view the islets in a laboratory, or in vitro [38]. In addition, the Diabetes Research Institute and scientists in Stockholm, Sweden, are transplanting human islets into diabetic mice. In a paper, The Anterior Chamber of the Eye Allows Studying Human Islet Cell Biology in Vivo, researchers report the following [38]:

- After transplantation of 500 islet equivalents per eye, recipient mice achieved and maintained normal blood sugar levels for over 150 days.
- Within one month of the transplant, new blood vessels formed around the islet cells to deliver necessary nutrients (a process called "neovascularization").
- As more blood vessels grew around the islet cells, the mouse's diabetes gradually reversed.

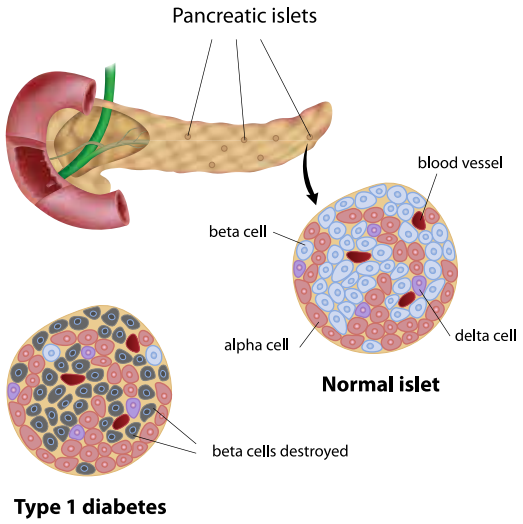
However, according to the ADA, islet cell transplants were being conducted with great success in laboratory mice in the 1970s [16]. But the excitement that those experiments generated soon turned to frustration, as initial attempts to reproduce that success in humans were largely disappointing. For many years, progress was slow and few transplant recipients were able to stay diabetes-free for more than a few months before the transplanted islet cells failed. However, in recent years, scientists have begun to make rapid advances in transplant technology, and some of the most exciting new research comes from researchers at the University of Alberta in Edmonton, Canada. The scientists there have used a new procedure called the Edmonton Protocol to treat patients with type 1 diabetes, which is elaborated upon in the following [16]:

> In this procedure, researchers use specialized enzymes to remove islets from the pancreas of a deceased donor. For an average-size person (70 kg), a typical transplant requires about 1 million islets, equal to two donor organs. Since the islet cells are extremely fragile, transplantation occurs immediately after they are removed. The transplant itself is easy and takes less than an hour to complete. The surgeon uses ultrasound to guide placement of a small plastic catheter through the upper abdomen and into the liver. The islets are then injected through the catheter into the liver. It takes some time for the cells to attach to new blood vessels and begin releasing insulin. The doctor will order many tests to check blood glucose levels after the transplant, and insulin may be needed until control is achieved.

According to Science Daily (2007), reporting in the proceedings of the National Academy of Sciences, that has greatly boosted the number of immune T-cells able to shield transplanted pancreatic islet cells from attack by the immune system [64]. In addition, over the nine-week study of islet transplantation in diabetic mice, no pharmacologic immunosuppression therapy was administered, and the transplanted islet cells stayed healthy and produced insulin throughout the study. This is promising data that may allow physicians to perform islet cell transplants in type 1 diabetics, especially if immunosuppressant therapy is not required because at this time, once a patient has received any type of a transplant, they need immunosuppresants therapy for the rest of their life to prevent their body from rejecting the donor cells.

According to the ADA, pancreatic transplants may treat type 1 diabetics if scientists can develop safe immunosuppresants that always work for the patient. Until safe immunosuppresants are fabricated and delivered, many doctors believe islet cell transplants are a better option.

> Islets are clusters of cells that make up 1 to 2 percent of the total pancreas that make insulin. In the patient with type 1 diabetes, islet cells are destroyed. With pancreatic islet transplantation, cells are taken from a donor pancreas and transferred into another person. Once implanted, the new islets begin to make and release insulin. Researchers hope that islet transplantation will help people with type 1 diabetes live without daily injections of insulin.



According to the National Institutes of Health (2009), there are different methods to obtain islet cells depending on whether it is fetal, adult or embryonic tissue [66]:

- **Fetal tissue as source for islet cells**. Several groups of researchers are investigating the use of fetal tissue as a potential source of islet progenitor cells. For example, using mice, researchers have compared the insulin content of implants from several sources of stem cells – fresh human fetal pancreatic tissue, purified human islets and cultured islet tissue – and they have found that insulin content was initially

higher in the fresh tissue and purified islets. However, with time, insulin concentration decreased in the whole tissue grafts, while it remained the same in the purified islet grafts. When cultured islets were implanted, however, their insulin content increased over the course of three months. The researchers concluded that precursor cells within the cultured islets were able to proliferate (continue to replicate) and differentiate (specialize) into functioning islet tissue, but that the purified islet cells (already differentiated) could not further proliferate when grafted. Importantly, the researchers found, however, that it was also difficult to expand cultures of fetal islet progenitor cells in culture.

- **Adult tissue as source for islet cells**. Many researchers have focused on culturing islet cells from human adult cadavers for use in developing transplantable material. Although differentiated beta cells are difficult to proliferate and culture, some researchers have had success in engineering such cells to do this.

  Fred Levine and his colleagues at the University of California, San Diego, have engineered islet cells isolated from human cadavers by adding to the cells' deoxyribonucleic acid (DNA) special genes that stimulate cell proliferation. However, because once such cell lines that can proliferate in culture are established, they no longer produce insulin. The cell lines are further engineered to express the beta islet cell gene, Pdx1 (pancreatic and duodenal homeobox 1), also known as insulin promoter factor 1(PDX-1), which stimulates the expression of the insulin gene. The specific cell lines have been shown to propagate in culture and can be induced to differentiate to cells, which produce insulin. When transplanted into immune-deficient mice, the cells secrete insulin in response to glucose. The researchers are currently investigating whether these cells will reverse diabetes in an experimental diabetes model in mice.

The investigators report that these cells do not produce as much insulin as normal islets, but it is within an order of magnitude. The major problem in dealing with these cells is maintaining the delicate balance between growth and differentiation. Cells that proliferate well do not produce insulin efficiently, and those that do produce insulin do not proliferate well. According to the researchers, the major issue is developing the technology to be able to grow large numbers of these cells that will reproducibly produce normal amounts of insulin.

Another promising source of islet progenitor cells lies in the cells that line the pancreatic ducts. Some researchers believe that multipotent (capable of forming cells from more than one germ layer) stem cells are intermingled with mature, differentiated duct cells, while others believe that the duct cells themselves can undergo a differentiation, or a reversal to a less mature type of cell, which can then differentiate into an insulin-producing islet cell.

Susan Bonner-Weir and her colleagues reported in 2008 that when ductal cells isolated from adult human pancreatic tissue were cultured, they could be induced to differentiate into clusters that contained both ductal and endocrine cells. Over the course of three to four weeks in culture, the cells secreted low amounts of insulin when exposed to low concentrations of glucose, and higher amounts of insulin when exposed to higher glucose content. The researchers have determined by immunochemistry and ultra structural analysis that these clusters contain all of the endocrine cells of the islets.

- **Embryonic stem cells**. The discovery of methods to isolate and grow human embryonic stem cells in 1998 renewed the hopes for doctors, researchers and diabetic patients and their families that a cure for type 1 diabetes and perhaps type 2 diabetes as well, may be within striking distance. In theory, embryonic stem cells could be cultivated and coaxed into developing into the insulin-producing islet cells of the pancreas. With a ready supply of cultured stem cells at hand, the theory is that a line of embryonic stem cells could be grown up as needed for anyone requiring a transplant. The cells could be engineered to avoid immune rejection. Before transplantation, they could be placed into nonimmunogenic material so that they would not be rejected and the patient would avoid the devastating effects of immunosuppressant drugs. There is also some evidence that differentiated cells derived from embryonic stem cells might be less likely to cause immune rejection. Although having a replenishable supply of insulin-producing cells for transplant into humans may be a long way off, researchers have been making remarkable progress in their quest for it. While some researchers have pursued the research on embryonic stem cells, other researchers have focused on insulin-producing precursor cells that occur naturally in adult and fetal tissues.

In 2001, several teams of researchers continued the initial embryonic research, continuing to believe the possibility that human embryonic stem cells could be developed as a therapy for treating diabetes. Recent studies in mice show that embryonic stem cells can be coaxed into differentiating into insulin-producing beta cells, and new reports indicate that this strategy may be possible using human embryonic cells as well. Last year, researchers in Spain reported using mouse embryonic stem cells that were engineered to allow researchers to select for cells that were differentiating into insulin-producing cells.

Bernat Soria and his colleagues at the Universidad Miguel Hernandez in San Juan, Alicante, Spain, added DNA containing part of the insulin gene to embryonic cells from mice. The insulin gene was linked to another gene that rendered the mice resistant to an antibiotic drug. By growing the cells in the presence of an antibiotic, only those cells that were activating the insulin promoter were able to survive. The cells were cloned and then cultured under varying conditions. Cells cultured in the presence of low concentrations of glucose differentiated and were able to respond to changes in glucose concentration by increasing insulin secretion nearly sevenfold. The researchers then implanted the cells into the spleens of diabetic mice and found that symptoms of diabetes were reversed.

Another researcher, Manfred Ruediger of Cardion Inc. in Erkrath, Germany, use the approach developed by Soria

and his colleagues to develop insulin-producing human cells derived from embryonic stem cells. By using this method, the noninsulin-producing cells would be killed off and only insulin-producing cells should survive. This is important in ensuring that undifferentiated cells are not implanted that could give rise to tumors.

Utilizing stem cell research is intriguing and provides a lot of hope for diabetic patients. However, at this time is important for researchers to define the protocols, exact mechanisms and potential need for immunosuppressive therapy. Ultimately, type 1 diabetes may prove to be especially difficult to cure, because the cells are destroyed when the body's own immune system attacks and destroys them. The autoimmunity must be overcome if researchers intend to use transplanted cells to replace the damaged ones. Many researchers believe that at least initially, immunosuppressive therapy similar to that used in the Edmonton protocol will be beneficial. A potential advantage of embryonic cells is that, in theory, they could be engineered to express the appropriate genes that would allow them to escape or reduce detection by the immune system. Others have suggested that a technology should be developed to encapsulate or embed islet cells derived from islet stem or progenitor cells in a material that would allow small molecules such as insulin to pass through freely, but would not allow interactions between the islet cells and cells of the immune system. Such encapsulated cells could secrete insulin into the blood stream, but remain inaccessible to the immune system.

Before any cell-based therapy to treat diabetes makes it to the clinic, many safety issues must be addressed. A major consideration is whether any precursor or stem-like cells transplanted into the body might revert to a more pluripotent state and induce the formation of tumors. These risks would seemingly be lessened if fully differentiated cells are used in transplantation. However, before any kind of human islet-precursor cells can be used therapeutically, a renewable source of human stem cells must be developed. Although many progenitor cells have been identified in adult tissue, few of these cells can be cultured for multiple generations. Embryonic stem cells show the greatest promise for generating cell lines that will be free of contaminants and that can self renew. However, most researchers agree that until a therapeutically useful source of human islet cells is developed, all avenues of research should be exhaustively investigated, including both adult and embryonic sources of tissue [66].

Closing

Although diabetes remains a prevalent chronic disease process, nurses can make an enormous difference in the life of the patient and family dealing with it. According to the American College of Physicians (ACP) (2006), many times physicians do not have the time, resources and appropriate levels of patient participation to effectively treat diabetes. Therefore, patients end up struggling with the understanding and complexity of the disease [4]. The ACP and the American College of Physicians Foundation (ACPF) are concerned about the dangers and enormous cost to America, so in 2006 they collaborated to pursue a three-year project engaging both physicians and patients to improve diabetes care in the United States [4].

In 2010, one can hope to analyze their investigation in hope of finding more educational options for patients and their families when they are encountering diabetes. In the meantime, it is important to understand there are enormous treatment modalities and options available for the patient. It is just imperative that the patient realize the significant role diabetes plays on the body and the complications that arise over time by not adhering to the recommendations. In addition, although the patient may realize the significance, the nurse needs to assess whether the patient is eager and capable of making the changes.

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DIABETES: A COMPREHENSIVE OVERVIEW

Self Evaluation Exercises

Select the best answer for each question and check your answers at the bottom of the page.

You do not need to submit this self-evaluation exercise with your participant sheet.

1. Diabetes has reached epic proportions worldwide, making it the seventh leading cause of death in the United States.

   ○ True ○ False

2. There are about 25.8 million children and adults (8.3% of the total United States population) living with diabetes.

   ○ True ○ False

3. Insulin is a hormone that increases the blood glucose levels any time the blood sugar is low..

   ○ True ○ False

4. Type 1 diabetes is a multifactorial disease caused by an autoimmune destruction of insulin-producing pancreatic beta cells.

   ○ True ○ False

5. Type 2 diabetes accounts for 10 percent of all cases.

   ○ True ○ False

6. Impaired fasting glucose (IFG) is a condition in which the fasting blood sugar level is 100 to 125 milligrams per deciliter (mg/dL) after an overnight fast.

   ○ True ○ False

7. The classic signs and symptoms of diabetes in general include the “three polys” (polyuria, polydipsia, and polyphagia).

   ○ True ○ False

8. Diabetes is diagnosed by a fasting blood glucose level greater than 126 mg/dl on two or more occasions.

   ○ True ○ False

9. The first line treatment of all diabetic patients is insulin.

   ○ True ○ False

10. The ADA recommends that all diabetics maintain their daily blood glucose levels as follows: Preprandial, 70 to 130 mg/dl; postprandial, less than 180 mg/dl.

    ○ True ○ False

Answers:	1.T 2.T 3.F 4.T 5.F 6.T 7.T 8.T 9.F 10.T