A NATIONAL EPIDEMIC

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About the Authors

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Purpose and Goals

The goal of this course is to provide nurses and other healthcare professionals with a comprehensive overview of type 1 diabetes mellitus and type 2 diabetes mellitus, including the impact, etiology, management and complications associated with diabetes.

Instructional Objectives

1. Explain the epidemiology of type 1 and type 2 diabetes.
2. Differentiate between type 1 diabetes and type 2 diabetes.
3. Describe the pathophysiology of diabetes.
4. Know the risk factors for type 1 and type 2 diabetes.
5. Describe the pathogenesis of type 1 and type 2 diabetes.
6. Review other types of diabetes.
7. Analyze diagnostic criteria for diabetes.
8. Identify treatment options for type 1 and type 2 diabetes.
9. Outline management options for type 1 and type 2 diabetes.
10. Determine nursing interventions for complications associated with type 1 and type 2 diabetes.

Epidemiology and Impact

The statistics on diabetes are staggering. The U.S. Department of Health and Human Services recently reported that diabetes affects 29.1 million people of all ages and 9.3% of the American population. Currently, there are 18.8 million diagnosed cases. Another alarming statistic reveals that there are currently 8.1 million undiagnosed cases in the United States. This indicates that a substantial portion of the population with diabetes go untreated. Finally, diabetes remains the seventh leading cause of death in the United States.

The future for diabetes incidence is also alarming. According to the WHO, by 2030 the estimated worldwide prevalence of diabetes will be an astounding 366 Million. The greatest increase is expected to occur in countries such as Africa and Asia. Researchers believe that this trend is partly due to lifestyle changes and migration of the western-style diet to those parts of the world.

Comorbidities

The significant health impact of diabetes can’t be ignored. The following list of conditions and associated complications was published in the most recent version of the National Diabetes Fact Sheet:

- **Kidney disease** - Diabetes is the leading cause of kidney failure that accounts for 44% of all new cases as of 2011.
- **Heart disease** - People with diabetes suffered myocardial infarctions related death 1.7 times greater than adults without diabetes.
- **Stroke** - Stroke hospitalization rates for people with diabetes are 1.5 times higher than for non-diabetics.
- **Blindness** - Diabetes is the leading cause of blindness among adults.
- **Amputations** - Diabetes is responsible for over 60% of lower limb amputations.
- **Nervous system disease** - Almost 30% of people with diabetes suffer from impaired sensations in the feet.
- **Dental disease** - Approximately 30% of people with diabetes have severe periodontal disease.
- **Pregnancy complications** - Uncontrolled diabetes can cause birth defects in approximately 5% to 10% of pregnancies and spontaneous abortions in 15% to 20% of pregnancies.
- **Associated complications** - Diabetes are more susceptible to other illnesses [1](#economic-factors .anchor) or Economic Factors

The economic impact of diabetes remains a tremendous burden in the United States. The total economic encumbrance is more than $256 billion annually. The majority of the expenses, $176 billion, were for direct medical costs such as hospital care, emergency room visits, physician office visits and home health visits. The other $69 billion was attributed toward indirect costs such as disability, loss of workplace productivity and premature mortality. According to recent data published by the National Institute of Diabetes and Digestive and Kidney Diseases and the CDC, most of the expenses associated with diabetes are for long-term complications, such as heart failure, renal failure, hypertension, amputation and blindness. The average cost of health care for a person with diabetes in over $13,000 per year. In fact, according to the CDC (2017) medical expenses for people with diabetes are 2.3 times higher than for people without diabetes.

Population Variances

The majority of people with diabetes are older than 45 years of age. Type 2 diabetes accounts for about 95% of known cases, and type 1 diabetes affects approximately 5% of all diagnosed cases. Gestational diabetes during pregnancy occurs in about 3% to 10% of pregnant women. There is also a dramatic increase in rates of diabetes in the aging population. The prevalence of diabetes is approximately seven times higher among people over 65 years of age. Type 1 diabetes is more common in children and adolescents. The American Diabetes Association recently reported that from 2008-2009 that the number of youth diagnosed with diabetes type 1 was 18,436. About 1 in 400 youth less than 20 years of age have diabetes.

Race and ethnicity also play a role as potential risk factors for diabetes. The most recent CDC (2017) report revealed Hispanic Americans and African Americans have a greater prevalence of diabetes than Caucasians (12.8% and 13.2% versus 7.6%). Asian Americans with diabetes make up about 9.0% of all diagnosed cases. Diabetes prevalence in American Indians and Alaska Natives in the United States is over twice as high compared to Caucasians (15.9%).

History of Diabetes

Diabetes is one of the first classes of diseases to be studied and defined. As far back as antiquity, ancient healers scried features of the disease on Egyptian papyrus. In the 2nd century AD, Aretaeus of Cappadocia coined the term “diabetes” from the Greek word for a siphon or “pass through.” He described the disease as the “melting down...
of the flesh and limbs into urine.” During ancient times, the symptoms were described as unquenchable thirst, nausea, excessive urine and a short painful life.

Between 400 and 500 BC, Hindu physicians wrote about the sweetness of diabetic urine. At the time, tasting the urine and noticing that ants congregated in and around diabetic urine helped to identify the diagnosis. Physicians of the time also noticed that the disease was more prevalent in overweight and obese populations. Treatments consisted of exercise and large quantities of vegetables.

During the 18th century, physician Matthew Dobson of Liverpool, England wrote about the first known description of hyperglycemia. He talked about his patient who passed 28 pints of urine daily. According to Dobson’s reports, he evaporated the urine to “a white cake (which) smelled sweet like brown sugar.” Dr. Dobson finished his studies by advising that the sugar in the urine came from the serum of the blood and was excreted through the kidneys. Toward the end of the 18th century, European surgeon John Rollo was the first to use the word “melitus,” which was borrowed from the Latin word meaning “honey.” Rollo prescribed, what was known at the time, as the “animal diet” to diabetic patients. This diet consisted of “game or old meats.” Rollo believed that consuming vegetables caused sugar to form in the stomach, therefore, his solution was to abstain from vegetables.

At the beginning of the 19th century, physicians understood that sugar in the urine was glucose. For the better sake of science, doctors no longer needed to taste diabetic urine, due to the advent of chemical tests. During this time, the medical community also started to understand the role of the pancreas in diabetes. Diabetes was also now understood to be a heterogeneous disorder consisting of various subtypes. At the dawn of the 20th century, physicians proposed that the pancreas, thyroid and adrenals were responsible for the regulation of carbohydrate metabolism. By the latter part of the 19th century, scientists also gained a better understanding of other comorbidities, such as the 19th century, scientists also gained a better understanding of other comorbidities, such as kidney disease and neuropathic symptoms.

**Discovery of Insulin**

Between 1889 and 1921, researchers attempted countless times to isolate the definable internal secretion of the pancreas. These endeavors failed because blood glucose was not measured, or due to the fact that hypoglycemia was misinterpreted as some kind of toxic reaction. In 1921, Canadian surgeon Frederick Banting and his assistant Charles Best came up with a way to remove destructive enzymes within the pancreas. For the first time, Banting and Best isolated the pancreatic secretion known as insulin. The first clinical trial of this new life-saving serum took place on January 11, 1922 through the course of several injections of insulin resulted in marked clinical improvement. The diabetic patient in this case benefited from the elimination of glycosuria and ketonuria. Furthermore, by the end of the 10-day course of treatment, the patient’s blood glucose level normalized.

**History of Diabetic Nursing Education**

During the early 20th century, nurses were instructed to provide care, known as “starvation therapy” to decrease acidosis and glycosuria in diabetic patients. Prior to 1936, many people with very limited or no training were practicing as nurses. In 1923, a landmark study, known as the Winslow-Goldmark Report, provided valuable insight on the state of nursing education and care in the United States. This investigation showed that hospitals controlled nursing schools, however, they lacked proper funding to educate nurses.

After the discovery of insulin and the widely publicized results of the Winslow-Goldmark Report, the urgency of adequate education for nurses became a priority. One well-known Harvard physician, Dr. Elliott Joslin, became a pioneer in diabetes education for nurses. He worked hand-in-hand with nurses to develop diabetes education models. By 1936, nursing care had evolved into diabetes specialties, which are similar to paradigms in place today for treating diabetes. Thanks to the invention of insulin and the work of Dr. Joslin, the diabetes nurse’s management program advanced from the “starvation method” to a regimen, which included diet counseling, exercise, insulin administration, urine glucose monitoring, foot care and other diabetes specialty services.

**Pathophysiology of Diabetes**

Diabetes mellitus, or simply diabetes, is a metabolic disorder that affects the way the body uses food for energy. Most of the food we consume is broken down by the digestive system into sugar called glucose. During normal digestion, glucose travels through the bloodstream, and is utilized by cells as a primary source of energy in the body. For this physiological process to transpire, insulin must be present. This essential peptide hormone is produced by the pancreas during digestion and is indispensable for proper blood glucose regulation. Insulin helps to regulate the body’s fuel homeostasis in muscles, tissue and in the liver where it stores fat, carbohydrates and amino acids.

The mean range of glucose levels in the body is 70 - 100mg/dL. When plasma concentration exceeds 180 mg/dL, glucose is removed from the body in the urine. This condition is known as glycosuria. Normally, the kidneys can reduce the excess levels in the blood however, when this fails tissue damage can occur. The kidneys typically excrete glucose when plasma glucose levels exceed 275 mg/dL. For this reason, kidneys can’t act alone to regulate blood sugar levels in the prevention of diabetes.

In the case of diabetes, the pancreas fails to produce enough insulin, or cells in the body do not respond to the insulin produced. This causes an accumulation of glucose in the blood, which leads to a condition known as hyperglycemia. This excess glucose is stored in the liver and muscles in the form of glycogen. Glycogen stored in the liver is released when the body’s blood sugar is low. In healthy people, the amount of glucose released by the liver is proportionate to the amount of glucose used by the tissues. In the diabetic, the renal tubes are unable to absorb the excess glucose filtered by the glomeruli. Hyperglycemia is defined as blood glucose levels of greater than 130 mg/dL or less than 180 mg/dL.

Hypoglycemia is the opposite of hyperglycemia and is characterized by an abnormally low level of blood glucose (below 70 mg/dL). During typical episodes of low blood glucose in people without diabetes, a hormone made by the pancreas, known as glucagon, signals the liver to break down glycogen and release glucose into the blood. However, people with diabetes can have impaired glucagon response. Insulin regulation also plays a role in hypoglycemia. Excessive doses of insulin are frequently associated with hypoglycemic episodes.

This renal excretion of glucose causes excretion of water, which causes excessive urination, known as polyuria. This condition is typically classified in adults as the production of over 2.5 liters of urine daily. Polyuria can lead to dehydration, dry skin and blurred vision. This reduction in fluid is responsible for an increase in serum polarity. This stimulates the thirst center of the hypothalamus, and causes excessive thirst. This condition is called polydipsia. The third hallmark symptom of high blood sugar is known as polyphagia. This condition results in excessive hunger and is caused by the body’s inability to transmit glucose through receptors into the cells. Without the adequate amount of glucose, the cells...
The three types of diabetes are classified by the Centers for Disease Control and Prevention as:
- Type 1 Diabetes Mellitus
- Type 2 Diabetes Mellitus
- Gestational Diabetes

The majority of diabetes cases fall within the first two categories, however, a closer examination of all three types will be explored later in this course.

**Pathogenesis of Type 1 Diabetes**

Historically, type 1 diabetes (T1DM) was labeled as insulin dependent diabetes or juvenile diabetes. The WHO and the American Diabetes Association changed the nomenclature due to the evolution of treatment options. The classification is now based on pathogenesis instead of the requirement of insulin therapy.

Over 95% of people with T1DM develop the disease before the age of 25. The annual incidence in children under the age of 10 is 19.7 per 100,000. According to the American Diabetes Association, 1 in every 400 children in the United States currently has type 1 diabetes. Recent literature published in the *Journal of Hormone Research in Pediatrics* helped to explain why T1DM typically develops earlier in life. This report advised that several studies proved that autoantibodies that play a role in T1DM emerge prior to the age of three months.

Type 1 diabetes is considered an autoimmune disorder and occurs when the pancreas fails to produce insulin. During T1DM, the pancreatic beta cells are damaged or destroyed, which causes insulin to decline. This lack of insulin causes blood glucose levels to rise. During this state, known as hyperglycemia, glucose levels are elevated in the blood and the unutilized sugar is excreted in the urine. This accumulation of glucose in the bloodstream prevents it from going into the cells (Figure 1). This results in the body’s inability to convert the glucose into energy and eventually leads to the symptoms of T1DM.

While the complete etiology of beta cell destruction is unknown, several studies have indicated that autoantibodies that appear in early childhood are predictive for the onset of T1DM. A variety of prospective cohort studies have also examined certain risk factors that contribute to the beta cell autoimmunity and the associated progression of T1DM. One promising study, known as the SEARCH for Diabetes in Youth, is an ongoing study seeking to understand the classification, occurrence and progression of diabetes among children and adolescents. This project is sponsored by the Centers for Disease Control and Prevention (CDC) and the National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK). The study currently has more than 20,000 enrolled participants and is scheduled through 2015.

**Risk Factors for Type 1 Diabetes**

The risk for developing T1DM can be calculated by measuring molecules, known as biomarkers, which reveal potential autoimmune reactions in the pancreas. Although the exact cause of the autoimmune response in T1DM is unknown, evidence suggests several risk factors, which may enhance the susceptibility for the disease.

**Genetic Factors**

Researchers have located certain genetic chromosomes and genes, which are related to T1DM. The majority of these genes are referred to as human leukocyte antigen (HLA) genes, they also are responsible for approximately half the genetic risk for T1DM. These HLA genes encode proteins known as the major histocompatibility complex and can have an effect on the body’s immune system. Another genetic factor of the disease pathogenesis is the DNA responsible for the regulation of the insulin gene. Specific variations in this gene can either promote or suppress T1DM.

Familial clustering and twin studies reported by the NIH in 2012, have indicated that approximately 80% to 90% of people with T1DM do not have any affected parent or sibling. However, according to this research first-degree relatives are still at an increased risk of developing the disease. In the United States, the risk is increased to 1:50 in offspring who have mothers with T1DM and 1:15 in offspring with fathers who have T1DM, suggesting that children are more likely to inherit the disease from their father. The sibling risk is about 1:12 to 1:35. These odds are also greater in siblings who have identical HLA. The greatest genetic risk exists among identical twins.

**Environmental Factors**

Research published by the National Center for Biotechnology Information, U.S. National Library of Medicine revealed that the environment plays a role on the development and progression of T1DM. Because of the increasing incidence of T1DM, researchers have conducted extensive retrospective and cross-sectional studies, which have suggested certain environmental triggers for the disease.

Certain viruses have been linked to the onset of T1DM. Mumps, rubella and herpes viruses appear to increase the chance of autoimmunity, which precipitates T1DM in people with predisposed autoimmunity. Recent cohort studies have revealed a relationship between human enterovirus (HEV) infections and the induction of beta cell autoimmunity, which is strongly linked to T1DM. HEVs are small virus particles with more than 100 known different strains.

HEVs can be transmitted by touch, contaminated food or other objects containing virus particles. The virus gains access to the circulation through the lymphatic system and ultimately to organs such as the pancreas. Autopsies have revealed the presence of HEVs in the pancreas of T1DM patients.
ers have concluded that this is direct evidence concerning the affect viral exposure has on the pathogenesis of T1DM.

One interesting study by Roivainen and Klingel investigated the link between day care exposure and the incidence of T1DM. Although communal places such as day care facilities, preschool and playgrounds strikes fear in many “germaphobe” parents, several well-designed case control studies have indicated that early social mixing, as in the case at day care, offers protective mechanisms against childhood diabetes. Researchers in these studies believe that exposure to infections in infancy help to strengthen immunoregulatory functions that have a protective effect against diabetes in children before the age of five. Another environmental factor concerning T1DM has to do with the so-called “hygiene hypothesis,” which proposes that T1DM incidence in most industrialized societies is less due to the reduced risk of parasites.

Current evidence has also shown a correlation between dietary factors and the development of T1DM. For example, investigators found that feeding infants cow’s milk influences the autoimmune response, which leads to T1DM. Animal studies have indicated that cow’s milk introduced during the weaning period caused insulinitis and diabetes. A more recent systematic review proposed that the lack of breastfeeding possibly increased the risk for developing T1DM later in life. During this review, eight studies suggested that breastfeeding has a protective role against T1DM. Seven additional studies advised that a short duration or the complete lack of breastfeeding increased the risk for eventual development of T1DM. Finally, recent studies have also showed that vitamin D supplementation in infancy may have a protective role against T1DM.

Pathogenesis of Type 2 Diabetes

Type 2 diabetes mellitus (T2DM) occurs most frequently in adults over 40 years of age. Prior to being changed by the WHO, T2DM was previously known as adult-onset diabetes, insulin-insensitive diabetes and non-insulin dependent diabetes. In recent years there has been a dramatic increase in the incidence of T2DM. Recent statistics published by the CDC extrapolate that over 26 to 27 million people currently have T2DM in the United States. In fact, T2DM is the most common non-communicable disease in the world.

T2DM is characterized by elevated levels of glucose in the blood. Insulin resistance is the primary contributor toward T2DM. During T1DM, the islet cells in the pancreas are destroyed resulting in the depletion of insulin. Conversely, during T2DM there may be a normal amount of circulating insulin in the body, however, the body’s muscles, fat and liver become resistant or do not use insulin effectively. This condition is called insulin resistance. Insulin resistance can begin in the components of the intercellular cascade, which connects the insulin receptors in the cell membranes. Unlike in the case with T1DM, at least initially, people with T2DM typically do not need insulin to survive.

In normal glucose regulation, insulin stimulates peripheral glucose uptake through the muscles and fat. At the same time, it inhibits glucose production in the liver. During this process, the gastrointestinal (GI) tract helps to transport the glucose into the body during digestion. Conversely, in an environment of impaired glucose tolerance, GI absorption of glucose aggravates already compromised glucose regulatory systems and overpowers the body’s other organs. Because the organs are overwhelmed, they are unable to excrete the surplus glucose, which results in hyperglycemia. T2DM also frequently causes hyperinsulinemia, which is characterized by excess insulin in the bloodstream. Also, during T2DM, the liver both overproduces and underuses glucose. Hyperglycemia causes injury to the beta cells in the pancreas, which makes it even more difficult for the pancreas to regulate the elevated levels of blood glucose. Over time, this process causes harm to blood vessels, nerves, eyes, kidneys and other organs of the body.

Risk Factors for Type 2 Diabetes

T2DM is a heterogeneous disease and can be attributed to a variety of causes. For example, many patients with T2DM are obese, and obesity alone often causes insulin resistance. The potential for developing T2DM increases certain genetic influences and environmental factors such as obesity, dietary factors, age, sedentary lifestyle, smoking and excessive alcohol consumption.

Genetic Factors

According to the National Diabetes Clearinghouse, genetics play a significant role in people with diabetes. Having certain inheritable genes can definitely increase susceptibility to T2DM. Current research has identified over 36 genes that have been associated with the development of T2DM. These genes influence insulin production rather than insulin resistance. One gene identified through research is known as the TCF7L2 gene. People with this gene have an 80% increased risk of developing T2DM compared to those who do not carry the gene. In fact, TCF7L2 is considered the most dominate gene related to the vulnerability of T2DM. Twin studies have also shown that if one identical twin has diabetes, the likelihood of the other twin contracting the disease is approximately 90%. One landmark study of identical twins revealed a 100% concordance rate for the disease.

The pathogenesis of genetic susceptibility is typically related to genetic irregularities related to glucose metabolism. Candidate genes involved in insulin secretion of beta cells play a role in these genetic abnormalities. Researchers have also linked genetic risk to mitochondrial genes and insulin receptor genes. One recent genome-wide association study by Morgan (2012) has recognized the mutation in the KCNQ1 gene, which is related to insulin secretion. The results of this study concluded that this specific gene played a significant role as a genetic risk factor for T2DM in Asian ethnic groups, who were the participants in the investigation.

Obesity

Obesity is one of the strongest risk factors related to insulin resistance and T2DM. According to research reported in The Journal of Clinical Endocrinology & Metabolism, obesity is present in the majority of patients
with T2DM. In fact, obesity accounts for almost 85% of the overall risk of developing T2DM. Obesity can be attributed toward eating and lifestyle habits, genetics, or a combination of all three factors. However, one thing is certain - obesity is a major dilemma in the United States and it is cited as one of the leading major health issues over the past couple decades. According to the U.S. CDC, over one third (35.7%) of adults in America are obese. The classification of obesity is defined by the measurement of the body mass index (BMI). The chart below (Figure 2) published by the WHO identifies the current standard for obesity in the United States.

<table>
<thead>
<tr>
<th>Classification</th>
<th>BMI (kg/m²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Underweight</td>
<td>&lt;18.5</td>
</tr>
<tr>
<td>Normal</td>
<td>18.5 - 24.9</td>
</tr>
<tr>
<td>Overweight</td>
<td>25.0 - 29.9</td>
</tr>
<tr>
<td>Obesity</td>
<td>30.0 - 34.9</td>
</tr>
<tr>
<td>Extreme Obesity</td>
<td>&gt;35</td>
</tr>
</tbody>
</table>

The CDC has estimated that the occurrence of T2DM is exploding at an alarming rate of between two to four million cases annually. A wide variety of recent literature has attributed this steep incline in T2DM incidence to the abundance of high calorie foods and the associated prevalence of a sedentary lifestyle in the western world. Countless reports have documented the problem of childhood obesity due to fast food, soda along with countless hours in front of video games and television sets. The statistics are frightening. According to recent government reports, childhood obesity has tripled over the past 30 years, partly due to the lack of activity. Less than 25% of high school students are engaged in the proper amount of moderate-intensity physical activity. Considerable progress has been made in understanding the causes of T2DM, it is now time to address positive lifestyle changes that will interrupt this disturbing trend.

Visceral Fat Obesity

An abundance of visceral fat is directly associated with insulin resistance. This type of fat is characterized by intra-abdominal fat or “belly fat,” rather than subcutaneous fat or gynecoid “pear shaped” fat, which usually manifests in the thighs and gluteal regions of the body. Visceral fat cells are more sensitive to neural signals than other fat cells, however, they are less responsive to insulin. The intraperitoneal fat drains to the liver and leads to excessive free fatty acid in the bloodstream. This process is one of the causes of insulin resistance.

When the level of free fatty acids becomes excessive in the blood, the liver excretes additional glucose in the blood. This causes a lack of glucose in the muscles and liver and ultimately results in hyperglycemia. At this point, the beta cells can no longer compensate for the glucose abnormalities. Once the pancreatic cells become activated, hyperglycemia causes the pancreas to release more insulin. This higher amount of circulating insulin that is present in the body is known as hyperinsulinemia.

Diet as a Risk Factor

A proper nutrition program for the diabetic patient will be explored later in the course, however, let’s take a moment to discuss how diet can affect the development of T2DM. The glycemic index of food is the measurement of the effects certain foods have on the blood glucose level after ingestion. Foods with a high glycemic index cause a spike in blood glucose. Conversely, food with lower glycemic index scores cause less of a spike in blood glucose after a meal. The following chart is an example of low-glycemic index foods:

<table>
<thead>
<tr>
<th>Pasta</th>
<th>Oats</th>
<th>Barley</th>
</tr>
</thead>
<tbody>
<tr>
<td>Beans</td>
<td>Legumes</td>
<td>Lentils</td>
</tr>
<tr>
<td>Apples</td>
<td>Bulgur</td>
<td>Oranges</td>
</tr>
<tr>
<td>Milk</td>
<td>Yogurt</td>
<td>Pumpernickel</td>
</tr>
</tbody>
</table>

The research regarding the prevention of T2DM from a diet composed of foods with a lower glycemic index has been mixed. Some studies have suggested a lower risk of developing T2DM, while others have failed to show any difference. Nevertheless, some of the foods with a lower glycemic index are also high in fiber, which has favorable outcomes in reducing the risk of diabetes. (Websites such as www.the-gi-diet.org/glycemindexchart/ can be a valuable resource in learning more about low, medium and high glycemic index foods).

According to a recent study published in the American Journal of Lifestyle Medicine, several investigations have proven that increased consumption of fiber and whole grains may have significant benefits in the prevention of T2DM. The components of vegetables related to lower risk of T2DM may include antioxidants such as tocopherols and carotenoids.

While dietary fiber may reduce the risk if T2DM, saturated fat may have the opposite effect. Several prospective studies have indicated that there is a strong link between meat consumption and a higher incidence of T2DM. In one recent study, participants who consumed meat had a 40% increase in insulin sensitivity compared to their vegetarian counterparts. Researchers in the study believe that the higher intake of saturated fat increases the amounts of fatty acids in the cell membranes of the skeletal muscle. This action creates insulin production by altering insulin receptor binding as well as affecting cell signaling.

Other Risk Factors

Although obesity and genetics are the primary risk factors for the development of T2DM, other health and environmental issues, such as hypertension, smoking, alcohol and sleep deprivation also play a role. The association between hypertension and T2DM has been the subject of several studies in the medical community. This link is based on the role that angiotensin plays in the body. Angiotensin, produced by the blood, can affect blood pressure and interfere with normal metabolic signaling of insulin.

Smoking has also been linked to the development of T2DM through several recent prospective studies. In a 20-year study, known as The Nurses’ Health Study, women who smoked at least 25 cigarettes a day were compared to non-smokers for the risk of disease. The researchers concluded that these women, absent of any other known risk factors, still had a greater risk of developing T2DM than the non-smokers. Another prospective study sampled 275,000 men and 434,000 women. The men who smoked at least 2 packs per day had a 45% greater risk for diabetes than the non-smokers, and the women had a 75% higher risk for the disease than the non-smoking group.

When it comes to alcohol as a risk for T2DM, there are two separate classifications to consider - moderate use of alcohol vs. excessive use. Like many things in life, moderate use does not appear to be a risk factor, in fact, it actually may have protective benefits. Excessive use of alcohol does appear to be a potential risk factor for T2DM. Recent systematic review and meta-analysis confirmed previous studies regarding the role of moderate alcohol consumption. This research revealed that when users consumed 22 grams of alcohol daily, protective benefits were seen. On the contrary, when users consumed more than 50 grams of alcohol per day, the protective benefits were negated and harmful effects occurred. In another recent investigation, researchers at the Diabetes Obesity and Metabolism Institute
advised that long-term binge drinking causes insulin resistance, which ultimately leads to T2DM.

Scientists are investigating lack of sleep as another recent emerging risk factor for T2DM. In a piece by Ley et al (2016) that discussed findings from the Data from the Nurse’s Health Study (2012), it was assessed that both quantity and quality of sleep were factors in the development of diabetes; especially long and short durations of sleep as well as extended periods of night shift work.

**Metabolic Syndrome**

Metabolic syndrome is defined by the World Health Organization (WHO) as a cluster of metabolic and hemodynamic risk factors frequently found together. This combination of health problems have also been called “insulin resistance syndrome,” “the deadly quartet,” hypertension syndrome,” and “syndrome x.” This lethal cocktail of health problems includes:

- Insulin resistance (hyperinsulinemia)
- Dyslipidemia (abnormal amounts of lipids in the blood)
- Hypertension
- Advanced atherosclerosis

Along with the above health concerns, obesity is also present in many cases of metabolic syndrome. Other risk factors include:

- Decreased HDL cholesterol
- Increased visceral fat
- High triglycerides
- High fasting glucose
- Increased uric acid

A diagnosis of metabolic syndrome is made if at least three of the aforementioned factors are present.

Studies have shown that individuals with metabolic syndrome have five times the risk of developing T2DM than people without the disorder. Furthermore, according to the National Heart, Lung and Blood Institute, metabolic syndrome is on the way to overtaking smoking as the leading risk factor for heart disease. Genetic influences such as family history of the disease and environmental factors (such as progressive weight gain, sedentary lifestyle and smoking) all play a contributing role toward the development of metabolic syndrome.

**Other Types of Diabetes**

In addition to T1DM and T2DM, there are other types that are universally recognized by the International Diabetes Federation and the American Diabetes Association.

These other forms of the disease include:

- Prediabetes
- Gestational diabetes
- Drug-induced diabetes

**Prediabetes**

During prediabetes, patients exhibit some of the diagnostic symptoms for diabetes, however, the indicators present don’t meet the formal criteria for diabetes (Figure 4). Individuals with prediabetes have impaired fasting glucose and/or impaired glucose tolerance. According to the American Diabetes Association, these abnormal levels include:

- Fasting plasma glucose (FPG) of 100 to 125 mg/dL
- Impaired glucose tolerance (IGT) of 140 to 199 mg/dL

Research conducted on the pathophysiology of prediabetes by M. Abdul-Ghani and R. DeFronzo in Current Diabetes Reports (2009), has indicated that people with prediabetes are at a greater risk for developing T2DM. In fact, over a 10-year period, approximately half of the people with prediabetes develop T2DM. Along with the increased risk of T2DM, prediabetes also enhances the potential for developing cardiovascular disease. Several clinical trials have documented that prediabetes increases the likelihood of later cardiovascular conditions by approximately 50%. Scientists involved in these studies have attributed insulin resistance as the likely risk factor for atherosclerosis.

Recent studies have shown alarming statistics regarding the prevalence of prediabetes in children. The epidemic of obesity in the United States is fueling this trend. It’s estimated that approximately 10% of obese children have symptoms that meet the diagnostic criteria for prediabetes. One recent longitudinal study followed 102 obese children for two years. The results of this study revealed that approximately 30% of these children, who had prediabetes, eventually developed T2DM. Researchers concluded that significant weight gain was the greatest risk for the development of T2DM in these children.

**Gestational Diabetes**

In the United States, gestational diabetes affects approximately 3% to 10% of pregnancies. According to the CDC, gestational diabetes increases a woman’s chance of developing T2DM by 40% to 60%, 5 to 10 years after pregnancy. Moreover, if a woman has gestational diabetes during pregnancy, the chance of experiencing it in future pregnancies is as high as 68%. Race and ethnicity also play a role in the potential development of gestational diabetes. While less than 2% of Caucasian women develop gestational diabetes, over 15% of Native American women acquire the disorder.

Gestational diabetes occurs from abnormal increases in glucose levels during pregnancy. During gestational diabetes, insulin receptors malfunction causing a marked increase in insulin sensitivity. Hormones present during pregnancy, such as progesterone, prolactin, estrogen and human placental lactogen have been shown to have an adverse influence on pancreatic beta cells. There are also certain risk factors that may contribute toward a greater risk of diabetes. These potential causes include:

- Family history of diabetes
- Hypertension
- Previously given birth to a baby that weighed over 9 pounds
- Obesity
- Excessive amount of amniotic fluid present
- Belonging to a susceptible ethnicity
- Older maternal age
- Previous unexplained stillbirth or miscarriage

Women with gestational diabetes are often asymptomatic or have very few distinguishable symptoms. After delivery, glucose levels typically return to normal. Nevertheless, women with gestational diabetes should be monitored closely after birth to screen for signs of diabetes due to the increased risk factor of developing the disease later in life.

**Drug-Induced Diabetes**

Drug-induced diabetes is defined as the development of a hyperglycemic state caused by the ingestion of drugs. Several types of drugs have been implicated as potential underlying causes of T2DM. In most circumstances, the specific protagonist drug has triggered an already abnormal carbohydrate metabolism in the body. In predisposed glucose intolerant patients and those with a familial history of diabetes, certain drugs will shift these people from prediabetic state to a diabetic one.

Drugs that play a role as potential risk factors for diabetes are classified into four categories that can induce diabetes. These include drugs that:

1. Interfere with insulin production
2. Block insulin action
3. Interfere with insulin secretion and action
4. Increase glucose by means independent of insulin’s action

Figure 3 Illustrates the separate classifications according to the mechanism by which they contribute toward diabetes along with the associated drug in that category.
Diabetes Screening and Diagnosis

In the early stages of prediabetes, and diabetes, there is often little to no indication. However, in the latter stages of diabetes, symptoms often appear suddenly without warning. A proper screening program for vulnerable patients helps to investigate certain risk factors associated with diabetes. After doctors identify potential factors, they can address them through a patient education program that can help delay the onset of diabetes.

Hyperglycemia is one of the most frequent causes of symptoms and related health issues associated with diabetes. During the diagnostic stage, laboratory analysis is directed toward measuring glucose levels in the blood to investigate for the potential of any hyperglycemic conditions.

Along with lab work, doctors also conduct standard patient medical history assessments for patients who may have diabetes. This helps doctors identify potential subtle symptoms that may indicate the presence of diabetes. During the assessment, doctors also conduct thorough examinations of the patient to check for any potential complications associated with the disease.

Screening

Because of the high prevalence of diabetes in the United States, the medical community has become increasingly reliant on nationwide education campaigns to help combat the issue. The American Diabetes Association has recommended screening guidelines for susceptible individuals with established risk factors. The risk factors for diabetes screening include:

- Family history of diabetes
- People older than 45 years of age
- Sedentary lifestyle
- Body Mass Index (BMI) greater than 25
- Hypertension
- Polycystic ovarian syndrome
- History of gestational diabetes
- Abnormal lipid levels (HDL levels < 35 mg/dL and/or triglyceride level > 250 mg/dL)

The American Diabetes Association has published two online risk questionnaires to help screen for an individual’s risk for diabetes. The first tool (“*Diabetes Risk Test*”) requires the user to input information regarding weight, height, age, exercise history and family history. The other online tool (“*My Health Advisor*”) is more detailed and requires other biomedical information, such as cholesterol readings and blood pressure, which make it more applicable in a healthcare setting. These questionnaires are available at www.diabetes.org/diabetes-basics/prevention/diabetes-risk-test/

The U.S. Preventive Services Task Force recommends screening for diabetes every three years for adults older than 45 years of age and every two years if any of the above risk factors are present. Educating the patient can mitigate several of the above risk factors, such as sedentary lifestyle, hypertension and a high BMI. Proper screening goes hand-in-hand with an instructional program that can help to reduce risk factors reported in the diabetes screening guidelines.

Diagnosis and Clinical Examination

Diabetes can be diagnosed through laboratory analysis, medical history assessment and physical examination. Once a positive diagnosis of diabetes is confirmed, routine laboratory and physical examinations are conducted to monitor the effectiveness of treatment and to screen for the presence of potential comorbidities. Other associated conditions, such as hypertension, should be monitored closely, due to the fact that they can exacerbate the presence of diabetes.

If other related health issues are identified, patients are often referred to healthcare professionals that specialize in the specific existing condition. The healthcare team routinely consists of the primary care physician, certified diabetes educator and dietitian. Depending on the comorbidities, podiatrists, ophthalmologists, dentists and mental health professionals may be needed.

Diagnostic Testing

Blood glucose testing is currently the most widely used tool for the diagnosis of diabetes. Testing for blood glucose consists of four different types of exams, which include:

- Random Blood Glucose test (RBG)
- Fasting Plasma Glucose test (FPG)
- Oral Glucose Tolerance test (OGTT)
- Glycosylated Hemoglobin test (HbA1c)

These tests enable healthcare providers to investigate for evidence of diabetes, however, they do not differentiate between T1DM and T2DM. It should also be noted that laboratory test results concerning glucose frequency vary depending on activity levels, meals and stress. Therefore, a multifaceted diagnostic approach of blood work, along with physical examination and medical history assessment is always warranted.

Random Blood Glucose Test

RBGT is also known as a simple blood test and is taken regardless of when food was last consumed. It measures the blood glucose level and a RBG result of greater than 200 mg/dL, along with the presence of other symptoms, may indicate diabetes. If this test yields a positive result, a diagnosis of diabetes is then verified using one of the other blood glucose tests.

Fasting Plasma Glucose Test

The FPG test is administered after at least an eight hour overnight fast. This is considered a first line method of testing because of its simplicity and cost-effectiveness. This tests the body’s ability to retain glucose after digestion and is most accurate in the morning.

FPG result glucose levels classify as follows:

- **Routine**: less than 99 mg/dL
- **Prediabetes**: between 100 and 125 mg/dL
- **Diabetic**: exceeding 126 mg/dL
One major drawback of the FPG test is that 30% to 40% of individuals with undiagnosed diabetes test in the normal range for FPG levels. If an FPG test does reveal levels above 126 mg/dL, it must be confirmed by retesting on another day, due to the fact that FBG levels vary daily.

**Oral Glucose Tolerance Test**

At one time, the OGTT was the gold standard for the diagnosis of diabetes. While it has proven more accurate than FPG tests, it is expensive and fairly inconvenient. In fact, the American Diabetes Association recognized the difficulties in performing the OGTT, and now recommends the FPG as a preferred method in routine clinical practice.

The OGTT measures blood glucose levels after at least an eight hour fast. The test is administered two hours after the subject has consumed liquid containing 75 grams of glucose. The OGTT examines the body’s ability to endure a large amount of glucose. The OGTT measures blood glucose levels after at least an eight hour fast. The test is administered two hours after the subject has consumed liquid containing 75 grams of glucose. The OGTT examines the body’s ability to endure a large amount of glucose.

OGTT result glucose levels classify as follows:

- **Routine:** less than 139 mg/dL
- **Prediabetes:** between 140 and 199 mg/dL
- **Diabetic:** exceeding 200 mg/dL

Test results can be altered in patients taking medications that affect glucose tolerance, such as glucocorticoids, diuretics and contraceptives.

**Glycosylated Hemoglobin Test**

The HbA1c test, also known as the A1C test, is used to measure the percentage of hemoglobin to which molecules have become attached. During typical circumstances of hyperglycemia, a large amount of glucose is prevalent in red blood cells. These cells also contain the oxygen-carrying protein hemoglobin. When glucose invades the red blood cells, it links to the hemoglobin and forms glycosylated hemoglobin. The A1C test measures the average of an individual’s blood glucose levels for a three month period. As glucose levels increase, so does the amount of glycosylated hemoglobin.

In recent years, significant improvements in the standardization of the A1C test have led to both the WHO and the American Diabetes Association recommending it as a viable option for diagnosing diabetes. The clear advantage of this test over the FPG and OGTT is that there is no fasting required. Because of its convenience, the American Diabetes Association recommends that current diabetics have the test at least twice a year to monitor their treatment programs.

A1C percentage results classify as follows:

- **Routine:** below 5.7%
- **Prediabetes:** between 5.7% and 6.4%
- **Diabetic:** exceeding 6.5%

It is important to note that exact levels of “normal” sometimes vary from laboratory to laboratory. Moreover, the A1C test can be inaccurate in individuals with genetic hemoglobin mutations, such as anemia, thalassemia and hemolysis.

**Other Tests**

Once laboratory tests indicate the presence of diabetes, it is necessary to diagnose the patient with either T1DM of T2DM to determine the relevant treatment options. T1DM and T2DM can usually be differentiated through the findings associated with lab tests, medical history assessment and physical examination.

If T1DM is expected, a urinalysis can be used to assay for ketones. This by-product is present during the breakdown of fat molecules when the body does not have enough insulin to use available glucose. Excessive ketones can result in diabetic ketoacidosis, which occurs more frequently in T1DM.

Tests can also be administered to measure C-peptide levels, which are usually low in people with T1DM. C-peptide is a molecule created during proinsulin’s conversion to insulin. A C-peptide reading of greater than 0.6 ng/mL typically indicates diabetes.

Blood lipid tests that measure triglycerides and cholesterol levels are also helpful in identifying and reducing some of the risk factors associated with T2DM. The normal range for triglycerides is less than 150 mg/dL, borderline is between 150 to 199 mg/dL and high is over 200 mg/dL.

Because diabetes is a major cause of renal disease, renal function tests are often administered in diabetic patients. **Blood urea nitrogen (BUN) levels and glomerular filtration rate (GFR)** are monitored to closely examine kidney function.

**Medical History**

Along with the typical laboratory analysis, it is important for members of the healthcare community to utilize standard questions in the patient assessment, which may be relevant to some of the known risk factors for diabetes. Since genetic factors have been shown to pose as a risk factor, the patient’s family history of diabetes should be determined.

During the patient assessment, medical staff should inquire about potential previous health history of prediabetes or diabetes. Other historical health factors to consider are current or past heart problems, hypertension, polyuria, polydipsia, nocturia or previous lab work that revealed high blood sugar.

Inquire about the patient’s current health status during the assessment as it may relate to potential symptoms of diabetes. Ask the patient about sudden weight gain, increased thirst, unusual hunger, increased urination, nausea, numbness or tingling in the feet, weakness and fatigue.

Because several types of drugs can increase plasma glucose levels, medication history is another important factor to consider during the assessment. Screen for known medications that affect glucose levels, such as:

- Antipsychotic drugs
- Antiretroviral drugs
- H2 blockers
- Glucocorticoids and steroids.
Physical Assessment

The physical examination also plays a vital role in the diagnosis stage for diabetes. During the exam, the goal is to investigate for symptoms that can potentially establish a diagnosis of diabetes. A diabetes-focused physical exam involves assessment of vital signs, funduscopic, cardiovascular and foot examinations as well as a detailed inspection of the mouth and skin.

Vital Signs

Diabetes can cause orthostatic hypotension and autonomic neuropathy. Measuring orthostatic blood pressure (just after a patient has stood up) helps to assess volume status and will assist in knowing if the patient has autonomic neuropathy. The pulse reading also corroborates the potential of damage to the nerves. For example, relative tachycardia is a routine occurrence in autonomic neuropathy. In diabetic patients, a continual resting blood pressure of less than 130/85 mmHg is typically an indication of potential future complications.

Funduscopic Examination

Diabetes has been known to cause eye problems such as glaucoma, cataracts and retinopathies. The eye exam should include a detailed observation of the optic disc and macula. Symptoms of concern include small round spots (retinal hemorrhages), white or gray areas with fluffy borders or tiny spots with sharp edges (exudates). If any of these symptoms are present, the patient should be referred to an ophthalmologist for a more detailed examination.

Foot Examination

Diabetes can cause lower-extremity complications such as foot ulcers, reduced blood flow, poor healing, chronic infections and peripheral neuropathy. If untreated, these can and often do lead to amputation. The foot examination includes palpation of the dorsalis pedis and posterior tibial pulses. The feet should also be carefully examined for any signs of ulcers or infections. Documentation of a toenail capillary refill test should also be noted.

Other Examinations

Examine the body for signs of infection or wounds that are slow to heal. Pay close attention to the peripheral extremities. Because diabetes increases the chance of dental disease, examine the teeth and gums for signs of decay and disease.

Diagnosing Gestational Diabetes

Gestational diabetes screening usually involves using the OGTT. At risk women include those with a history of gestational diabetes, family history of diabetes and women with a BMI of higher than 25 prior to pregnancy. The OGTT is usually performed during the second trimester - between 24 and 28 weeks of pregnancy (Figure 5).

Recent research has revealed the growing importance of screening for gestational diabetes in pregnant women. One recent international, multicenter study, known as the Hyperglycemia and Adverse Pregnancy Outcome (HAPO) demonstrated that increased levels of blood glucose had a direct adverse effect on pregnancy. According to the study, pregnancy complications transpired at glucose levels previously considered normal. Based on the results of this study, the American Diabetes Association adopted new screening guidelines for gestational diabetes (Figure 6).

There is no easy cure or simple treatment program for diabetes. The management and treatment of diabetes involves a multifaceted approach centered on patient education, glucose monitoring, insulin therapy, pharmacological management, surgical intervention, weight management, proper nutrition and exer-
cise. In the past, diabetes was poorly understood and the known management options were limited. Before 1921, people with T1DM died just a couple of years after the disease first appeared. Today, the medical community has a thorough grasp on the risk factors as well as the associated treatment options for diabetes.

One of the primary goals of the medical community is to prevent the onset of diabetes in susceptible people. This is the first step in the management program. While there is no cure for T1DM, the known environmental factors related to T2DM can be minimized. As discussed previously in the section on screening, research has shown that people considered high-risk (obese and/or prediabetic) can either delay or avoid the onset of T2DM through intensive lifestyle modification - low-fat diet and frequent physical activity.

The Diabetes Prevention Program Research Group has recently conducted research that corroborates the importance of lifestyle management for diabetes in vulnerable individuals. During this large, randomized clinical trial study, 3,234 subjects under the age of 25 with impaired fasting glucose (IFG) or impaired glucose tolerance (IGT) were assigned to either:

1. Intensive Lifestyle Modification program, consisting of a minimum of 150 minutes of weekly physical activity and a low-fat diet.
2. Standard Lifestyle Modification program with metformin therapy (an oral medication for treating type 2 diabetes that lowers blood glucose)

The results of this trial revealed that subjects assigned to the Intensive Lifestyle Modification program showed a 58% reduction in the incidence of diabetes compared to a 31% reduction in the Standard Lifestyle Modification program with metformin treatment group.

Once the disease manifests, however, management programs will concentrate on the regulation of blood sugars, reducing the severity of symptoms and preventing complications through a multidisciplinary team approach.

Patient Education

A successful diabetes management program includes patient education and participation. Research has proven that patients who stay informed about the disease can manage their blood sugar levels easier. These patients also better understand the warning signs regarding the complications associated with diabetes. Diabetes educators, physicians, nurses, occupational therapists and physical therapists all play a vital role concerning health promotion and lifestyle changes that can assist the patient with diabetes. In addition to the medical team, the patient must also take charge of their health.

Figure 7

Patient Self-Management Topics
- Blood glucose monitoring program
- Signs of hyperglycemia
- Symptoms of diabetes
- Nutritional guide
- Exercise schedule
- Long-term complication prevention
- Management of associated health problems
- Medication schedule
- Patient resource guides

The American Diabetes Association provides information regarding diabetes education throughout the United States as well as comprehensive information on management programs for diabetes. This can be a valuable resource for both patients and other members of the healthcare team when it comes to topics for patient education. (Figure 7).

Blood Glucose Monitoring

An important part of an effective diabetes self-care plan is to instruct patients on how to monitor their blood glucose levels at home. Self-Monitoring of Blood Glucose (SMBG) provides the patient with immediate feedback on glucose levels in regards to activity, food and medications. This information allows medical staff to make any needed adjustments to the patient healthcare plan.

Another natural by-product of glucose monitoring is that it gives the diabetic patient a sense of self-empowerment and control over the disease.

Patients that have control over their blood sugar levels start to recognize the warning signs of hypoglycemia. At first, patients on insulin are asked to perform SMBG at least four times a day until an optimal range is reached. Once glucose levels are stable, patients can reduce the frequency to two to three times daily. Patients should also be instructed to keep a detailed record of their blood glucose levels to help reach target glycemic levels. T2DM patients who do not take insulin can monitor glucose levels once daily. Patients should also vary the monitoring schedule so that they check levels each week first thing in the morning, before lunch, before dinner, one to two hours after each meal and before bedtime.

Three types of glucose monitors are:
- Handheld monitors
- Continuous glucose monitors
- Urine glucose monitors

Handheld Monitor

Portable glucose monitors were first introduced in 1970 for patients to monitor glucose at home. Glucose meters are handheld electronic devices that typically measure glucose concentration from a finger prick. The measurement is taken from a drop of whole capillary blood from the finger rather than the glucose concentration in plasma from venous blood, which is the clinical procedure in laboratories. Patients read the glucose level on the monitor’s screen (Figure 8).

There are many types of handheld glucose meters on the market today. Some meters take blood samples from other areas of the body such as the forearm, upper arm or thigh. Blood glucose monitors are reliable; however, the accuracy of the meters can be influenced by poor readings caused by dirty meters, outdated test strips or a meter that is not properly calibrated.

Like many electronic devices, the technology for glucose meters continues to evolve. In the latest version of The Diabetes Educator, an article described a new handheld glucose monitor that provided bilingual audio readings.
within 6 seconds. Every year, the American Diabetes Association publishes a guide on the latest and greatest diabetes glucose monitors. The most recent guide can be found at http://www.forecast.diabetes.org/meters-jan2013.

Continuous Glucose Monitoring Systems

Continuous glucose monitors are small sensors that are inserted under the skin of the abdomen (Figure 9). These tiny sensors monitor glucose levels in tissue fluid by using a transmitter that relays data regarding glucose levels through radio waves to a small wireless monitor. The sensor usually stays in place for several days to a week and then must be replaced. While these devices are useful, they are not meant to take the place of the traditional finger prick glucose meters.

Continuous glucose monitoring is typically used to help T1DM patients control insulin therapy. The research has shown mixed results regarding the accuracy of continuous glucose monitoring. One recent study published in Diabetes Research and Clinical Practice revealed that sensors have an almost 21% inaccuracy rate when compared to conventional plasma glucose values. However, this same article did report that patients who used continuous blood glucose monitoring did spend less time in hypoglycemic episodes. One of the noted benefits of this type of test was its convenience. Patients can send results instantly to their doctor’s office through the internet. This enhances the psychological benefit of allowing patients to be in control of their own health. (Figure 10)

Urine Glucose Monitoring

Urine tests look for the presence of ketones in the urine, which are produced due to shortages of insulin in the blood. Ketones are a sign that the body’s breaking down fat for energy instead of glucose due to the lack of insulin. They are also more commonly seen in patients with T1DM.

Urine glucose monitoring test kits are available at most pharmacies. The simple kits usually include test strips. After the patient passes the urine onto the test strip, it is compared to a color chart on the strip bottle. This provides a range of the amount of ketones that may be present in the urine.

Research published recently in Diabetes Research and Clinical Practice compared blood glucose monitoring to urine glucose monitoring. The results of this investigation revealed that these two forms of glucose monitoring shared “comparable efficacy in glycemic control” in non-insulin T2DM patients.

Hypoglycemia

Diabetes Monitor

Management

Hypoglycemia occurs when blood levels drop below the normal range. In patients with T2DM, hypoglycemia is a major contributor toward meeting target glycemic goals. The hallmark symptoms of hypoglycemia include:

- Hunger
- Nervousness
- Dizziness
- Confusion
- Weakness
- Sleepiness
- Anxiety

Hypoglycemia can be extremely dangerous. An extreme decrease in blood glucose can cause a person to pass out. Patients should be aware of what may trigger low blood glucose levels and should have a plan in place in the event of a hypoglycemic episode. At-risk patients should keep rations of sugary foods or glucose tablets nearby in the event of an emergency. Susceptible individuals should also be aware of the normal glucose blood levels.

<table>
<thead>
<tr>
<th>Normal and Target Blood Glucose Ranges</th>
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<tbody>
<tr>
<td>Normal glucose levels in non-diabetics</td>
</tr>
<tr>
<td>Upon waking/fasting 70 to 99 mg/dL</td>
</tr>
<tr>
<td>After meals 70 to 140 mg/dL</td>
</tr>
<tr>
<td>Target glucose levels in diabetics</td>
</tr>
<tr>
<td>Before meals 70 to 130 mg/dL</td>
</tr>
<tr>
<td>1 to 2 hours after the start of a meal below 180 mg/dL</td>
</tr>
</tbody>
</table>

Source: American Diabetes Association, Standards of Medical Care in Diabetes, Diabetes Care.

Hypoglycemia is often caused by several medications that are commonly used to treat diabetes. One recent study by Amed et al. showed that almost 20% of T2DM patients who were 40 to 65 years of age suffered from hypoglycemic symptoms, which were caused by oral sulfonylurea. Other retrospective studies have reported that sulfonylureas ingested by diabetic patients were responsible for over 65% of hypoglycemic cases.

Management of patients with hypoglycemia has to be carefully balanced with the need for the diabetic patient to the medication to increase insulin production. The first goal of treatment is to determine the exact cause of the hypoglycemia. Then the severity of the symptoms must be assessed. And finally, a careful change in treatment plan may be warranted. This may include less medication or a different kind of medication along with a change in meal plan and/or activity level.

Insulin Therapy

After 75 years of use, insulin remains a core treatment for diabetic patients. The primary goal of insulin intervention is to mimic physiological insulin and regulate glycemic control and glycosylated hemoglobin concentrations. While medication can also be prescribed to work in conjunction with insulin, insulin is the first-line treatment.

Since patients with T1DM cannot produce insulin, they must receive it through injections. The American Diabetes Association currently recommends that most T1DM patients be treated with 3 to 4 multi-dose insulin injections daily depending on the patient’s condition.

Patients with T2DM can also be treated with insulin if the disease cannot be controlled through diet, exercise and oral medication. Insulin therapy for T2DM patients begins when the pancreas secretes 20% to 30% of the typical amount of insulin. Research has shown that the majority of people with T2DM start to receive
Types of Insulin

The various types of insulin are categorized by the following characteristics:
- **Onset** - the amount of time it takes to reach the blood stream
- **Peak** - the time period in which the insulin is working to lower blood sugar
- **Duration** - the amount of time the insulin is working in the blood stream

Historically, there were various species of insulin, which were beef, pork and a combination of beef and pork. However, recombinant human insulin - a form of insulin made from recombinant DNA that is identical to human insulin is used almost exclusively. The types of insulin include:
- **Rapid-Acting insulin**
- **Intermediate-Acting insulin**
- **Long-Acting insulin**

Some insulin therapies include a combination of short and long-acting insulin to help mimic the physiological cycle of insulin release.

Rapid-Acting Insulin

Rapid-acting insulin starts to work approximately 15 minutes after injection and peaks in about 1 hour. The duration is 2 to 4 hours. The types of fast acting insulin available are: insulin glulisine (Apidra), insulin aspart (NovoLog) and insulin lispro (Humalog).

Regular (Short-Acting) Insulin

Regular-Acting insulin starts to work about 30 minutes after injection and peaks anywhere from 2 to 4 hours after injection. The duration is 3 to 6 hours. The types of regular-acting insulin available are: Novolin R and Humulin R.

Intermediate-Acting Insulin

Intermediate-Acting insulin begins to work 2 to 4 hours after injection and peaks 4 to 12 hours later. The duration is 12 to 18 hours. The types of intermediate-acting insulin include: Novolin N and Humulin N.

Long-Acting Insulin

Long-Acting insulin works slowly and is usually administered in the morning because it lowers glucose levels evenly throughout a 24-hour period. The types of long-acting insulin include: Insulin glargine (Lantus) and Insulin detemir (Levemir).

Table 1: A quick reference guide to types of insulin.

<table>
<thead>
<tr>
<th>Insulin Type</th>
<th>Onset</th>
<th>Peak</th>
<th>Duration</th>
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<tbody>
<tr>
<td>Rapid-Acting insulin</td>
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<tr>
<td>Intermediate-Acting insulin</td>
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<tr>
<td>Long-Acting insulin</td>
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Insulin Administration and Delivery

Insulin comes suspended in liquids or dissolves and is measured in units. The units are measured in weight and 24 units are equal to 1 mg. The standard weight is U-100, which means there are 100 units of insulin in 1cc of solution. U-500 insulin is available for people who are greatly resistant to insulin.

The dose of insulin administered depends on the patient’s weight. The preprandial glucose target level is 80 to 150 mg/dL. The insulin regime typically varies from 1 to 4 injections daily. Factors related to dosage include the patient’s activity levels, type of diabetes, the amount of insulin the pancreas is producing, diet and sensitivity to insulin.

There are two primary components to insulin administration, known as basal and bolus insulin. **Basal administration** of insulin provides a steady amount of insulin throughout the day. **Bolus insulin administration** refers to the boost of insulin present after a meal in response of the glucose taken from food. Samples of basal insulin include Insulin glargine (Lantus) and Insulin detemir (Levemir) and faster-acting basal insulin consist of insulin glulisine (Apidra), insulin aspart (NovoLog) and insulin lispro (Humalog). Oral administration of insulin is not possible because it would be broken down during digestion.

The two methods of delivery are subcutaneous injection or intravenous administration. Many people use syringes and insulin pens that contain cartridges of insulin that are injected through a needle. Another option of insulin delivery is through the use of a subcutaneous insulin pump. The pump delivers both basal rate and bolus rate insulin through a catheter. The goal of the pump is to mimic the action of normal beta cells. The basal administration is preprogrammed for continuous delivery and bolus doses are programmed prior to meals or snacks.

Pharmacological Management of Diabetes

While lifestyle modifications and patient education are the cornerstone of diabetes management, the pharmacological intervention is necessary in many cases. If diet and exercise fail to control blood levels, doctors can prescribe several classes of medication to help combat diabetes. A pharmacotherapy approach can consist of the following medications:
- Biguanides
- Sulfonylureas
- Meglitinides
- Thiazolidinediones
- Dipeptidyl-peptidase 4 inhibitors
- Alpha-glucosidase inhibitors
- Amylinomimetics
- Dopamine agonists

These medications can also be prescribed in a combination of oral medication preparations. They can also be taken with insulin, or insulin can be used alone.

Table 2 A quick reference to T2DM medications, benefits and possible side effects.
<table>
<thead>
<tr>
<th>Type of Insulin</th>
<th>Brand Name</th>
<th>Generic Name</th>
<th>Onset</th>
<th>Peak</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rapid-acting</td>
<td>NovoLog</td>
<td>Insulin aspart</td>
<td>15 minutes</td>
<td>30 to 90 minutes</td>
<td>3 to 5 hours</td>
</tr>
<tr>
<td></td>
<td>Apidra</td>
<td>Insulin glulisine</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Humalog</td>
<td>Insulin lispro</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Short-acting</td>
<td>Humulin R</td>
<td>Regular (R)</td>
<td>30 to 60 minutes</td>
<td>2 to 4 hours</td>
<td>5 to 8 hours</td>
</tr>
<tr>
<td></td>
<td>Novolin R</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intermediate-acting</td>
<td>Humulin N</td>
<td>NPH (N)</td>
<td>1 to 3 hours</td>
<td>8 hours</td>
<td>12 to 16 hours</td>
</tr>
<tr>
<td></td>
<td>Novolin N</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Long-acting</td>
<td>Leveimir</td>
<td>Insulin detemir</td>
<td>1 hour</td>
<td>Peakless</td>
<td>20 to 26 hours</td>
</tr>
<tr>
<td></td>
<td>Lantus</td>
<td>Insulin glargine</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre-mixed NPH (intermediate-acting) and regular (short-acting)</td>
<td>Humulin 70/30</td>
<td>70% NPH and 30% regular</td>
<td>30 to 60 minutes</td>
<td>Varies</td>
<td>10 to 16 hours</td>
</tr>
<tr>
<td></td>
<td>Novolin 70/30</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Humulin 50/50</td>
<td>50% NPH and 50% regular</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre-mixed insulin lispro protamine suspension (intermediate-acting) and insulin lispro (rapid-acting)</td>
<td>Humalog Mix 75/25</td>
<td>75% insulin lispro protamine and 25% insulin lispro</td>
<td>10 to 15 minutes</td>
<td>Varies</td>
<td>10 to 16 hours</td>
</tr>
<tr>
<td></td>
<td>Humalog Mix 50/50</td>
<td>50% insulin lispro protamine and 50% insulin lispro</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre-mixed insulin aspart protamine suspension (intermediate-acting) and insulin aspart</td>
<td>NovoLog Mix 70/30</td>
<td>70% insulin aspart protamine and 30% insulin aspart</td>
<td>5 to 15 minutes</td>
<td>Varies</td>
<td>10 to 16 hours</td>
</tr>
</tbody>
</table>

Table 1

Source: National Diabetes Information Clearinghouse, 2013
### Medicines for Type 2 Diabetes - Benefits

<table>
<thead>
<tr>
<th>Type of Medicine</th>
<th>Benefits</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Generic Name</strong></td>
<td><strong>Brand Name</strong></td>
</tr>
<tr>
<td><strong>Biguanides - Block the liver from making sugar</strong></td>
<td></td>
</tr>
<tr>
<td>Metformin</td>
<td>Glucophage®</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Sulfonylureas - Raise the amount of insulin in the body</strong></td>
<td></td>
</tr>
<tr>
<td>Glimepiride</td>
<td>Amaryl®</td>
</tr>
<tr>
<td>Glipizide</td>
<td>Glucotrol®</td>
</tr>
<tr>
<td>Glyburide</td>
<td>Diabeta®; Glynase Prestab®; Micronase®</td>
</tr>
<tr>
<td><strong>Meglitinides - Raise the amount of insulin in the body</strong></td>
<td></td>
</tr>
<tr>
<td>Repaglinide</td>
<td>Prandin®</td>
</tr>
<tr>
<td>Nateglinide</td>
<td>Starlix®</td>
</tr>
<tr>
<td><strong>Thiazolidinediones (TZDs) - Help the body use insulin better</strong></td>
<td></td>
</tr>
<tr>
<td>Pioglitazone</td>
<td>Actos®</td>
</tr>
<tr>
<td>Rosiglitazone</td>
<td>Avandia®</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Dipeptidyl Peptidase-4 (DPP-4) Inhibitors - Raise the amount of insulin in the body after a meal</strong></td>
<td></td>
</tr>
<tr>
<td>Sitagliptin</td>
<td>Januvia®</td>
</tr>
<tr>
<td>Saxagliptin</td>
<td>Onglyza®</td>
</tr>
<tr>
<td>Alogliptin</td>
<td>Nesina®</td>
</tr>
<tr>
<td>Linagliptin</td>
<td>Tradjenta®</td>
</tr>
<tr>
<td><strong>Glucagon-Like Peptide-1 (GLP-1) Receptor Agonists - Raise the amount of insulin in the body</strong></td>
<td></td>
</tr>
<tr>
<td>Exenatide</td>
<td>Byetta®</td>
</tr>
<tr>
<td>Liraglutide</td>
<td>Victoza®</td>
</tr>
<tr>
<td><strong>Combinations</strong></td>
<td></td>
</tr>
<tr>
<td>Glyburide/metformin</td>
<td></td>
</tr>
<tr>
<td>Metformin/pioglitazone</td>
<td></td>
</tr>
<tr>
<td>Metformin/sitagliptin</td>
<td></td>
</tr>
<tr>
<td>Metformin/saxagliptin</td>
<td></td>
</tr>
<tr>
<td>Metformin + GLP-1 receptor agonists</td>
<td></td>
</tr>
<tr>
<td>Metformin + basal insulin</td>
<td></td>
</tr>
<tr>
<td>Metformin + premixed insulin</td>
<td></td>
</tr>
</tbody>
</table>

Table 2 continued on page 17
<table>
<thead>
<tr>
<th>Type of Medicine</th>
<th>Generic Name</th>
<th>Brand Name</th>
<th>Possible Side Effects</th>
</tr>
</thead>
</table>
| **Biguanides**   | Metformin    | Glucophage® | Some risk for low blood sugar  
|                  |              |            | Less weight gain than other medicines  
|                  |              |            | Higher risk for stomach problems (gas, diarrhea)  |
| **Sulfonylureas**| Glimepiride  | Amaryl®    | May cause weight gain  |
|                  | Glipizide    | Glucotrol® | 3 to 5 times more likely to cause low blood sugar  
|                  | Glyburide    | Diabeta®; Glynase Prestab®; Micronase® | May cause stomach problems  |
| **Meglitinides** | Repaglinide  | Prandin®   | May cause weight gain  |
|                  | Nateglinide  | Starlix®   | Risk for low blood sugar  |
| **Thiazolidinediones (TZDs)** | Pioglitazone | Actos® | May cause weight gain  
|                  | Rosiglitazone | Avandia® | Some risk for low blood sugar  
|                  |               |           | Can add to risk of heart failure  
|                  |               |           | Increases the risk for fracture in women  |
| **Dipeptidyl Peptidase-4 (DPP-4) Inhibitors** | Sitagliptin | Januvia® | Headaches and UTI infections  |
|                  | Saxagliptin  | Onglyza®  |                    |
|                  | Alogliptin   | Nesina®   |                    |
|                  | Linagliptin  | Tradjenta®|                    |
| **Glucagon-Like Peptide-1 (GLP-1) Receptor Agonists** | Exenatide | Byetta® | Not enough is known about the side effects of these medicines  |
|                  | Liraglutide  | Victoza®  |                    |
| **Combinations** | Glyburide/metformin | | Some combinations with drugs such as sulfonylureas may increase the risk of low blood sugar  |
|                  | Metformin/pioglitazone | | Pioglitazone combinations may cause more weight gain than other medicines  |
|                  | Metformin/sitagliptin | | Pioglitazone combinations can add to the risk of hip and non-hip fractures, especially for women  
|                  | Metformin/saxagliptin | | Some combinations with metformin increase the risk of stomach problems, but not as much as metformin alone  |
|                  | Metformin + GLP-1 receptor agonists | |  |
|                  | Metformin + basal insulin | |  |
|                  | Metformin + premixed insulin | |  |
Biguanides
These oral medications are considered the first line of therapy for T2DM. Biguanides are considered antihyperglycemic agents because they reduce basal and postprandial plasma glucose levels by lowering the amount of glucose produced by the liver. The secondary effect of biguanides is an increase of peripheral insulin sensitivity as well as an augmentation of peripheral insulin-mediated glucose uptake. Unlike some other diabetes medications, biguanides do not cause weight gain or increase the risk of hypoglycemia and hyperinsulinemia.

Metformin (Glucophage) is the only agent available in this class of diabetes medications. Metformin is frequently used in patients with T2DM who are unable to control blood glucose levels through diet and exercise. Other potential candidates include people with insulin resistance syndrome, polycystic ovary syndrome and impaired glucose tolerance. Landmark clinical trials have revealed the efficacy of this medication for the treatment of T2DM patients. During one study, patients receiving metformin had a 31% reduction in any diabetes related complications, such as heart failure, angina, amputation, renal failure and death from hyperglycemia or hypoglycemia.

Metformin is available in immediate-release and extended-release tablets. Typical dosing schedules for adults are 500 mg twice daily or 850 mg once daily. Doses may also be titrated up every 1 to 2 weeks with a maximum safe dose of 2500 mg daily. For best results, dosing should be initiated in the evening.

The most common side effect of metformin is diarrhea and nausea, which occurs in approximately 20% to 30% of users. However, because it can increase the risk of lactic acidosis, metformin therapy can be contraindicated in patients with hepatic dysfunction. Routine liver function testing is advised during the administration of this drug.

Sulfonylureas
Sulfonylureas have been used as oral insulin secretagogues for over 60 years. These drugs are used to stimulate the beta cells in the pancreas to release more insulin. They are categorized as either first or second generation and their duration can be rapid acting, intermediate acting or long acting. Treatment with these agents is only effective for patients with functioning pancreatic beta cells, therefore, they are not used for the treatment of T1DM.

The difference between first and second-generation sulfonylureas is how well they connect to the sulfonylurea receptors. During the development of these drugs, each progressive generation has bound tighter and thus requires a lower dose to bring about the same amount of insulin secretion. The first generation sulfonylureas include chlorpropamide, tolbutamide and tolazamide. Second generation agents are more frequently used in the treatment of T2DM. The second-generation sulfonylureas are glipizide, glyburide and glimepiride.

Weight gain is the most common side effect of sulfonylureas. On average, users may gain between 10 to 15 pounds. Other potential adverse reactions include headaches, drowsiness, skin rash, gastrointestinal disturbances and dizziness. Sulfonylureas may interact with some medications, such as beta blockers, thiazide diuretics, antifungals, cyclic antidepressants and corticosteroids.

Meglitinides
These agents are quicker-acting insulin secretagogues than sulfonylureas. Meglitinides affect postprandial glucose levels and antagonize the same pancreatic beta cell potassium channel as the sulfonylureas, but at a different binding location. Meglitinides should be administered prior to meals for best results. Repaglinide and nateglinide are the two drugs in this category.

Potential side effects of meglitinides include hypoglycemia, bronchitis, nausea, diarrhea, constipation, headaches, back pain, UTI and dizziness. Meglitinides may interact with some medications, such as beta blockers, thiazide diuretics, anti-inflammatory drugs, cyclic antidepressants and corticosteroids and other medications. It should also be noted that repaglinide should be used cautiously in patients with hepatic dysfunction. Routine liver monitoring is advised during the administration of this drug.

Thiazolidinediones
This class of drugs, also called TZDs, improves insulin production in the muscle and fat and decrease insulin production in the liver. TZDs act by binding to the Peroxisome Proliferator Activator Receptor-y (PPAR). This receptor affects the separation of fibroblasts into adipocytes, the PPAR also lowers free fatty acid levels. PPAR receptors are located in tissues, such as adipose tissue, skeletal muscle, vascular endothelium, macrophages and the liver.

TZDs have become a popular option for diabetic patients as a result of their ability to alter insulin insensitivity, hyperglycemia and dyslipidemia without the risk of hypoglycemia. However, the success of TZDs depends on the presence of insulin in the pancreatic beta cells.

TZDs can be used in monotherapy or in combination with insulin, sulfonylureas and biguanides. The first medication, troglitazone, was taken off the market because of adverse reactions on the liver. Today, there are two TZD agents available - pioglitazone and rosiglitazone. The efficacy of both agents for patients with T2DM has been shown in several multicenter double-blind placebo-controlled clinical trials. During one recent study, patients were randomized to receive pioglitazone or a placebo. At the conclusion of the study, researchers found that patients who were administered TZDs had significant decreased levels of fasting plasma glucose along with improvements in HbA1c and increased beta cell function.

Potential side effects of TZDs may include a reduction of hematocrit, neutrophil counts and hemoglobin as a result of plasma volume expansion. TZDs are not recommended for patients with heart failure because of the increase in plasma volume. Other adverse effects may include back pain, headaches, fluid retention and weight gain. TZDs may interact with other drugs, such as hormonal contraceptives, atorvastatin and ketoconazole.

Dipeptidyl-Peptidase-4 Inhibitors
These agents, also known as DPP-4 inhibitors, are a newer class of medications approved in the treatment of T2DM patients. The mechanism of action behind DPP-4 inhibitors is their positive influence on A1C without causing hypoglycemia. DPP-4s work by preventing the breakdown of glucagon-like peptide-1 (GLP-1) and glucose-dependant insulintrophic polypeptide (GIP) in the body. These naturally occurring compounds cause an increase in insulin synthesis, and they are also responsible for stimulating pancreatic beta cells in response to normal or increased glucose levels. DPP-4 therapy also prolongs the activation of incretin hormones and allows GLP-1 and GIP to be stored in the body longer, thus extending their action.

Several investigations have been carried out to evaluate the effects of DPP-4 inhibitors in people with diabetes. These studies have revealed an increase in both GLP-1 and insulin, as well as a decrease in blood glucose and glucagon levels after meals with use of the DPP-4 inhibitors. Examples of current FDA-approved DPP-4 inhibitors include - sitagliptin, saxagliptin, linagliptin and alogliptin.

The most commonly reported side effects are headaches and upper respiratory tract infections. However, they have an excellent record regarding their tolerability in older adults. While nausea is a common complaint for patients who take biguanides, it occurs much less frequent in patients who take DPP-4s.

Glucagon-Like Peptide 1 (GLP-1) Receptor Agonists
The Glucagon-Like Peptide 1 receptor (GLP-1) agonists are another class of medication that mimics the action of GLP-1 to reduce glucagon and increase the incretin...
effect in T2DM patients. Clinical evidence shows that these agents stimulate insulin, while decreasing DPP-4 inhibitors, which slow the breakdown of GLP-1 by increasing its concentration. Glucagon-like peptide-1 (GLP-1) receptor agonists are an alternative pharmacological approach to stimulate insulin release, while reducing the release of glucagon.

One recent study published in the European Journal of Internal Medicine compares the efficacy of DPP-4 inhibitors with GLP-1 agonists. The results indicated that both drugs offer advantages over other types of diabetes medications, such as the absence of weight gain and reduced risk of hypoglycemia. However, the marked advantage of DPP-4 inhibitors over GLP-1 agonists is due to the ease of use, tolerance and expense. Further long-term comparative controlled trials are warranted to investigate potential therapeutic strengths and weaknesses.

The two available GLP-1 agonists on the market are liraglutide and exenatide. Current research has indicated that both drugs have been proven beneficial on postprandial lipid and fasting levels in patients with T2DM. Current research is underway to investigate the cardiovascular safety of these medications.

**Alpha-Glucosidase Inhibitors**

These drugs help to lower blood glucose levels by preventing the breakdown of starches (such as pastas, bread and potatoes) in the intestines. This response has a direct effect on postprandial glycemic control. Patients with T2DM should take alpha-glucosidase inhibitors while eating (after the first bite of food) to inhibit the increase in blood glucose after meals.

These agents may be used in monotherapy or in combination with other drugs or with insulin. Research has indicated that these drugs have been successful in lowering blood sugar levels in people with postprandial hyperglycemia. Studies have shown that alpha-glucosidase inhibitors lower A1C by 0.5% to 0.8%. However, these agents are typically less effective than other diabetes drugs, such as sulfonylureas.

The two FDA approved alpha-glucosidase inhibitors are acarbose and miglitol. Both agents can be used in monotherapy or in combination with other treatment modalities. The most common adverse effect of these medications include gastrointestinal distress including feeling bloated, stomach pain, and flatulence.

**Surgical Interventions**

When diet, exercise, insulin therapy and oral medications fail to improve complications related to diabetes, surgical intervention becomes a viable option. The classification of diabetes dictates often the type of surgery. For example, some people with T1DM may benefit from pancreas transplantation (Figure 11) and/or kidney transplantation or islet cell transplantation to reduce the need for insulin and improve blood glucose regulation.

### Transplant

Conversely, some patients with T2DM are potential candidates for bariatric surgery to reduce obesity and improve quality of life. This next section will explore the surgical options for patients with either T1DM or T2DM.

### Pancreas Transplantation

The first clinical transplantation was performed in 1966 at the University of Minnesota. In the beginning, this procedure had few success rates; however, it is now a common procedure worldwide due to advances in surgical techniques. Today, there are approximately 1,200 pancreas transplants performed annually in the United States. Most pancreas transplantations are performed on patients with T1DM due to lack of insulin production. Kidney and pancreas transplantations are routinely performed together (approximately 75% pancreas transplantations) because renal failure is typically present. In rare cases, the pancreas is removed alone. The remaining 15% of pancreas transplantations are carried out after previously successful kidney transplantation, known as pancreas-after-kidney-transplantation.

The goal of these two simultaneous surgical procedures is to prevent the progress of any complication associated with diabetes, maintain normal glucose levels without taking insulin and to protect the transplanted kidney from hyperglycemia. Most pancreas grafts are taken from cadaver donors, however, in some instances a portion of the pancreas is donated by a living related donor in a procedure known as a hemipancreatectomy. During the surgery, the pancreas is placed laterally in the pelvis. The arterial flow is provided by the iliac artery and venous return is conducted through the iliac vein. The pancreatic duct outlet and duodenal segment are oversewn onto the urinary bladder. In some cases, the drainage is diverted through the bowel. The insulin released from the pancreas graft is secreted in the blood stream and typically produces approximately 800 to 1000 mL of daily fluid. The excess fluid is diverted into either the bowel or bladder. Because the graft comes from another person, the recipient’s body may reject it. To prevent this problem, healthcare staff must administer immunosuppression medications daily.

A successful procedure produces a normal glycemic state and can significantly improve the patient’s quality of life. According to research, positive outcomes of pancreas transplantation for T1DM patients almost always results in insulin independence and euglycemia. Moreover, successful pancreas transplantations can permanently restore counter regulatory functions.

### Pancreatic Islet Cell Transplantation

Successful pancreas transplantation has the valued benefit of sustained insulin dependence, however, the risk of pancreas graft rejection does exist. Islet cell transplantation offers an appealing less invasive option for some T1DM patients. During islet cell transplantation, surgeons isolate islets from a donor pancreas and transplant them to the recipient (Figure 12). After successful integration, the islets produce insulin. These pancreatic islets, also known as islets of Langerhans, are clusters of cells in the pancreas that produce insulin. Islet cells contain β-cells, which manufacture insulin, an important hormone that regulates blood sugar. Islet cells work together with the liver and the other cells of the pancreas to control the amount of glucose in the blood.

**Donor Recipient**

- **Liver**: The liver is responsible for producing glucose from glycogen, a storage form of glucose.
- **Pancreas**: The pancreas produces insulin and glucagon, hormones that regulate blood sugar levels.
- **Infusion of islet**

Islet cell transplantation is a complex procedure that involves isolating islets from the donor pancreas, ensuring they are suitable for transplantation, and then transplanting them into the recipient's body. Islet cell transplantation offers several advantages over pancreas transplantation, including decreased risk of graft rejection and less invasive surgery. However, it also has limitations, such as limited availability of suitable donor islets and the need for lifelong immunosuppression to prevent rejection.

**Figure 12** Source: National Diabetes Information Clearinghouse

© National Center of Continuing Education - nursece.com Diabetes: Managing the Sugar Highs and Lows
prise a small percentage of cells (1% to 2%) in the pancreas, and these cells are destroyed in cases of T1DM.

During this procedure, researchers use specialized enzymes, known as collagenase, to isolate the pancreatic islets from the donor. They then inject collagenase into the pancreatic duct. At this point, expansion occurs in the pancreas, which causes the separation of the islets. After distension, the donor pancreas is placed in a chamber, which consists of a lower cylinder and an upper conical section. Once in the chamber, the pancreas is immersed in the enzyme solution and a pump is used to progressively release the islets. This separation of the islets from the exocrine tissue is a process called purification.

During this process, surgeons use ultrasound to guide placement of a catheter through the upper abdomen into the portal vein of the liver. The islets are then transferred through the catheter into the liver.

After the transplantation, doctors administer immunosuppression agents to assist the immune system from destroying the transplanted islets. Similar to cases of complete pancreas transplantation, patients must take immunosuppressive drugs for life. Examples of these medications include:

- Sirolimus (Rapamune)
- Tacrolimus (Prograf)
- Daclizumab (Zenapax)
- Basiliximab (Simulect)
- Alemtuzumab (Campath)

Immunosuppressive drugs have side effects that include gastrointestinal issues, hypertension, decreased kidney function and increased susceptibility to viral and bacterial infections. In addition to the adverse effects of these drugs, other limitations exist regarding pancreatic islet cell transplantation. The primary obstacle is the shortage of islets from donors. A strict selection criterion prevents many donated pancreases from being used. Researchers are currently pursuing options to address this concern, such as using islets from pigs and using only a small portion of the pancreas from human donors. Despite the limitations, recent studies have yielded short-term positive results concerning this procedure. The Collaborative Islet Transplant Registry conducted a study regarding 571 patients who received pancreatic islet cell transplantations. The results of this study revealed that 60% of these recipients achieved insulin independence for at least 14 days. The results also indicated that eventually many of the recipients did have to take insulin again, however, the transplanted islets did improve the amount of insulin required after loss of independence.

Bariatric Surgery

Doctors who treat patients with T2DM continue to investigate the emerging role of bariatric surgery for the management of the disease. These bariatric procedures target the growing dilemma of obesity that is prevalent in patients with T2DM. Bariatric surgery is a surgical procedure to produce substantial weight loss. The term “bariatric” is derived from the Greek word “baros” meaning weight. There are currently a variety of bariatric stomach-shrinking procedures available for patients with T2DM.

The most basic procedure is known as adjustable gastric banding (AGB). During this procedure, surgeons place a laparoscopic band around the stomach to help reduce its size by restricting it to produce early satiety during meals. Another procedure, known as sleeve gastrectomy, can also decrease the size of the stomach. During sleeve gastrectomy, surgeons remove approximately 75% of the stomach, which results in a thin, crescent-shaped stomach. One of the most common bariatric surgeries is called Roux-en-Y gastric bypass (RYGB). During this procedure, surgeons use a surgical stapler to make a small gastric pouch, which creates a gastrojejunostomy between the pouch and the jejunum. This causes ingested food to bypass 95% of the stomach. Nutrients and bile combine in the jejunum and are absorbed through the remaining section of the small bowel.

Several recently published studies have investigated the benefits of bariatric surgery for patients with T2DM. One study published in the Journal of the American Medical Association involved 60 obese patients who had T2DM. Half of the patients were given conventional therapy that included diet, exercise, and medication. The other half received conventional treatment along with gastric banding surgery. The researchers in the study concluded that approximately three-quarters of those who received surgery, all symptoms of diabetes disappeared, while only 13% of the conventional therapy group had total remissions. Moreover, the surgically treated patients had a 20.7% reduction in weight, compared to a dismal 1.7% reduction in the conventionally treated group. Other recent reviews in The Cochrane Library corroborated the effective weight loss outcomes of this study, and also showed improvements in other comorbidities such as hypertension.

The risks associated with bariatric surgery include wound infections, surgical site leaks, hemorrhage and potential pulmonary events. However, a recent study conducted by the U.S. Agency for Healthcare Research and Quality reported a marked decline in surgery-related complications due to improved surgical techniques. This study revealed that post-surgery infection dropped by 58% and other complications, such as staple leakage declined from 29% to 50%.

Based on the evidence, The Journal of Diabetes recently published the International Diabetes Federation’s position regarding bariatric surgery for patients with T2DM. Due to the popularity of these surgical procedures for treating obesity, the International Diabetes Federation convened a multidisciplinary working group to develop this position statement. One of the primary goals of this group was to suggest practical recommendations for clinicians. The evidence suggests that bariatric surgery can result in drastic improvements in glycemic control, reduction in diabetic-associated complications, as well as a potential total remission of diabetes. The International Diabetes Federation recognized the clear benefits, but also recommended future research to evaluate the efficacy of different bariatric procedures and necessary regimens for diabetes management after bariatric surgery.

Diet and Nutrition

Proper diet and nutrition are crucial for successful management of all types of diabetes. People with T1DM must pay close attention to a meal plan that limits consumption of carbohydrates. T2DM patients also need to watch what they eat to avoid both hypoglycemia and hyperglycemia. A well-balanced diet plays a vital role in the regulation of blood glucose levels as well as the prevention of complications associated with diabetes. The value of a healthy diet for people with diabetes has been the subject of many studies. One large study, known as the Diabetes Prevention Program Trial concluded that, “Diet and lifestyle reduced diabetes incidence by 58% and was more effective than metformin, which conferred a 31% risk reduction.” Additional epidemiologic investigations, such as the Nurses Health Study found that 91% of the risks associated with T2DM could be attributed toward four primary diet and lifestyle factors, which included BMI of less than 25 kg/m, consumption of fiber, low-glycemic diet and routine exercise. The evidence is clear regarding the importance of a well-designed diet plan for patients.

The interdisciplinary medical team needs to understand the importance of meal size, nutritional value, timing and frequency to better educate their patients. The safest and most basic method to maintain a healthy weight is to limit caloric intake from fats and sugar while increasing fiber intake, and avoiding cholesterol and sodium latent foods. Vitamin supplementation can also play an important
role in the diabetic diet.

A large clinical trial is currently investigating the potential vitamin D has on preventing T2DM. Past studies have indicated that vitamin D does have beneficial qualities that help to ward off T2DM. This current study, funded by the National Institutes of Health, is being conducted at 20 sites across the country and will involve 2,500 participants. Stay tuned for the results!

While designing a nutritional plan, healthcare professionals need to be aware of psychosocial factors that the patient may exhibit, which may include stress, support systems, food as a comfort mechanism and preconceived notions of “diet.” Goal setting, regarding individual meal plans tailored to the patient, can be an extremely valuable tool in nutritional therapy. The American Diabetes Association currently recommends individualizing a diet plan based on the patient’s specific desired medical outcome. Some specific guidelines to facilitate successful eating behaviors may include:

• Setting realistic goals and begin slowly.
• Focusing on permanent positive lifestyle changes and avoid the term “diet.”
• Having the patient keep a food diary to track progress.
• Educating the patient regarding caloric intake, frequency of meals and healthy alternatives.
• Creating a behavior-based award system.

**Dietary Choices**

The proper blend of nutrients from healthy food choices begins with a basic understanding of fundamental nutritional terms such as carbohydrates, fat, protein, fiber, and starches. Once the patient understands the components of healthy eating, valuable resources concerning diet samples and meal plans are available both through the healthcare team and online. One such example, published by the American Diabetes Association, is called “My Meal Advisor.” This free online tool can be found at [https://www.choosemyplate.gov/health-professionals](https://www.choosemyplate.gov/health-professionals). The simple, but effective USDA dietary tips include the following points:

• Enjoy your food, but eat less
• Avoid oversized portions
• Eat more vegetables, fruits, whole grains
• Make half your plate fruits and vegetables
• Switch to fat-free or low-fat (1%) milk
• Have whole grains make up half of your grain intake
• Cut back on foods high in solid fats, added sugars, and salt
• Compare sodium in foods
• Drink water instead of sugary drinks

Many diabetes educators are familiar with the plate, however, the question that begs to be answered is - does the USDA’s plate apply to people with diabetes. A recent article published by the Joslin Diabetes Center reported:

> “Many of us in the diabetes community are already familiar with the plate. Although, the USDA’s plate is set up a bit differently, it can also work well for people with diabetes; its recommendations are sensible and achievable. Is it too high in carbohydrates? Perhaps, but not outrageously so for a 2,000 calorie diet approximately 56% comes from carbohydrate sources including vegetables. This is a bit higher than we usually recommend, but that is what glucose monitoring is for. Checking your blood glucose two hours after eating will let you know if you need to adjust your portions a bit.”

While patients with diabetes can clearly benefit from the common sense approach to the USDA guidelines, the American Diabetes Association recently published a more specific version that can be applied toward people with diabetes. The following chart is a useful tool published by the American Diabetes Association to assist people in establishing a diabetic meal plan. This tool called “Create Your Plate” asks users to draw an imaginary line down the middle of the plate and then simply select healthy foods of the proper portion sizes (Table 3). Creating your plate lets diabetics choose adequate portion sizes so they are consuming larger portions of non-starchy vegetables and smaller portions of starchy foods.

### Carbohydrates

Carbohydrates are important sources of energy for the body; however, they also contribute toward an increase in blood glucose levels. Patients can keep track of the amount of carbohydrates they consume by paying attention to the serving labels on most foods. The typical goal of a person with diabetes is to consume less than 130 grams of carbohydrates daily. The three main types of carbohydrates are from starches, sugars and fats. On the serving labels, the term “total carbohydrates” refers to all three types of carbohydrates. Foods high in starch include:

<table>
<thead>
<tr>
<th>potatoes</th>
<th>peas</th>
<th>corn</th>
</tr>
</thead>
<tbody>
<tr>
<td>beans</td>
<td>oats</td>
<td>lentils</td>
</tr>
<tr>
<td>barley</td>
<td>rice</td>
<td>bread</td>
</tr>
<tr>
<td>crackers</td>
<td>pasta</td>
<td>lentils</td>
</tr>
</tbody>
</table>

### Protein

Proteins are composed of amino acids and are used to repair the body and create new tissue formation. According to the American Diabetes Association, proteins should contribute approximately 15% to 20% of daily calories. Research has proven that protein consumption does increase insulin secretion, which may help a person with diabetes reduce post meal blood sugar spikes. Examples of foods that contain protein include:

<table>
<thead>
<tr>
<th>fish</th>
<th>meat</th>
<th>poultry</th>
</tr>
</thead>
<tbody>
<tr>
<td>beans</td>
<td>eggs</td>
<td>milk</td>
</tr>
<tr>
<td>cheese</td>
<td>legumes</td>
<td>veal</td>
</tr>
</tbody>
</table>

### Fats

Dietary fat slows the digestion of food in the body. Current dietary guidelines suggest that fats should make up about 20% to 35% of a person’s daily caloric intake. Certain polyunsaturated fats are an important part of a healthy diet. However, a high intake of saturated fat has the opposite effect. A high-fat diet leads to the formation of atherosclerotic plaque and increases unhealthy lipid levels. Research has shown that a high-fat diet increases the risk of heart disease in people with T2DM and also causes unfavorable changes in HDL cholesterol levels and triglycerides.
### Six Easy Steps to “Create Your Plate”

1. Using your dinner plate, put a line down the middle of the plate. Then on one side, cut it again so you will have 3 sections on your plate.

2. Fill the largest section with **non-starchy vegetables** such as: spinach, carrots, lettuce, greens, cabbage, bok choy, green beans, broccoli, cauliflower, tomatoes, vegetable juice, salsa, onion, cucumber, beets, okra, mushrooms, peppers, turnips.

3. Now in one of the small sections, put **starchy foods** such as: whole grain breads, such as whole wheat or rye whole grain, high-fiber cereal cooked cereal such as oatmeal, grits, hominy or cream of wheat rice, pasta, tortillas cooked beans and peas, such as pinto beans or black-eyed peas, potatoes, green peas, corn, lima beans, sweet potatoes, winter squash low-fat crackers and snack chips, pretzels and fat-free popcorn.

4. And then on the other small section, put your **protein** such as: chicken or turkey without the skin, fish such as tuna, salmon, cod or catfish, other seafood such as shrimp, clams, oysters, crab or mussels, lean cuts of beef and pork such as sirloin or pork loin tofu, eggs, low-fat cheese.

5. Add an 8 oz glass of **non-fat or low-fat milk**. If you don't drink milk, you can add another small serving of a carb such as a 6 oz. container of light yogurt or a small roll.

6. Add a piece of fruit or a 1/2 cup fruit salad and you have your meal planned. Examples are fresh, frozen, or canned in juice or frozen in light syrup.

Source: American Diabetes Association
Key Fat Recommendations

- Consume less than 10% of calories from saturated fatty acids and less than 300 mg/day of cholesterol, and keep trans fatty acid consumption as low as possible.
- Keep total fat intake between 20 to 35 percent of calories, with most fats coming from sources of polyunsaturated and monounsaturated fatty acids, such as fish, nuts, and vegetable oils.
- When selecting and preparing meat, poultry, dry beans, and milk or milk products, make choices that are lean, low-fat, or fat-free.
- Limit intake of fats and oils high in saturated and/or trans fatty acids, and choose products low in such fats and oils.

Fiber

Fiber plays an important role in a healthy diet. Fibers are divided into soluble (gel forming) and insoluble (structural). Soluble fiber slows the absorption of food. Insoluble fiber speeds the movement of food through the digestive tract.

The current dietary recommendations are 14 grams of fiber per 1000 calories. Patients with diabetes should consume up to 50 grams of fiber daily regardless of the caloric intake. Research has shown that large amounts of fiber can lower concentrations of plasma glucose and reduce insulin resistance as well as hyperglycemia. Clinical trials have also shown that continual consumption of fiber can also reduce LDL blood cholesterol levels. Examples of food containing fiber include:

<table>
<thead>
<tr>
<th>Oil</th>
<th>Butter</th>
<th>Salad dressing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Olives</td>
<td>Bacon</td>
<td>Avocado</td>
</tr>
<tr>
<td>Cream cheese</td>
<td>Margarine</td>
<td>Mayonnaise</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Fruits</th>
<th>Vegetables</th>
<th>Cereal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Whole-grain breads</td>
<td>Bran</td>
<td>Oatmeal</td>
</tr>
<tr>
<td>Bulgar</td>
<td>Seeds</td>
<td>Nuts</td>
</tr>
</tbody>
</table>

Read the Labels

In the world of marketing, advertisement companies are constantly trying to make boastful claims of healthy products. Have you ever picked up a box of sugary children’s cereal and compared the healthy claims on the front to the fine print on the nutritional label? A well-informed consumer should know the difference between the claims of - “low in sugar” compared to what really constitutes low sugar in a certain product. The U.S. government has defined certain claims that are allowed on food packaging. The following chart, published by the American Diabetes Association, helps to shine some light on these popular claims.

Claims for Calories

- Calories free: This means there are less than 5 calories per serving
- Low calorie: This translates to 40 calories or less per serving

Claims for Total, Saturated and Trans Fat

- Fat free: Means that there is less than 0.5 g of fat
- Saturated fat free: Equates to less than 0.5 g of saturated fat
- Trans fat free: Translates to less than 0.5 grams trans fat
- Low fat: Means that there is 3 g or less of total fat
- Low saturated fat: 1 g or less of saturated fat present
- Reduced fat or less fat: Means that there is at least 25% less fat than the regular version

Claims for Sodium

- Sodium free or salt free: Means there is less than 5 mg of sodium per serving
- Very low sodium: Translates to 35 mg of sodium or less
- Low sodium: Means there is 140 mg of sodium or less
- Reduced sodium or less sodium: Equates to at least 25% less sodium than the regular version

Claims for Cholesterol

- Cholesterol free: Means less than 2 mg per serving
- Low cholesterol: 20 mg or less present
- Reduced cholesterol or less cholesterol: This means that there is at least 25% less cholesterol than the regular version

Claims for Sugar

- Sugar free: This means less than 0.5 grams (g) of sugar per serving
- Reduced sugar: This lets you know that there is at least 25% less sugar per serving than the regular version
- No sugar added or without added sugars: This informs the consumer that no sugar or sugar-containing ingredients were added during the processing

Claims for Fiber

- High fiber: Means 5g or more of fiber per serving
- Good source of fiber: Equates to 2.5 g to 4.9 g of fiber per serving

Source Used: American Diabetes Association (2017), Last Reviewed: August 1, 2013, Last Edited: August 8, 2014

Physical Activity for Diabetic Patients

Since ancient times, physical activity has been prescribed as a central component in the treatment of diabetes. It is still recognized today as a principal modality in diabetic treatment programs for patients with all types of diabetes. Unfortunately, those with T2DM, typically have a lower level of fitness than non-diabetics. Fitness levels are generally classified in VO2max measurements. Any physical activity that utilizes oxygen, which is delivered to muscles in the body, VO2max is the measurement that calculates the volume of oxygen used by the body per kilogram of body weight in a minute. This supply of oxygen also depends on how often the heart beats. In simple terms, VO2max is a widely-accepted measurement of fitness levels. Research has revealed that increased fitness levels in diabetics provide favorable changes in blood glucose levels and improved insulin sensitivity.

The United States Department of Health and Human Services currently recommends at least 150 minutes of weekly exercise distributed over 3 to 4 days. If physically possible, an exercise routine should consist of a combination of aerobic activity (Figure 14), resistance training and stretching. The American Diabetes Association has also stated that people with diabetes should also follow this advice. While evidence has supported the role of physical activity in diabetic patients, the challenge is in the public health approach directed toward these patients. Nursing staff should stay well informed regarding what type of exercise approach can be incorporated into daily lifestyle changes, as well as the associated health benefits for patients with both T1DM and T2DM. Finally, it is also important to understand the possible complications that exercise may potentially aggravate in the diabetic patient.

Benefits of Exercise for T1DM

While much of the published literature has revealed the obvious benefits of exercise regarding T2DM, patients with T1DM can also benefit from lifestyle changes, which include a disciplined exercise program. When addressing the potential benefits of physical activity for patients with diabetes, recent high quality studies have established positive results for blood pressure, lipid levels, cardiovascular events, improvement in insulin sensitivity, changes in blood glucose levels, mortality rates and quality of life (Page 23)
Benefits of Exercise for T2DM

The potential benefits of exercise for patients with T2DM are substantial. Not only does it play a pivotal role in therapeutic options to treat T2DM complications, current evidence suggests that a routine exercise program may protect against the development of T2DM. The preventative role is especially true in susceptible populations, such as those with a family history and people who have had gestational diabetes or are glucose intolerant.

One recent literature review published in the Journal of Human Nutrition and Dietetics examined the potential effect of diet and exercise has on the prevention of T2DM. During this literature search, four Cohort studies were identified. These studies included 4,864 high-risk subjects that were monitored for a period of up to six years after a prescribed regime of routine exercise and healthy eating habits. The results of these four studies showed that lifestyle changes, such as diet and exercise, may reduce the incidence of T2DM by 28% to 59%. Moreover, lifestyle intervention had a lasting effect on these subjects. Follow-up investigations suggested that the onset of diabetes continued to be low even many years after the discontinuation of the exercise and diet program. The researchers in the review reported, “After 20 years there was a 43% lower incidence of type 2 diabetes among those who participated in the diet plus exercise intervention than the control group.” The evidence clearly suggests that exercise has a marked benefit on the incidence of diabetes. Researchers have also conducted investigations concerning the specific metabolic effects exercise has on patients with T2DM (Figure 16).

As discussed in the previous section on the benefits of exercise for carbohydrate metabolism and improved insulin sensitivity in T1DM patients, the same principal holds true for patients with T2DM. Several long-term studies have shown that routine exercise helps to regulate blood glucose levels, while simultaneously improving insulin sensitivity. During these studies, subjects exercised at 50% to 80% of their VO2max three to four times a week for 30 to 60 minutes per session. The results revealed that these subjects showed a 10% to 20% improvement in their HbA1c levels after exercise. Some studies also showed that exercise increases both splanchnic and peripheral insulin sensitivity for as long as 24
hours after exercise in T2DM patients.

The reduction in blood glucose due to exercise is due to the reduction of hepatic glucose production and increase in muscle glucose. It should be noted that evidence suggests mild-to-moderate intensity exercise lowers blood glucose during exercise and has a sustained effect after exercise. Conversely, short-term, high-intensity exercise has been shown to increase blood glucose levels in people who are obese or have T2DM with hyperinsulinemia. This increase in blood glucose typically lasts for about one hour after rigorous physical activity. This type of exercise is referred to as anaerobic, which requires quick bursts of oxygen. This results in a significant increase in blood lactate levels, which prevents the muscles from using the complete supply of available carbohydrates. During this process, higher amounts of glucose are required to perform the exercise. Once the activity ceases, insulin levels continue to rise resulting in hyperglycemic conditions.

Another benefit of exercise for patients with T2DM is the reduction of complications, such as cardiovascular risk, associated with the disease. Routine physical activity decreases triglycerides, LDL cholesterol, hypertension and increases HDL cholesterol. A recent meta-analysis of 54 randomized controlled trials revealed that aerobic exercise reduced blood pressure in people with hypertension and those who had normal blood pressure. Other recent random controlled trials published in the Journal of Diabetes Care indicated that exercise showed desirable changes in triglycerides, and cholesterol levels in patients with T2DM.

It is well established that obesity and T2DM go hand-in-hand. Exercise has also been clearly linked to weight reduction. However, what is more specifically relevant to the T2DM patient is the reduction of intra-abdominal fat. This is the fat that has been most closely associated with some of the known complications linked to T2DM. Studies have also shown that weight loss leads to decreased insulin resistance, which may be helpful in the early onset of T2DM when insulin secretion is still sufficient.

**Risks of Exercise for Diabetes**

Before counseling patients regarding the benefits of exercise, the healthcare team should screen patients for potential health problems that could be aggravated by physical activity. The medical examination should include diagnostic studies to investigate for the presence of macrovascular and microvascular complications. The exam should focus on symptoms of disease related to the heart, blood vessels, eyes and nervous system.

Prior to the start of an exercise program, a graded stress test can investigate for the presence of cardiovascular disease. A test that indicates cardiovascular problems does not necessarily preclude the patient from physical activity. Supervised low-intensity programs, such as walking, may be a safe substitute in certain occasions. High blood pressure should also be treated prior to initiating an exercise program.

If a patient is taking insulin, the lowered level of plasma glucose caused by exercise may have a dramatic effect. This could result in a hypoglycemic state. The typical solution is for the patient to ingest carbohydrates prior to activity and incorporate disciplined self-monitoring techniques.

Research has shown that strenuous activity may cause vitreous hemorrhage or retinal detachment in diabetic patients with proliferative retinopathy. Patients with this condition should consult an ophthalmologist prior to starting an exercise program.

Peripheral neuropathy is the inability to sense injury to the legs, ankles and feet and autonomic neuropathy is damage to the autonomic nervous system. Diabetic patients with peripheral neuropathy should limit weight-bearing exercise to minimize potential damage to the joints. Patients with autonomic neuropathy are at a greater risk of an adverse cardiac event during exercise. These patients should undergo a complete cardiac exam prior to exercise.

**Types of Physical Activity**

There are a wide variety of exercises available for people with diabetes. The main classification of physical activity applicable to diabetic patients falls into either aerobic activity or strength training. Aerobic activity is typically categorized into low, medium or high intensity. During low or medium intensity exercise, a person should still be able to carry on a conversation. At this intensity, the heart rate is between one half and three quarters of the maximum rate. Examples of aerobic activities include:

- Walking
- Jogging
- Bicycling
- Dancing
- Cross-country skiing
- Rowing
- Swimming
- Stair climbing

The American Diabetes Association recommends at least 30 minutes of moderate to high intensity aerobic activity at least 5 days a week. The exercise should be spread out evenly throughout the week for a total of 2.5 hours. A warm-up of at least 10 minutes and a stretching routine is also recommended to complement aerobic training.

Strength training, also known as resistance training should also be incorporated into the weekly exercise schedule. Resistance training uses muscles to move weights. Benefits of strength training include stronger muscles and bones. Strength training can also lower glucose levels and make the body more sensitive to insulin. Examples of resistance training include:

- Weights
- Weight machines
- Body weight (push-ups, pull-ups, lunges)
- Resistance bands

The American Diabetes Association recommends resistance training at least two to three days a week on non-consecutive days. As in the aerobic program, a proper warm-up and stretching routine is recommended.
Diabetes Complications & Nursing Interventions

Over an extended time period, symptoms of diabetes, such as chronic elevated blood glucose, can cause several complications. The deleterious effects of diabetes are classified into acute or chronic. Acute complications include diabetic ketoacidosis, hyperglycemia, hyperglycemic, lactic acidosis and infections. Examples of chronic complications include cardiovascular disease, hypertension, dyslipidemia, diabetic nephropathy, diabetic retinopathy, diabetic neuropathy and foot problems.

Despite the large amount of potential complications associated with diabetes, the good news is that because of active diabetes educational programs, the rate of serious complications has decreased over the last couple of decades. As a healthcare professional, a thorough understanding of the following diabetic complications can help you recognize and treat the early warning signs.

Hyperglycemia

Hyperglycemia Hyperosmolar State (HHS) is a serious metabolic condition that most commonly occurs in patients with T2DM, but can also occur in T1DM. HHS was formerly called hyperosmolar hyperglycemic nonketotic (HHNK) coma, however, experts changed the term because coma only occurred in less than 20% of the patients. HHS is caused when the body has enough insulin to utilize glucose as fuel, but there is not enough insulin to keep blood glucose levels within a safe range.

HHS is often triggered by illness, infection and by medications that lower glucose tolerance. It can also be triggered by lack of fluids. High blood glucose levels over 600 mg/dL are usually seen in patients with HHS. In some instances, glucose levels over 1000 mg/dL are present. The American Diabetes Association also reports the following diagnostic features associated with HHS:

- Bicarbonate concentration higher than 15 mEq/L
- Serum pH greater than 7.30
- Excessive dehydration
- Plasma glucose levels greater than 600 mg/dL
- Slight alteration in consciousness
- Slight ketonuria
- Minimal or no ketonemia present

Emergency treatment for HHS is often necessary and consists of insulin supplementation to reduce hyperglycemia and fluids to combat dehydration. Assessment of vital signs is also important. Monitor the patient for tachycardia, which is an early indication of dehydration or hypotension, which suggests excessive dehydration. Perform a detailed skin examination to investigate for skin turgor. Sunken eyes or dry mouth may also be a clue indicating dehydration. Changes in muscle tone and eye deviation may also be present due to neurological issues associated with HHS.

The ultimate goal in treatment of HHS patients is to rehydrate the patient, correct hyperglycemia, treat underlying diseases and monitor pulmonary, cardiovascular, CNS and renal function. Dehydration remains the leading cause of mortality in HHS patients; therefore adequate hydration remains a key element in the treatment process.

Diabetic Ketoacidosis

Diabetic Ketoacidosis (DKA) is another potentially life-threatening acute complication of diabetes. It occurs most commonly in patients with T1DM, however, it can also occur in patients with T2DM. DKA is a complex state that occurs due to a lack of insulin. Insufficient insulin levels cause the body to break down fats rather than glucose for energy. Acidic ketones are present as a result of fat metabolism. This acidosis is characterized by a decrease in blood pH below 7.3. This condition ultimately results in a deep, sighing pattern of respiration, known as Kussmaul breathing.

DKA can be the result of the patient not taking prescribed insulin, an unsuitable insulin schedule, a sickness that alters the balance of glucose and insulin, stress, infection, trauma and alcohol abuse. The onset typically occurs within 24 hours and the following typical symptoms may be present:

- Nausea and vomiting
- Deceased appetite
- Abdominal pain
- Weakness
- Altered consciousness
- Rapid weight loss
- Kussmaul breathing
- Thirst and dry mouth
- Fruity-scented breath

The primary evidence regarding the diagnosis of DKA is the presence of ketones in the urine. Another characteristic of DKA is hyperglycemia over 300 mg/dL and bicarbonate levels less than 15 mEq/L along with the aforementioned pH level less than 7.3. Ketonemia and ketonuria are also present in patients with DKA. Close attention to laboratory data assists with some of the underlying conditions in this condition.

When treated properly, DKA produces a low (2%) mortality rate. Effective approaches to treatment include admission to an ICU during the first 24 hours. During this time, the goals are to correct fluid loss and electrolyte imbalance through IV therapy, reverse hyperglycemia through insulin supplementation, correction of acid-base balance and treatment of any potential infection. Current guidelines regarding the fluids recommend the administration of isotonic sodium chloride solution or lactated Ringer solution. When blood glucose reaches levels under 180 mg/dL, dextrose should be administered in place of isotonic sodium chloride. Approximately one hour after IV fluid replacement, insulin should be administered. During treatment, also watch out for other potential concomitant conditions, such as deep venous thrombosis, myocardial infarction, sepsis and cerebrovascular issues. Patients are typically discharged after pH returns to normal (7.35 - 7.45), bicarbonate has increased above 18 mEq/L and they are able to resume their daily insulin routine.

Cardiovascular Complications

People with diabetes are at an increased risk for cardiovascular complications including stroke, heart disease and hypertension. Approximately 80% of people with T2DM eventually die from some form of cardiovascular disease and patients with T1DM are ten times more likely to develop heart disease than their healthy counterparts. Atherosclerosis is the mitigating factor, which links cardiovascular complications to diabetes. Patients with diabetes are susceptible to the formation of atherosclerotic plaques because of increased oxidative stress and glycosylation of proteins. Another factor that contributes toward thickening of artery walls is the activation of the enzyme protein kinase C. These fatty deposits that accumulate in the arteries eventually impair the blood flow in the arteries causing cardiac stress. Atherosclerosis may be asymptomatic until a cardiac event occurs. This “silent killer” is responsible for strokes, congestive heart failure and heart attacks. Diabetic patients that exhibit warning signs of cardiac complications should be administered stress tests.

Coronary artery disease (CAD) and stroke are the two primary types of cardiovascular disease. Approximately two-thirds of the people with diabetes have CAD. CAD is also known as ischemic heart disease and is caused by the thickening of the artery walls. A stroke results when the brain’s blood supply is suddenly blocked. This occurs when a blood vessel in the neck or brain bursts or is obstructed. Diabetic patients are susceptible to strokes caused by blood clots, which are a common
cause of many strokes.

Strokes require immediate treatment, which usually requires medication that dissolves clots that block the flow of blood from the heart to the brain. These drugs need to be administered within three hours to be effective. Due to this fleeting time window, it is important for patients to recognize early warning signs of a stroke, such as:

- Sudden confusion
- Difficulty speaking
- Difficulty walking
- Headache
- Vision problems

One of the symptoms of heart disease is angina, which occurs after narrowing of the blood vessel to the heart. This narrowing causes a reduction in the heart’s blood supply. Angina does not cause permanent damage, however, it does increase the risk for heart attack. A heart attack occurs when the blood vessel to the heart is blocked, which results in permanent damage. Symptoms of heart attack include:

- Chest pain or discomfort
- Shortness of breath
- Pain in arms, back, jaw, stomach or neck
- Sweating
- Nausea

If patients with diabetes have control of their glucose levels, they can greatly reduce the incidence of developing cardiovascular complications associated with diabetes. The risk of adverse cardiac events can also be minimized further by controlling hypertension, which is often common in patients with T2DM. Experts recommend that diabetics maintain a blood pressure reading of 130/80 mmHg, which is just below normal levels of 140/90 recommended for the general population. Other common sense lifestyle modifications, such as eating healthy and exercise also pay dividends when it comes to preventing the onset of cardiovascular disease.

Diabetic Neuropathy

Diabetic neuropathy is a common condition for people with diabetes. It occurs in approximately 50% of diabetic patients. During diabetic neuropathy, nerves are damaged by the presence of excessive amounts of glucose in the body. This often results in a lack of sensation. One of the early signs, known as peripheral neuropathy or distal symmetrical polyneuropathy, is characterized by a tingling sensation or numbness in the toes. As neuropathy progresses, the tingling sensation travels to the foot, ankles and lower legs. The inability to feel hot or cold also becomes an issue as well as a loss of balance and coordination. As the condition progresses, the hands and lower arms are also affected by numbness and tingling.

Diabetes can also damage the autonomic nerves. This condition is called autonomic neuropathy and affects the internal organs of the body. Symptoms include:

- Decrease in systolic blood pressure of at least 20 mmHg upon standing
- Resting tachycardia less than 100 bpm
- Recurrent UTI due to inability to empty the bladder
- Difficulty swallowing
- Reduced sweating of extremities
- Diarrhea

Treatment of patients with neuropathy consists of addressing the neurological symptoms, progression of the disease and pain-relieving measures. Educate the patient on proper glycemic regulation, which may reduce additional nerve damage. Because the nerves that have already been damaged cannot be fixed, a major goal in treating patients with neuropathy is pain relief. Pain specialists often prescribe a regime of medications to help relieve nerve pain.

Diabetic Neuropathy Pain Relieving Agents

**Anticonvulsant Agents**

- pregabalin (Lyrica)
- gabapentin (Neurontin)
- gabapentin (extended-release) (Gralise)
- carbamazepine (Tegretol, Carbatriol, Epitol)
- phenytoin (Dilantin)

**Tricyclic Antidepressants**

- desipramine (Pertofrane, Norpramin)
- nortriptyline (Pamelon, Sensoral, Norpress, Aventyl HCL, Allegron)
- amitriptyline (Elavil, Tryptomer)
- imipramine (Tofranil)

**Selective Serotonin/Norepinephrine Reuptake Inhibitor (SSNRI)**

- Duloxetine (Cymbalta)
- Venlafaxine (Effexor)
- Paroxetine (Paxil)
- Citalopram (Celexa)

Several recent studies have suggested that lidocaine patches and gel have helped relieve nerve pain associated with diabetic neuropathy. One recent placebo-controlled study showed dramatic pain relief in patients that used 5% lidocaine gel compared to the placebo group.

**Foot Complications**

Diabetic foot ulcers are responsible for more hospitalizations than any other complication of diabetes. Diabetes-related amputations due to foot complications are the leading cause of non-traumatic amputations in the United States. According to the National Institute of Diabetes and Digestive and Kidney Diseases, 15% of diabetics develop foot ulcers, and approximately 12% to 24% of those with foot ulcers will eventually require amputation. Foot complications in patients with diabetes are a result of issues, such as loss of protective sensation and coordination associated with peripheral neuropathy. Other known causes of foot problems related to diabetes are atherosclerotic peripheral arterial disease and mechanical changes of the bony structure of the foot.

Damage to the nerves of the foot can also cause it to become dry, which may result in cracking. When patients are unaware of the injury due to lack of sensation, infection can occur. Other contributing factors include poor blood circulation in the feet caused by chronic damage to capillaries and small blood vessels. The accumulation of pressure due to poor circulation can also cause calluses in the feet. Eventually, these calluses rupture and open ulcers become susceptible to infection. For more information order course #720 Wound Care.

The treatment of diabetic foot ulcers requires a thorough examination of the patient’s feet, skin, joints and toenails. Test ankle and foot pulses for sensory ability. Special care should also be emphasized regarding the patient’s footwear. Poor fitting shoes and motor neuropathy can cause muscle weakness and instability in the foot, which result in chronic unnoticed fractures. These fractures may result in a convex-shaped foot, known as Charcot foot. Educate the patient regarding foot ulcer preventative measures including:

- Daily foot inspection
- Adequate foot hygiene
- Application of skin moisturizer
- Foot wear inspection
- Protection of feet from hot and cold
- Exercise to improve circulation
- Glycemic control

If foot ulcers are present, the patient should be assessed by a podiatric or vascular surgeon. Surgical management may be necessary in cases of chronic infection. Surgeons typically use available surgical methods, such as vascular reconstruction, debridement, bony architecture revisional surgery and soft tissue coverage using skin grafts.

Some specialists have increasingly prescribed hyperbaric oxygen therapy for chronic wounds. During this therapy, patients are placed into an air-tight hyperbaric chamber and infused oxygen for a predetermined amount of time. The theory is that the direct infusion of oxygen helps to hasten the healing process. One recent study assessed patients who were treated for 85 minutes, five days a week for 8 weeks with hyperbaric oxygen. The results of the study indicated that 52% of these patients...
benefited from a complete healing of chronic foot ulcers.

**Diabetic Nephropathy**

According to the American Diabetes Association, approximately 20% to 40% of people with diabetes develop kidney damage, known as diabetic nephropathy. This common complication arises from progressive loss of kidney function, which in many cases can advance to end-stage renal disease (ESRD). In fact, diabetic nephropathy is the leading cause of ESRD in the United States.

Kidney disease occurs when chronic elevated glucose levels cause organs to filter high volumes of blood. This event overworks the kidneys, which results in protein excretion from the blood through capillaries. The protein is eventually filtered out of the body through the urine. This presence of protein in the urine is known as microalbuminuria. When the levels of protein increase toward the advanced stage of kidney disease, the condition is called macroalbuminuria.

Early intervention is essential in treating kidney disease. According to the American Diabetes Association, once symptoms of microalbuminuria are present, strict regulation of blood glucose levels may potentially reduce the progression of macroalbuminuria by almost 50%. In addition to the administration of a disciplined glucose management program, patient’s blood should be tested for serum creatinine levels to assess the glomerular filtration rate, and urine should be tested for protein excretion. Once kidney disease progresses to ESRD, dialysis treatment is often necessary to remove the surplus of toxins from the blood.

**Diabetic Retinopathy**

The National Institutes of Health - National Eye Institute reported that diabetic retinopathy is the leading cause of blindness in America. Diabetic retinopathy is a term used to describe health complications of the retina caused by diabetes. Diabetic retinopathy starts with small aneurysms and swelling of the retinal tissue. Along with several of the aforementioned complications, high glucose levels are also responsible for damage to the retina in patients with diabetes. Diabetic retinopathy has four stages:

1. **Mild non-proliferative retinopathy** - Small balloon-like pouches form in the retina’s blood vessels.
2. **Moderate non-proliferative retinopathy** - Retina blood vessels are starting to become blocked by the tiny balloon-like bulges.
3. **Severe non-proliferative retinopathy** - Additional blockage of capillaries behind the eye occur depriving the retina of much-needed blood supply.
4. **Proliferative retinopathy** - During this stage, capillaries are so blocked that the retinal vessel closes, causing growth of abnormal blood vessels over the retina. They also develop along the surface of the clear, vitreous gel that fills the inside of the eye. By themselves, these blood vessels do not cause symptoms or vision loss. However, they have thin, fragile walls. If they leak blood, severe vision loss and even blindness can result.

Symptoms of retinopathy include:
- blurry or double vision
- difficulty reading
- pain in the eyes
- red eyes
- seeing spots
- the feeling of pressure in the eyes.

Cataracts and glaucoma can also develop along with retinopathy in diabetic patients. During routine assessments of patients with diabetes, full eye examinations are necessary to investigate for the presence of retinopathy.

**Coping Strategies for Patients with Diabetes**

Living with diabetes is a major lifestyle adjustment for most patients. Diabetes, in particular, involves adherence of strict medical regimes to control the disease. The reality of the disease can take an emotional and psychological toll on many patients. Each individual patient presents a variety of social, environmental and even cultural influences that influence how they cope with diabetes. According to the American Association of Diabetes Educators, the level of a patient’s psychological distress can have a direct influence on their motivation to keep their diabetes under control. Many patients have different emotional responses throughout the evolution of the disease. After the initial diagnosis, some patients may have difficulty accepting that they have diabetes despite the evidence, and some may be left feeling overwhelmed and confused. Others may feel downright angry and can be indigent toward treatment. Depression is another common psychological symptom that needs to be considered in patients diagnosed with diabetes.

To help combat the variety of emotional and psychological problems that can accompany diabetes, health professionals need to become educated on successful coping strategies that they can convey to their patients. Successful coping strategies are essential in every field of nursing, but particularly when patients are suffering from complications associated with a chronic illness like diabetes. A recent literature review revealed over 200 current articles on the topic of coping with diabetes. Several of these studies concluded that patients can benefit significantly by learning successful coping methods. Key skills for healthy coping generally include social problem solving, cognitive behavior modifications, communication skills training and conflict resolution. Coping skills training should focus on each one of the aforementioned skills.

- **Social problem solving** - This skill is designed to assist patients during the decision making process. This skill helps patients identify a problem, determine goals, generate solutions, assess consequences, pick a solution and evaluate the outcome. For example, when a diabetic patient feels the urge to overeat, this technique can be used to make an informed choice to use moderation instead of indulgence.

- **Cognitive behavior modification** - This type of therapy involves the recognition of feelings, problem solving methods and self-dialogue. Reflection plays a huge role in this skill. This coping mechanism can help patients mentally talk their way through certain inherent fears associated with the disease. Cognitive behavior modification can also help the patient to contemplate if their perception of certain disease complications, such as a catastrophic hyperglycemic event, is based on fear or reality. The goal of this skill is to motivate the patient to educate themselves about potential symptoms, complications and outcomes of the disease.

- **Communication skills training** - This skill assists patients to express themselves to other people regarding the necessary lifestyle changes that accompany their disease. For example, some patients may find it difficult to perform blood glucose testing in front of their colleagues at work. During communication skills training, instruction regarding assertiveness will be utilized in role-playing episodes, which can be applied to a variety of diabetes-specific scenarios.

- **Conflict resolution** - This skill allows the patient to focus on positive, rather than negative outcomes. During this skill, patients identify a problem and then focus on a resolution. For example, a patient may be diagnosed with kidney failure at some point during the disease process. The conflict may present itself in choosing the medical options of dialysis or transplantation. With the assistance of the clinician, the patient will need to choose the correct resolution that best suits their problem.

The above skills go a long way in helping
the diabetic patient endure daily management of diabetes. However, they also need a nurse who is empathetic, caring and patient. By developing your own coping skills, you will be able to effectively apply this knowledge toward your patient care program. When you recognize how you cope with certain stressful situations, this understanding can be directly applied to your patients through an insightful coping skills training program.

Resources

American Diabetes Association
1701 North Beauregard Street
Alexandria, VA 22311
Phone: 1-800-DIABETES (342-2383)
Email: AskADA@diabetes.org
Internet: www.diabetes.org

American Urological Association Foundation
1000 Corporate Boulevard
Linthicum, MD 21090
Phone: 1-800-828-7866 or 410-689-3700
Fax: 410-689-3998
Email: auafoundation@auafoundation.org
Internet: www.UrologyHealth.org

American Podiatric Medical Association
9312 Old Georgetown Road
Bethesda, MD 20814-1621
Phone: 1-800-FOOTCARE (366-8227) or 301-581-9200
Fax: 301-530-2752
Email: askapma@apma.org
Internet: www.apma.org

Centers for Disease Control and Prevention
National Center for Chronic Disease Prevention and Health Promotion
Division of Diabetes Translation
4770 Buford Highway NE, Mail Stop K-10
Atlanta, GA 30341-3717
Phone: 1-800-CDC-INFO (236-4636) or 770-488-5000
Email: cdcinfo@cdc.gov
Internet: www.cdc.gov/diabetes

Juvenile Diabetes Research Foundation
International
26 Broadway, 14th Floor
New York, NY 10004
Phone: 1-800-533-CURE (2873)
Fax: 212-785-9595
Email: info@jdrf.org
Internet: www.jdrf.org

Lower Extremity Amputation Prevention Program
Health Resources and Services Administration
5600 Fishers Lane
Rockville, MD 20857
Phone: 1-888-ASK-HRSA (275-4772)
Internet: www.hrsa.gov/leap

National Diabetes Education Program
1 Diabetes Way
Bethesda, MD 20892-3560
Phone: 1-800-438-5383
Fax: 703-738-4929
Email: ndep@mail.nih.gov
Internet: www.ndep.nih.gov

National Digestive Diseases Information Clearinghouse
2 Information Way
Bethesda, MD 20892-3570
Phone: 1-800-891-5389
Fax: 703-738-4929
Email: nddic@info.niddk.nih.gov
Internet: www.digestive.niddk.nih.gov

National Heart, Lung, and Blood Institute Information Center
P.O. Box 30105
Bethesda, MD 20824-0105
Phone: 301-592-8573
Fax: 240-629-3246
Email: nhlbiinfo@nhlbi.nih.gov
Internet: www.nhlbi.nih.gov

National Institute of Neurological Disorders and Stroke
P.O. Box 5801
Bethesda, MD 20824
Phone: 1-800-352-9424 or 301-496-5751
Internet: www.ninds.nih.gov

National Kidney and Urologic Diseases Information Clearinghouse
3 Information Way
Bethesda, MD 20892-3580
Phone: 1-800-891-5390
Fax: 703-738-4929
Email: nkudic@info.niddk.nih.gov
Internet: www.kidney.niddk.nih.gov

Pedorthic Footwear Association
2025 M Street NW, Suite 800
Washington, DC 20036
Phone: 1-800-673-8447 or 202-367-1145
Fax: 202-367-2145
Email: info@pedorthics.org\ Internet: www.pedorthics.org

References and Suggested Readings

American Diabetes Association Website:
Site accessed May, 2017.


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