DIABETES MELLITUS IN THE ADULT

Dana Bartlett is a professional nurse and author. His clinical experience includes 16 years of ICU and ER experience and over 20 years of as a poison control center information specialist. Dana has published numerous CE and journal articles, written NCLEX material and textbook chapters, and done editing and reviewing for publishers such as Elsevier, Lippincott, and Thieme. He has written widely on the subject of toxicology and was recently named a contributing editor, toxicology section, for Critical Care Nurse journal. He is currently employed at the Connecticut Poison Control Center and is actively involved in lecturing and mentoring nurses, emergency medical residents and pharmacy students.

ABSTRACT

Type 1 and type 2 diabetes are two primary kinds of diabetes mellitus. There are significant differences between them however both are caused by defects in insulin production, and/or insulin resistance. Adults diagnosed with diabetes mellitus benefit greatly from education on the pathophysiology of the disease as well as lifestyle and medication management to improve outcomes. The worldwide incidence of type 2 diabetes has dramatically increased due to the rise in obesity. Diabetes mellitus is associated with serious co-morbid conditions such as heart disease, stroke and neuropathies. The pathogenesis, complications and treatment of type 1 and type 2 diabetes in the adult are discussed.
Continuing Nursing Education Course Director & Planners
William A. Cook, PhD, Director; Douglas Lawrence, MS, Webmaster;
Susan DePasquale, CGRN, MSN, FPMHNP-BC, Lead Nurse Planner

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Statement of Need
Nurses as members of the health team need to understand the
pathogenesis, complications and treatment of type 1 and 2 diabetes to
better identify risks early on and to support prevention and treatment.

Course Purpose
To provide nurses and health associates knowledge about the cause,
complications and treatment of type 1 and 2 diabetes in the adult.
Learning Objectives

1. Explain the role of insulin in the body.
2. Identify the two primary types of diabetes.
3. Identify risk factors for the development of diabetes.
4. Identify signs and symptoms of diabetes.
5. Identify acute complications caused by diabetes.
6. Identify chronic complications caused by diabetes.
7. Identify at least three medications used to treat diabetes.

Target Audience
Advanced Practice Registered Nurses, Registered Nurses, Licensed Practical Nurses, and Associates

Course Author & Director Disclosures
Dana Bartlett, RN, BSN, MA, MSN, William S. Cook, PhD,
Douglas Lawrence, MS, Susan DePasquale, CGRN, MSN, FPMHNP-BC
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Please take time to complete the self-assessment Knowledge Questions before reading the article. Opportunity to self-assess knowledge learned will be provided at the end of the course.
1. Type 1 diabetes is characterized by:
   a. a complete lack of endogenous insulin
   b. a chronically low blood sugar
   c. a relative lack of endogenous insulin production
   d. decreased insulin sensitivity

2. Type 2 diabetes is characterized by:
   a. decreased insulin sensitivity
   b. a relative lack of insulin production
   c. inappropriate secretion of glucagon
   d. increased efficiency of pancreatic beta cells

3. HbA1c provides a measurement of:
   a. post-prandial blood sugar
   b. fasting blood sugar
   c. the average serum glucose over several months
   d. the average serum glucose after an insulin injection

4. Type 1 diabetes is considered to be primarily caused by:
   a. an autoimmune process
   b. insulin resistance
   c. lifestyle factors
   d. chronic hyperglycemia

5. Type 2 diabetes is considered to be primarily caused by:
   a. an autoimmune process
   b. an infectious process affecting pancreatic beta cells
   c. drugs that damage the pancreatic beta cells
   d. genetic susceptibility and lifestyle factors
6. *Commonly reported signs and symptoms of diabetes include:*
   a. anorexia, weight loss, and diarrhea
   b. excessive thirst and hunger, excessive urination
   c. persistent headache, shortness of breath, fever
   d. abdominal pain, dysuria, and peripheral neuropathy

7. **Which of the following are **common** acute complications of diabetes?**
   a. Chest pain, pulmonary embolism
   b. Pneumonia, urinary tract infection
   c. Hypoglycemia, DKA
   d. Nephrolithiasis, pulmonary edema

8. **Which of the following are **common** chronic complications of diabetes?**
   a. Nephropathy, neuropathy, and retinopathy
   b. Coagulopathies, DVT, and nephrolithiasis
   c. Hepatitis, congestive heart failure, and Parkinsonism
   d. Dementia, gastric reflux, and osteoporosis

9. The first choice drug for treating patients who have type 2 diabetes is:
   a. insulin
   b. glyburide
   c. metformin
   d. exenatide
10. **Patients who have diabetes should be very closely monitored for:**
   
a. urinary tract infections  
b. infected foot ulcers  
c. upper respiratory tract infections  
d. gastroenteritis
INTRODUCTION

Diabetes mellitus is a metabolic disorder that is characterized by hyperglycemia. There are two primary types of diabetes mellitus, type 1 and type 2. There are significant differences between type 1 and type 2 diabetes mellitus, but both are caused by defects in insulin production, and/or insulin resistance.

Diabetes can also be caused by pregnancy (gestational diabetes), exposure to chemicals or drugs, endocrinopathies (e.g., Cushing’s syndrome, hyperthyroidism, pheochromocytoma), and large number of genetic disorders and insulin resistance syndromes. These types of diabetes are typically called secondary diabetes. These forms of the disease are relatively uncommon when compared to diabetes mellitus and they will not be discussed in this module.

Diabetes is one of the most common chronic diseases in the United States and worldwide. The Centers for Disease Control and Prevention (CDC) has estimated that there are 29.1 million people in the United States that have diabetes.\(^1\) Diabetes is the seventh leading cause of death, and it is also a significant contributing cause, or direct cause of amputation, blindness, cardiovascular disease, kidney disease, peripheral neuropathy, and stroke.\(^1\) The prevalence of diabetes worldwide has dramatically increased in the past two decades and because of the epidemic of obesity, and longer life spans, this increase is expected to continue.\(^2\)

Diabetes mellitus is typically called diabetes. In more formal contexts the full term is used to distinguish diabetes mellitus from diabetes insipidus, a neurological or renal disease that does not affect glucose
metabolism and homeostasis. The terms insulin-dependent, non-insulin-dependent, adult-onset, and juvenile-onset diabetes are considered obsolete.

**INSULIN, GLUCOSE, AND HYPERGLYCEMIA**

Glucose is one of the primary sources of energy that is used for metabolic processes. Glucose molecules are too large to diffuse through cell membranes, and insulin is needed so that glucose can be moved into the cells.

Insulin is a polypeptide hormone that is produced by the beta cells in the islets of Langerhans in the pancreas. The primary function of insulin is to facilitate the entry of glucose into cells where it can be used for energy or stored as glycogen. When foods are digested and glucose is produced, insulin is released and it binds to an insulin receptor on the cell membrane. This binding stimulates the activity of glucose transporters, specialized proteins that carry glucose across the cell membranes.

Normal insulin secretion and proper utilization of insulin are very important because: 1) prolonged, high levels of serum glucose are quite harmful, and; 2) glucose is a primary nutrient of the skeletal muscles, the brain, and other tissues. Parenthetically, glucose is essentially the only nutrient that can be used by the brain, but the brain does not depend on insulin for glucose utilization. Insulin has other functions in the brain and central nervous system aside from glucose utilization.
The defining characteristic of diabetes is hyperglycemia. Hyperglycemia is the primary cause of the acute and chronic complications of type 1 and type 2 diabetes and the higher the level of serum glucose and the longer hyperglycemia persists, the greater the risk of developing diabetic complications. The mechanisms by which hyperglycemia negatively affects cells and tissues are very complex and not completely understood, but the end result of chronic hyperglycemia is damage to large and small blood vessels. These micro-vascular and macro-vascular changes are widespread but they are more common in the eyes, the kidneys, and the peripheral nerves as these tissues are especially vulnerable to the deleterious effects of a persistently elevated serum glucose.

**PATHOGENESIS OF TYPE 1 DIABETES**

Type 1 diabetes accounts for approximately 10% of all cases of the disease, but the prevalence of type 1 diabetes varies considerably depending on population demographics. Most cases of type 1 diabetes are caused by an autoimmune process, which over a period of months and/or years irreversibly destroys the pancreatic beta cells. This autoimmune process occurs in genetically susceptible people, and it is thought to be triggered by an environmental or infectious stimulus. The onset of type 1 diabetes is usually in childhood or early adolescence, but onset of the disease can occur in adulthood. The patient who is developing, or has type 1 diabetes often complains of fatigue, excessive hunger and thirst, and excessive urination. However, many cases of type 1 diabetes are undiagnosed until the patients develop diabetic ketoacidosis.
The interaction between genetic pre-disposition to type 1 diabetes and the infectious and environmental stimuli is not completely understood. However, genetic susceptibility is quite pronounced. If both parents have type 1 diabetes there is a 30% chance that their children will develop the disease, and there are certain ethnic groups that are particularly susceptible to type 1 diabetes. However, given the recent increase in the prevalence of type I diabetes in young adults (an increase too dramatic to be caused by changes in the gene pool) it is also clear that infectious and environmental factors play a prominent role in the development of type 1 diabetes.

The most commonly suspected environmental triggers that may cause type 1 diabetes are viruses. The mumps virus, enteroviruses, the rubella virus, rotavirus, Epstein-Barr virus, and cytomegalovirus have been identified as possible causes of type 1 diabetes and of these, the association between infection and the development of type 1 diabetes is strongest for the childhood enteroviruses and rubella.

**PATHOGENESIS OF TYPE 2 DIABETES**

The pathogenesis of type 2 diabetes involves a complex interaction between genetic and environmental factors that causes impaired beta cell function and insulin resistance. Insulin resistance is a condition in which the cells do not respond as expected to a specific amount of insulin. The mechanisms of action of insulin resistance are both inherited and caused by environmental and lifestyle factors.

In type 2 diabetes, insulin resistance is probably the first metabolic abnormality. Insulin resistance in turn causes elevated serum glucose, and hyperglycemia causes hyper-secretion of insulin by the
pancreas. When hyperglycemia is chronic and prolonged the beta cells of the pancreas become impaired and they cease to function. The amount of insulin that can be produced and the degree of insulin resistance varies for each individual that has type 2 diabetes.

**Genetic factors**

It is clear that genetic susceptibility is important in the development of type 2 diabetes as a family history of type diabetes in a first-degree relative is considered to be a significant risk factor for developing the disease. Genetic susceptibility for type 2 diabetes is thought to contribute to the development of the disease by causing insulin resistance or by negatively affecting pancreatic islet development and/or function. It has been estimated that <10% of the risk for developing type 2 diabetes has been identified in genetic studies and the genetic susceptibility for type 2 diabetes is poorly understood.

**Environmental factors**

Obesity, sedentary life style, hypertension, dyslipidemia, age > 45, and ethnicity (African American, Hispanic) are risk factors for the development of type 2 diabetes. These risk factors frequently co-exist in people who have type 2 diabetes and because they interact and influence one another, it can be difficult to determine 1) when these individual risk factors are a cause of type 2 diabetes, 2) when they are an effect of the disease, or 3) when they are both. For example, hypertension is thought to increase insulin resistance independent of the presence of type 2 diabetes, and obesity is thought to cause insulin resistance by creating a pro-inflammatory state, disrupting the signaling mechanisms by which insulin initiates glucose transport across cell membranes, and several other pathogenic mechanisms.
SIGNS, SYMPTOMS, AND THE DIAGNOSIS OF DIABETES

Patients who have type 1 diabetes will typically present with polydipsia, polyphagia, and polyuria, and they frequently complain of blurred vision, fatigue, nausea, weight loss, and vomiting. If these signs and symptoms are not correctly attributed to type 1 diabetes or medical treatment is not sought, the patient may develop diabetic ketoacidosis and this is a common scenario. Approximately 15%-70% of patients who have type 1 diabetes have diabetic ketoacidosis at the time of diagnosis.\(^\text{12}\)

Patients who have type 2 diabetes may have the signs and symptoms that are common to patients who have type 1 diabetes, e.g., polyuria, fatigue, and blurred vision, etc. However, many patients who have type 2 diabetes are asymptomatic for many years, and the diagnosis of type 2 diabetes is often made when serum glucose is measured during a routine screening exam or when there is evidence of a complication such as diabetic neuropathy.

The diagnostic criteria for diabetes are:\(^2\)

1. Fasting plasma glucose of 126 mg/dL or higher. The fasting plasma glucose test is a measurement of plasma glucose that is performed after the patient has been fasting for at least eight hours. The test should be repeated twice to confirm the presence of diabetes. The normal fasting plasma glucose is considered to be < 100 mg dL.

2. A 2-hour plasma glucose level ≥ 200 mg/dL during an oral glucose challenge test. The patient should be fasting for eight hours prior to the test. A plasma glucose level is obtained and if
it is < 140 mg/dL, the patient is given 75 grams of an oral glucose solution. Two hours after administration of the glucose solution the plasma glucose is measured, and the result should be < 140 mg/dL. (Note: The level considered to be normal varies somewhat with age).

3. A hemoglobin A1c (HbA1c) level of > 6.5%. The HbA1c, also known as the glycosated hemoglobin level, measures glucose that is attached to hemoglobin and it provides an indication of what the average blood glucose has been for several months prior to the test.

Pre-diabetes, also known as impaired glucose tolerance is a condition in which the patient has abnormally high plasma glucose and HbA1c levels but these levels are below what are considered to be diagnostic for diabetes. Between 18%-30% of people who have pre-diabetes will develop type 2 diabetes within five years if they do not make significant changes to their health habits, life style, and diet. The diagnostic criteria for pre-diabetes are: 1) HbA1c 5-7%-6.4%; 2) fasting plasma glucose 100-125 mg/dL, and; 3) a 2-hour plasma glucose level of 140-199 mg/dL.13

**CHRONIC COMPLICATIONS OF DIABETES**

Diabetes can cause serious complications. The pathogenesis of these complications is explained in part by chronic hyperglycemia and by the co-morbidities of diabetes such as dyslipidemia, and hypertension. However, research indicates that hyperglycemia and co-morbidities are just part of the reason why people who have diabetes develop diabetic complications.
**Cardiovascular complications**

Cardiovascular disease is very common in people who have diabetes. It has been estimated that for the patient who has type 2 diabetes there is a two to four-fold increase in the occurrence of coronary artery disease and stroke and a two to eight-fold increase in the occurrence of heart failure.\(^{14}\) Cardiovascular disease is the primary cause of mortality in patients with type 2 diabetes and approximately two thirds of all patients who have type 2 diabetes will die of a stroke or heart disease.\(^{8}\)

Cardiovascular disease is a common complication of type 1 diabetes, as well. People who have type 1 diabetes have a higher risk of developing coronary artery disease, myocardial infarction, peripheral artery disease, and stroke than the non-diabetic population, and these complications occur at an earlier age for the type 1 diabetic than for the general population or for people who have type 2 diabetes.\(^{15}\)

Many patients who have type 1 or type 2 diabetes have risk factors such as cigarette smoking, dyslipidemia, and hypertension that contribute to the development of atherosclerosis and cardiovascular disease. However, diabetes itself appears to be an independent factor that increases the risk of developing atherosclerosis and cardiovascular disease. Sub-clinical inflammation, one of the primary pathophysiologic processes of atherosclerosis, is more common in diabetics and chronic hyperglycemia has been associated with the development of atherosclerosis.\(^{15}\)
**Diabetic retinopathy**

Diabetic retinopathy is the leading cause of blindness in adults 20-74 years of age.\(^4\) Diabetic retinopathy develops slowly over a period of years and within 10 years of the onset of the disease, nearly all those who have type 1 diabetes and approximately 60% of those who have type 2 diabetes will have some evidence of diabetic retinopathy.\(^16\) The mechanism, or mechanisms by which diabetes causes damage to the microvasculature of the eye are not completely understood, but there is a well established relationship between the level and duration of hyperglycemia and the development of diabetic retinopathy.\(^16\) Hypertension and dyslipidemia also increase the risk of developing diabetic retinopathy.

**Diabetic peripheral neuropathy**

Sensorimotor polyneuropathy is one of the most common complications of diabetes,\(^17,18\) occurring in 10%-54% of patients with either type 1 or type 2 diabetes.\(^17\) The symptoms of diabetic peripheral neuropathy vary from patient to patient but the following are typically reported: abnormal response to painful stimuli; burning sensations in the hands and/or feet; numbness; pain; pins and needles sensation, and; spasms. Diabetic peripheral neuropathy, as with the other chronic complications of the disease, develops over a period of years and most patients will have a sub-clinical phase before symptoms appear. In an advanced stage, difficulty walking and persistent pain greatly interfere with quality of life, and patients who have diabetic peripheral neuropathy are at risk for foot ulcers and amputation.

Diabetic peripheral neuropathy is diagnosed through completion of the patient history, a clinical exam, and quantitative neurological testing
such as electrophysiological studies. The duration of diabetes and poor glycemic control are directly related to the development of diabetic peripheral neuropathy; the longer someone has the disease and the higher the blood sugar, the greater is the risk.\textsuperscript{4}

**Diabetic nephropathy**

Diabetic nephropathy is a very serious complication of diabetes. It is the most common cause of end-stage renal disease,\textsuperscript{19} and patients who have diabetic nephropathy are at high risk for cardiovascular morbidity and mortality that is independent of other risk factors for cardiovascular disease.\textsuperscript{20,21} The pathogenesis of diabetic nephropathy is not completely understood, and it is a slow, progressive disorder. It is unusual to see evidence of this complication within 10 years of the diagnosis of diabetes.

Most people develop diabetic nephropathy after 10 to 20 years of having the disease.\textsuperscript{22} Clinically, diabetic nephropathy is characterized by: 1) albuminuria of $> 300$ mg/day on at least two occasions, 3 to 6 months apart; 2) a progressive decline in the glomerular filtration rate, and; 3) persistently elevated blood pressure.\textsuperscript{22} As with the other chronic complication of diabetes, the longer the duration of the disease and higher the serum glucose level the greater the chances of developing diabetic nephropathy. This relationship is supported by evidence that the incidence and progression of diabetic nephropathy can be significantly decreased by strict control of serum glucose and intensive diabetes therapy.\textsuperscript{2,23}
Impaired wound healing

Impaired wound healing is a significant and serious chronic complication of diabetes mellitus. Diabetes affects the wound healing process in many ways, including but not limited to: peripheral neuropathy that decreases pain sensation and predisposes the diabetic to repeated trauma/stress; hyperglycemia that causes a sub-clinical inflammation and damage to the micro-vasculature; local tissue hypoxia, and; interference with collagen synthesis. Unfortunately, these changes frequently lead to the development of foot ulcers, and infected foot ulcers are quite common in patients who have diabetes.

It has been estimated that approximately 15%-25% of patients who have diabetes will develop a foot ulcer at least once and > 50% of those ulcers will become infected. Patients who require hospitalization for an infected foot ulcer often require lengthy hospital stays and approximately 50% will require some degree of amputation. Factors that increase the risk of developing an infected foot ulcer include: an ulcer that has been open for greater than 30 days; advanced age; obesity; male gender; duration of diabetes; peripheral neuropathy; peripheral artery disease, and; a history of skin ulcers.

ACUTE COMPLICATIONS OF DIABETES MELLITUS

Diabetic ketoacidosis

Diabetic ketoacidosis (DKA) is an acute, potentially life-threatening complication of diabetes mellitus. It is characterized by hyperglycemia, an anion gap metabolic acidosis with a serum pH < 7.30, a bicarbonate level < 15 mEq/L, ketonemia and ketonuria. Diabetic
Ketoacidosis occurs more commonly in patients with type 1 diabetes than in patients with type 2, and it occurs more often in children and adolescents than in adults.\textsuperscript{28} It is the most important cause of diabetes-related morbidity and mortality in children who have diabetes.\textsuperscript{29}

The underlying cause of DKA is insulin deficiency. This can be an absolute deficiency when no insulin is produced or no insulin is administered, or it can be a relative deficiency, when the amount of insulin available is not sufficient to the body’s metabolic needs. In either case, the lack of insulin causes hyperglycemia, and hyperglycemia initiates changes in energy production and metabolism that have profound effects.\textsuperscript{29}

The first of these is an increased production and release of specific hormones. The insulin deficiency of DKA causes a decrease in glucose uptake and utilization. The body responds to lack of an energy source by an increased production of cortisol, epinephrine, glucagon, and growth hormone. These hormones, in turn, stimulate gluconeogenesis, glycogenolysis, proteolysis, and lipolysis, the last being defined as the use of fats for energy.\textsuperscript{29,30}

These changes in energy production are beneficial in some ways. However, they also have intensely harmful effects and consequences, and perhaps the most important of these is the production of ketones from lipolysis. Lipolysis generates free fatty acids and when free fatty acids are oxidized, ketones are produced. These ketones, acetone, acetoacetate and beta-hydroxybutyrate, are weak acids that dissociate and produce hydrogen ions. Normally, the degree of lipolysis and the
production of ketones are such that the hydrogen ions that are produced are easily buffered. But in DKA an enormous number of ketones are produced and the resulting acid load is overwhelming.

The increased production and release of hormones and the changes in energy production, along with a very high serum glucose level, are the basis for the signs and symptoms that are characteristic of DKA.

1. Dehydration:
   Dehydration is one of the most prominent clinical signs of DKA and there are several reasons why it occurs. Nausea, vomiting and tachypnea are common in patients that have DKA and these all cause fluid losses. The high serum glucose acts as an osmotic diuretic as do the serum ketones. And there are alternate ways of providing energy, e.g., gluconeogenesis and lipolysis, all increase the metabolic rate and result in an increase in body heat that increases fluids loss.

2. Acidosis:
   The acidosis of DKA causes tachypnea, tissue hypoxia, central nervous system depression, confusion, and a shift of potassium from the intracellular space to the extracellular space.

3. Hypokalemia:
   DKA causes hypokalemia for several reasons. Acidosis initiates a movement of potassium out of the cells, and the osmotic diuresis and vomiting deplete body potassium by renal and gastrointestinal losses.
4. Ketonuria and ketonemia:
   Elevated urine levels of ketones and elevated serum level of ketones cause diuresis. An elevated serum ketone level causes the characteristic fruity odor of the breath that is so often noted in patients who have DKA, as some of the excess ketones are excreted by respiration.

Diabetic ketoacidosis happens for many reasons but the most common causes are an infectious process such as urinary tract infection or pneumonia, poor compliance with insulin therapy, or new onset of type 1 diabetes. Diabetic ketoacidosis can also occur during periods of stress (e.g., during or after a surgical procedure, or during or after a myocardial infarction) or DKA may be caused by medications such as atypical antipsychotics and corticosteroids. Regardless of the etiology, the underlying pathologic processes are the same, and the signs/symptoms of DKA are excessive thirst, increased urination, nausea, vomiting, weakness, confusion, coma, hypotension, an acetone-like odor on the breath, shallow, rapid breathing, Kussmaul respirations, hypokalemia, and hyponatremia.

The diagnostic criteria of DKA in children are slightly different than for adults (the serum glucose must be > 200 mg/dL), but the pathologic process is the same. However, DKA in children is a potentially very dangerous condition, more so than for adults. Children who have a DKA for a long period of time and/or have significantly depressed sensorium on presentation are at risk for cerebral edema. Cerebral edema accounts for the great majority of deaths caused by DKA. Fortunately with early identification and aggressive treatment of DKA,
cerebral edema is an unusual complication of DKA. Cerebral edema in adults as a complication of DKA is very uncommon.

**Hypoglycemia**

Hypoglycemia is defined in diabetic patients as a serum glucose \( \leq 70 \) mg/dL, and it is a serious and commonly experienced problem for this population. Most patients who have type 1 diabetes have numerous asymptomatic episodes of hypoglycemia every year, several episodes of symptomatic hypoglycemia a week, and at least one episode each year of hypoglycemia that is so profound as to be temporarily disabling. Hypoglycemia is less common in patients who have type 2 diabetes, but it is still a significant problem.

The American Diabetes Association and the Endocrine Society Workgroup on Hypoglycemia classifies hypoglycemic episodes that occur in diabetic patients as severe, documented symptomatic, asymptomatic, probable symptomatic, and pseudohypoglycemia.

1. **Severe hypoglycemia:**
   The patient has a degree of hypoglycemia so severe that he/she requires assistance and treatment with carbohydrates or glucagon.

2. **Documented symptomatic hypoglycemia:**
   The patient has signs and symptoms characteristic of hypoglycemia and the measured serum glucose is \( \leq 70 \) mg/dL.

3. **Asymptomatic hypoglycemia:**
   The serum glucose is \( \leq 70 \) mg/dL but the patient is asymptomatic.
4. Probable symptomatic hypoglycemia:
The patient has signs and symptoms that are characteristic of hypoglycemia but the serum glucose was not measured.

5. Relative hypoglycemia:
The diabetic patient has the characteristic signs and symptoms of hypoglycemia but the serum glucose is measured at > 70 mg/dL. This event can happen to people who have poor glycemic control as these people may have symptomatic hypoglycemia when the blood sugar is > 70 mg/dL.

The basic cause of hypoglycemia is a relative excess of insulin. However, a relative excess of insulin and a diminished ability of the compensatory mechanisms that increase serum glucose in response to hypoglycemia cause most episodes of hypoglycemia. In people who do not have diabetes or in people who have recently developed the disease, hypoglycemia causes a decrease in the production and release of insulin, the release of glucagon from the pancreas, and the release of epinephrine and blood sugar are increased. In people who have type 1 or type 2 diabetes these compensatory mechanisms are absent or attenuated, and as the disease progresses there is a decreased ability to respond to hypoglycemia and hypoglycemic episodes become more frequent.\textsuperscript{32,33} The consequences of hypoglycemia include a difficulty in maintaining euglycemia and increased morbidity and mortality.\textsuperscript{32,33}

**PREVENTION OF DIABETES AND SCREENING**

Type I diabetes can be a devastating disease. The only available effective treatment is insulin therapy and insulin must be continued forever. Although insulin therapy and good medical care help reduce
the incidence of diabetic complications or delay the onset of complications, it cannot eliminate them entirely.\textsuperscript{35} Unfortunately, although it is now far easier to predict who is at risk for developing type 1 diabetes, efforts aimed at preventing the disease and/or changing the course of the disease have not been successful.\textsuperscript{36}

Type 2 diabetes, however, can be prevented. Screening for the risk factors that increase the chances of progression of pre-diabetes to diabetes is the first step: these risk factors include advanced age, dyslipidemia, ethnicity, family history of diabetes, obesity, sedentary lifestyle, and the severity of the pre-diabetes condition.\textsuperscript{37} Detection of diabetes is one of the crucial steps in preventing complications and ensuring that those who have diabetes receive the proper care.

Asymptomatic adults should be tested for diabetes if:\textsuperscript{38}
1. The patient is 45 years of age or older. If the first screening tests are normal they should be repeated at three-year intervals, at least.

2. The patient has a body mass index (BMI) of \( \geq 25 \text{ mg/m}^2 \) and any of the following apply:
   a. Sedentary life style;
   b. First-degree relatives with diabetes;
   c. High-risk ethnic background, \textit{i.e.}, African American, Latino, Native American, Asian American, Pacific Islander;
   d. A woman who delivered a baby weighing > 9 lbs or who had gestational diabetes;
   e. Hypertension \( \geq 140/90 \text{ mm Hg} \) or being treated with anti-hypertensives;
f. HbA1c ≥ 5.7%, impaired glucose tolerance or abnormal fasting glucose;
g. Cardiovascular disease;
h. HDL cholesterol <35 mg/dL or triglyceride level >250 mg/dL;
i. Polycystic ovary disease;
j. Any medical condition associated with insulin resistance.

Modification and/or elimination of these risk factors, along with the judicious use of medications including metformin, thiazolidinediones, and an alpha-glucosidase inhibitor acarbose, has been shown to significantly decrease the risk of developing type 2 diabetes.\textsuperscript{37} Patients who have impaired glucose tolerance, abnormal fasting glucose, or an HbA1c of 5.7%-6.4% should be encouraged to lose weight and start an exercise program, and consideration should be given to initiating therapy with metformin, especially if the patient has a BMI of > 32 kg/m\textsuperscript{2}, is 60 years of age or older, or is a woman who has had gestational diabetes.\textsuperscript{38}

**TREATMENT OF TYPE 1 AND TYPE 2 DIABETES**

Treatment of diabetes requires insulin therapy, the use of oral or other injectable medications that lower serum glucose, life style changes, close monitoring for complications, and treatment of complications. Self-care is an essential part of treatment of diabetes so patient education and the use of support systems is vital. There is no cure for type 1 diabetes. It is possible for some people who have type 2 diabetes to reach a point where they no longer need anti-diabetic medications. The disease can be managed with life style changes and dietary manipulation, but it cannot be said that type 2 diabetes can be cured.
The focus of treatment of complications associated with chronic diabetes is often on restoring glycemic control, symptomatic and supportive care and will not be discussed in this module. The treatment of DKA and hypoglycemia will be briefly discussed.

**Insulin therapy**

People who have type 1 diabetes must use insulin. Insulin is delivered by subcutaneous injection or via an implanted insulin pump that delivers the drug subcutaneously. Inhaled insulin products and transdermal insulin products have been developed, but at the time of this writing they are not commercially available.

There are many different types of injectable insulins. They all have the same mechanism of action, but they differ in onset of action, peak effect, and duration of action. They are usually separated into the following categories:

- Rapid-acting
- Short-acting
- Intermediate-acting
- Intermediate to long-acting
- Long-acting
- Combination products

For example, insulin lispro (brand name HumaLOG®) is rapid-acting insulin. It has an onset of action of 25-30 minutes, the peak glycemic effect occurs in 30 minutes to 2.5 hours, and the duration of action is ≤ 5 hours. NovoLIN® is a combination product that contains 30% of a short-acting insulin, regular insulin, and 70% of an intermediate-acting insulin, NPH insulin. NovoLIN has an onset of action of 30 minutes, the
peak glycemic effect occurs between 2-12 hours after injection, and the duration of action is 18-24 hours.

There are two approaches to the use of insulin for the treatment of patients who have type 1 diabetes: the conventional approach and the intensive insulin therapy approach. Intensive insulin therapy has become the preferred approach and it involves three or more injections a day of insulin or the use of an insulin pump.\(^3\) The goals of intensive insulin therapy are to maintain blood sugar and HbA1c levels as close as possible to non-diabetic levels while avoiding hypoglycemic episodes and by doing so, prevent/reduce cardiovascular disease, nephropathy, neuropathy, and retinopathy: the HbA1c level should be < 7\%, the capillary glucose before meals should be between 70-130 mg/dL, and the peak postprandial capillary glucose should be < 180 mg/dL.\(^3\) Persistent hyperglycemia is considered to be the cause of these diabetic complications, and there is a large body of evidence that strongly suggests that aggressively reducing blood sugar levels will prevent them or decrease their severity if they have been established.\(^3\),\(^4\)

Intensive insulin therapy is the standard of care for treating patients who have type 1 diabetes.\(^3\)\(^-\)\(^4\) However, reviews of intensive insulin therapy have noted:

- this approach has not had a significant effect on all-cause mortality when compared with conventional insulin therapy;
- intensive insulin therapy does appear to reduce the risk of developing macrovascular complications and diabetic nephropathy;
• the evidence for its effect in preventing diabetic retinopathy and diabetic ketoacidosis is inconsistent;
• the parameters of glycemic control differ from source to source;
• the risk of severe hypoglycemia is increased;
• the benefits of intensive insulin therapy seem to be decreased if diabetic complications are already present; and,
• the effectiveness of intensive insulin therapy may differ with age.\textsuperscript{40,42}

Intensive insulin therapy also requires the patient to exercise a high degree of self-care but despite these requirements and potential drawbacks it is, as mentioned, the preferred approach to the use of insulin.

**Lifestyle changes**

Blood glucose monitoring, proper diet, and exercise are critically important part of the life style changes needed for the treatment and self-care of patients who have type 1 or type 2 diabetes. It can be done by self-monitoring of blood glucose (SMBG) or by continuous glucose monitoring (CGM) via an implanted insulin sensor. Self-monitoring of blood glucose is more commonly used than CGM, and it requires the patient to perform testing as many as six to eight times a day if he/she is using intensive insulin therapy. Typically blood glucose should be monitored: 1) before meals and snacks; 2) postprandially; 3) before going to sleep; 4) if the patient has signs or symptoms of low blood sugar; and, 5) before performing any task during which a hypoglycemic episode would have dangerous consequences, e.g., driving. The HbA1c should be measured at least twice a year, and it should be measured more frequently in patients who have difficulty
stabilizing the blood sugar and/or if there is a change in their drug therapy.\(^\text{38}\)

The glycemic goals of insulin therapy in non-pregnant adults are: 1) an HbA1c of < 7.0%; 2) a preprandial capillary glucose of 70-130 mg/dL; and, 3) a peak postprandial capillary glucose of 180 mg/dL.\(^\text{38}\) The goals can be adjusted for age, the presence of co-morbid conditions, the duration of the diabetes, and other factors.

Proper diet is essential for maintaining an optimal glucose level and decreasing the risk for developing complications. The following dietary recommendations are from the American Diabetes Association 2014 standards of medical care.\(^\text{38}\)

- The ideal distribution of caloric intake from carbohydrates, fats, and proteins has not been determined, so an individual dietary assessment should be made in order to decide the proper distribution of these nutrients for each patient.

- Monitoring of carbohydrate intake is very important for attaining glycemic control. Dairy products, fruits, legumes, vegetables, and whole grains are the preferred source of carbohydrates. Carbohydrate sources that contain added fats, sodium, and sugar should be avoided.

- The intake of sugar-sweetened beverages is discouraged. Ingestion of these makes glycemic control difficult and increases the risk of weight gain.
• People who have diabetes are at an increased risk for developing cardiovascular disease. However, there is no evidence at this point that suggests that the intake of cholesterol containing foods, trans fats, and/or saturated fats should be appreciably less for diabetics than for the general population.

• There is no evidence that people with diabetes would benefit from vitamin, mineral, or micro-nutrient supplementation.

• Alcohol should be used sparingly: one drink a day or less for adult women; two drinks a day or less for adult men.

Exercise helps maintain ideal weight and there is evidence that it helps with glycemic control and improves insulin sensitivity. Adults are encouraged to perform at least 150 minutes a week of aerobic exercise at an intensity that raises the heart rate to 50%-70% maximum, and there should be no more than two consecutive days without exercise. Resistance training should be performed at least twice a week.

Smoking cessation is an important part of diabetes management. It should be mandatory.

**Monitoring For Complications**

The following guidelines for monitoring for diabetic complications are applicable to patients who have either type 1 or type 2 diabetes. Obviously, the specific frequency of, and the methods used for monitoring will vary for each individual.
**Blood pressure**

Blood pressure is measured using the same guideline as for a non-diabetic individual, and abnormally high values should be confirmed by re-measuring the blood pressure on separate occasions. A systolic blood pressure of ≤ 140 mm Hg and a diastolic blood pressure of ≤ 80 mm Hg are considered desirable. These levels have been associated with a reduction in CVD, nephropathy, and stroke in patients who have diabetes.

**Fasting lipid profile**

The fasting lipid profile should be measured at least once a year. An LDL cholesterol level of < 100 mg/dL in patients who do not have CVD and an LDL cholesterol level of < 70 mg/dL in patients with overt CVD are the desired goals. The triglyceride level should be < 150 mg/dL and the HDL cholesterol level should be < 40 mg/dL in men and < 50 mg/dL in women.

**Kidney disease**

All patients who have diabetes should have serum creatinine, serum potassium, and urinary albumin excretion measured at least once a year. If the glomerular filtration rate (GFR) is below 60 mL/min/1.73m² the patient should be referred to a nephrologist if there is a possibility of non-diabetic kidney disease. In addition, this patient population should have periodic measurements of serum electrolytes, serum bicarbonate, serum calcium, serum phosphorus, and hemoglobin, and GFR.

The frequency with which these tests should be performed should be increased as the GFR decreases. Refer the patient for dietary
counseling to make sure that his/her diet contains a sufficient amount of vitamin D, and consider bone density testing.

**Diabetic retinopathy**

Patients who have type 1 or type 2 diabetes should have a comprehensive, dilated eye exam at the time they are diagnosed. If there is no evidence of retinopathy then an eye exam can be done every other year. If there is evidence of retinopathy the eye exam should be repeated every year.

Women who have diabetes and who are pregnant or planning to become pregnant should have a comprehensive eye exam. Women who are pregnant should have an eye exam during the first trimester, and they should be followed closely during the course of the pregnancy and for one-year post-partum.

**Diabetic peripheral neuropathy**

Patients who type 1 diabetes should be examined for the presence of peripheral neuropathy starting at five years post-diagnosis. Patients who have type 2 diabetes should have this examination at the time the diagnosis is made.

**Impaired wound healing**

Every patient who has diabetes should have a yearly foot exam. The exam should include evaluation of pulses, testing for loss of protective sensation and re-education, if necessary, in the basics of diabetic foot care.
Assessment of co-morbid conditions

People who have diabetes have an increased risk for developing medical and psychiatric disorders including, but not limited to anxiety, depression, cancers, cognitive decline and dementia, hearing impairment, hip fracture, non-alcoholic liver disease, obstructive sleep apnea, and periodontal disease. People at risk should be identified and the appropriate screening tests applied and preventive measures taken.

Medications For The Treatment of Type 2 Diabetes

There are many medications used to treat type 2 diabetes. The mechanisms of actions of these will be discussed in the sections below, dosing and side effects will not.

\(\alpha\)-glucosidase inhibitors

\(\alpha\)-glucosidase enzymes digest carbohydrates in the brush border of the small intestine. The \(\alpha\)-glucosidase inhibitors acarbose (Precose®) and miglitol (Glyset®) are competitive antagonists of these enzymes and they act by delaying glucose absorption. They are available as oral tablets and they can be used alone or with other diabetes medications.

Biguanides

The biguanides metformin and phenformin were first used for the treatment of diabetes in the late 1950s. Phenformin was withdrawn from commercial use in the 1970s because it caused lactic acidosis and death. Metformin is the biguanide in use today.
Metformin is not a hypoglycemic drug. It is an antihyperglycemic drug that lowers blood sugar when hyperglycemia is present, but it will not lower blood sugar below the normal range. It has four mechanisms of action: decreased glucose absorption by the gut, increased glucose uptake by the tissues, decreased glucose production by the liver, and decreased insulin requirements for glucose disposal. Metformin is available as an oral tablet and it is often combined with a sulfonyurea and occasionally with other diabetic medications.

**Dipeptidyl peptidase 4 inhibitors (DPP-4)**

The DPPD-4 inhibitor sitagliptin (Januvia®) increases insulin secretion by inhibiting the action of DPP-4. DPP-4 is an enzyme that breaks down incretins (such as glucagon-like peptide-1, aka GLP-1. Incretins are gastrointestinal hormones that reduce blood glucose by increasing the production and release of insulin by the pancreas. Sitagliptin is available as an oral tablet.

**Exenatide**

Exenatide (Bydureon®; Byetta ®) is an analogue of the endogenous incretin GLP-1. The incretin GLP-1 - and its analogue exenatide - lowers blood glucose by increasing glucose-independent insulin release. Exenatide also decreases inappropriate glucagon secretion, increase beta cell growth and replication, decreases food intake, and slows gastric emptying. Exenatide is administered as a subcutaneous injection.
**Meglitinide derivatives**

The meglitinide derivatives repaglinide and nateglinide (Prandin® and Starlix ®) stimulate the release of insulin by the pancreas by facilitating the movement of calcium across cell membranes. They are available as oral tablets and repaglinide is also available as a combination product with metformin (Prandimet®).

**Pramlintide**

Pramlintide (SymlinPen®) is an injectable anti-diabetic drug that can be used to treat patients who have type I or type 2 diabetes. Pramlintide lowers blood glucose by slowing the absorption of glucose through the gut, reducing postprandial glucagon secretion, and acting as an appetite suppressant.

**Thiazolidinediones**

The thiazolidinediones rosiglitazone and pioglitazone (Avandia® and Actos®) lower blood sugar by decreasing insulin resistance. These drugs are available as oral tablets, and they are also available as a combination product with metformin, a DPP-4 inhibitor, or a sulfonylurea.

**Sulfonylureas**

This class of drug includes glyburide, glipizide, and glimepiride. There are many brand names of these drugs. They are very commonly used as anti-diabetic drugs and they lower blood sugar by stimulating the secretion of insulin by the pancreas, increasing insulin sensitivity, and reducing glucose output by the liver. They are available as oral tablets and they are often combined with metformin.
**Insulin**

Insulin was discussed in a previous section. Initially, people who have type 2 diabetes can be successfully treated with oral medications and lifestyle changes. However, many patients who have type 2 diabetes will eventually need insulin.

Metformin is the first-line drug for the treatment of patients who have type 2 diabetes. If metformin, lifestyle changes, and dietary manipulations are not successful in lowering the serum glucose and the HbA1c to the desired levels within several months, a second drug such as exenatide, insulin, sitagliptin, a sulfonylurea, or a thiazolidinedione can be added. A third drug can be added as needed.

**TREATMENT OF DIABETIC KETOACIDOSIS**

The most important treatments for DKA are fluid replacement, insulin therapy, and correction of electrolyte imbalances. Obtain baseline serum electrolytes, BUN, creatinine, serum glucose, an arterial or venous blood gas (either one is appropriate), a complete blood count, an ECG, and a CXR. Consider other laboratory studies if the cause of DKA is thought to be an underlying disease.

The first step in treating DKA is IV hydration, followed by correction of any potassium deficit. Insulin therapy should be started only after IV hydration has begun and any potassium deficit has been corrected. Although the patient who has DKA may be quite acidotic, sodium bicarbonate is not routinely administered.
**Fluid replacement**

Fluid replacement is *paramount* in the initial treatment of the patient of DKA.\(^{43,44}\) Fluid replacement will replace the fluid deficit, help decrease the blood glucose level, increase insulin sensitivity, and maintain renal function. If the patient is severely hypovolemic, an intravenous infusion of 0.9% sodium chloride 1 liter over 60 minutes should be given. This rate of infusion may need to be continued over several hours.

After the initial fluid volume correction, the serum sodium level should be checked. If the serum sodium is normal or high, the infusion should be changed to 0.45% sodium chloride IV and infused at a rate of 250-500 mL/h. If the serum sodium continues to be low, then 0.9% sodium chloride IV should be resumed at a rate of 250-500 mL/hour. Once the blood glucose is approximately 200 mg/dL, 5% dextrose/0.45% sodium chloride IV should be started and infused at 150-250 mL/hour.\(^43\)

**Correcting electrolyte imbalances**

If the serum potassium is < 3.3 mEq/L, insulin therapy should not be started and 20-30 mEq of potassium per hour should be given until the serum level is >3.3 mEq/L. If the serum potassium is > 3.3 mEq/L but < 5.3 mEq/L, 20-30 mEq of potassium should be added to each liter of IV fluid until the serum potassium is between 4-5 mEq/L. If the serum potassium is ≥ 5.3 mEq/L, there is no need to give supplemental potassium.\(^{43}\)
**Insulin therapy**

Insulin is a critical part of the treatment of DKA. It can be given as an IV infusion or frequent subcutaneous injections; they are equally effective. The IV infusion is often preferred because the onset of action is quicker and the half-life is shorter with the IV route than that of the subcutaneous route, and this allows for closer monitoring of therapy.

The American Diabetes Association recommends giving an initial IV bolus of regular insulin, 0.1 U/kg. However, if the initial serum potassium is < 3.3 mEq/L, insulin should *not* be given until potassium replacement has been started. Following the bolus dose, it is recommended that a continuous IV infusion of regular insulin be started at a rate of 0.1 U/kg/h. An alternative is to omit the bolus dose and to start a continuous IV infusion of regular insulin at a rate of 0.14 U/kg/h.

If the serum glucose does not decrease by 10% within an hour of starting the insulin, 0.14 U/kg should be given as a bolus dose, then the IV infusion continued. Once the serum glucose is < 200 mg/dL, the insulin dose should be reduced to 0.02-0.05 U/kg/h or subcutaneous doses of rapid-acting insulin 0.1 U/kg given every two hours. The goal at this point is to keep the serum glucose between 150-200 mg/dL.43

The algorithm, page 38, helps to illustrate the treatment steps recommended to maintain the serum glucose within the desired range of 150-200 mg/dL.
0.1 U/kg IV bolus
↓
0.1 U/kg/hr *
↓
When serum glucose < 200 mg/dL, decrease infusion to 0.02-0.05 U/kg/h
↓
Keep serum glucose between 150-200 mg/dL until DKA resolves

* If serum glucose doesn’t decrease by 50-70 mg/dL in the first hour, double the IV insulin dose.

Assess the need for bicarbonate

If the serum pH is < 6.90, 100 mEq of sodium bicarbonate and 20 mEq of potassium chloride in 400 mL of sterile water should be administered, infused over two hours. If the serum pH is > 7.0, sodium bicarbonate should not be given.43

Serum glucose should be checked every hour until the patient is stable. Blood urea nitrogen, creatinine, electrolytes, and venous pH should be checked every two to four hours until the patient is stable. DKA is considered to be resolved when: 1) the anion gap is less than 12 mEq/L; 2) the venous pH is > 7.30; 3) the serum bicarbonate is ≥ 15 mEq/L; and, 4) the patient is able to eat. At that point subcutaneous insulin should be started but IV insulin continued for one to two more hours.
TREATMENT OF HYPOGLYCEMIA

The first step in treating hypoglycemia is recognizing the signs and symptoms of low blood sugar: anxiety, blurred vision, confusion, hunger, incoordination, palpitations, sweating, tremor, and weakness. If the hypoglycemic episode occurs at home and it is less than severe, the patient can be given 15-20 grams of glucose, e.g., a tablespoon of honey or sugar, four ounces of juice. If the hypoglycemic episode occurs at home and considered to be severe, 0.5-1.0 mg of glucagon can be given subcutaneously.

Glucagon injections are available by prescription for home use. Administering oral glucose to an unconscious person is not recommended. Prolonged and profound hypoglycemia can cause brain damage and myocardial infarction, and a physician should evaluate someone who has severe hypoglycemia. In a hospital setting, patients who have hypoglycemia should be treated with intravenous boluses of 25%-50% dextrose and hypertonic dextrose infusion.

DIABETIC FOOT CARE

Diabetic foot care is one of the most important aspects of self-care for the patient who has diabetes. In order to avoid foot ulcers, infections, and amputation, the diabetic patient must know how to recognize potential and actual problems and know how to properly care for her/his feet.

Warning signs and symptoms that should prompt the patient to seek medical attention include:
• Difficulty walking
• Numbness and/or tingling
• Pen wounds, especially those with drainage
• Pain
• Red areas of the skin
• Skin that is abnormally cold or warm to the touch
• Swelling

A daily inspection of the feet should be considered mandatory for all patients who have diabetes and if any of the signs and symptoms previously mentioned are present, a physician or other healthcare provider should be notified. Self-care measures for avoiding foot ulcers and infections include:

• Exercise: Regular exercise can help improve peripheral circulation.
• Hygiene: The feet should be bathed daily in warm water.
• Proper footwear: A podiatrist or other healthcare provider should be consulted when making choices about footwear but in general, shoes should fit loosely and comfortably and should never be tight or restricting. Tight fitting socks should not be worn.
• Smoking cessation
• Toenail trimming: Patients should receive instruction on the proper method for trimming toenails. An ingrown toenail should be evaluated and treated as soon as possible.

Diabetic ulcers should be treated by a healthcare professional that has experience with the care of these wounds. The affected extremity should be off-loaded as much as possible. The wound itself should be
covered with a dressing. The choice of dressing will depend on the nature of the wound, *i.e.*, whether it is dry, infected, or has an exudate. A vascular surgeon or an experienced podiatrist should be consulted, as debridement may be necessary.

Other treatments that can be used for a diabetic foot ulcer include platelet-derived growth factors such as becaplermin gel (Regranex®) that help recruit the cells involved in wound healing, clostridial collagenase ointment, and hyperbaric oxygen. There is some clinical evidence that supports the use of these treatments, but at this point there is a lack of high-level, unequivocal clinical evidence supporting them.

**SUMMARY**

Diabetes mellitus is a chronic disease characterized by chronic, persistent hyperglycemia. There are two primary types of diabetes mellitus, type 1 and type 2. Type 1 diabetes typically has an onset in childhood or early adolescence. It is an autoimmune disease caused by an environmental or infectious stimulus in a genetically susceptible adult, and type 1 diabetes causes complete destruction of the insulin-producing beta cells of the pancreas.

Type 2 diabetes typically has an onset during adulthood. Type 2 diabetes is thought to be caused by insulin resistance, a process that is initiated in genetically susceptible people but that is also, in part, caused by lifestyle factors including, but not limited to, dyslipidemia, ethnic risk factors, hypertension, and obesity. Insulin resistance causes chronic hyperglycemia and eventually this high level of serum glucose permanently impairs some but not all the pancreatic beta
cells. People who have type 2 diabetes still produce insulin, but in the later stages of the disease they will require exogenous insulin.

Glucose is one of the primary sources of energy that is used for metabolic processes. Glucose molecules are too large to diffuse through cell membranes, and insulin is needed so that glucose can be moved into the cells. Insulin is a polypeptide hormone that is produced by the beta cells in the islets of Langerhans in the pancreas. The primary function of insulin is to facilitate the entry of glucose into cells where it can be used for energy or stored as glycogen. Because the patient who has diabetes has a complete or relative lack of insulin, serum glucose levels will abnormally high unless the patient is treated.

The classic signs and symptoms of diabetes are polyphagia, polydipsia, and polyuria. Clinically, diabetes is diagnosed if: 1) the fasting plasma glucose of 126 mg/dL or higher; 2) a 2-hour plasma glucose level is ≥ 200 mg/dL during an oral glucose challenge test, and; 3) the hemoglobin A1c (HbA1c) level is > 6.5%. The HbA1c, also known as the glycosated hemoglobin level, measures glucose that is attached to hemoglobin, and it provides an indication of what the average blood glucose has been for several months prior to the test.

Diabetes can cause many serious complications: cardiovascular disease, diabetic nephropathy, diabetic peripheral neuropathy, diabetic retinopathy, foot ulcers, diabetic ketoacidosis, and hypoglycemia. In addition, the presence of diabetes increases the risk for developing many co-morbid conditions, such as cancer, dementia, and psychiatric disorders.
Diabetes cannot be cured. People who have type 1 diabetes will always need to take insulin. Type 2 diabetes can be prevented with dietary manipulations and lifestyle changes, and the patient who has type 2 diabetes and can assiduously control risk factors may be able to manage the disease without medications, but it cannot be said that type 2 diabetes can be cured. The basics of care for both type 1 and type 2 diabetes are: 1) elimination or control of lifestyle factors that accelerate the disease process and/or cause diabetic complications; 2) the use of injectable and/or oral anti-diabetic medications, and; 3) monitoring for diabetic complications.

Please take time to help NurseCe4Less.com course planners evaluate the nursing knowledge needs met by completing the self-assessment Knowledge Questions after reading the article, and providing feedback in the course evaluation.

Correct Answers, pg. 46
1. **Type 1 diabetes is characterized by:**  
   a. a complete lack of endogenous insulin  
   b. a chronically low blood sugar  
   c. a relative lack of endogenous insulin production  
   d. decreased insulin sensitivity

2. **Type 2 diabetes is characterized by:**  
   a. decreased insulin sensitivity  
   b. a relative lack of insulin production  
   c. inappropriate secretion of glucagon  
   d. increased efficiency of pancreatic beta cells

3. **HbA1c provides a measurement of:**  
   a. post-prandial blood sugar  
   b. fasting blood sugar  
   c. the average serum glucose over several months  
   d. the average serum glucose after an insulin injection

4. **Type 1 diabetes is considered to be primarily caused by:**  
   a. an autoimmune process  
   b. insulin resistance  
   c. lifestyle factors  
   d. chronic hyperglycemia

5. **Type 2 diabetes is considered to be primarily caused by:**  
   a. an autoimmune process  
   b. an infectious process affecting pancreatic beta cells  
   c. drugs that damage the pancreatic beta cells  
   d. genetic susceptibility and lifestyle factors
6. *Commonly reported signs and symptoms of diabetes include:*
   a. anorexia, weight loss, and diarrhea  
b. excessive thirst and hunger, excessive urination  
c. persistent headache, shortness of breath, fever  
d. abdominal pain, dysuria, and peripheral neuropathy

7. **Which of the following are common acute complications of diabetes?**
   a. Chest pain, pulmonary embolism  
b. Pneumonia, urinary tract infection  
c. Hypoglycemia, DKA  
d. Nephrolithiasis, pulmonary edema

8. **Which of the following are common chronic complications of diabetes?**
   a. Nephropathy, neuropathy, and retinopathy  
b. Coagulopathies, DVT, and nephrolithiasis  
c. Hepatitis, congestive heart failure, and Parkinsonism  
d. Dementia, gastric reflux, and osteoporosis

9. **The first choice drug for treating patients who have type 2 diabetes is:**
   a. insulin  
b. glyburide  
c. metformin  
d. exenatide
10. Patients who have diabetes should be very closely monitored for:
   a. urinary tract infections
   b. infected foot ulcers
   c. upper respiratory tract infections
   d. gastroenteritis

CORRECT ANSWERS:

1. A
2. B
3. C
4. A
5. D
6. B
7. C
8. A
9. C
10. B
Footnotes:


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