Medical Nutrition Therapy for Diabetes Mellitus and Hypoglycemia of Nondiabetic Origin

KEY TERMS

A1C an evaluation of a combination of all fractions of the hemoglobin molecule; a measurement of the glycosylation of the “c” fraction and the recommended assay method (simplified to A1C). An A1C of 6% reflects an average plasma glucose level of ≈120 mg/dl. In general, each 1% increase in A1C is a reflection of an increase in average glucose levels of ≈30 mg/dl.

autonomic symptoms symptoms of hypoglycemia that are adrenergically based and that arise from the action of the autonomic nervous system.

carbohydrate counting a commonly used method for diabetes food and meal planning based on research showing that carbohydrate is the primary nutrient affecting postprandial blood glucose and insulin levels and that all carbohydrates affect blood glucose levels approximately the same when eaten in similar gram amounts; carbohydrates can be measured in grams or carbohydrate servings—one carbohydrate serving is a portion of food that contains 15 g of carbohydrate

combination therapy a form of therapy for diabetes using combinations of oral glucose-lowering medications or a combination of oral glucose-lowering medication(s) and insulin(s) or other injectable medications

correction factor (CF) a factor determined from the “1700 rule,” which defines how many milligrams per deciliter a unit of rapid-acting or short-acting insulin will lower blood glucose levels over a 2- to 4-hr period in an individual.

counterregulatory (stress) hormones hormones, including glucagon, epinephrine (adrenaline), norepinephrine, cortisol, and growth hormone, released during stressful situations, that have the opposite effect of insulin and cause the liver to release glucose from stored glycogen (glycogenolysis) and the adipose

cells to release fatty acids (lipolysis); these hormones also counterbalance declining glucose levels

dawn phenomenon a natural increase in morning blood glucose levels and insulin requirements that occurs in people with and without diabetes but tends to be more marked in people with diabetes; possibly caused by a diurnal variation in growth hormone, cortisol, or catecholamines

Diabetes Control and Complications Trial (DCCT) a 10-year study in people with type 1 diabetes who were treated with either conventional or intensive therapy; follow-up evaluations proved that intensive blood glucose control reduces the risk of diabetic microvascular and macrovascular complications.

diabetic ketoacidosis (DKA) severe, uncontrolled diabetes resulting from insufficient insulin, in which ketone bodies (acids) build up in the blood; if left untreated (with immediate administration of insulin and fluids), DKA can lead to coma and even death.

exchange lists foods grouped into six lists: starch, fruit, milk, vegetables, meat and meat substitutes, and fat; each list is a group of measured foods of approximately the same nutritional value; therefore foods on the same list can be “exchanged” or substituted for one another.

fasting (food-deprived) hypoglycemia low blood glucose concentrations in response to no food intake for 8 hours or longer.

gastroparesis impaired gastric motility; results in delayed or irregular contractions of the stomach, leading to various gastrointestinal symptoms such as feelings of fullness, bloating, nausea, vomiting, diarrhea, or constipation.

gestational diabetes mellitus (GDM) glucose intolerance, the onset or first recognition of which occurs during pregnancy.
glycemic index (GI) a measurement of the relative area under the postprandial glucose curve of 50 g of digestible carbohydrates compared with 50 g of a standard food, either glucose or white bread

glycemic load (GL) the estimated GL of foods, meals, and dietary patterns is calculated by multiplying the glycemic index by the amount of carbohydrate in each food and then totaling the values for all foods in a meal or dietary pattern

glucagon a hormone produced by the α-cells of the pancreas that causes an increase in blood glucose levels by stimulating the release of glucose from liver glycogen stores

glucose-lowering medications drugs administered orally that are used to control or lower blood glucose levels, including first- and second-generation sulfonylureas, nonsulfonylureas, secretagogues, biguanides, α-glucosidase inhibitors, and thiazolidinediones

glucotoxicity β-cells chronically exposed to hyperglycemia become progressively less efficient in responding to a glucose challenge

glycosylated hemoglobin a blood test that reflects the blood glucose concentration over the life span of red blood cells (≈120 days), expressed as a percentage of total hemoglobin with glucose attached; also may be called glycated hemoglobin or glycohemoglobin

honeymoon phase the period after the initial diagnosis of type 1 diabetes when there may be some recovery of β-cell function and a temporary decrease in exogenous insulin requirement

hyperglycemia excessive glucose in the blood (generally 180 mg/dl or above) caused by too little insulin, insulin resistance, or increased food intake; symptoms include frequent urination, increased thirst, weight loss, and often tiredness or fatigue

hyperglycemic hyperosmolar state (HHS) extremely high blood glucose levels with an absence of or only slight ketosis and profound dehydration

hypoglycemia (or insulin reaction) low blood glucose level (70 mg/dl or less) caused by the administration of excessive insulin or insulin secretagogues, too little food, delayed or missed meals or snacks, increased exercise or other physical activity, or alcohol intake without food

hypoglycemia of nondiabetic origin low levels of blood glucose that lead to neuroglycopenia symptoms that are ameliorated by the ingestion of carbohydrates

immune-mediated diabetes mellitus a form of type 1 diabetes resulting from autoimmune destruction of the β-cells of the pancreas

incretins hormones released during nutrient absorption, which increase glucose-dependent insulin secretion

injectable glucose-lowering medications drugs administered by injection that are used to control or lower blood glucose levels; incretin mimetics that have the same glucose-lowering effects as the body’s naturally occurring incretins; synthetic amylin—a polypeptide hormone normally co-secreted with insulin by the β-cells of the pancreas in response to food intake

insulin a hormone released from the β-cells of the pancreas that enables cells to metabolize and store glucose and other fuels

insulin resistance an impaired biologic response (sensitivity) to either exogenous or endogenous insulin; involved in the etiology of type 2 diabetes

insulin secretagogues oral medications that stimulate insulin release from the β-cell of the pancreas, such as sulfonylureas and nonsulfonylurea secretagogues (i.e., repaglinide and nateglinide)

insulin sensitizers oral medications that enhance insulin action and include biguanides (metformin) and thiazolidinediones

macrovascular diseases diseases of the large blood vessels, including coronary artery disease, cardiovascular disease, and peripheral vascular disease

metabolic syndrome characterized by central obesity and insulin resistance with increased risk for cardiovascular disease and type 2 diabetes; associated risk factors include dyslipidemia, hypertension, presence of prothrombotic factors, and impaired glucose tolerance

microvascular diseases diseases of the small blood vessels, including retinopathy and nephropathy

neuropathic symptoms neurologic symptoms of hypoglycemia that are related to an insufficient supply of glucose to the brain

polydipsia excessive thirst

polyuria excessive urination

postprandial (after a meal) blood glucose blood glucose level 1 to 2 hours after eating

postprandial (reactive) hypoglycemia low blood glucose within 2 to 5 hours after eating

pre-diabetes (impaired glucose homeostasis) blood glucose concentrations that are higher than normal but not yet high enough to be diagnosed as diabetes; sometimes referred to as impaired glucose tolerance (IGT) or impaired fasting glucose (IFG); risk factor for future diabetes and cardiovascular disease

preprandial (fasting) blood glucose blood glucose level before eating

self-monitoring of blood glucose (SMBG) individuals testing their own blood glucose levels using a chemically treated strip and visually comparing the strip to a color chart or by inserting the strip into a meter that measures the glucose level

Somogyi effect hypoglycemia followed by “rebound” hyperglycemia; originates during hypoglycemia with the secretion of counterregulatory hormones (glucagon, epinephrine, growth hormone, and cortisol) and is usually caused by excessive exogenous insulin doses

target blood glucose goals levels for capillary blood glucose tests that are as near normal as possible and that can be achieved without risk of serious hypoglycemia

type 1 diabetes a type of diabetes that usually occurs in persons younger than 30 years of age but can occur at any age; previously known as insulin-dependent diabetes mellitus (IDDM) or juvenile-onset diabetes

type 2 diabetes a type of diabetes usually occurring in persons older than 30 years of age, previously known as noninsulin-dependent diabetes mellitus (NIDDM) or maturity-onset diabetes; now also frequently diagnosed in youth and young adults
Diabetes mellitus is a group of diseases characterized by high blood glucose concentrations resulting from defects in insulin secretion, insulin action, or both. Abnormalities in the metabolism of carbohydrate, protein, and fat are also present. Persons with diabetes have bodies that do not produce or respond to insulin, a hormone produced by the β-cells of the pancreas that is necessary for the use or storage of body fuels. Without effective insulin, hyperglycemia (elevated blood glucose) occurs, which can lead to serious complications and premature death; but, people with diabetes can take steps to control the disease and lower the risk of complications.

In 2005 total prevalence of diabetes in the United States, all ages, was 20.8 million people or 7% of the population. Of these, 14.6 million are diagnosed, and 6.2 million undiagnosed. About 10.9 million men and 9.7 million women 20 years of age or older had diagnosed diabetes, or 9.6% of all people in this age-group, representing an increase from 4.9% of the adult population in 1990 and 7.3% in 2000. Diabetes prevalence increases with age, affecting 10.3 million people age 60 years or older, or 21% of all people in this age-group. Furthermore, in 2005 1.5 million people age 20 years or older were newly diagnosed with diabetes (Centers for Disease Control and Prevention, 2005).

Much of the increase is because type 2 diabetes is no longer a disease that affects mainly older adults. Between 1990 and 1998 the prevalence of diabetes increased by 76% among people in their thirties (Mokdad et al., 2001). Among children with newly diagnosed diabetes, the prevalence of type 2 diabetes also increased dramatically in the past decade, growing from less than 4% in the years preceding 1990 to as high as 45% in certain racial and ethnic groups in recent years (American Diabetes Association [ADA], 2000).

The prevalence of type 2 diabetes is highest in ethnic groups in the United States. Non-Hispanic blacks are 1.8 times as likely to have diabetes as non-Hispanic whites; Hispanic Latinos, 1.7 times; American Indians and Alaska Natives, 2.2 times, and Asian-Americans and Pacific Islanders 1.5 times (Centers for Disease Control and Prevention, 2005) (see Focus On: Diabetes Does Discriminate!)

In addition, another 41 million people are estimated to have pre-diabetes, which includes impaired glucose tolerance (IGT) (2-hour postchallenge glucose of 140–199 mg/dl) and impaired fasting glucose (IFG) (fasting plasma glucose 100 to 125 mg/dl) (Centers for Disease Control and Prevention, 2005). Persons with pre-diabetes are at high risk for conversion to type 2 diabetes and cardiovascular disease (CVD) if lifestyle prevention strategies are not implemented.

Diabetes mellitus contributes to a considerable increase in morbidity and mortality rates, which can be reduced by early diagnosis and treatment. In 2002 diabetes costs in the United States were $132 billion. Direct medical expenditures such as inpatient care, outpatient services, and nursing home care totaled $92 billion, or an average annual total direct cost of medical care of $13,243 per person with diabetes compared with $2,560 per person without diabetes. Indirect costs, totaling $40 billion, were associated with lost productivity, including premature death and disability (ADA, 2003a).

### Pathophysiology

Assigning a type of diabetes to an individual often depends on the circumstances present at the time of diagnosis, and many individuals do not easily fit into a single category. Thus it is less important to label the particular type of diabetes than it is to understand the pathogenesis of the hyperglycemia and to treat it effectively (ADA, 2006a). What is clear, however, is the need to intervene early with lifestyle interventions, beginning with pre-diabetes and continuing through the disease process. In 1997 recommendations were made to eliminate the terms insulin-dependent diabetes mellitus (IDDM) and noninsulin-dependent diabetes mellitus (NIDDM) and to use the terms type 1 and type 2 diabetes and to use Arabic rather than Roman numerals (Table 30-1).

#### Pre-diabetes

A stage of impaired glucose homeostasis that includes IFG and GT is called pre-diabetes. People with pre-diabetes have IFG, IGT, or both. Individuals with pre-diabetes are at high risk for future diabetes and CVD.

#### Type 1 Diabetes

At diagnosis, people with type 1 diabetes are often lean and experience excessive thirst, frequent urination, and significant weight loss. The primary defect is pancreatic β-cell destruction, usually leading to absolute insulin deficiency and resulting in hyperglycemia, polyuria (excessive urination), polydipsia (excessive thirst), weight loss, dehydration, electrolyte disturbance, and ketoacidosis. The rate of β-cell destruction is quite variable, proceeding rapidly in some persons (mainly infants and children) and slowly in others (mainly adults). The capacity of a healthy pancreas to secrete insulin is far in excess of what is needed normally; therefore, the clinical onset of diabetes may be preceded by an extensive asymptomatic period of months to years, during which β-cells are undergoing gradual destruction (see Pathophysiology and Care Management Algorithm: Type 1 Diabetes Mellitus).

Type 1 diabetes accounts for 5% to 10% of all diagnosed cases of diabetes. Persons with type 1 diabetes are dependent on exogenous insulin to prevent ketoacidosis and
Diabetes strikes particularly hard in certain ethnic populations. Certain environmental or lifestyle factors may increase the risk of developing type 2 diabetes in susceptible populations. For example, an increase in the prevalence is observed in populations who have migrated to more urbanized locations compared with people of the same group who remained in their traditional home. Urbanization is usually related to major changes in diet, physical activity, and socioeconomic status, as well as increased obesity.

One theory that might explain the increased prevalence of diabetes and insulin resistance among Native people is the “thrifty” gene. Years of subsistence living have created a thrifty genotype that allows Native people to extract a lot of energy and fat from small amounts of food. In an era of store-bought processed food, that gene backfires to induce obesity and diabetes. Adoption of a “Western” lifestyle (which may include a diet high in fat and a sedentary way of life) has been associated with a dramatically increased rate of type 2 diabetes in the Pima Indians of Arizona (ADA, 2001). Among the Pima Indians of Arizona, about 55% of adults older than 35 years of age have type 2 diabetes. This disease is increasingly being diagnosed in Native Americans younger than 30 years of age and has been diagnosed in some as young as 7 years.

Ravussin and colleagues (1994) surveyed a closely related population of Pima Indians living in Maycoba, a small village in a remote, mountainous region of northwestern Mexico. They found that individuals in this community ate a diet lower in fat than is typically consumed in Arizona, and both men and women were very physically active. The men and women of Maycoba weighed, on average, 50 lb less than a comparable group of Pimas from the Phoenix area. More importantly, diabetes was diagnosed in about 10% of the Maycoba Pimas compared with almost 50% of the Arizona Pimas.

The main staples of the Maycoba Pimas’ diet are beans, corn (as tortillas), and potatoes. Several essential nutrients are lacking because of the relative absence of fruits and vegetables. Diet analysis reveals a diet composed of 13% protein, 23% fat, 63% carbohydrate, and less than 1% alcohol and containing more than 50 g of fiber. This is in sharp contrast to the present diet of the Arizona Pimas. Even more striking than the low-fat diet of the Maycoba population, however, was the high level of physical activity in this population; more than 40 hours a week were spent engaged in hard physical work (Ravussin et al., 1994).

Interventions involving increased physical activity and a reduced fat and energy diet slowed the progression to type 2 diabetes in high-risk populations (Diabetes Prevention Program Research Group, 2002). Health promotion activities through community-based exercise programs and a return to more traditional diets also may help to reduce the diabetes epidemic that affects many developing countries and ethnic groups in industrialized nations.

### TABLE 30-1

<table>
<thead>
<tr>
<th>Classification</th>
<th>Distinguishing Characteristics</th>
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<tbody>
<tr>
<td>Type 1 diabetes</td>
<td>Affected persons are usually children and young adults, although it can occur at any age, and are dependent on exogenous insulin to prevent ketoacidosis and death. Type 1 diabetes accounts for 5% to 10% of all diagnosed cases of diabetes.</td>
</tr>
<tr>
<td>Type 2 diabetes</td>
<td>Affected persons are often older than 30 yr at diagnosis, although it is now occurring frequently in young adults and children. The disease is slowly progressive, and treatment necessary to control hyperglycemia varies over time. Individuals are not dependent on exogenous insulin for survival but often require it for adequate glycemic control. Complications of diabetes may be present at diagnosis.</td>
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<tr>
<td>Gestational diabetes</td>
<td>Diabetes diagnosed in some women during pregnancy.</td>
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<tr>
<td>Other specific types</td>
<td>Diabetes that results from specific genetic syndromes, surgery, drugs, malnutrition, infections, or other illnesses.</td>
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<tr>
<td>Pre-diabetes</td>
<td>Fasting or glucose tolerance test results above normal, but not diagnostic of diabetes. These persons should be monitored closely because they have an increased risk of developing diabetes.</td>
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</table>

death. Although it may occur at any age, even in the eighth and ninth decades of life, most cases are diagnosed in people younger than 30 years of age, with a peak incidence at around ages 10 to 12 years in girls and ages 12 to 14 years in boys.

Type 1 diabetes has two forms: immune-mediated diabetes mellitus and idiopathic diabetes mellitus. Immune-mediated diabetes mellitus results from an autoimmune destruction of the β-cells of the pancreas, the only cells in the body that make the hormone insulin that regulates
blood glucose. Idiopathic type 1 diabetes mellitus refers to forms of the disease that have no known etiology. Although only a minority of persons with type 1 diabetes fall into this category, of those who do, most are of African or Asian origin (ADA, 2006a).

Risk factors for type 1 diabetes may be genetic, autoimmune, or environmental. The genetic predisposition to type 1 diabetes is the result of the combination of HLA-DQ coded genes for disease susceptibility offset by genes that are related to disease resistance (ADA, 2006a). However, the genetic factors that confer susceptibility or protection remain unclear. A 50% discordance rate of type 1 diabetes exists between identical twins, suggesting that specific genes are necessary but not sufficient for its development. A trigger, likely environmental, is necessary for the expression of the genetic propensity. There are no known means to prevent type 1 diabetes.

Regardless of the trigger, early type 1 diabetes is first identified by the appearance of active autoimmunity directed against pancreatic β-cells and their products. At diagnosis, 85% to 90% of patients with type 1 diabetes have one or more circulating autoantibodies to islet cells, endogenous insulin, or other antigens that are constituents of islet cells. Antibodies identified as contributing to the destruction of β-cells are (1) islet cell autoantibodies; (2) insulin autoantibodies, which may occur in persons who have never received insulin therapy; (3) antibodies against islet tyrosine phosphatase (known as IA2 and IA2); and (4) autoantibodies to glutamic acid decarboxylase (GAD), a protein on the surface of β-cells. GAD autoantibodies appear to provoke an attack by the T cells (killer T lymphocytes), which may be what destroys the β-cells in diabetes. The clinical onset of diabetes may be abrupt, but the pathophysiologic insult is a slow, progressive process. Hyperglycemia and symptoms develop only after greater than 90% of the secretory capacity of the β-cell mass has been destroyed.

Frequently, after diagnosis and the correction of hyperglycemia, metabolic acidosis, and ketoacidosis, endogenous insulin secretion recovers. During this **honeymoon phase** exogenous insulin requirements decrease dramatically for up to 1 year; however, the need for increasing exogenous insulin replacement is inevitable, and within 5 to 10 years after clinical onset, β-cell loss is complete, and circulating islet cell antibodies can no longer be detected.

### Type 2 Diabetes

**Type 2 diabetes** may account for 90% to 95% of all diagnosed cases of diabetes and is a progressive disease that, in many cases, is present long before it is diagnosed. Hyperglycemia develops gradually and is often not severe enough in the early stages for the patient to notice any of the classic symptoms of diabetes. Although undiagnosed, these individuals are at increased risk of developing macrovascular and microvascular complications.

Risk factors for type 2 diabetes include genetic and environmental factors, including a family history of diabetes, older age, obesity, particularly intraabdominal obesity, physical inactivity, a prior history of gestational diabetes, pre-diabetes, and race or ethnicity. Adiposity and a longer duration of obesity are powerful risks factors for type 2 diabetes, and even small weight losses are associated with a change in glucose levels toward normal in persons with pre-diabetes. Nevertheless, type 2 diabetes is found in persons who are not obese, and many obese persons never develop type 2 diabetes. Obesity combined with a genetic predisposition may be necessary for type 2 diabetes to occur. Another possibility is that a similar genetic predisposition leads independently to both obesity and insulin resistance, which increases the risk for type 2 diabetes (ADA, 2001). A sedentary lifestyle has also been linked to an increased propensity to develop type 2 diabetes (see *Pathophysiology and Care Management Algorithm: Type 2 Diabetes Mellitus*).

In most cases type 2 diabetes results from a combination of insulin resistance and β-cell failure, but the extent to which each of these factors contributes to the development of the disease is unclear. Endogenous insulin levels may be normal, depressed, or elevated; but they are inadequate to overcome concomitant **insulin resistance** (decreased tissue sensitivity or responsiveness to insulin); as a result, hyperglycemia ensues. Insulin resistance is first demonstrated in target tissues, mainly muscle, liver, and adipose cells. Initially there is a compensatory increase in insulin secretion, which maintains normal glucose concentrations; but, as the disease progresses, insulin production gradually decreases. Hyperglycemia is first exhibited as an elevation of **postprandial (after a meal) blood glucose** caused by insulin resistance at the cellular level and is followed by an elevation in fasting glucose concentrations. As insulin secretion decreases, hepatic glucose production increases, causing the increase in **preprandial (fasting) blood glucose** levels. Compounding the problem is the deleterious effect of hyperglycemia itself—**glucotoxicity**—on both insulin sensitivity and insulin secretion; hence the importance of achieving near-euglycemia in persons with type 2 diabetes.

Insulin resistance is also demonstrated at the adipocyte level, leading to lipolysis and an elevation in circulating free fatty acids. In particular, excess intraabdominal obesity, characterized by an excess accumulation of visceral fat around and inside abdominal organs, results in an increased flux of free fatty acids to the liver, leading to an increase in insulin resistance. Increased fatty acids also cause a further decrease in insulin sensitivity at the cellular level, impair pancreatic insulin secretion, and augment hepatic glucose production (lipotoxicity) (Bergman and Adler, 2000). The above defects contribute to the development and progression of type 2 diabetes and are also primary targets for pharmacologic therapy.

Persons with type 2 diabetes may or may not experience the classic symptoms of uncontrolled diabetes, and they are not prone to develop ketoacidosis. Although persons with type 2 diabetes do not require exogenous insulin for survival, about 40% or more will eventually require exogenous insulin for adequate blood glucose control. Insulin may also be required for control during periods of stress-induced hyperglycemia, such as during illness or surgery.
Type 2 Diabetes Mellitus

Etiology
- Genetic factors
- Risk factors (physical inactivity, older age, obesity)
- Environmental factors
- Intake of excessive calories

Pathophysiology
- Type 2 Diabetes Mellitus (insulin resistance; insulin deficiency)

Clinical Findings
- Abnormal pattern of insulin secretion and action
- Decreased cellular uptake of glucose and increased postprandial glucose
- Increased release of glucose by liver (gluconeogenesis) in early morning hours

Symptoms (variable)
- Hyperglycemia
- Excessive thirst
- Frequent urination
- Polyphagia
- Weight loss

Medical Management
- Diagnosis
  - FBG >126 mg/dl
  - Nonfasting glucose >200 mg/dl (with symptoms)
  - Oral GTT >200 mg/dl
- Monitoring
  - Blood glucose
  - A1C testing
- Medication
  - Sulfonylureas
  - Non-sulfonylurea secretagogues
  - Biguanides
  - α-Glucosidase inhibitors
  - Thiazolidinediones
  - Incretins

Nutrition Management
- Lifestyle strategies (food/eating and physical activity) that improve glycemia, dyslipidemia, and blood pressure
- Nutrition education (carbohydrate counting and fat modification)
- Energy restriction to promote 5%–10% weight loss
- Blood glucose monitoring to determine adjustments in food or medications

**Gestational Diabetes Mellitus**

*Gestational diabetes mellitus (GDM)* is defined as any degree of glucose intolerance with onset or first recognition during pregnancy. It occurs in about 7% of all pregnancies, resulting in more than 200,000 cases annually (ADA, 2001). Women with known diabetes mellitus before pregnancy are not classified as having GDM. GDM is usually diagnosed during the second or third trimester of pregnancy. At this point, insulin-antagonist hormone levels increase, and insulin resistance normally occurs. During pregnancy gestational diabetes requires treatment to normalize maternal blood glucose levels to avoid complications in the infant.

**Other Types of Diabetes**

This category includes diabetes associated with specific genetic syndromes (such as maturity-onset diabetes of youth), surgery, drugs, malnutrition, infections, and other illnesses. Such types of diabetes may account for 1% to 5% of all diagnosed cases of diabetes.

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### Diagnostic and Screening Criteria

Diagnostic criteria for diabetes are summarized in Table 30-2. Three diagnostic methods may be used to diagnose diabetes and each, in the absence of unequivocal hyperglycemia, must be confirmed, on a subsequent day, by any of the following three methods (ADA, 2006a). At this time, hemoglobin A1C (A1C) is not recommended for diagnosis. In pregnant women different criteria are applied in establishing the diagnosis of gestational diabetes.

- Symptoms of diabetes plus a casual plasma glucose value of \( \geq 200 \text{ mg/dl} \) (11.1 mmol/l). *Casual* is defined to any time of the day without regard to time since last meal. The classic symptoms of diabetes include polyuria, polydipsia, and unexplained weight loss.

- A fasting plasma glucose (FPG) value \( \geq 126 \text{ mg/dl} \) (7 mmol/L). Fasting is defined as no caloric intake for at least 8 hr.

- A 2-hour postload glucose \( \geq 200 \text{ mg/dl} \) (11.1 mmol/L) during an oral glucose tolerance (OGT) test involving administration of 75 g of glucose

Testing or screening for diabetes should be considered in all individuals at age 45 years and above, particularly in those with a body mass index (BMI) of 25 kg/m\(^2\) or more, and, if normal, should be repeated at 3-year intervals (ADA, 2006b). Testing should be considered at a younger age or be carried out more frequently in individuals who are overweight (BMI \( > 25 \text{ kg/m}^2 \)) and have additional risk factors:

- Are habitually physically inactive
- Have a first-degree relative with diabetes
- Are members of a high-risk ethnic population (e.g., black, Latino, Native American, Asian-American, and Pacific Islander)
- Have delivered a baby weighing more than 9 lb or have been diagnosed with gestational diabetes
- Are hypertensive (blood pressure \( \geq 140/90 \text{ mm Hg} \))
- Have a high-density lipoprotein (HDL) cholesterol level \( < 35 \text{ mg/dl} \) (0.9 mmol/L) and/or a triglyceride level \( > 250 \text{ mg/dl} \) (2.82 mmol/L)
- Have polycystic ovary syndrome (PCOS)
- On previous testing, had IGT or IFG
- Have other clinical conditions associated with insulin resistance (e.g., PCOS or acanthosis nigricans [i.e., gray-brown skin pigmentations])
- Have a history of vascular disease

The incidence of type 2 diabetes in children and adolescents is increasing dramatically and is consistent with screening recommendations for adults: children and youth at increased risk for type 2 diabetes should be tested. Youth who are overweight (BMI \( > 85\text{th percentile for age and sex} \)) and have any two of the following risk factors should be screened: family history of type 2 diabetes, members of high-risk

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**TABLE 30-2**

<table>
<thead>
<tr>
<th>Diagnosis of Diabetes Mellitus and Impaired Glucose Homeostasis</th>
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<tbody>
<tr>
<td><strong>Diagnosis</strong></td>
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<tr>
<td>---------------------</td>
</tr>
<tr>
<td>Diabetes</td>
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<td></td>
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<td></td>
</tr>
<tr>
<td>Pre-diabetes</td>
</tr>
<tr>
<td>Impaired fasting glucose</td>
</tr>
<tr>
<td>Impaired glucose tolerance</td>
</tr>
<tr>
<td>Normal</td>
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<td></td>
</tr>
</tbody>
</table>


CPG, Casual plasma glucose; FPG, fasting plasma glucose; 2hPG, 2-hour plasma glucose level (measured 2 hours after an oral glucose tolerance test with administration of 75 g of glucose).
ethnic populations, signs of insulin resistance (acanthosis nigricans, hypertension, dyslipidemia, or PCOS [see Focus On: Polycystic Ovary Syndrome in Chapter 21]), or maternal history of diabetes or gestational diabetes. The age of initiation of screening is age 10 years or onset of puberty, and the frequency is every 2 years (ADA, 2006b).

Screening and diagnosis for GDM are discussed later in this chapter.

**Management of Pre-Diabetes**

Based on early observational and intervention studies, the Finnish Diabetes Prevention Study (Tuomilehto et al., 2001) and the Diabetes Prevention Program (DPP) (DPP Research Group, 2002) were designed to investigate the effects of lifestyle interventions on prevention of diabetes in those at high risk (presence of IGT). The development of type 2 diabetes is strongly related to lifestyle factors, thus suggesting to researchers that it might be a preventable disease. Observational studies, as well as early intervention trials addressing physical activity, weight loss, and dietary intake, including whole grains and fiber and dietary fat, provided evidence for factors that might delay or prevent type 2 diabetes, but all had methodologic limitations.

In the Finnish study 522 middle-age, overweight subjects with IGT were randomized to receive either brief diet and exercise counseling (control group) or intensive individualized instruction on how to reduce weight (goal of 5% weight reduction), reduce total intake of fat (goal of <30% of energy intake) and saturated fat (goal of <10% of energy intake), and increase fiber intake (goal of 15 g/1000 kcal) and physical activity (goal of >150 minutes weekly). After an average follow-up of 3.2 years, there was a 58% reduction in the incidence of type 2 diabetes in the intervention group compared with the control group. The reduction in the incidence of diabetes was directly associated with the ability of the subjects to achieve one or more of the lifestyle strategies.

The Diabetes Prevention Program (DPP) randomized 3234 persons (45% from minority groups) with IGT to placebo, metformin, or lifestyle intervention. Subjects in the placebo and medication arms received standard lifestyle recommendations that included written information and an annual 20- to 30-minute individual session. Subjects in the lifestyle arm were expected to achieve and maintain a weight loss of at least 7% and to perform 150 minutes of physical activity per week. Subjects were seen weekly for the first 24 weeks, followed by monthly sessions. After an average follow-up of 2.8 years, a 58% decrease in the progression to diabetes was observed in the lifestyle group, and a 31% relative reduction was observed in the metformin group. On average, 59% of the lifestyle group achieved the goal of 7% or greater weight reduction, and 74% maintained at least 150 minutes per week of moderately intense activity (Fujimoto et al., 2007).

Three diabetes prevention trials in individuals with IGT using pharmacologic therapy also reported a lowering of the incidence of diabetes. Metformin reduced the risk of diabetes by 31% (DPP Research Group, 2002), the α-glucosidase inhibitor acarbose reduced risk by 32% (Chiasson et al., 2002), and the thiazolidinedione troglitazone reduced risk by 56% (Buchanan et al., 2002). In a group of obese individuals with or without IGT, orlistat added to lifestyle delayed the risk of diabetes by 45% in subjects with IGT but had no effect in those without IGT (Torgerson et al., 2004). Studies comparing lifestyle modifications to medication strongly provide support for the greater benefit of weight loss and physical activity as the first choice to prevent or delay diabetes. Modest weight loss (5% to 10% of body weight) and modest physical activity (30 minutes daily) are the recommended goals. Follow-up counseling is necessary to accomplish these objectives. There is insufficient evidence to support the use of drug therapy as a substitute for lifestyle modifications (ADA).

**Medical Nutrition Therapy for Pre-Diabetes**

Over the next decade the number of persons with diabetes and at risk for diabetes and CVD is expected to grow by 25%, largely driven by the rising prevalence of obesity and inactivity. Unless action is taken to change the predicted estimates of diabetes, the disease will also become a huge economic burden because of both direct health care costs and indirect costs resulting from a decline in workplace productivity. It is essential that individuals at risk for diabetes be identified and treatment interventions implemented. In no other disease does the role of lifestyle—healthy and appropriate food choices and physical activity—play a more important role in both prevention and treatment than in diabetes.

Goals of medical nutrition therapy (MNT) for pre-diabetes emphasize the importance of lifestyle in decreasing the risk of type 2 diabetes by increasing physical activity and promoting food choices that facilitate moderate weight loss (Box 30-1). Because of the effects of obesity on insulin resistance, weight loss is an important goal for persons with pre-diabetes or metabolic syndrome. Structured programs that emphasize lifestyle changes, including education, reduced fat (=30% of total energy) and energy intake, regular physical activity, and regular participant contact have been shown to result in long-term weight loss of 5% to 7% of starting weight. In the DPP, weight loss of 3 kg from baseline was also associated with an improved lipid profile (Ratner et al., 2005).

Although desired by the public, there is no “miracle diet” for weight loss. Low-carbohydrate and low-fat, energy-restricted diets both result in weight loss for up to 1 year. The low-carbohydrate diets have potential favorable effects on triglyceride and HDL cholesterol values; however, this should be weighed against potential unfavorable effects in low-density lipoprotein (LDL) cholesterol values from low-carbohydrate diets (Nordmann et al., 2006). Of prime importance is a reduced-energy diet that an individual can follow long term. Lifestyle modifications are reported to produce weight loss of approximately 10%, or 10 to 12 kg, over 3 to 6 months (Douketis et al., 2005) and can maintain...
Goals of Medical Nutrition Therapy for Diabetes Mellitus

Goals of Medical Nutrition Therapy That Apply to Persons at Risk for Diabetes or With Pre-Diabetes:

1. To decrease risk of diabetes and cardiovascular disease by promoting healthy food choices and physical activity leading to moderate weight loss that is maintained.

Goals of Medical Nutrition Therapy for Persons With Diabetes:

1. To the extent possible, achieve and maintain:
   - Blood glucose levels in the normal range or as close to normal as is safely possible.
   - A lipid and lipoprotein profile that reduces the risk for vascular disease.
   - Blood pressure levels that reduce the risk for vascular disease.

2. To prevent, or at least slow the rate of development of the chronic complications of diabetes by modifying nutrient intake and lifestyle as appropriate.

3. To address individual nutrition needs, taking into account personal and cultural preferences and willingness to change.

4. To limit food choices based on evidence and to maintain the pleasure of eating.

Goals of Nutrition Therapy That Apply to Specific Situations:

1. For youth with type 1 diabetes, youth with type 2 diabetes, pregnant and lactating women, and older adults with diabetes, to meet the nutritional needs of these unique times in the life cycle.

2. For individuals treated with insulin or insulin secretagogues, to provide self-management training for safe conduct of exercise, prevention and treatment of hypoglycemia, and treatment of acute illness.


Management of Diabetes

Two classic clinical trials have demonstrated beyond a doubt the clear link between glycemic control and the development of complications in persons with type 1 and type 2 diabetes, as well as the importance of nutrition therapy in achieving control. The Diabetes Control and Complications Trial (DCCT) was a long-term, prospective, randomized, controlled, multicenter trial that studied approximately 1400 young adults (ages 13 to 39 years) with type 1 diabetes who were treated with either intensive therapeutic regimens (multiple injections of insulin or use of insulin infusion pumps guided by blood glucose monitoring results) or conventional regimens (one or two insulin injections per day). Intensively treated patients experienced a 50% to 75% reduction in the risk of progression to retinopathy, nephropathy, and neuropathy after 8 to 9 years (DCCT Research Group, 1993). Furthermore, average A1C values in patients who adhered to the prescribed meal plan and adjusted food and insulin in response to hyperglycemia were 0.25% to 1% lower than among patients who did not follow these behaviors (Delahanty and Halford, 1993).

After a mean 17 years of follow-up, intensive diabetes therapy implemented during the trial was shown to reduce the risk of any CVD event by 42% and the risk of nonfatal myocardial infarction, stroke, or death from CVD by 57% (DCCT, 2005). Thus, in persons with type 1 diabetes, intensive therapy not only reduces the risk of microvascular complications, but also reduces the risk of macrovascular complications.

The reports of the United Kingdom Prospective Diabetes Study (UKPDS) demonstrated conclusively that elevated blood glucose levels cause long-term complications in type 2 diabetes, just as in type 1 diabetes (United Kingdom Prospective Diabetes Study Group [UKPDS], 1998a). The UKPDS recruited and followed up on 5102 newly diagnosed type 2 diabetic patients for an average of 10 to 11 years. Subjects were randomized into a group treated conventionally, primarily with nutrition therapy, and compared...
with subjects randomized into an intensively treated group, initially treated with sulfonylureas. In the intensive therapy group the microvascular complication rate decreased significantly by 25%, and the risk of macrovascular disease decreased by 16%. Combination therapy (combining insulin or metformin with sulfonylureas) was needed in both groups to meet glycemic goals as loss of glycemic control was noted over the 10-year trial. Aggressive treatment of even mild-to-moderate hypertension was also beneficial in both groups (UKPDS, 1998b).

The importance of implementing nutrition therapy at diagnosis was clearly demonstrated. Before randomization into intensive or conventional treatment, subjects received individualized intensive nutrition therapy for 3 months. During this period the mean A1C decreased by 1.9% (~9% to ~7%) (the greatest reduction in A1C during the trial) with only a modest average weight loss of 3.5 kg (8 lb). UKPDS researchers concluded that in determining FPG, the reduction of energy intake was at least as important, if not more important, than the actual weight lost.

This study illustrates the progressive nature of type 2 diabetes. An important lesson learned from the UKPDS is that therapy needs to be intensified over time and that, as the disease progresses, MNT alone is not enough to keep most patients’ A1C level at 7% or less. Medication(s), and for many patients, eventually insulin, needs to be combined with nutrition therapy. The “diet” doesn’t fail; the pancreas fails to secrete enough insulin to maintain adequate glucose control.

The management of diabetes includes MNT, physical activity, monitoring, medications, and self-management education. An important goal of treatment is to provide the patient with the necessary tools to achieve the best possible control of glycemia, lipidemia, and blood pressure to prevent, delay, or arrest the microvascular and macrovascular complications while minimizing hypoglycemia and excess weight gain (ADA, 2006b). Optimal control of diabetes also requires the restoration of normal carbohydrate, protein, and fat metabolism. Insulin is both anticatabolic and anabolic and facilitates cellular transport (Table 30-3). In general, the counterregulatory (stress) hormones (glucagon, growth hormone, cortisol, epinephrine, and norepinephrine) have the opposite effect of insulin.

Glycemic treatment goals for persons with diabetes are listed in Table 30-4. Achieving goals requires open communication and appropriate self-management education. Patients can assess day-to-day glycemic control by self-monitoring of blood glucose (SMBG) and measurement of urine or blood ketones. Longer-term glycemic control is assessed from the results of glycated hemoglobin (simplified as A1C) tests. When hemoglobin and other proteins are exposed to glucose, the glucose becomes attached to the protein in a slow, nonenzymatic, and concentration-dependent fashion. Measurements of A1C reflect a weighted average of plasma glucose concentration over the preceding weeks, thereby complementing day-to-day testing. In nondiabetic persons A1C values are 4%.

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**Table 30-3**

<table>
<thead>
<tr>
<th>Effect</th>
<th>Carbohydrates</th>
<th>Protein</th>
<th>Fat</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anticatabolic (prevents breakdown)</td>
<td>Decreases breakdown and release of glucose from glycogen in the liver</td>
<td>Inhibits protein degradation, diminishes gluconeogenesis</td>
<td>Inhibits lipolysis, prevents excessive production of ketones and ketoacidosis</td>
</tr>
<tr>
<td>Anabolic (promotes storage)</td>
<td>Facilitates conversion of glucose to glycogen for storage in liver and muscle</td>
<td>Stimulates protein synthesis</td>
<td>Facilitates conversion of pyruvate to free fatty acids, stimulating lipogenesis</td>
</tr>
<tr>
<td>Transport</td>
<td>Activates the transport system of glucose into muscle and adipose cells</td>
<td>Lowers blood amino acids in parallel with blood glucose levels</td>
<td>Activates lipoprotein lipase, facilitating transport of triglycerides into adipose tissue</td>
</tr>
</tbody>
</table>

**Table 30-4**

<table>
<thead>
<tr>
<th>Glycemic Control</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>A1C</td>
<td>&lt;7.0%*</td>
<td></td>
</tr>
<tr>
<td>Preprandial capillary plasma glucose</td>
<td>90-130 mg/dl (5.0-7.2 mmol/l)</td>
<td></td>
</tr>
<tr>
<td>Peak postprandial capillary plasma glucose†</td>
<td>&lt;180 mg/dl (&lt;10.0 mmol/l)</td>
<td></td>
</tr>
</tbody>
</table>


*Referenced to a nondiabetic range of 4%-6% using a DCCT-based assay.
†Peak levels in patients with diabetes.
to 6%; these values correspond to mean blood glucose levels of about 90 mg/dl (or about 5 mmol/L). An A1C of 6% reflects an average plasma glucose level of 120 mg/dl. In general, each 1% increase in A1C is a reflection of an increase in average glucose levels of 30 mg/dl.

Lipid levels and blood pressure must also be monitored (Table 30.5). Lipids should be measured annually, and blood pressure at every diabetes management visit (ADA, 2006b).

### Medical Nutrition Therapy for Diabetes

MNT is integral to total diabetes care and management. To integrate MNT effectively into the overall management of diabetes requires a coordinated team effort, including a registered dietitian (RD) who is knowledgeable and skilled in implementing current principles and recommendations for diabetes. MNT requires an individualized approach and effective nutrition self-management education and counseling. Monitoring glucose, A1C and lipid levels, blood pressure, weight, and quality-of-life issues is essential in evaluating the success of nutrition-related recommendations. If desired outcomes from MNT are not met, changes in overall diabetes care and management should be recommended (ADA, 2007).

The ADAs nutrition guidelines underscore the importance of individualizing nutrition care. Before 1994 nutrition recommendations attempted to define optimal percentages for macronutrient intake. Then, by determining a person’s energy needs based on theoretic calorie requirements and using the ideal percentages for carbohydrate, protein, and fat, a nutrition prescription was developed (e.g., 1800 calories, 225 g of carbohydrate [50%], 90 g of protein [20%], and 60 g of fat [30%]). The problem with this approach is that the prescribed “diet” cannot really be individualized; and it often lacks relevance to the patient’s personal lifestyle, culture, or socioeconomic status. Furthermore, this approach is not supported by scientific evidence and usually does not produce successful outcomes. Beginning in 1994 the ADA recommended that an individualized nutrition prescription be based on metabolic profiles, treatment goals, and changes that the person with diabetes is willing and able to make and not on rigid, predetermined calorie levels and macronutrient percentages (Franz et al., 1994). This approach continues with the 2002 and 2007 ADA nutrition recommendations for persons with diabetes (ADA, 2007; Franz, 2002).

Although numerous studies have attempted to identify the optimal percentages of macronutrients for the diet of persons with diabetes, it is unlikely that one such combination of macronutrients exists. The best mix appears to vary, depending on individual circumstances. If guidance is needed, the dietary reference intakes (DRIs) may be helpful to meet the body’s daily nutritional needs while at the same time minimizing risk for chronic diseases (ADA, 2007). The DRIs recommend that adults should consume 45% to 65% of total energy from carbohydrate, 20% to 35% from fat, and 10% to 35% from protein (Institute of Medicine, 2002).

### Goals and Outcomes of Medical Nutrition Therapy for Diabetes

The goals for MNT for diabetes emphasize the role of lifestyle in improving glucose control, lipid and lipoprotein profiles, and blood pressure. Improving health through food choices and physical activity is the basis of all nutrition recommendations for the treatment of diabetes (see Box 30-1).

Besides being skilled and knowledgeable in assessing and implementing MNT, RDs must also be aware of expected outcomes from nutrition therapy, when to assess outcomes, and what feedback, including recommendations, should be given to referral sources. Research supports MNT as an effective therapy in reaching diabetes treatment goals. Outcomes studies demonstrate that MNT provided by an RD as MNT alone or as MNT in combination with diabetes self-management training is associated with a decrease in A1C of approximately 1% in patients with type 1 diabetes and 1% to 2% in type 2 diabetes, depending on the duration of diabetes (Pastors et al., 2003; DAFNE Study Group, 2002; Lemon et al., 2004). These outcomes are similar to those from oral glucose-lowering medications. Interventions include reduced energy or reduced carbohydrate or fat intake, basic nutrition education with healthy food choices for improved glycemia, and matching insulin doses to carbohydrate intake. Furthermore, the effect of MNT on A1C will be known by 6 weeks to 3 months, at which time the RD must assess whether the goals of therapy have been met by changes in lifestyle or whether changes or additions of medications are needed (Franz et al., 1995).

Metaanalysis of studies in nondiabetic free-living subjects and expert committees report that MNT reduces LDL cholesterol by 15 to 25 mg/dl (Yu-Poth et al., 1999, NCEP, 2001). After initiation of MNT, improvements were apparent in 3 to 6 months. Metaanalysis and expert committees also support the role of lifestyle modifications in treatment of hypertension (Chobanian et al., 2003) (see Chapter 33).

### TABLE 30-5

<table>
<thead>
<tr>
<th>Recommendations for Lipid and Blood Pressure for Adults With Diabetes</th>
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<tbody>
<tr>
<td><strong>Lipids</strong></td>
</tr>
<tr>
<td>LDL cholesterol</td>
</tr>
<tr>
<td>HDL cholesterol</td>
</tr>
<tr>
<td>Men</td>
</tr>
<tr>
<td>Women</td>
</tr>
<tr>
<td>Triglycerides</td>
</tr>
<tr>
<td>Blood Pressure</td>
</tr>
</tbody>
</table>


HDL, High-density lipoprotein; LDL, low-density lipoprotein.
Carbohydrates and Diabetes

**Sugars, starch, and fiber** are the preferred terms for carbohydrates. Foods that contain carbohydrates from whole grains, fruits, vegetables, and low-fat milk are excellent sources of vitamins, minerals, dietary fiber, and energy; therefore these foods are important components of a healthy diet for all Americans, including those with diabetes. Although low-carbohydrate diets might seem to be a logical approach to lowering postprandial glucose, the ADA specifically states that “low-carbohydrate diets (restricting total carbohydrate to <130 g/day) are not recommended in the management of diabetes” (ADA, 2006b).

Historically it was a long-held belief that sucrose must be restricted based on the assumption that sugars such as sucrose (see Chapter 3) are more rapidly digested and absorbed than starches and thus aggravate hyperglycemia; however, scientific evidence does not justify restricting sugars or sucrose based on this belief. In approximately 20 studies in which sucrose was substituted for other carbohydrates, sucrose did not increase glycemia to a greater extent than isocaloric amounts of starch (ADA, 2007; Franz et al., 2002a). The glycemic effect of carbohydrate foods cannot be predicted based on their structure (i.e., starch versus sugar) owing to the efficiency of the human digestive tract in reducing starch polymers to glucose. Starches are rapidly metabolized into 100% glucose during digestion, in contrast to sucrose, which is metabolized into glucose and fructose. Fructose has a lower glycemic index (GI), which has been attributed to its slow rate of absorption and its storage in the liver as glycogen (see Chapters 3 and 9, and Appendix 43).

Numerous factors influence glycemic responses to foods, including the amount of carbohydrates, type of sugar (glucose, fructose, sucrose, lactose), nature of the starch (amylose, amylopectin, resistant starch), cooking and food processing, particle size, and food form, as well as the fasting and preprandial glucose concentrations, severity of the glucose intolerance, and the second meal or lente effect of carbohydrates. Although both the amount (grams) and type of carbohydrates in a food influence the blood glucose levels, monitoring total grams of carbohydrates, whether by use of carbohydrate counting or exchanges, remains a key strategy in achieving glycemic control (ADA, 2007). Numerous studies have reported that, when subjects are allowed to choose from a variety of starchy and sugars, the glycemic response is identical if the total amount of carbohydrate is similar (Franz, 2002). In studies comparing low- and high–GI diets, total carbohydrate is first of all kept consistent (Rizkalla et al., 2004). However, some individuals may note improvements in postprandial glucose responses with use of the GI or glycemic load (GL) factors when choosing foods or meals.

An important priority for food and meal planning is the total amount of carbohydrates that the person with diabetes chooses to have for meals or snacks. A variety of methods can be used to estimate the nutrient content of meals, including carbohydrate counting, exchange lists, and experience-based estimation (ADA, 2007). In carbohydrate counting food portions contributing 15 g of carbohydrates (regardless of the source) are considered to be one carbohydrate serving.

Testing premeal and postmeal glucose levels is important for making adjustments in either food intake or medication to achieve glucose goals. In using exchange lists foods are grouped into six lists—starch, fruit, milk, vegetables, meat and meat substitutes, and fat—and each list is a group of measured foods of approximately the same nutritional value. Therefore foods on the same list can be “exchanged” or substituted for one another. In addition, the exchange lists have an “other carbohydrates” list of sweets and snack foods and identify foods that are good sources of fiber; high in sodium; and combination foods such as casseroles, pizza, and soups, which fit into more than one exchange group.

**Glycemic Index and Glycemic Load**

Glycemic indexing of food was developed to compare the physiologic effects of carbohydrates on glucose. The glycemic index (GI) measures the relative area under the postprandial glucose curve of 50 g of digestible carbohydrates compared with 50 g of a standard food, either glucose or white bread. When bread is the reference food, the GI value for the food is multiplied by 0.7 to obtain the GI value that is comparable to glucose being used as the reference food (GI of glucose = 100; GI of white bread = 70). The GI does not measure how rapidly blood glucose levels increase. When reported, the peak glucose response for individual foods (Crapo et al., 1977) and meals, either high or low GI (Rizkalla et al., 2004), occurs at approximately the same time. Low-GI foods are usually defined as having a GI less than 55, moderate as GI 55 to 70, and high as GI more than 70. Refined starches often have a high GI; sugars such as fructose, lactose, and sucrose and fats a moderate to low GI (Foster-Powell et al., 2002).

The estimated glycemic load (GL) of foods, meals, and dietary patterns is calculated by multiplying the GI by the amount of carbohydrates in each food and then totaling the values for all foods in a meal or dietary pattern.

Low GI diets have been reported to improve glycemic control compared with high GI diets in persons with diabetes (Brand-Miller et al., 2003). However, there are substantial inconsistencies in study outcomes (Pi-Sunyer, 2002; Franz, 2003). Most people likely already consume a moderate GI diet (Rizkalla et al., 2004), and it is unknown whether further lowering of the dietary GI can be achieved long term.

A major problem with the GI is the variability of response to a specific carbohydrate food. For example, Australian potatoes are reported to have a high GI, whereas potatoes in the United States and Canada have moderate GIs (Fernandes et al., 2005). The concept of the GI can be best used for fine-tuning postprandial responses after focusing on total carbohydrate. See Appendix 43 for GI and GL factors for foods.

**Fiber**

Early short-term studies using large amounts of fiber (>30 g daily) in small numbers of subjects suggested a positive effect on glycemia; however, results from later studies have shown mixed effects. In subjects with type 1 diabetes, a
high-fiber diet (56 g daily) had no beneficial effects on glycemic control (Lafrance et al., 1998). Another study of subjects with type 1 diabetes showed positive effects from 50 g of fiber on glucose concentrations but no beneficial effects on lipids (Giacco et al., 2000). In persons with type 2 diabetes, increasing fiber from 11 to 27 g/1000 kcal did not improve glyceria, insulinemia, or lipemia (Hollenbeck et al., 1986); whereas another study comparing 24 g of fiber per day with 50 g of fiber reported improved glycemic control, reduced hyperinsulinemia, and decreased plasma lipids (Chandalia et al., 2000). Therefore it appears that ingestion of large amounts of fiber (>50 g/day) is necessary to have beneficial effects.

It is unknown whether free-living individuals can maintain such high levels of fiber and whether this amount would be acceptable to most people. As for the general public, people with diabetes are encouraged to choose a variety of fiber-containing foods such as legumes, fiber-rich cereals (≥5 g of fiber per serving), fruits, vegetables, and whole grain products. However, evidence is lacking to recommend a higher fiber intake for people with diabetes than for the population as a whole (ADA, 2007).

**Sweeteners**

Even though sucrose restriction cannot be justified on the basis of its glycemic effect, it is still good advice to suggest that persons with diabetes be careful in their consumption of foods containing large amounts of sucrose. Besides often being high in total carbohydrate content, these foods may also contain significant amounts of fat. If sucrose is included in the food and meal plan, it should be substituted for other carbohydrate sources or, if added, be adequately covered with insulin or other glucose-lowering medications. Sucrose and sucrose-containing foods also should be eaten in the context of a healthy diet, and care taken to avoid excess energy intake (ADA, 2007).

There appears to be no significant advantage of alternative nutritive sweeteners such as fructose over sucrose. Fructose provides 4 kcal/g, as do other carbohydrates, and even though it does have a lower glycemic response than sucrose and other starches, large amounts (15% to 20% of daily energy intake) of fructose have an adverse effect on plasma lipids (Bantle et al., 2000). However, there is no reason to recommend that persons with diabetes avoid fructose, which occurs naturally in fruits and vegetables as well as in foods sweetened with fructose (ADA, 2007).

Reduced calorie sweeteners approved by the U.S. Food and Drug Administration (FDA) include sugar alcohols (erythritol, sorbitol, mannitol, xylitol, isomalt, lactitol, and hydrogenated starch hydrolysates) and tagatose. They produce a lower glycemic response and have a lower caloric content than sucrose and other carbohydrates. Sugar alcohols contain, on average, approximately 2 calories per gram. With foods containing sugar alcohols, one half of sugar alcohol grams can be subtracted from total carbohydrate grams, particularly when using carbohydrate counting for meal planning. Although their use appears to be safe, it is unlikely that sugar alcohols in the amounts likely to be ingested in individual food servings or meals will contribute to significant reduction in total energy or improvement in glycemia (ADA, 2007). Some people report gastric discomfort after eating foods sweetened with these products, and consuming large quantities may cause diarrhea, especially in children.

Saccharin, aspartame, neotame, acesulfame potassium, and sucralose are nonnutritive sweeteners currently approved for use by the FDA. All such products must undergo rigorous testing by the manufacturer and scrutiny from the FDA before they are approved and marketed to the public. For all food additives, including nonnutritive sweeteners, the FDA determines an acceptable daily intake (ADI), defined as the amount of a food additive that can be safely consumed on a daily basis over a person's lifetime without risk (see Chapter 11). The ADI includes a 100-fold safety factor and greatly exceeds average consumption levels. For example, aspartame actual daily intake in persons with diabetes is 2 to 4 mg/kg of body weight daily, well below the ADI of 50 mg/kg daily (Butchko and Stargel, 2001). All FDA-approved nonnutritive sweeteners, when consumed within the established daily intake levels, can be used by persons with diabetes, including pregnant women (ADA, 2007). However, clinical studies in subjects without diabetes provide no evidence that nonnutritive sweeteners in foods cause weight loss or gain (Raben et al., 2002).

**Protein**

The rate of protein degradation and conversion of protein to glucose in type 1 diabetes depends on the state of insulinization and the degree of glycemic control. With less than optimal insulinization, conversion of protein to glucose can occur rapidly, adversely influencing glycemic control. In poorly controlled type 2 diabetes, gluconeogenesis is also accelerated and may account for most of the increased glucose production in the postabsorptive state. However, in those with controlled type 2 diabetes (Gannon et al., 2001; Nuttall et al., 1984) and well-controlled type 1 diabetes (Peters and Davidson, 1993), ingested protein did not increase plasma glucose concentrations. Although nonessential amino acids undergo gluconeogenesis, it is unclear why the glucose produced does not appear in the general circulation after ingestion of protein. Furthermore, protein does not slow the absorption of carbohydrates (Nuttall et al., 1984), and adding protein to the treatment of hypoglycemia does not prevent subsequent hypoglycemia (Gray et al., 1996). In patients with type 2 diabetes who are still able to produce insulin, ingested protein is just as potent a stimulant of insulin secretion as carbohydrate (Gannon et al., 2001; Nuttall et al., 1984).

For persons with diabetes and normal renal function, there is insufficient evidence to suggest that usual protein intake (10% to 20% of energy) should be modified (ADA, 2007). Intake of protein in this range does not appear to be associated with the development of diabetic nephropathy; however, the long-term effects of consuming more than 20% of energy as protein on the development of nephropathy has not been adequately studied.
Short-term studies with small numbers of subjects with diabetes suggest that diets with protein contents greater than 20% of total energy may improve glucose and insulin concentrations, reduce appetite, and improve satiety (Gannon et al., 2003; Gannon et al., 2004). However, such diets appear to be difficult to follow outside of a research setting (Brinkworth et al., 2004). The effects of protein on regulation of energy intake, satiety, and long-term weight loss have not been adequately studied.

Dietary Fat
Studies in persons with diabetes demonstrating the effects of specific percentages of dietary saturated and trans-fatty acids and specific amounts of cholesterol on CVD risk are not available. However, those with diabetes are considered to be at risk similar to those with a past history of CVD. Therefore, because of a lack of specific information, the goal for dietary fat intake (amount and type) for persons with diabetes is the same as for those without diabetes with a history of CVD. It is recommended that total fat be 25% to 35% of total energy, and saturated fatty acids less than 7%. Intake of trans-fat should be minimized or eliminated (ADA, 2007). Diets high in polyunsaturated fatty acids appear to have effects on lipids similar to those from diets rich in monounsaturated fatty acids (Summer et al., 2002). Therefore, to lower LDL cholesterol, energy derived from saturated fatty acids should be replaced with either monounsaturated or polyunsaturated fatty acids. In persons without diabetes, reducing saturated and trans-fatty acids decreases total and LDL cholesterol but may also reduce HDL cholesterol. However, importantly, the ratio of LDL to HDL cholesterol is not adversely affected.

In metabolic studies in which energy intake is maintained so that subjects do not lose weight, diets high in either carbohydrates or monounsaturated fat lower LDL cholesterol equivalently, but the concern has been the potential of a high-carbohydrate diet (greater than 55% of energy intake) to increase triglycerides and postprandial glucose compared with a high–monounsaturated fat diet (Garg et al., 1994). However, in other studies when energy intake is reduced, the adverse effects of high-carbohydrate diets are not observed (Heilbronn et al., 1999; Parker et al., 2002). Therefore energy intake appears to be a factor in determining the effects of a high-carbohydrate versus a high–monounsaturated fat diet.

There is evidence from the general population that foods containing very long omega-3 polyunsaturated fatty acids are beneficial, and two to three servings of fish per week are recommended. Although most studies in persons with diabetes have used omega-3 supplements and show beneficial lowering of triglycerides, an accompanying rise in LDL cholesterol also has been noted (Montori et al., 2000). If supplements are used, the effects on LDL cholesterol should be monitored. The omega-3 supplements may be most beneficial in the treatment of severe hypertriglyceridemia (Patti et al., 1999). In addition, two or more servings of fish per week (with the exception of commercially fried fish filets) can be recommended (ADA, 2007).

Plant sterol and stanol esters block the intestinal absorption of dietary and biliary cholesterol. In the general public (Hallikainen et al., 1999) and in persons with type 2 diabetes (Lee et al., 2000), intake of 2 to 3 g of plant stanols or sterols per day is reported to decrease total and LDL cholesterol levels by 9% to 20% (see Chapter 32).

Alcohol
The same precautions that apply to alcohol consumption for the general population apply to persons with diabetes. Abstention from alcohol should be advised for people with a history of alcohol abuse or dependence; for women during pregnancy; and for people with medical problems such as liver disease, pancreatitis, or advanced neuropathy. If individuals choose to drink alcohol, daily intake should be limited to one drink or less for adult women and two drinks or less for adult men (1 drink = 12 oz beer, 5 oz of wine, or 1½ oz of distilled spirits). Each drink contains ≈15 g alcohol. The type of alcoholic beverage consumed does not make a difference (ADA, 2007).

Moderate amounts of alcohol ingested with food have minimum, if any, acute effect on glucose and insulin levels (Howard et al., 2004). However, alcoholic beverages should be considered an addition to the regular food and meal plan for all persons with diabetes. No food should be omitted, given the possibility of alcohol-induced hypoglycemia and the fact that alcohol does not require insulin to be metabolized. Excessive amounts of alcohol (three or more drinks per day) on a consistent basis, contribute to hyperglycemia. This hyperglycemia improves as soon as alcohol use is discontinued (Howard et al., 2004).

In persons with diabetes, light-to-moderate amounts of alcohol (1 to 2 drinks per day; 15 to 30 g of alcohol) are associated with a decreased risk of coronary heart disease (Howard et al., 2004), perhaps because of the concomitant increase in HDL cholesterol and improved insulin sensitivity associated with alcohol consumption. Long-term, prospective studies are needed to confirm these observations (ADA, 2007) (see Chapter 32). In observational studies and in short-term studies, moderate amounts of alcohol did not increase triglyceride levels in hypertriglyceridemic individuals (Pownall et al., 1999) and had beneficial effects on blood pressure and triglyceride levels in postmenopausal women (Davies et al., 2002). Ingestion of light-to-moderate amounts of alcohol does not raise blood pressure; whereas excessive, chronic ingestion of alcohol does raise blood pressure and may be a risk factor for stroke (Chobanian et al., 2003). The contribution of alcohol to excessive energy intake in the overweight individual always has to be considered.

Micronutrients
No clear evidence has been established for benefits from routine vitamin or mineral supplements in persons with diabetes who do not have underlying deficiencies. Exceptions include folate for the prevention of birth defects (ADA, 2007). Since diabetes may be a state of increased oxidative stress, there has been interest in prescribing antioxidant vitamins in people with diabetes. Large observational studies and several pla-
Exercise consists of rhythmic, repeated, and continuous movement of one or more components of physical fitness. Aerobic exercise involves bodily movement performed to improve or maintain cardiovascular outcomes. Routine supplementation with antioxidants such as vitamins E and C and β-carotene is not advised because of lack of evidence of effectiveness and concern related to long-term safety (ADA, 2007).

Because the response to supplements is determined largely by a person’s nutrition status, persons with micronutrient deficiencies are most likely to respond favorably. Although difficult to ascertain, if deficiencies of vitamins or minerals are identified, supplementation can be beneficial. Those at greatest risk of deficiency who may benefit from prescription of vitamin and mineral supplements include patients who consume obviously poor diets or extreme calorie-restricted diets, strict vegetarians, older adults, pregnant or lactating women, those taking medication known to alter micronutrient metabolism (see Chapter 16), patients in poor metabolic control (glycosuria), and patients in critical care environments.

Several small studies have suggested a role for chromium supplementation in the management of glucose intolerance, gestational diabetes, body weight, and corticosteroid-induced diabetes. In two randomized, placebo-controlled studies in Chinese subjects with diabetes, chromium supplementation did have beneficial effects on glycemia (Anderson et al., 1997; Cheng et al., 1999); however, the study population may have had marginal baseline chromium status because the chromium status was not evaluated either at baseline or after supplementation. According to a recent FDA statement there is insufficient evidence to support any of the proposed health claims of chromium supplementation. The FDA concluded that, although a small study suggested that chromium picolinate may reduce the risk of insulin resistance, the existence of such a relationship between chromium picolinate and either insulin resistance or type 2 diabetes is highly uncertain. In addition, a metaanalysis of randomized controlled trials suggests no benefit of chromium picolinate supplementation in reducing body weight (Pittler et al., 2003). Therefore chromium supplementation is not recommended unless the individual’s intake of chromium does not meet the DRI of 20 to 25 mcg/day (ADA, 2006b).

**Physical Activity/Exercise**

Physical activity involves bodily movement produced by the contraction of skeletal muscles that requires energy expenditure in excess of resting energy expenditure. Exercise is a subset of physical activity: planned, structured, and repetitive bodily movement performed to improve or maintain one or more components of physical fitness. Aerobic exercise consists of rhythmic, repeated, and continuous movements of the same large muscle groups for at least 10 minutes at a time. Examples include walking, bicycling, jogging, swimming, and many sports. Resistance exercise consists of activities that use muscular strength to move a weight or work against a resistive load. Examples include weight lifting and exercises using resistance-providing machines.

Physical activity should be an integral part of the treatment plan for persons with diabetes. Exercise helps all persons with diabetes improve insulin sensitivity, reduce cardiovascular risk factors, control weight, and improve well-being (ADA, 2004a). Furthermore, regular exercise may prevent type 2 diabetes in high-risk individuals. Given appropriate guidelines, the majority of people with diabetes can exercise safely. The exercise plan will vary, depending on interest, age, general health, and level of physical fitness. Despite the increase in glucose uptake by muscles during exercise, glucose levels change little in individuals without diabetes. Muscular work causes insulin levels to decline while counterregulatory hormones (primarily glucagon) rise. In this way, increased glucose use by the exercising muscle is matched precisely with increased glucose production by the liver. This balance between insulin and counterregulatory hormones is the major determinant of hepatic glucose production, underscoring the need for insulin adjustments in addition to adequate carbohydrate intake during exercise for people with diabetes.

In persons with type 1 diabetes, the glycemic response to exercise may vary, depending on overall diabetes control, plasma glucose, and insulin levels at the start of exercise; timing, intensity and duration of the exercise; previous food intake; and previous conditioning. An important variable is the level of plasma insulin during and after exercise. Hypoglycemia can occur because of insulin-enhanced muscle glucose uptake by the exercising muscle. In contrast, insulin deficiency in a poorly controlled (underinsulinized) exercised results in increases in glucose concentrations, and free fatty acid release continues with minimum uptake. This can result in large increases in plasma glucose and ketone levels (Wasserman and Zinman, 1994).

In persons with type 2 diabetes, blood glucose control can improve with exercise, largely because of decreased insulin resistance and increased insulin sensitivity, which results in increased peripheral use of glucose not only during but also after the activity. This exercise-induced enhanced insulin sensitivity occurs independent of any effect on body weight (Boulé et al., 2001). Exercise also decreases the effects of counterregulatory hormones; this, in turn, reduces the hepatic glucose output, contributing to improved glucose control.

Exercise regimens at an intensity of 50% to 80% $V_{O2max}$ three to four times a week for 30 to 60 minutes a session can result in a 10% to 20% baseline improvement in A1C and are most beneficial in persons with mild type 2 diabetes and in those who are likely to be the most insulin resistant. Regular exercise also has consistently been shown to be effective in reducing triglyceride levels in persons with type 2 diabetes; however, the effect of exercise on HDL cholesterol levels is unclear. Reductions in blood pressure...
and improvements in impaired fibrinolysis have also been noted (Sigal et al., 2004).

Potential Problems With Exercise
Hypoglycemia is a potential problem associated with exercise in persons taking insulin or insulin secretagogues. Hypoglycemia can occur during, immediately after, or many hours after exercise. Hypoglycemia has been reported to be more common after exercise, especially exercise of long duration, strenuous activity or play, or sporadic exercise, than during exercise (MacDonald et al., 1987). This is because of increased insulin sensitivity after exercise and the need to replete liver and muscle glycogen, which can take up to 24 to 30 hours (see Chapter 23). Hypoglycemia can also occur during or immediately after exercise. Blood glucose levels before exercise reflect only the value at that time, and it is unknown if this is a stable blood glucose level or a blood glucose level that is dropping. If blood glucose levels are dropping before exercise, adding exercise can contribute to hypoglycemia during exercise. Furthermore, hypoglycemia on the day before exercise is reported to increase the risk of hypoglycemia on the day of exercise as well (Davis et al., 2000).

Hyperglycemia can also result from exercise. When a person exercises at what for him or her is a high level of exercise intensity, there is a greater-than-normal increase in counter-regulatory hormones. As a result, hepatic glucose release exceeds the rise in glucose use. The elevated glucose levels may also extend into the postexercise state (Purdon et al., 1993). Hyperglycemia and worsening ketosis can also result in persons with type 1 diabetes who are deprived of insulin for 12 to 48 hours and are ketotic. Vigorous activity should probably be avoided in the presence of ketosis (ADA, 2006b). The latter cause of hyperglycemia is not as likely to occur as the first.

Exercise Guidelines
The variability of glucose responses to exercise contributes to the difficulty in giving precise nutrition (and insulin) guidelines. Frequent blood glucose monitoring before, during, and after exercise helps individuals identify their response to physical activities. To meet their individual needs, patients must modify general guidelines to reduce insulin doses before (or after) or ingest carbohydrates after (or before) exercise.

Carbohydrate for Insulin or Insulin Secretagogue Users
During moderate-intensity exercise, glucose uptake is increased by 8 to 13 g/hr (Wasserman and Zinman, 1994), and this is the basis for the recommendation to add 15 g carbohydrate for every 30 to 60 minutes of activity (depending on the intensity) over and above normal routines. Moderate exercise for less than 30 minutes usually does not require any additional carbohydrate or insulin adjustment. Added carbohydrates should be ingested if preexercise glucose levels are less than 100 mg/dl (5.6 mmol/L). Supplementary carbohydrate is generally not needed in individuals who are not treated with insulin or insulin secretagogues (ADA, 2006b).

In all persons, blood glucose levels decline gradually during exercise, and ingesting a carbohydrate feeding during prolonged exercise can improve performance by maintaining the availability and oxidation of blood glucose. For the exerciser with diabetes whose blood glucose levels may drop sooner and lower than the exerciser without diabetes, ingesting carbohydrate after 40 to 60 minutes of exercise is important and may also assist in preventing hypoglycemia. Drinks containing 6% or less of carbohydrates empty from the stomach as quickly as water and have the advantage of providing both needed fluids and carbohydrates (see Chapter 23). Consuming carbohydrates immediately after exercise optimizes repletion of muscle and liver glycogen stores. For the exerciser with diabetes, this takes on added importance because of increased risk for late-onset hypoglycemia (Franz, 2002).

Insulin Guidelines
It is often necessary to adjust the insulin dosage to prevent hypoglycemia. This occurs most often with moderate to strenuous activity lasting more than 45 to 60 minutes. For most persons a modest decrease (of about 1 to 2 units) in the rapid- or short-acting insulin during the period of exercise is a good starting point. For prolonged vigorous exercise, a larger decrease in the total daily insulin dosage may be necessary. After exercise insulin may also need to be decreased. In addition to these acute reductions in insulin dosages, individuals who participate in a regular, long-term fitness program often find their usual total dosage of insulin decreasing by as much as 15% to 20% (Wasserman and Zinman, 1994).

Insulin doses should be reduced in anticipation of exercise after a meal, depending on the duration and intensity of the exercise. In persons with type 1 diabetes, Rabasa-Lhoret and colleagues (2001) validated that exercise at 25% VO_{2max} for 60 minutes required a 50% reduction in mealtime rapid-acting insulin and exercise at 50% VO_{2max} for 30 and 60 minutes required a 50% and 75% reduction in mealtime rapid-acting insulin, respectively. Such reductions in mealtime rapid-acting insulin for postprandial exercise resulted in a 75% decrease in exercise-induced hypoglycemia.

Precautions for Persons With Type 2 Diabetes
Persons with type 2 diabetes may have a lower VO_{2max} and therefore need a more gradual training program. Rest periods may be needed; this does not impair the training effect from physical activity. Autonomic neuropathy or medications, such as for blood pressure, may not allow for increased heart rate, and individuals must learn to use perceived exertion as a means of determining exercise intensity. Blood pressure may also increase more in persons with diabetes than in those who do not have diabetes, and exercise should not be undertaken if systolic blood pressure is greater than 180 to 200 mm Hg.

Exercise Prescription
The ADA recommends that a graded exercise test with electrocardiogram should seriously be considered before undertaking aerobic physical activity with intensity exceeding the
Medications

Glucose-Lowering Medications

The use of the newer glucose-lowering medications, alone or in combination, provides numerous options for achieving euglycemia in persons with type 2 diabetes. Some persons with hyperglycemia that is not adequately controlled by MNT alone can be treated with MNT and glucose-lowering medications—frequently combination therapy using two, and occasionally even three, medications. If glycemic control cannot be attained with MNT and glucose-lowering medications, insulin, either alone or in combination with other medications, is required.

The transition to insulin often begins with a long-acting or premixed insulin given at bedtime to control fasting glucose levels. However, eventually many patients with type 2 diabetes will require a more physiologic insulin regimen to achieve control. If large doses of insulin are required, oral medications such as insulin sensitizers are often combined with the insulin regimen. Recently two new injectable medications—exenatide and pramlintide—have become available for use in combination therapies. Although these two drugs have some similarities in action, they are recommended for use in different populations.

Currently four classes of oral medications exist: (1) insulin secretagogues, which include the sulfonylureas (first- and second-generation) and the meglitinides (repaglinide and nateglinide); (2) biguanides (metformin); (3) thiazolidinediones (TZDs) (e.g., pioglitazone, rosiglitazone); and (4) α-glucosidase inhibitors (acarbose, miglitol). Each class has a different mechanism of action: in the pancreas insulin secretion is stimulated; at the cellular level (muscle and adipose tissue) insulin resistance is decreased, and glucose uptake enhanced; in the liver hepatic glucose output is decreased, especially overnight, improving fasting glucose levels; or in the intestine glucose absorption is slowed, improving postprandial glucose concentrations. Because of the different sites of action, the medications can be used alone or in combination. Because combination therapy is so common, drug companies now market combination pills (Table 30-6).

Insulin secretagogues (sulfonylureas and meglitinides) promote insulin secretion by the β-cells of the pancreas. First- and second-generation sulfonylurea drugs differ from one another in their potency, pharmacokinetics, and metabolism. Disadvantages of their use include weight gain and the potential to cause hypoglycemia. The meglitinides differ from the sulfonylureas in that they have short metabolic half-lives, which result in brief episodic stimulation of insulin secretion. As a result, a frequent dosing schedule is required with meals, postprandial glucose excursions are less, and because less insulin is secreted several hours after a meal, there is a decreased risk of hypoglycemia between meals and overnight. Nateglinide only works in the presence of glucose and is a somewhat less potent secretagogue (Inzucchi, 2002).

Insulin sensitizers enhance insulin action and include biguanides (metformin) and TZDs. Both classes require the presence of insulin, exogenous or endogenous, to be effective. Metformin (Glucophage) suppresses hepatic glucose production and lowers insulin resistance, but it does not stimulate insulin secretion. It is not associated with hypoglycemia, may cause small weight losses when therapy begins, and improves lipid levels. Adverse effects include gastrointestinal distress such as abdominal pain, nausea, and diarrhea in up to 50% of patients. The frequency of these adverse effects can be minimized with food consumption and slow titration of dose. A rare side effect is severe lactic acidosis, which can be fatal. Acidosis usually occurs in patients who use alcohol excessively, have renal dysfunction, or have liver impairments (Inzucchi, 2002). Biguanides can be used alone or in combination with other diabetes medications. Other available agents include metformin extended release (Glucophage XR); a liquid metformin (Riomet); and metformin combined with the sulfonylurea gliburide (Glucovan), with the sulfonylurea glipizide (Metaglip), and with the TZD rosiglitazone (Avandamet).

The TZDs decrease insulin resistance in peripheral tissues and thus enhance the ability of muscle and adipose cells to take up glucose. TZDs also have certain lipid benefits. HDL cholesterol increases, and triglycerides frequently decrease with TZD therapy. Although LDL cholesterol may increase because of a shift from small and dense to large and buoyant LDL particles, these types of particles are less atherogenic; thus the increase in LDL cholesterol may not be a concern (Inzucchi, 2002). Adverse effects include weight gain and edema, and these effects are more common in patients who receive TZDs along with insulin. Patients with advanced forms of congestive heart disease or hepatic impairment should not receive TZDs. Troglitazone (Rezulin), the first approved TZD, was removed from the market because of hepatocellular injury. Rosiglitazone (Avandia) and pioglitazone (Actos) are the two TZD drugs currently available and have not been associated with liver injury. However, a
recent meta-analysis suggests that rosiglitazone may be associated with an increased risk of myocardial infarction in those who use it (Nissen and Wolski, 2007). This medication may become unavailable as further inquiry into its side effects is carried out. Also available is rosiglitazone combined with glimepiride (Avandaryl).

**Glucose-Lowering Medications for Type 2 Diabetes**

<table>
<thead>
<tr>
<th>Class and Generic Names</th>
<th>Recommended Dose</th>
<th>Principal Action</th>
<th>Mean Decrease in A1C</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sulfonylureas</strong> (Second-generation)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Glipizide (Glucotrol)</td>
<td>2.5-20 mg single or divided dose; single dose for XL</td>
<td>Stimulate insulin secretion from the β-cells</td>
<td>1% to 2%</td>
</tr>
<tr>
<td>Glipizide (Glucotrol XL)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Glyburide (Glynase Prestabs)</td>
<td>12 mg daily</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Glimepiride (Amaryl)</td>
<td>4-8 mg daily</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Meglitinide</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Repaglinide (Prandin)</td>
<td>0.5-4 mg before meals</td>
<td>Stimulate insulin secretion from the β-cells</td>
<td>1% to 2%</td>
</tr>
<tr>
<td>Nateglinide (Starlix)</td>
<td>120 mg before meals</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Biguanide</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Metformin (Glucophage)</td>
<td>500-850 mg tid or 1000 mg bid</td>
<td>Decrease hepatic glucose production</td>
<td>1.5% to 2%</td>
</tr>
<tr>
<td>Metformin Extended Release (Glucophage XR)</td>
<td>500-2000 mg once daily</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Thiazolidinediones</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pioglitazone (Actos)</td>
<td>15-45 mg daily</td>
<td>Improve peripheral insulin sensitivity</td>
<td>1% to 2%</td>
</tr>
<tr>
<td>Rosiglitazone (Avandia)</td>
<td>2-8 mg daily</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Alpha Glucosidase Inhibitors</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acarbose (Precose)</td>
<td>25-100 mg three times daily with meals</td>
<td>Delay carbohydrate absorption</td>
<td>0.5% to 1%</td>
</tr>
<tr>
<td>Miglitol (Glyset)</td>
<td>25-100 mg three times daily with meals</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Injectables**

There are two new injectable glucose-lowering medications. Exenatide (Byetta) is an incretin mimetic or incretin-like agent. Incretins are hormones (glucagon-like peptide-1 [GLP-1]) released during nutrient absorption from the cells of the gut and from pancreatic islet cells, which increase glucose-dependent insulin secretion, slow gastric emptying, decrease glucagon production, and decrease appetite. Exenatide is associated with reduction in A1C and modest weight loss (Kendall et al., 2005). It is approved for use in people with type 2 diabetes not achieving optimal glucose control with a sulfonylurea and/or metformin. Typically exenatide is injected twice a day, at breakfast and at the evening meal.

Pramlintide (Symlin) is a synthetic form of amylin, a hormone normally co-secreted with insulin by the β-cells in response to food intake and deficient in people with type 1 and type 2 diabetes. Pramlintide was developed to counter the effects of amylin deficiency and is given at each meal in addition to an insulin bolus. It is approved for use in adults with type 1 or type 2 diabetes who have not achieved optimal glucose control (Buse et al., 2002).

**Insulin**

Persons with type 1 diabetes depend on insulin to survive. In persons with type 2 diabetes, insulin may be needed to restore glycemia to near normal. Circumstances that require the use of insulin in type 2 diabetes include the failure to achieve adequate control with administration of oral medications; periods of acute injury, infection, or surgery; pregnancy; and allergy or serious reactions to sulfonylurea agents.

Insulin has three characteristics: onset, peak, and duration (Table 30-7). U-100 is the concentration of insulin available in the United States. This means it has 100 units of insulin per milliliter of fluid (100 units/ml). U-100 syringes deliver...
**TABLE 30-6**

**Glucose-Lowering Medications for Type 2 Diabetes—cont’d**

<table>
<thead>
<tr>
<th>Class and Generic Names</th>
<th>Recommended Dose</th>
<th>Principal Action</th>
<th>Mean Decrease in A1C</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Incretin Mimetics</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Exenatide (Byetta)</td>
<td>Initially dosed at 5 mcg twice a day—at breakfast and lunch; increased to 10 mcg twice a day</td>
<td>Enhances glucose-dependent insulin secretion and suppresses postprandial glucagon secretion</td>
<td>0.5% to 0.9%</td>
</tr>
<tr>
<td><strong>Amylinomimetic</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pramlintide (Smylin)</td>
<td>Initially dosed at 60 mcg before meals; dose increased directly to 120 mcg if no clinically significant nausea occurs after 3-7 days</td>
<td>Decreases glucagon production, which decreases mealtime hepatic glucose release and prevents postprandial hyperglycemia</td>
<td>0.4% to 0.7%</td>
</tr>
<tr>
<td><strong>Combination Medications</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Glyburide/metformin (Glucovance) (1.25 mg/250 mg)</td>
<td>2.5/500 mg to 5/500 mg daily</td>
<td>Combined action of each medication</td>
<td>≈2%</td>
</tr>
<tr>
<td>Glipizide/metformin (MetaGlip) (2.5 mg/250 mg)</td>
<td>2.5 mg/500 mg to 4 mg/500 mg daily</td>
<td>≈2%</td>
<td></td>
</tr>
<tr>
<td>Rosiglitazone/metformin (Avandamet) (1 mg/500 mg)</td>
<td>1 mg/500 mg to 4 mg/500 mg daily</td>
<td>≈2%</td>
<td></td>
</tr>
<tr>
<td>Rosiglitazone/glimepiride (Avandaryl) (4 mg/1, 2, or 4 mg)</td>
<td>4 mg/1.0 mg to 4 mg/4.0 mg daily</td>
<td>Unknown</td>
<td></td>
</tr>
</tbody>
</table>


**TABLE 30-7**

**Action Times of Human Insulin Preparations**

<table>
<thead>
<tr>
<th>Type of Insulin</th>
<th>Onset of Action</th>
<th>Peak Action</th>
<th>Usual Effective Duration</th>
<th>Monitor Effect In</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Rapid-Acting</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Insulin lispro (Humalog)</td>
<td>&lt;15 min</td>
<td>1-2 hr</td>
<td>3-4 hr</td>
<td>2 hr</td>
</tr>
<tr>
<td>Insulin aspart (NovoLog)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Insulin glulisine (Apidra)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Short-Acting</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Regular</td>
<td>0.5-1 hr</td>
<td>2-3 hr</td>
<td>3-6 hr</td>
<td>≈4 hr</td>
</tr>
<tr>
<td><strong>Intermediate-Acting</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NPH</td>
<td>2-4 hr</td>
<td>4-10 hr</td>
<td>10-16 hr</td>
<td>8-12 hr</td>
</tr>
<tr>
<td><strong>Long-Acting</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Insulin glargine (Lantus)</td>
<td>2-4 hr</td>
<td>Peakless</td>
<td>20-24 hr</td>
<td>10-12 hr</td>
</tr>
<tr>
<td>Insulin determir (Levemir)</td>
<td>2-4 hr</td>
<td>Peakless</td>
<td>18-24 hr</td>
<td>10-12 hr</td>
</tr>
<tr>
<td><strong>Mixtures</strong></td>
<td>70/30 (70% NPH, 30% regular)</td>
<td>0.5 to 1 hr</td>
<td>Dual</td>
<td>10 to 16 hr</td>
</tr>
<tr>
<td>75/25 (75% neutral protamine lispro [NPL], 25% lispro)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>70/30 (70% neutral protamine aspart [NPA], 30% aspart)</td>
<td></td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

U-100 insulin; however, insulin pens are now being used more frequently as an alternative to the traditional syringe-needle units.

**Rapid-acting insulins** include insulin lispro (Humalog), insulin aspart (Novolog), and insulin glulisine (Apidra) and are used as bolus (mealtime) insulins. They are insulin analogs that differ from human insulin in amino acid sequence but bind to insulin receptors and thus function in a manner similar to human insulin. All have an onset of action within 15 minutes, a peak in activity at 60 to 90 min, and a duration of action of 3 to 5 hours. They result in fewer hypoglycemic episodes compared with regular insulin.

**Regular** is a short-acting insulin with an onset of action 15 to 60 minutes after injection and a duration of action ranging from 5 to 8 hours. For best results the slow onset of regular insulin requires it to be taken 30 to 60 minutes before meals. NPH is the only available **intermediate-acting insulin** (Lente insulin has been discontinued). Its appearance is cloudy, and its onset of action is about 2 hours after injection, with a peak effect from 6 to 10 hours.

**Long-acting insulins** are insulin glargine (Lantus) and insulin determir (Levemir) (Ultralente has been discontinued). Insulin glargine is an insulin analog that because of its slow dissolution at the injection site results in a relatively constant and peakless delivery over 24 hours. Because of its acidic pH, it cannot be mixed with any other insulin in the same syringe before injection and is usually given at bedtime. However, glargine can be given before any meal, but, whichever time is chosen, it must be given consistently at that time. Insulin determir is absorbed from the subcutaneous tissue relatively quickly but then binds to albumin in the bloodstream, resulting in a prolonged action time of approximately 17 hours. Therefore it may need to be given twice a day. Basal insulin analogs decrease the chances of hypoglycemia, especially nocturnal hypoglycemia (Rosenstock et al., 2005).

**Premixed insulins** are also available: 70% NPH/30% regular, 75% lispro protamine (NPL [addition of neutral protamine to lispro to create an intermediate-acting insulin])/25% lispro, and 70% protamine (addition of neutral protamine to aspart to create an immediate-acting insulin)/30% aspart. Persons using premixed insulins must eat at specific times and be consistent in carbohydrate intake to prevent hypoglycemia.

Insulin administered via the pulmonary route (inhaled insulin) has been approved by the FDA and also may be used as bolus insulin. The very thin alveolar-capillary barrier in the lungs allows for rapid uptake of insulin into the bloodstream after inhalation, similar to rapid-acting analogs or even faster (Rave et al., 2005). The overall bioavailability of inhaled insulin is generally 10% to 15% of the total dose used. Exubera, involving a spray-dried insulin powder contained in a blister packet and a simple inhalation device, is currently approved, but other inhaled insulin systems are under development (Patton et al., 2004). Inhaled insulin may facilitate early introduction of insulin therapy to people who need it but are averse to insulin injections.

All persons with type 1 diabetes and those with type 2 diabetes who no longer produce adequate endogenous insulin need replacement of insulin that mimics normal insulin action. After individuals without diabetes eat, their plasma glucose and insulin concentrations increase rapidly, peak in 30 to 60 minutes, and return to basal concentrations within 2 to 3 hours. To mimic this, rapid-acting (or short-acting) insulin is given before meals, and this is referred to as bolus or mealtime insulin. Mealtime insulin doses are adjusted based on the amount of carbohydrate in the meal. An insulin-to-carbohydrate ratio can be established for an individual that will guide decisions on the amount of mealtime insulin to inject. Basal or background insulin dose is that amount of insulin required in the postabsorptive state to restrain endogenous glucose output primarily from the liver. Basal insulin also limits lipolysis and excess flux of free fatty acids to the liver. Long-acting insulins are used for basal insulin.

The type and timing of insulin regimens should be individualized, based on eating and exercise habits and blood glucose concentrations. For persons with type 2 diabetes, there are three primary insulin regimens: (1) long-acting insulin and oral glucose-lowering agents; (2) premixed insulin; and (3) a basal long-acting insulin with rapid-acting insulin for meals (Raskin et al., 2005). Options one and two may suffice for persons with type 2 diabetes who still have significant endogenous insulin production. However, for persons with type 1 diabetes and many patients with type 2 diabetes, a more physiologic insulin regimen such as option three is preferred (Figure 30-1).

These types of insulin regimens allow increased flexibility in the type and timing of meals. For normal-weight persons with type 1 diabetes, the required insulin dosage is about 0.5 to 1 unit/kg of body weight per day. About 50% of the total daily insulin dose is used to provide for basal or background insulin needs. The remainder (rapid-acting insulin) is divided among the meals either proportionately to the carbohydrate content or by giving about 1 to 1.5 units of insulin per 10 to 15 g of carbohydrates consumed. The larger amount is usually needed to cover breakfast carbohydrates as a result of the presence in the morning of higher levels of counterregulatory hormones (Rahasa-Lhoret et al., 1999). Persons with type 2 diabetes may require insulin doses in the range of 0.5 to 1.2 units/kg of body weight daily. Large doses, even more than 1.5 units/kg of body weight daily, may be required at least initially to overcome prevailing insulin resistance.

Insulin pump therapy provides basal rapid-acting or short-acting insulin pumped continuously by a mechanical device in micro amounts through a subcutaneous catheter that is monitored 24 hours a day. Both lispro and aspart work well in insulin pumps, resulting in improved glycemia and less hypoglycemia than with regular insulin (Bode and Strange, 2001). Boluses of the insulin are given before meals. Pump therapy requires a committed and motivated person who is willing to do a minimum of four blood glucose tests per day, keep blood glucose and food records, and learn the technical features of pump use.
Monitoring

Self-monitoring of blood glucose (SMBG) is used on a day-to-day basis to manage diabetes effectively and safely; however, laboratory measurement of glycated hemoglobin provides the best available index of overall diabetes control. The health care team, including the individual with diabetes, should work together to implement blood glucose monitoring and establish individual target blood glucose goals (see Table 30-4 for a listing of these goals). The frequency of monitoring depends on the type of diabetes and overall therapy.

Patients can perform SMBG up to eight times per day—before breakfast, lunch, and dinner; at bedtime; 1 to 2 hours after meals; and during the night or whenever needed to determine causes of hypoglycemia or hyperglycemia. For most patients with type 1 diabetes, SMBG is recommended four or more times a day, before each meal and at bedtime. SMBG in patients with type 2 diabetes should be sufficient to facilitate reaching glucose goals and is often performed one to four times a day; often before breakfast and before and 2 hours after the largest meal but only 3 or 4 days per week. When adding to or modifying therapy, type 1 and type 2 patients with diabetes should test more often than usual especially 2 hours after meals (ADA, 2004b).

Because the accuracy of SMBG is instrument and user dependent, it is important for health care providers to evaluate each patient's monitoring techniques, both initially and at regular intervals thereafter. Comparisons between results from patient self-testing in the clinic and simultaneous laboratory testing are useful to assess the accuracy of patient results. Meters now automatically convert the capillary whole-blood test to plasma glucose values so comparisons can readily be made with laboratory values.

It is important that the results of SMBG be written in a record book and that patients be taught how to adjust their management program based on these results. The first step in using such records is to learn how to identify patterns in blood glucose levels and how to adjust basic insulin doses. For example, if blood glucose levels are consistently (generally 3 days in a row) elevated at a specific testing time, adjustments are made in the insulin or medication acting at that time. Algorithms for insulin dose changes to compensate for an elevated or low glucose value also can be used. A commonly used formula determines the insulin sensitivity, or correction factor (CF), which defines how many milligrams per deciliter a unit of rapid- or short-acting insulin will lower blood glucose levels over a 2- to 4-hr period (Bode, 2004). The CF is determined by using the “1700 rule,” in which 1700 is divided by the total daily dose (TDD) of insulin. For example, if the TDD is 50 units of insulin, the CF = 1700/50 = 35. In this case, 1 unit of insulin should lower the patient’s blood glucose level by 35 mg/dl (2 mmol/L).

In using blood glucose monitoring records, it should be remembered that factors other than food affect blood glucose concentrations. An increase in blood glucose can be the result of insufficient insulin or insulin secretagogue; too much food; or increases in glucagon and other counterregulatory hormones as a result of stress, illness, or infection. Factors that contribute to hypoglycemia include too much insulin or insulin secretagogue, not enough food, unusual amounts of exercise, and skipped or delayed meals. Urine glucose testing, frequently used in the past, has so many limitations that it should not be used.

It is now possible to do continuous ambulatory blood glucose monitoring to determine 24-hour blood glucose patterns and to detect unrecognized hypoglycemia. One such system consists of a subcutaneous sensor that monitors interstitial glucose levels for up to 72 hours. Data can be downloaded in the physician’s office after completion of the prescribed cycle. Another device is worn on the wrist and can provide up to three glucose readings each hour for a maximum of 12 hours. It works through a process called reverse iontophoresis, in which a low-level electric current passes through intact skin and extracts glucose molecules.

Urine or blood testing can be used to detect ketones. Testing for ketonuria or ketonemia should be performed regularly during periods of illness and when blood glucose levels consistently exceed 240 mg/dl (13.3 mmol/L). The
presence of persistent, moderate, or large amounts of ketones, along with elevated blood glucose levels, requires insulin adjustments. Persons with type 2 diabetes rarely have ketosis; however, ketone testing should be done when the person is seriously ill.

Self-Management Education
Diabetes management is a team effort. Persons with diabetes must be at the center of the team because they have the responsibility for day-to-day management. RDs, nurses, physicians, and other health care providers contribute their expertise to developing therapeutic regimens that help the person with diabetes achieve the best metabolic control possible. The goal is to provide patients with the knowledge, skills, and motivation to incorporate self-management into their daily lifestyles.

Nutrition Therapy Interventions for Specific Populations

Medical Nutrition Therapy Interventions for Type 1 Diabetes
For persons requiring insulin therapy, the first priority is to integrate an insulin regimen into their usual eating habits and physical activity schedule. With the many insulin options now available (rapid- and long-acting insulins), an insulin regimen usually can be planned that will conform to an individual’s preferred meal routines and food choices (ADA, 2007; Franz et al., 2002a). It is no longer necessary to create unnatural or artificial divisions of meals and snacks.

Physiologic insulin regimens involve multiple injections (three or more insulin injections per day) or use of an insulin infusion pump and mimic natural insulin secretion. Approximately half of the required insulin dose is given as a basal or background insulin, and the other half is divided and given before meals (bolus or mealtime insulin). These types of insulin regimens allow increased flexibility in choosing when and what to eat. The total carbohydrate content of meals is the major determinant of the mealtime rapid-acting insulin dose and postprandial glucose response (Rabasa-Lhoret et al., 1999, DAFNE Study Group, 2002). Thus individuals can be taught how to adjust mealtime insulin doses based on the carbohydrate content of the meal. For persons who receive fixed insulin regimens such as with the use of premixed insulins or those who do not adjust their mealtime insulin doses, daily consistency in the timing and amount of carbohydrates eaten is recommended.

Attention must also be paid to total energy intake as well as carbohydrate intake. Weight gain has the potential to adversely affect glycemia, lipids, blood pressure, and general health; thus prevention of weight gain is desirable.

Medical Nutrition Therapy Interventions for Type 2 Diabetes
For persons with type 2 diabetes, the first priority is to adopt lifestyle interventions that improve the associated metabolic abnormalities of glycemia, dyslipidemia, and hypertension. Lifestyle interventions independent of weight loss that can improve glycemia include reduced energy intake and increased energy expenditure through physical activity. Because many persons also have dyslipidemia and hypertension, limited consumption of saturated and trans-fatty acids, cholesterol, and sodium is recommended. These interventions should be implemented as soon as the diagnosis of diabetes is made.

MNT interventions for established type 2 diabetes differ in several aspects from interventions for prevention. Because of the progressive nature of type 2 diabetes, MNT interventions progress from prevention of obesity, to the prevention or delay of type 2 diabetes, to strategies for improved metabolic control. Modest weight loss is beneficial in persons with insulin resistance, but, as the disease progresses to insulin deficiency, medications usually need to be combined with MNT. Emphasis should be on blood glucose control, improved food choices, increased physical activity, and moderate energy restriction rather than weight loss alone.

Teaching which foods are carbohydrates (fruits, grains, starchy vegetables, milk, sweets), average portion sizes, and how many servings to select at meals (and snacks, if desired) is the first step in food and meal planning. Limiting fats, especially saturated and trans-fats, encouraging physical activity, and using blood glucose monitoring to adjust food and eating patterns and medications are also important components of successful MNT for type 2 diabetes. Frequent follow-up with an RD can provide the problem-solving techniques, encouragement, and support that lifestyle changes require.

Physical activity improves insulin sensitivity, acutely lowers blood glucose in persons with diabetes and may also improve cardiovascular status; but by itself it has only a modest effect on weight. However, it is essential for long-term weight maintenance. Cardiorespiratory fitness in persons with diabetes appears to be more important than thinness in relation to all-cause and cardiovascular mortality (Church, 2004). During an average 14-year follow-up of about 2000 men with diabetes, fit men had greater longevity than unfit men, regardless of their body composition or risk factor status. Fitness had a strong and independent inverse association with mortality, independent of BMI and percentage of body fat. This again highlights the importance of counseling persons with diabetes to increase physical activity and fitness levels.

Weight-loss drugs may be beneficial in the treatment of overweight persons with type 2 diabetes and can help achieve a 5% to 10% weight loss when combined with lifestyle modifications. They should be used only in people with a BMI greater than 27. Gastric reduction surgery can be an effective weight-loss treatment for severely obese patients with type 2 diabetes and can result in marked im-
provenments in glycemias. However, it should be considered only in patients with a BMI greater than 35 because long-term benefits and risks of bariatric surgery in persons with diabetes have not been adequately studies (ADA, 2007) (see Chapter 21).

**Medical Nutrition Therapy Interventions for Children and Adolescents with Diabetes**

Involvement of a multidisciplinary team, including a physician, RD, nurse, and behavioral specialist, all trained in pediatric diabetes, is the best means of achieving optimal diabetes management in youth. However, the most important team members are the child or adolescent and his or her family.

**Type 1 Diabetes in Youth**

A major nutrition goal for children and adolescents with type 1 diabetes is maintenance of normal growth and development. Possible causes of poor weight gain and linear growth include poor glycemic control, inadequate insulin, and overrestriction of calories. The last may be a consequence of the common erroneous belief that restricting food, rather than adjusting insulin, is the way to control blood glucose. Other reasons unrelated to diabetes management include thyroid abnormalities and malabsorption syndromes. Excessive weight gain can be caused by excessive caloric intake, overtreatment of hypoglycemia, or overinsulization. Other causes include low physical activity levels and hypothyroidism (accompanied by poor linear growth) (Silverstein et al., 2005).

The nutrition prescription is based on the nutrition assessment. Newly diagnosed children often present with weight loss and hunger; as a result, the initial meal plan must be based on adequate calories to restore and maintain appropriate body weight. In about 4 to 6 weeks the initial caloric level may need to be modified to meet more usual caloric requirements. Nutrient requirements for children and adolescents with diabetes appear to be similar to those of children and adolescents without diabetes. The DRIs can be used to determine energy requirements (Institute of Medicine, 2002). However, it may be preferable to use a food and nutrition history of typical daily intake, providing that growth and development are normal, to determine an individual child’s or adolescent’s energy needs. Since energy requirements change with age, physical activity, and growth rate, an evaluation of height, weight, BMI, and the nutrition plan is recommended at least every year. Good metabolic control is essential for normal growth and development (for growth charts see Appendices 9 through 16). However, withholding food or having the child eat consistently without an appetite for food in an effort to control blood glucose should be discouraged. Calories should be adequate for growth and restricted if the child becomes overweight. Consultation with an RD to develop and discuss the medical nutrition plan is encouraged (Silverstein et al., 2005).

Individualized food and meal plans, insulin regimens using basal (background) and bolus (mealtime) insulins, and insulin algorithms or insulin pumps can provide flexibility for children with type 1 diabetes and their families. This approach accommodates irregular meal times and schedules and varying appetites and activity levels (ADA, 2007). Daily eating patterns in young children generally include three meals and two or three snacks, depending on the length of time between meals and the child’s physical activity level. Children often prefer smaller meals and snacks. Snacks can prevent hypoglycemia between meals and provide adequate calories. Older children and teens may prefer only three meals. Blood glucose monitoring data are then used to integrate an insulin regimen into the meal, snack, and exercise schedules.

Realistic blood glucose goals should be determined and discussed with the youth and family. Youth with diabetes are also more likely than their age- and sex-matched nondiabetic peers to be at risk for CVD. Therefore it is essential to reduce the risk factors in youth with type 1 diabetes. Lipid levels should be monitored regularly, and National Cholesterol Education Program treatment guidelines for children and adolescents should be followed (ADA, 2003b) (see Chapter 32).

After the appropriate nutrition prescription has been determined, the meal planning approach can be selected. A number of meal planning approaches can be used. Carbohydrate counting for food planning provides youth and their families with guidelines that facilitate glycemic control while still allowing the choice of many common foods that children and adolescents enjoy. However, whatever approach to food planning is used, the youth and family must find it understandable and applicable to their lifestyle. Blood glucose records are essential to assist the RD and other team members in making appropriate changes in insulin regimens.

**Type 2 Diabetes in Youth**

Childhood obesity has been accompanied by an increase in the prevalence of type 2 diabetes among children and adolescents. IGT has been shown to be highly prevalent in obese youth, irrespective of ethnic group, and is associated with insulin resistance. Once type 2 diabetes develops, β-cell failure is also a factor (Sinha et al., 2002). Thus type 2 diabetes in youth appears to follow a progressive pattern similar to type 2 diabetes in adults.

Successful lifestyle treatment of type 2 diabetes in children and adolescents involves cessation of excessive weight gain, promotion of normal growth and development, and the achievement of blood glucose and A1C goals (ADA, 2000). Nutrition guidelines should also address comorbidities such as hypertension and dyslipidemia. Behavior modification strategies to decrease intake of high-caloric, high-fat, and high-carbohydrate foods (e.g., extra-large desserts) and drinks (e.g., regular soda and other high sugar beverages) while encouraging healthy eating habits and regular physical activity for the entire family should be considered. Unfortunately successful lifestyle treatment regimens for youth with type 2 diabetes have not been defined (ADA, 2007). Dietary intake in a large cohort of youth with diabetes substantially failed to meet current
recommendations. Less than 50% met recommendations for total fat, fiber, fruits, vegetables, and grains (Mayer-Davis et al., 2006b). A multivitamin/mineral supplement with DRI amounts of chromium, magnesium, zinc and especially of vitamin D may be warranted.

Metformin is often used when lifestyle strategies alone have not achieved target glucose goals and has been shown to be safe and effective for the treatment of pediatric type 2 diabetes (Jones et al., 2002). Youth with type 2 diabetes may also require insulin therapy to achieve adequate glycemic control.

**Medical Nutrition Therapy Interventions for Pregnancy and Diabetes**

Normalization of blood glucose levels during pregnancy is very important for women who have preexisting diabetes or who develop GDM. MNT goals are to assist in achieving and maintaining optimal blood glucose control and to provide adequate maternal and fetal nutrition throughout pregnancy, energy intake for appropriate maternal weight gain, and necessary vitamins and minerals (ADA, 2007). Nutrition recommendations during pregnancy and lactation appear to be similar for women with and without diabetes; therefore the DRIs can be used to determine energy and nutrient requirements during pregnancy and for lactation (Institute of Medicine, 2002). Table 30-8 outlines blood glucose goals during pregnancy for preexisting diabetes and for GDM (ADA, 2006b; Jovanovic, 2000).

**Preexisting Diabetes and Pregnancy**

Preconception counseling and the ability to achieve near-normal blood glucose levels before pregnancy have been shown to be effective in reducing the incidence of anomalies in infants born to women with preexisting diabetes to nearly that of the general population.

As a result of hormonal changes during the first trimester, blood glucose levels are often erratic. Although caloric needs do not differ from those preceding pregnancy, the meal plan may need to be adjusted to accommodate the metabolic changes. Women should be educated about the increased risk of hypoglycemia during pregnancy and cautioned against overtreatment.

The need for insulin increases during the second and third trimesters of pregnancy. (This is why screening for GDM is done between weeks 24 and 28 of pregnancy.) At 38 to 40 weeks’ postconception, insulin needs and levels peak at two to three times prepregnancy levels. Pregnancy-associated hormones that are antagonistic to the action of insulin lead to an elevation of blood glucose levels. For women with preexisting diabetes, this increased insulin need must be met with increased exogenous insulin.

Meal plan adjustments are necessary to provide the additional calories required to support fetal growth, and weight should be monitored. During pregnancy the distribution of energy and carbohydrate intake should be based on the woman’s food and eating habits and blood glucose responses. Insulin regimens can be matched to food intake, but maintaining consistency of times and amounts of food eaten are essential to avoid hypoglycemia caused by the continuous fetal draw of glucose from the mother. Smaller meals and more frequent snacks are often needed. A late-evening snack is often necessary to decrease the likelihood of overnight hypoglycemia and fasting ketosis. Records of food intake and blood glucose values are essential for determining whether glycemic goals are being met and for preventing and correcting ketosis.

Regular follow-up visits are needed to also monitor caloric and nutrient intake, blood glucose control, and whether there is starvation ketosis. Urine or blood ketones during pregnancy may signal starvation ketosis that can be caused by inadequate energy or carbohydrate intake, omission of meals or snacks, or prolonged intervals between meals (e.g., more than 10 hours between the bedtime snack and breakfast). Ketonemia during pregnancy has been associated with reduced IQ scores in children, and women should be instructed to test for ketones periodically before breakfast.

**Gestational Diabetes Mellitus**

About 7% of all pregnancies are complicated by GDM, resulting in more than 200,000 cases annually (ADA, 2004c). After delivery about 90% of all women with GDM become normoglycemic but are at increased risk of developing GDM earlier in subsequent pregnancies and for developing type 2 diabetes. After pregnancy, 5% to 10% of women with gestational diabetes are found to have type 2 diabetes. Women who have had gestational diabetes have a 20% to 50% chance of developing diabetes in the next 5 to 10 years (Centers for Disease Control and Prevention, 2005). Lifestyle modifications aimed at reducing or preventing weight gain and increasing physical activity after pregnancy are recommended and can reduce the risk of subsequent diabetes.

Because fetal morbidity may be increased, a risk assessment for GDM should be done at the first prenatal visit, and women at high risk (those with marked obesity, previous history of GDM, glycosuria, or a strong family history of

**TABLE 30-8 Plasma Glucose Goals During Pregnancy**

<table>
<thead>
<tr>
<th>Test</th>
<th>Preexisting Diabetes (mg/dl)</th>
<th>Gestational Diabetes (mg/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fasting plasma glucose</td>
<td>65-100</td>
<td>65-95</td>
</tr>
<tr>
<td>Premeal</td>
<td>65-110</td>
<td></td>
</tr>
<tr>
<td>1 hr postprandial</td>
<td>&lt;=145</td>
<td>&lt;=140</td>
</tr>
<tr>
<td>2 hr postprandial</td>
<td>&lt;=135</td>
<td>&lt;=120</td>
</tr>
<tr>
<td>2 to 6 hr postprandial</td>
<td>65-135</td>
<td></td>
</tr>
</tbody>
</table>

diabetes) should be tested as soon as possible. An FPG of 126 mg/dl (7 mmol/L) or higher or a casual plasma glucose (CPG) of 200 mg/dl (11 mmol/L) meets the threshold for the diagnosis of diabetes and, if confirmed on a second day, requires no further testing (ADA, 2006b). High-risk women not found to have GDM at the initial screening and average risk women (a few women can be classified as low risk) should be tested between 24 to 28 weeks of gestation. An oral glucose challenge (which does not have to be preceded by fasting) with 50g glucose is performed, and an elevated plasma glucose level (>140 mg/dl [7.8 mmol/L]) 1 hour later is considered an indication of the need for diagnostic testing. The criteria for the diagnosis of GDM based on a 100-g OGT test are listed in Table 30-9. Low-risk women who do not need to be screened must meet all the following criteria: younger than 25 years of age; normal body weight; no family history of diabetes; no history of abnormal glucose tolerance; and not a member of an ethnic or racial group with a high prevalence of diabetes (ADA, 2006b).

MNT for GDM primarily involves a carbohydrate-controlled meal plan that promotes optimal nutrition for maternal and fetal health with adequate energy for appropriate gestational weight gain, achievement and maintenance of normoglycemia, and absence of ketosis. Specific nutrition and food recommendations are determined and modified based on individual assessment and blood glucose records. Monitoring blood glucose, fasting ketones, appetite, and weight gain can aid in developing an appropriate, individualized meal plan and in adjusting the meal plan throughout pregnancy.

Nutrition practice guidelines for gestational diabetes have been developed and field-tested (Reader et al., 2006). All women with GDM should receive MNT at diagnosis of GDM. Monitoring records guide nutrition therapy and are used to determine if insulin therapy is needed. Insulin therapy is added if glucose goals exceed target range (see Table 30-8) on two or more occasions in a 1- to 2-week period without some obvious explanation from food records or if glucose levels are consistently elevated because of patient's dietary indiscretions after MNT intervention. Weight gain or lack of weight gain and ketone testing can be useful in determining whether women are undereating to keep glucose levels within target range to avoid insulin therapy.

Carbohydrates should be distributed throughout the day into three small-to-moderate size meals and two to four snacks. All women require a minimum of 175 g of carbohydrates daily (Institute of Medicine, 2002). An evening snack is usually needed to prevent accelerated ketosis overnight. Carbohydrates are not as well tolerated at breakfast as they are at other meals because of increased levels of cortisol and growth hormones. To compensate for this, the initial food plan may have approximately 30 g of carbohydrate at breakfast. To satisfy hunger, protein foods, because they do not affect blood glucose levels as much, can be added.

Although caloric restriction must be viewed with caution, in obese women with GDM a 30% caloric restriction (an intake of about 1700 to 1800 kcal daily) may reduce hyperglycemia without ketonemia and reduce the rate of maternal weight gain. Intake below these levels is not advised. The pattern of weight gain during pregnancy for women with GDM should be similar to that of women without diabetes. Weight loss is not recommended for overweight and obese women with GDM; however, modest energy and carbohydrate restriction may be appropriate (ADA, 2007).

Exercise can also assist in overcoming peripheral resistance to insulin and in controlling fasting and postprandial hyperglycemia. It may be used as an adjunct to nutrition therapy to improve maternal glycemia. The ideal form of exercise is unknown, but a brisk walk after meals is often recommended.

Women with GDM should be encouraged to breast-feed because even a short duration of breast-feeding may retard the future onset of diabetes (McManus, 2001).

**Medical Nutrition Therapy Interventions for Older Adults**

The prevalence of diabetes and IGT increases dramatically as people age. Many factors predispose older adults to diabetes: age-related decreases in insulin production and

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**TABLE 30-9**

<table>
<thead>
<tr>
<th>Type of Test</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Screening during pregnancy—a 50-g oral glucose challenge (does not have to be fasting) at 24- to 28-weeks’ gestation</td>
<td>A plasma glucose level of 140 mg/dl (7.8 mmol/L) 1 hr later indicates the need for further diagnostic testing</td>
</tr>
<tr>
<td>Oral glucose tolerance test with an abnormal screen</td>
<td>After a 100-g oral glucose load, GDM may be diagnosed if two plasma glucose values equal or exceed:</td>
</tr>
<tr>
<td></td>
<td>Fasting: ≥95 mg/dl</td>
</tr>
<tr>
<td></td>
<td>1 hr: ≥180 mg/dl</td>
</tr>
<tr>
<td></td>
<td>2 hr: ≥155 mg/dl</td>
</tr>
<tr>
<td></td>
<td>3 hr: ≥140 mg/dl</td>
</tr>
</tbody>
</table>

increases in insulin resistance, adiposity, decreased physical activity, multiple prescription medications, genetics, and coexisting illnesses. A major factor appears to be insulin resistance. Controversy persists as to whether the insulin resistance is itself a primary change or whether it is attributable to reduced physical activity, decreased lean body mass (sarcopenia), and increased adipose tissue, which are all frequently seen in older adults. Furthermore, medications used to treat coexisting diseases may complicate diabetes therapy in older persons.

Despite the increase in glucose intolerance with age, aging per se should not be a reason for suboptimal control of blood glucose. Even if it is incorrectly assumed that preventing long-term diabetic complications is not relevant to the care of older adults, persistent hyperglycemia has deleterious effects on the body’s defense mechanisms against infection. It also increases the pain threshold by exacerbating neuropathic pain, and it has a detrimental effect on the outcome of cerebrovascular accidents.

Nutrition recommendations for older adults with diabetes must be extrapolated from what is known from the general population and should address nutrition-related cardiovascular risk factors common in older adults and encourage consumption of a variety of foods. Because of changes in body composition (loss of lean body mass) and exercise patterns, the energy requirements of older adults are 20% to 30% lower than those of younger adults (ADA, 2007). Physical activity can significantly reduce the decline in aerobic capacity that occurs with age, improve risk factors for atherosclerosis, slow the decline in age-related lean body mass, decrease central adiposity, and improve insulin sensitivity; thus it should be encouraged.

Malnutrition, osteoporosis, is often the more prevalent nutrition-related problem of older adults. It often remains subclinical or unrecognized because the result of malnutrition—excessive loss of lean body mass—resembles the signs and symptoms of the aging process. Until a primary disease develops or chronic problems are exacerbated by illness or some other stress, malnutrition may remain unrecognized. Both malnutrition and diabetes adversely affect wound healing and defense against infection, and malnutrition is associated with depression and cognitive deficits. The most reliable indicator of poor nutrition status in older adults is probably a change in body weight. In general, involuntary weight gain or loss of more than 10 pounds or 10% of body weight in less than 6 months indicates a need to evaluate whether the reason is nutrition related.

Because of concern over malnutrition, it is essential that older adults, especially those in long-term care settings, be provided a diet that meets their nutritional needs, enables them to attain or maintain a reasonable body weight, helps control blood glucose, and is palatable. The imposition of dietary restriction on older residents in long-term health facilities is not warranted. Residents should be served the regular (unrestricted) menu with consistency in the amount and timing of carbohydrates (ADA, 2007). A multivitamin/mineral supplement to meet the DRIs may be necessary.

In older adults acute hyperglycemia and dehydration can lead to a serious complication of diabetes: the hyperglycemic hyperosmolar state (HHS). Patients with HHS have a very high blood glucose level (ranging from 400 to 2800 mg/dl, [22.2-155.6 mmol/L] with an average of 1000 mg/dl [55.6 mmol/L]) without ketones. Patients are markedly dehydrated, and mental status often ranges from mild confusion to hallucinations or coma. Patients who have HHS have sufficient insulin to prevent lipolysis and ketosis. Treatment consists of hydration and small doses of insulin to control hyperglycemia.

THE NUTRITION CARE PROCESS

The nutrition care process (NCP) articulates the consistent and specific steps used to deliver MNT for diabetes. It is used to deliver MNT for some individuals in individual sessions and for others in group sessions. The NCP begins with developing rapport with the patient, and, whether provided individually or in groups, it involves a common process: nutrition assessment, nutrition diagnosis, nutrition intervention, and nutrition monitoring and evaluation (Lacey and Pritchett, 2003). When MNT is implemented individually, the intervention is individualized based on the needs of the patient and whether the intervention is for initial, continuing, or intensive care (see Chapter 17).

Providing nutrition interventions in groups is becoming increasingly more important, since reimbursement criteria for diabetes self-management education and for MNT recommend that, when possible, group sessions are preferable. It is helpful if the group participants are similar in their stage of diabetes management and if they all speak and understand the same language. Group education shifts more responsibility to the patient to provide the needed initial assessment information, to evaluate outcomes, and to decide about therapy changes (Franz et al, 2002b). However, group interventions must also allow for individualization of MNT and evaluation of outcomes. When compared with individual interventions, group interventions for diabetes self-management education have produced similar positive outcomes (Rickheim et al., 2002).

Nutrition Assessment

The nutrition assessment involves obtaining information before and during the encounter needed to identify nutrition-related problems. Within 30 days before the first encounter, the RD should obtain pertinent clinical data from the referral source or patient record or information system. Box 30-2 provides a summary of assessment data—minimum referral data and parameters to be assessed to develop an individualized nutrition care plan. Some of this information can be gathered from a patient questionnaire. By collecting the data before the first session, completion of the assessment and implementation of interventions can begin efficiently.

It is essential to learn about the patient’s lifestyle and eating habits. Food and eating histories can be done several ways, with the objective being to determine a schedule and
**CHAPTER 30 | Medical Nutrition Therapy for Diabetes Mellitus and Hypoglycemia of Nondiabetic Origin  791**

**Box 30-2**

**Nutrition Assessment**

**Minimum Referral Data Needed Before First Encounter**

- **Diabetes treatment regimen:** nutrition therapy alone; nutrition therapy and glucose-lowering medications; nutrition therapy and insulin (physiologic or fixed regimen); nutrition therapy and combination medications (type[s])
- **Laboratory data:** A1C, date of test; fasting or nonfasting plasma glucose; lipid fractionation (cholesterol, low-density lipoprotein and high-density lipoprotein cholesterol, triglycerides); urinary microalbumin (albumin-to-creatinine ratio); blood pressure
- **Goals for patient care:** target blood glucose levels (premeal and postmeal); target A1C; method and frequency of self-monitoring of blood glucose; plans for instruction and evaluation of glucose monitoring
- **Medical history:** other pertinent diagnoses—cardiovascular disease, hypertension, renal disease, autonomic neuropathy, especially gastrointestinal synchronies; (5) frequency with which meals are eaten in restaurants; (6) who usually prepares food; (7) eating problems (e.g., as related to dental, gastrointestinal or other problems); (8) alcoholic beverage intake; and (9) supplements used (see Chapter 18). It is also essential to learn about the patient’s daily routine and schedule. The following information is needed: (1) time of waking; (2) usual meal and eating times; (3) work schedule or school hours; (4) type, amount, and timing of exercise; and (5) usual sleep habits.

**Data to Be Obtained From a Food and Nutrition Assessment**

- **Anthropometric measures:** weight; height (for adults at initial visit and for youth at every visit); body mass index; waist circumference
- **Diabetes history:** previous diabetes education, use of blood glucose monitoring, diabetes problems/concerns (hypoglycemia, hyperglycemia, fear of insulin)
- **Food and nutrition history:** 24-hr recall or typical day’s intake; meal and snack eating times; schedule changes; travel frequency; exercise routine/sports (type, amount, and time of exercise); usual sleep habits; appetite/gastrointestinal issues; food allergies/food intolerance; alcohol use; weight history, weight goals
- **Social history:** occupation, hours worked away from home, living situation, financial issues
- **Medications/supplements:** medications taken, vitamin/mineral/supplement use, herbal supplements
- **Knowledge base:** motivation to change; readiness to change


pattern of eating that will be the least disruptive to the lifestyle of the individual with diabetes and, at the same time, will facilitate improved metabolic control. With this objective in mind, asking the individual either to record or report what, how much, and when he or she typically eats during a 24-hour period may be the most useful.

Another approach is to ask the patient to keep and bring a 3-day or 1-week food intake record. This request can be made when an appointment with the RD is scheduled. Assessment of the most typical daily pattern can then be made. The history can also reveal other useful information, including (1) usual caloric intake; (2) quality of the usual diet; (3) times, sizes, and contents of meals and snacks; (4) food idiosyncrasies; (5) frequency with which meals are eaten in restaurants; (6) who usually prepares food; (7) eating problems (e.g., as related to dental, gastrointestinal or other problems); (8) alcoholic beverage intake; and (9) supplements used (see Chapter 18). It is also essential to learn about the patient’s daily routine and schedule. The following information is needed: (1) time of waking; (2) usual meal and eating times; (3) work schedule or school hours; (4) type, amount, and timing of exercise; and (5) usual sleep habits.

**Nutrition Diagnosis**

The nutrition diagnosis describes factors in the patients’ nutrition status that RDs are responsible for treating independently (see Chapter 17). A nutrition diagnosis is written in the PES format that states the problem (P), the etiology (E), and signs and symptoms (S). Examples of P related to diabetes are listed in Box 30-3. The following are examples of nutrition diagnosis statements:

- Altered blood glucose values (P) related to insufficient insulin (E) as evidenced by hyperglycemia despite excellent eating habits (S)
- Inconsistent carbohydrate intake (P) related to inconsistent timing of meals (E) as evidenced by wide fluctuations in blood glucose levels (S)
- Inappropriate intake of food fats (P) related to inadequate knowledge (E) as evidenced by high intake of foods containing saturated fats (S)

**Nutrition Intervention**

Nutrition interventions include two distinct processes: planning nutrition interventions and implementing the nutrition intervention. An individualized food and meal plan and behavioral goals focused on the etiology of the problem are developed, along with a statement of specific expected outcomes and timeline for each. A list of materials is provided with referrals or resources used and recommendations to other health care team members to reinforce nutrition and physical activity goals, recheck laboratory data, and reevaluate medications and/or doses.

**Developing a Food/Meal Plan**

Using the assessment data and food and nutrition history information, a preliminary food and meal plan can then be designed, and, if the patient desires, sample menus provided.
Developing a food and meal plan does not begin with a set calorie or macronutrient prescription; instead, it is determined by modifying the patient’s usual food intake as necessary. The worksheet in Figure 30-2 can be used to record the usual foods eaten and to modify the usual diet as necessary. The macronutrient and caloric values for the exchange lists are listed on the form and in Table 30-10.

See Appendix 34 for portion sizes of the foods on the exchange lists. These tools are useful in evaluating nutrition assessments. Using the form in Figure 30-2, the RD begins by totaling the number of exchanges from each list and multiplying this number by the grams of carbohydrate, protein, and fat contributed by each. Next the grams of carbohydrate, protein, and fat are totaled from

### BOX 30-3

**Examples of Nutrition Diagnoses (Problems) Related to Diabetes Mellitus**

<table>
<thead>
<tr>
<th>Inadequate energy intake</th>
<th>Food medication interaction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Excessive energy intake</td>
<td>Underweight</td>
</tr>
<tr>
<td>Excessive alcohol intake</td>
<td>Involuntary weight loss</td>
</tr>
<tr>
<td>Excessive fat intake</td>
<td>Overweight/obesity</td>
</tr>
<tr>
<td>Inappropriate intake of food fats—specify</td>
<td>Excessive weight gain</td>
</tr>
<tr>
<td>Excessive protein intake</td>
<td>Involuntary weight-related knowledge deficit</td>
</tr>
<tr>
<td>Inadequate carbohydrate intake</td>
<td>Not ready for diet/lifestyle change</td>
</tr>
<tr>
<td>Excessive carbohydrate intake</td>
<td>Limited adherence to nutrition-related recommendations</td>
</tr>
<tr>
<td>Inappropriate intake of types of carbohydrate—specify</td>
<td>Physical inactivity</td>
</tr>
<tr>
<td>Inconsistent carbohydrate intake</td>
<td>inability or lack of desire to manage self-care</td>
</tr>
<tr>
<td>Inadequate fiber intake</td>
<td>Impaired ability to prepare foods/meals</td>
</tr>
<tr>
<td>Altered GI function</td>
<td>Involuntary weight loss</td>
</tr>
<tr>
<td>Inappropriate intake of types of carbohydrate—specify</td>
<td>Overweight/obesity</td>
</tr>
<tr>
<td>Inconsistent carbohydrate intake</td>
<td>Disordered eating pattern</td>
</tr>
<tr>
<td>Inadequate fiber intake</td>
<td>Limited adherence to nutrition-related recommendations</td>
</tr>
<tr>
<td>Altered nutrition-related laboratory value (i.e., glucose)</td>
<td>Physical inactivity</td>
</tr>
<tr>
<td>Altered nutrition-related laboratory value (i.e., glucose)</td>
<td>Inability or lack of desire to manage self-care</td>
</tr>
<tr>
<td>Altered nutrition-related laboratory value (i.e., glucose)</td>
<td>Impaired ability to prepare foods/meals</td>
</tr>
</tbody>
</table>


### TABLE 30-10

**Macronutrient and Caloric Values for Exchange Lists**

<table>
<thead>
<tr>
<th>Groups/Lists</th>
<th>Carbohydrate (g)</th>
<th>Protein (g)</th>
<th>Fat (g)</th>
<th>Calories</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Carbohydrate Group</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Starch</td>
<td>15</td>
<td>3</td>
<td>0-1</td>
<td>80</td>
</tr>
<tr>
<td>Fruit</td>
<td>15</td>
<td>—</td>
<td>—</td>
<td>60</td>
</tr>
<tr>
<td>Milk</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Skim</td>
<td>12</td>
<td>8</td>
<td>0-3</td>
<td>90</td>
</tr>
<tr>
<td>Reduced-fat</td>
<td>12</td>
<td>8</td>
<td>5</td>
<td>120</td>
</tr>
<tr>
<td>Whole</td>
<td>12</td>
<td>8</td>
<td>8</td>
<td>150</td>
</tr>
<tr>
<td>Other carbohydrates</td>
<td>15</td>
<td>Varies</td>
<td>Varies</td>
<td>Varies</td>
</tr>
<tr>
<td>Vegetables</td>
<td>5</td>
<td>2</td>
<td>—</td>
<td>25</td>
</tr>
<tr>
<td><strong>Meat and Meat Substitute Group</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Very lean</td>
<td>—</td>
<td>7</td>
<td>0-1</td>
<td>35</td>
</tr>
<tr>
<td>Lean</td>
<td>—</td>
<td>7</td>
<td>3</td>
<td>55</td>
</tr>
<tr>
<td>Medium-fat</td>
<td>—</td>
<td>7</td>
<td>5</td>
<td>75</td>
</tr>
<tr>
<td>High-fat</td>
<td>—</td>
<td>7</td>
<td>8</td>
<td>100</td>
</tr>
<tr>
<td><strong>Fat Group</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>—</td>
<td>—</td>
<td>5</td>
<td>45</td>
</tr>
</tbody>
</table>

*See Appendix 34.*
each column; the grams of carbohydrates and protein are then multiplied by 4 (4 kcal/g of carbohydrates and protein), and the grams of fat are multiplied by 9 (9 kcal/g of fat). Total calories and percentage of calories from each macronutrient can then be determined. Numbers derived from these calculations are then rounded off. Figure 30-3 provides an example of a preliminary food and meal plan. In this example the nutrition prescription would be the following: 1900 to 2000 calories, 230 g of carbohydrates (50%), 90 g of protein (20%), 65 g of fat (30%).

The number of carbohydrate choices for each meal and snack is the total of the starch, fruit, and milk servings. Vegetables, unless starchy or eaten in very large amounts (three or more servings per meal), are generally considered “free foods.” The carbohydrate choices are circled under each meal and snack column. Table 30-11 is an example of a sample meal plan and menu based on Figure 30-3.

The next step is to evaluate the preliminary meal plan. First and foremost, does the patient think it is feasible to implement the meal plan into his or her lifestyle? Second, is it appropriate for diabetes management? Third, does it encourage healthful eating?

To answer the first question concerning the feasibility of the food plan, the food and meal plan is reviewed with the patient in terms of general food intake. Timing of meals and snacks and approximate portion sizes and types of foods discussed. Later a meal-planning approach can be selected that will assist the patient in making his or her own food choices. At this point it needs to be determined whether this meal plan is reasonable for the patient with diabetes. To determine the appropriateness of the meal plan for diabetes management involves assessing if the distribution of the meals (and snacks, if desired) is appropriate based on the types of medications prescribed and treatment goals. Methods for determining caloric requirements are only approximate. Adjustments in calories can be made during follow-up visits. Parameters that should be taken into consideration are weight changes, feelings of satiety and hunger, and concerns about palatability.

For patients with type 2 diabetes receiving MNT alone, often the food and meal plan begins with three or four carbohydrate servings per meal for adult women and four or five for adult men and, if desired, one or two for a snack. Blood glucose monitoring before the meal and 2 hours after the meal is recommended. Results of the blood glucose monitoring and feedback from the patient are used to assess if these recommendations are feasible and realistic and to determine if target glucose goals are being achieved.

For patients who require insulin, the timing of eating is extremely important. Food consumption must be synchronized with the time actions of insulin (see “Medications” earlier in the chapter). If the eating pattern is determined

![Table 30-11](image)

**FIGURE 30-2** Worksheet for assessment and design of a meal or food plan.  
CHO, Carbohydrate.
first, an insulin regimen can be selected that will fit with it. To prevent overnight hypoglycemia, some patients may require a bedtime snack.

The best way to ensure that the meal plan encourages healthful eating is to encourage patients to eat a variety of foods from all the food groups. The Dietary Guidelines for Americans, with its suggested number of servings from each food group, can be used to compare the patient’s meal plan with the nutrition recommendations for all Americans (see Chapter 12).

Self-Management Training
This step involves selecting an appropriate meal-planning approach and identifying strategies for behavioral change that enhance motivation and adherence to necessary lifestyle changes. A number of meal-planning approaches are available, ranging from simple guidelines or menus to more complex counting methods (Table 30-12). No single meal-planning approach has been shown to be more effective than any other, and the meal-planning approach selected should allow individuals with diabetes to select appropriate foods for meals and snacks.

A popular approach to meal planning is carbohydrate counting. It can be used as a basic meal-planning approach or for more intensive management. Carbohydrate-counting educational tools are based on the concept that, after eating it is the carbohydrate in foods that is the major predictor of postprandial blood glucose levels. One carbohydrate serving contributes 15 g of carbohydrates. Basic carbohydrate counting emphasizes the following topics: basic facts about carbohydrates, primary food sources of carbohydrate, average portion sizes and the importance of consistency and measuring portions, amount of carbohydrates they should be eating, and label reading. Advanced carbohydrate counting emphasizes the importance of record keeping, pattern management, and calculating insulin-to-carbohydrate ratios.

Facilitating Behavioral Changes and Goal Setting
Optimal self-management of diabetes requires changes in existing behaviors in addition to the adoption of new ones. Successful behavioral change requires comprehensive education, skill development, and motivation. The transtheoretical model was proposed by Prochaska as a general model of intentional behavior change (Prochaska et al., 1994). It includes a sequence of stages along a continuum of behavioral change. Different intervention strategies may be needed for individuals at different stages of the change process. Motivational interventions may work best with patients in the earlier contemplative stages, whereas specific skill-training interventions may be most appropriate for persons who have decided to change. Relapse and recycling through the stages occur quite frequently as patients attempt to modify behaviors (see Chapter 19).

<table>
<thead>
<tr>
<th>Food Group</th>
<th>Breakfast</th>
<th>Snack</th>
<th>Lunch</th>
<th>Snack</th>
<th>Dinner</th>
<th>Snack</th>
<th>Total servings/day</th>
<th>CHO (g)</th>
<th>Protein (g)</th>
<th>Fat (g)</th>
<th>Calories</th>
</tr>
</thead>
<tbody>
<tr>
<td>Starches</td>
<td>2</td>
<td>1</td>
<td>2–3</td>
<td>1</td>
<td>2–3</td>
<td>1–2</td>
<td>10</td>
<td>15</td>
<td>3</td>
<td>1</td>
<td>80</td>
</tr>
<tr>
<td>Fruit</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>0–1</td>
<td>3</td>
<td></td>
<td>12</td>
<td>15</td>
<td>45</td>
<td></td>
<td>60</td>
</tr>
<tr>
<td>Milk</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>2</td>
<td></td>
<td></td>
<td>12</td>
<td>24</td>
<td>16</td>
<td>2</td>
<td>90</td>
</tr>
<tr>
<td>Vegetables</td>
<td>✓</td>
<td>✓</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>5</td>
<td>10</td>
<td>2</td>
<td>4</td>
<td>25</td>
</tr>
<tr>
<td>Meats/Substitutes</td>
<td>2–3</td>
<td>3–4</td>
<td>6</td>
<td>5</td>
<td>150</td>
<td></td>
<td>42</td>
<td>5(3)</td>
<td>30</td>
<td></td>
<td>75(55)</td>
</tr>
<tr>
<td>Fats</td>
<td>1</td>
<td>0–1</td>
<td>1–2</td>
<td>0–1</td>
<td>1–2</td>
<td>0–1</td>
<td>5</td>
<td></td>
<td>5</td>
<td></td>
<td>45</td>
</tr>
<tr>
<td>CHO Choices</td>
<td>1–2</td>
<td>1–2</td>
<td>1–2</td>
<td>1–2</td>
<td>1–2</td>
<td>1–2</td>
<td>5</td>
<td></td>
<td>5</td>
<td></td>
<td>45</td>
</tr>
</tbody>
</table>

FIGURE 30-3 An example of a completed worksheet from the assessment, the nutrition prescription, and a sample 1900- to 2000-calorie meal plan. CHO, Carbohydrate.
Short-term goals (days or weeks) are often behavioral goals and relate to lifestyle changes. Common self-management behavioral goals are consistent and appropriate carbohydrate servings, regular physical activity, correct medication dosage (if needed), and blood glucose monitoring as determined to be needed. Goals should be specific, realistic for the patient, and written in behavioral language.

**Nutrition Monitoring and Evaluation**

Before the patient leaves the session, plans for the next appointment should be identified. A timeline should be established for follow-up visits to monitor and evaluate responses to nutrition interventions. The patient is also given information on how to call or e-mail with questions and concerns. In making plans for the next encounter, the patient is asked to keep a 3- or 7-day or weekly food record with blood glucose-monitoring data.

Medical and clinical outcomes should be monitored after the second or third visit to determine whether the patient is making progress toward established goals. If no progress is evident, the individual and RD need to reassess and perhaps revise the nutrition care plan. If altering food intake alone is not achieving metabolic target ranges, the RD should recommend that medications be added or adjusted.

Documentation is essential for communication and reimbursement. An initial progress note is documented in the patient's medical record and information system according to the organization’s policy, and a copy of the progress note is sent to the referral source. Box 30-4 lists some of the areas of the nutrition intervention that require documentation (see Chapter 17).

**Follow-Up Encounters**

Successful nutrition therapy involves a process of assessment, problem solving, adjustment, and readjustment. Food records can be compared with the meal plan, which will help to determine whether the initial meal plan needs changing, and can be integrated with the blood glucose-monitoring records to determine changes that can lead to improved glycemic control. For patients receiving oral medications or insulin, it can then be determined whether blood glucose values that are outside target ranges can be corrected with adjustments in the meal plan or whether adjustments in medications are needed.

The knowledge and skills needed to implement nutritional recommendations cannot be acquired in one session; therefore continued nutrition education is essential and must be an ongoing component of diabetes care. After the basic food and nutrition interventions have been mastered, other aspects of nutrition education should be presented to increase flexibility in food choices and lifestyle while still maintaining glucose control (Box 30-5). Of particular importance is information about eating out and the use of information from food labels. Persons using insulin also need information about how to make adjustments in food intake or insulin when schedules are disrupted.

Nutrition follow-up visits should provide encouragement and ensure realistic expectations for the patient.

---

**TABLE 30-11**

**Sample Menu for 1900-2000 Kilocalorie Meal Plan**

<table>
<thead>
<tr>
<th>Meal/Timing</th>
<th>Food Selections</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Breakfast—7:30 AM</strong></td>
<td>Raisin bran cereal, ½ c</td>
</tr>
<tr>
<td>3–4 Carbohydrate choices (i.e., 2 starch, 1 fruit, 1 milk)</td>
<td>Bagel, ¼ (1 oz)</td>
</tr>
<tr>
<td>1 Fat</td>
<td>Cantaloupe (5-inch), ½</td>
</tr>
<tr>
<td></td>
<td>Skim milk, 1 c</td>
</tr>
<tr>
<td></td>
<td>Reduced-fat cream cheese, 1 Tbsp</td>
</tr>
<tr>
<td><strong>Snack—10.00 AM</strong></td>
<td>Bagel, ¼ (1 oz)</td>
</tr>
<tr>
<td>1 Carbohydrate choice (i.e., 1 starch or fruit)</td>
<td>Reduced-fat cream cheese, 1 Tbsp</td>
</tr>
<tr>
<td>0-1 Fat</td>
<td></td>
</tr>
<tr>
<td><strong>Lunch—Noon</strong></td>
<td>Whole-wheat bread, 2 slices</td>
</tr>
<tr>
<td>3–4 Carbohydrate choices (i.e., 2-3 starches, 1 fruit)</td>
<td>Vegetable-beef soup, 1 c</td>
</tr>
<tr>
<td></td>
<td>Apple, 1 small</td>
</tr>
<tr>
<td></td>
<td>Lettuce and tomato slices</td>
</tr>
<tr>
<td></td>
<td>Turkey, 2 oz</td>
</tr>
<tr>
<td></td>
<td>Reduced-fat mayonnaise, 1 Tbsp</td>
</tr>
<tr>
<td><strong>Vegetable</strong></td>
<td>Pretzels, ¼ oz</td>
</tr>
<tr>
<td>2-3 Meats</td>
<td></td>
</tr>
<tr>
<td>1-2 Fats</td>
<td></td>
</tr>
<tr>
<td><strong>Snack—3:00 PM</strong></td>
<td></td>
</tr>
<tr>
<td>1 Carbohydrate choice (i.e., 1 starch or fruit)</td>
<td>Baked potato, 1 medium</td>
</tr>
<tr>
<td>0-1 Fat</td>
<td>Dinner roll, 1</td>
</tr>
<tr>
<td><strong>Dinner—6:30 PM</strong></td>
<td>Mandarin oranges, ¼ c</td>
</tr>
<tr>
<td>4-5 Carbohydrate choices (i.e., 2-3 starch, 1 fruit, 1 milk)</td>
<td>Skim milk, 1 c</td>
</tr>
<tr>
<td></td>
<td>Broccoli spears, ½ c</td>
</tr>
<tr>
<td></td>
<td>Chicken breast, baked, 3 oz</td>
</tr>
<tr>
<td></td>
<td>Sour cream, regular, 2 Tbsp</td>
</tr>
<tr>
<td></td>
<td>Reduced-fat salad dressing, 2 Tbsp</td>
</tr>
<tr>
<td><strong>Vegetables</strong></td>
<td>Ice cream, light ½ c</td>
</tr>
<tr>
<td>3-4 Meats</td>
<td>Strawberries, 1¼ c</td>
</tr>
<tr>
<td>1-2 Fats</td>
<td></td>
</tr>
<tr>
<td><strong>Snack—10:00 PM</strong></td>
<td></td>
</tr>
</tbody>
</table>
## Table 30-12

<table>
<thead>
<tr>
<th>Food-Planning Approaches for Diabetes</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Approach</strong></td>
</tr>
<tr>
<td>Diabetes nutrition guidelines</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Menu approaches</td>
</tr>
<tr>
<td>Carbohydrate counting</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Exchange list approaches</td>
</tr>
</tbody>
</table>

A change in eating habits is not easy for most people, and they become discouraged without appropriate recognition of their efforts. Patients should be encouraged to speak freely about problems they are having with food and eating patterns. Furthermore, there may be major life changes that require changes in the meal plan. Job and schedule changes, travel, illness, and other factors all have an impact on the meal plan.

**Acute Complications**

Hypoglycemia and diabetic ketoacidosis are the two most common acute complications related to diabetes.

**Hypoglycemia**

A low blood glucose, or hypoglycemia (or insulin reaction), is a common side effect of insulin therapy, although patients taking insulin secretagogues can also be affected. Autonomic symptoms arise from the action of the autonomic nervous system and are often the first signs of mild hypoglycemia. Adrenergic symptoms include shakiness, sweating, palpitations, anxiety, and hunger. Neuroglycopenic symptoms, related to an insufficient supply of glucose to the brain, can also occur at similar glucose levels as autonomic symptoms but with different manifestations. The earliest signs of neuroglycopenia include a slowing down in performance and difficulty concentrating and reading. As blood glucose levels drop further, the following symptoms occur: frank mental confusion and disorientation, slurred or rambling speech, irrational or unusual behaviors, extreme fatigue and lethargy, seizures, and unconsciousness. Symptoms differ for different people but tend to be consistent from episode to episode for any one person.

Several common causes of hypoglycemia are listed in Box 30-6. In general, glucose of 70 mg/dl or lower should be treated immediately (Cryer et al., 2003). Treatment of hypoglycemia requires ingestion of glucose or carbohydrate-containing food. Although any carbohydrate will raise glucose levels, glucose is the preferred treatment. Commercially
Hyperglycemia can lead to diabetic ketoacidosis (DKA), a life-threatening but reversible complication characterized by severe disturbances in carbohydrate, protein, and fat metabolism. DKA is always the result of inadequate insulin for glucose use. As a result, the body depends on fat for energy, and ketones are formed. Acidosis results from increased production and decreased use of acetoacetic acid and 3-β-hydroxybutyric acid from fatty acids. These ketones spill into the urine; hence the reliance on testing for ketones.

DKA is characterized by elevated blood glucose levels (>250 mg/dl but generally <600 mg/dl) and the presence of ketones in the blood and urine. Symptoms include polyuria, polydipsia, hyperventilation, dehydration, the fruity odor of ketones, and fatigue. SMBG, testing for ketones, and medical intervention can all help prevent DKA. If left untreated, DKA can lead to coma and death. Treatment includes supplemental insulin, fluid and electrolyte replacement, and medical monitoring. Acute illnesses such as flu, colds, vomiting, and diarrhea, if not managed appropriately, can lead to the development of DKA. Patients need to know the steps to take during acute illness to prevent DKA (Box 30-8). During acute illness, oral ingestion of about 150 to 200 g of carbohydrates per day (45 to 50 g every 3 to 4 hr) should be sufficient, along with medication adjustments, to keep glucose in the goal range and to prevent starvation ketosis (ADA, 2007).

Fasting hyperglycemia is a common finding in persons with diabetes. The amount of insulin required to normalize blood glucose levels during the night is less in the predawn period (from 1:00 to 3:00 AM) than at dawn (4:00 to 8:00 AM). The increased need for insulin at dawn causes a rise in fasting blood glucose levels and is referred to as the dawn phenomenon. It may result if insulin levels decline between predawn and dawn or if overnight hepatic glucose output becomes excessive as is common in type 2 diabetes. To identify the dawn phenomenon, blood glucose levels are monitored at bedtime and at 2:00 to 3:00 AM. With the dawn phenomenon, predawn blood glucose levels will be in the low range of normal but not in the hypoglycemic range. For patients with type 2 diabetes, metformin is often used because of its effect on hepatic glucose output. For persons with type 1 diabetes, administering insulin that does not peak at 1:00 to 3:00 AM such as a long-acting insulin should be considered.

Hypoglycemia followed by “rebound” hyperglycemia is called the Somogyi effect. This phenomenon originates during hypoglycemia with the secretion of counterregulatory hormones (glucagon, epinephrine, growth hormone, and cortisol) and is usually caused by excessive exogenous fluid intake. In type 2 diabetes, patients have the potential to become insulin resistant because the liver produces less insulin in response to a meal. Thus, administering insulin at mealtime is often not sufficient. In such patients, basal insulin may be needed to maintain a target blood glucose level. Metformin is often required to control blood glucose levels.

Common Causes of Hypoglycemia

- Inadvertent or deliberate errors in insulin doses
- Excessive insulin or oral secretagogue medications
- Improper timing of insulin in relation to food intake
- Intensive insulin therapy
- Inadequate food intake
- Omitted or inadequate meals or snacks
- Delayed meals or snacks
- Unplanned or increased physical activities or exercise
- Prolonged duration or increased intensity of exercise
- Alcohol intake without food

Treatment of Hypoglycemia

- Immediate treatment with carbohydrates is essential. If the blood glucose level falls below 70 mg/dl (3.9 mmol/L), treat with 15 g of carbohydrates, which is equivalent to:
  - 3 glucose tablets
  - Fruit juice or regular soft drinks, ½ c
  - Saltine crackers, 6
  - Syrup or honey, 1 Tbsp
- Wait 15 minutes and retest. If the blood glucose level remains <70 mg/dl (<3.9 mmol/L), treat with another 15 g of carbohydrates.
- Repeat testing and treatment until the blood glucose level returns to within normal range.
- Evaluate the time to the next meal or snack to determine the need for additional food. If it is more than an hour to the next meal, test again 60 minutes after treatment to see if additional carbohydrates are needed.

The relationship between glycemic control and decreased risk of microvascular complications (DCCT Research Group, 1993; UKPDS, 1998), and between type 1 diabetes, and macrovascular disease (DCCT Research Group, 2005). Blood pressure control also benefited.

**Macrovascular Diseases**

Insulin resistance, which may precede the development of type 2 diabetes and macrovascular disease by many years, induces numerous metabolic changes known as the metabolic syndrome or the insulin resistance syndrome (see Chapters 9 and 32). It is characterized by intraabdominal obesity or the android distribution of adipose tissue (waist circumference greater than 102 cm [>40 in] in men and greater than 88 cm [>35 in] in women) and is associated with dyslipidemia, hypertension, glucose intolerance, and increased prevalence of macrovascular complications. Other risk factors include genetics, smoking, sedentary lifestyle, high-fat diet, renal failure, and microalbuminuria.

Macrovascular diseases, including coronary heart disease (CHD), peripheral vascular disease (PVD), and cerebrovascular disease are more common, tend to occur at an earlier age, and are more extensive and severe in people with diabetes. Furthermore, in women with diabetes the increased risk of mortality from heart disease is greater than in men, in contrast to the nondiabetic population, in which heart disease mortality is greater in men than in women (ADA, 2001). Diabetes itself has been elevated from a risk factor for cerebrovascular disease to a CHD risk equivalent in the National Cholesterol Education Panel Adult Treatment Program III (NCEP ATP III, 2001).

**Dyslipidemia**

Patients with diabetes have an increased prevalence of lipid abnormalities that contributes to higher rates of CVD. In type 2 diabetes the prevalence of an elevated cholesterol level is about 28% to 34%, and about 5% to 14% of patients with type 2 diabetes have high triglyceride levels; also, lower HDL cholesterol levels are common. Furthermore, patients with type 2 diabetes typically have smaller, denser LDL particles, which increase atherogenicity even if the total LDL cholesterol level is not significantly elevated. Lifestyle intervention, including MNT, increased physical activity, weight loss, and smoking cessation should always be implemented. MNT should focus on the reduction of saturated and trans-fatty acids and cholesterol.

Primary therapy is directed first at lowering LDL cholesterol levels with the goal in individuals without overt CVD of reducing LDL cholesterol concentrations to less than 100 mg/dl (2.6 mmol/L). In individuals with overt CVD a lower LDL cholesterol goal of less than 70 mg/dl (1.8 mmol/L) is suggested (ADA, 2006b). Pharmacologic therapy with a statin (HMG-CoA reductase inhibitors) is indicated if there is an inadequate response to lifestyle modifications and improved glucose control. For LDL lowering, statins are the drugs of choice with the goal being to achieve an LDL reduction of 30% to 40%. In addition, if the HDL cholesterol is less than 40 mg/dl, a fibrin acid derivative or niacin might be considered.

**Long-Term Complications**

Long-term complications of diabetes include macrovascular diseases, microvascular diseases, and neuropathy. Macrovascular diseases involve diseases of large blood vessels; microvascular diseases associated with diabetes involve the small blood vessels and include nephropathy and retinopathy. In contrast, diabetic neuropathy is a condition characterized by damage to the nerves.

MNT is important in managing several long-term complications of diabetes. Nutrition therapy is also a major component in reducing risk factors for chronic complications, especially those related to macrovascular disease. The DCCT and the UKPDS provided convincing evidence for...
be used (ADA, 2006b). Combination therapy using statins and other lipid-lowering agents may be necessary to achieve lipid targets. Aspirin therapy should be used in all adult patients with diabetes and macrovascular disease and for primary prevention in patients 40 years of age or older with diabetes and one or more cardiovascular risk factors (ADA, 2006b) (see Chapter 32).

**Medical Nutrition Therapy for Dyslipidemia.** Studies in persons with diabetes demonstrating the effects of specific percentages of dietary saturated and trans-fatty acids and specific amounts of dietary cholesterol at risk for coronary heart disease are not available. In nondiabetic individuals, reducing saturated and trans-fatty acids and cholesterol intake decreases total and LDL cholesterol but may also reduce HDL cholesterol. However, importantly, the ratio of LDL to HDL cholesterol is not adversely affected. The most recent guidelines from the National Cholesterol Education Program recommend that total fat be 25% to 35% of total calories and saturated fat less than 7%. Intake of trans-fat should be minimized (NCEP ATP III, 2001). Either monounsaturated or polyunsaturated fats may be substituted for saturated fats.

Individuals with triglyceride measurements of 1000 mg/dl should restrict all types of dietary fat (except omega-3 fatty acids) and be treated with medication to reduce triglycerides. Supplementation with omega-3 fatty acids may benefit those with resistant hypertriglyceridemia (ADA, 2007).

**Hypertension**

Hypertension is a common comorbidity of diabetes, with about 73% of adults with diabetes having blood pressure of 130/80 mm Hg or higher or using prescription medications for hypertension (Centers for Disease Control and Prevention, 2005). Treatment of hypertension in persons with diabetes should also be vigorous to reduce the risk of macrovascular and microvascular disease. Blood pressure should be measured at every routine visit with a goal for blood pressure control of less than 130/80 mm Hg. Patients with systolic blood pressure of 130 to 139 mm Hg or a diastolic blood pressure of 80 to 89 mm Hg should be given MNT for hypertension (see Chapter 33) for a maximum of 3 months. If targets are not achieved, they should also be treated with pharmacologic agents that block the renin-angiotensin system. Patients with a systolic blood pressure of 140 mm Hg or greater or a diastolic pressure of 90 mm Hg or greater should receive drug therapy in addition to MNT. Lowering of blood pressure with antihypertensive drugs, including angiotensin-converting enzyme (ACE) inhibitors, angiotensin receptor blockers (ARBs), β-blockers, diuretics, and calcium channel blockers, has been shown to be effective in reducing the number of cardiovascular events. Multiple drug therapy (two or more agents at proper doses) is generally needed to achieve blood pressure targets (ADA, 2006b). See Chapter 33 for MNT for hypertension.

**Microvascular Diseases**

**Nephropathy**

In the United States and Europe diabetic nephropathy has become the most common single cause of end-stage renal disease (ESRD) and accounts for about 40% of new cases of ESRD. About 20% to 40% of patients with diabetes develop evidence of nephropathy, but in type 2 diabetes a considerably smaller number progress to ESRD. However, because of the much greater prevalence of type 2 diabetes, such patients constitute over half of the patients with diabetes currently starting on dialysis (ADA, 2004d).

The earliest clinical evidence of nephropathy is the appearance of low but abnormal urine albumin levels (30 to 299 mg/24 hr), referred to as microalbuminuria or incipient nephropathy. Microalbuminuria is also a marker of increased cardiovascular disease risk. Without specific interventions, progression to overt nephropathy or clinical albuminuria (≥300 mg/24 hr) occurs over a period of years. An annual screening for microalbuminuria should be performed in patients who have had type 1 diabetes for more than 5 years and in all patients with type 2 diabetes starting at diagnosis and during pregnancy (ADA, 2006b). An analysis of a spot urine sample for the albumin-to-creatinine ratio is the preferred method. Two of three tests within a 6-month period should be abnormal before a patient is designated as having microalbuminuria. Table 30-13 defines abnormalities in albumin excretion based on spot urine collections.

Although diabetic nephropathy cannot be cured, persuasive data indicate that the clinical course of the disease can be modified. To reduce the risk or slow the progression of nephropathy, glucose and blood pressure control should be optimized. In the treatment of both microalbuminuria and macroalbuminuria, either ACE inhibitors or ARBs should be used except during pregnancy. Although there are no adequate head-to-head comparisons of the two drugs, evidence supports the following. In patients with type 1 diabetes, with hypertension and any degree of albuminuria, ACE

<table>
<thead>
<tr>
<th><strong>Table 30-13</strong></th>
<th><strong>Definitions of Abnormalities in Urine Albumin Excretion</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Category</strong></td>
<td><strong>Spot Collection (mg/µg Creatinine)</strong></td>
</tr>
<tr>
<td>Normal</td>
<td>&lt;30</td>
</tr>
<tr>
<td>Microalbuminuria</td>
<td>30–299</td>
</tr>
<tr>
<td>Macroalbuminuria</td>
<td>≥300</td>
</tr>
<tr>
<td>Exercise failure</td>
<td>24 hr</td>
</tr>
</tbody>
</table>

Exercise within 24 hr, infection, fever, coronary heart failure, marked hyperglycemia, and marked hypertension may elevate urinary albumin excretion over baseline values.

inhibitors delay the progression of nephropathy. In patients with type 2 diabetes, hypertension, microalbuminuria, and renal insufficiency, ARBs delay the progression of nephropathy. If one class is not tolerated, the other should be substituted, and their combination will decrease albuminuria more than use of either agent alone (ADA, 2006b).

**Medical Nutrition Therapy for Nephropathy.** Research on low-protein diets delaying the progression of renal disease has been controversial. The role of MNT in glucose and blood pressure control is clearly the first priority. However, there is some evidence that, once albuminuria is present, there may be beneficial effects for renal function with a reduction of protein to 0.8 to 1 g/kg of body weight per day (Franz and Wheeler, 2003). Several studies that attempted to reduce protein intake in persons with type 1 or type 2 diabetes and microalbuminuria achieved a protein reduction to about 1 g/kg of body weight. In a dose-response analysis (Pijls et al., 1999), a 0.1 g/kg of body weight per day decrease in the intake of protein was related to an improvement of 11.1% in albuminuria. In studies conducted in subjects with type 1 diabetes and macroalbuminuria (overt nephropathy), the achieved protein restriction ranged from 0.7 g/kg to 0.9 g/kg body weight daily, and slowed the rate of decline in the GFR significantly over 32 to 35 months (Zeller et al., 1991). However, one study raised concern that too low a protein intake may cause malnutrition (Meloni et al., 2002). Therefore protein must be reduced in the context of overall adequate energy and nutrient intake. In microalbuminuria there may be additional benefits in lowering phosphorus to 500 to 1000 mg/day along with the low-protein diet (Zeller et al., 1991). Although several studies have explored the potential of plant versus animal protein, the data are inconclusive (Wheeler et al., 2002). Despite the controversy, reduction of protein intake to 0.8 to 1 g/kg of body weight per day in individuals with diabetes and the earlier stages of chronic kidney disease (CKD) and to 0.8 g/kg of body weight per day in the later stages of CKD may improve measures of renal function such as urine albumin excretion rate and glomerular filtration rate (ADA, 2007) (see Chapter 36).

**Retinopathy**

Diabetic retinopathy is estimated to be the most frequent cause of new cases of blindness among adults 20 to 74 years of age. After 20 years of diabetes, nearly all patients with type 1 diabetes and more than 60% of patients with type 2 diabetes have some degree of retinopathy (ADA, 2004c). Laser photoocoagulation surgery can reduce the risk of further vision loss but usually does not restore lost vision—thus the importance for a screening program to detect diabetic retinopathy. Adults and adolescents with type 1 diabetes should have an initial dilated and comprehensive eye examination by an ophthalmologist or optometrist within 3 to 5 years after the onset of diabetes and patients with type 2 diabetes should be examined shortly after the diagnosis of diabetes. Subsequent examinations for both groups should be done annually. Less frequent examinations may be considered (every 2 to 3 years) if the eye examination is normal (ADA, 2006b).

There are three stages of diabetic retinopathy. The early stage of nonproliferative diabetic retinopathy (NPDR) is characterized by microaneurysms, a pouchlike dilation of a terminal capillary, lesions that include cotton-wool spots (also referred to as soft exudates), and the formation of new blood vessels as a result of the great metabolic need of the retina for oxygen and other nutrients supplied by the bloodstream. As the disease progresses to the middle stages of moderate, severe, and very severe NPDR, gradual loss of the retinal microvasculature occurs, resulting in retinal ischemia. Extensive intraretinal hemorrhages and microaneurysms are common reflections of increasing retinal nonperfusion.

The most advanced stage, termed **proliferative diabetic retinopathy** (PDR), is the final and most vision-threatening stage of diabetic retinopathy. It is characterized by the onset of ischemia-induced new vessel proliferation at the optic disk or elsewhere in the retina. The new vessels are fragile and prone to bleeding, resulting in vitreous hemorrhage. With time the neovascularization tends to undergo fibrosis and contraction, resulting in retinal traction, retinal tears, vitreous hemorrhage, and retinal detachment. Diabetic macular edema, which involves thickening of the central (macular) portion of the retina, and glaucoma, in which fibrous scar tissue increases intraocular pressure, are other clinical findings in retinopathy.

**Neuropathy**

Chronic high levels of blood glucose are also associated with nerve damage and affect 60% to 70% of patients with both type 1 and type 2 diabetes (ADA, 2001). Peripheral neuropathy usually affects the nerves that control sensation in the feet and hands. Autonomic neuropathy affects nerve function controlling various organ systems. Cardiovascular effects include postural hypotension and decreased responsiveness to cardiac nerve impulses, leading to painless or silent ischemic heart disease. Sexual function may be affected, with impotence the most common manifestation. Damage to nerves innervating the gastrointestinal tract can cause a variety of problems. Neuropathy can be manifested in the esophagus as nausea and esophagitis, in the stomach as unpredictable emptying, in the small bowel as loss of nutrients, and in the large bowel as diarrhea or constipation. Intensive treatment of hyperglycemia reduces the risk of developing diabetic neuropathy.

**Medical Nutrition Therapy for Gastroparesis.** Gastroparesis (impaired gastric motility) affects about 25% of this population and is perhaps the most frustrating condition that patients and RDs experience. It results in delayed or irregular contractions of the stomach, leading to various gastrointestinal symptoms such as feelings of fullness, bloating, nausea, vomiting, diarrhea, or constipation. Gastroparesis should be suspected in individuals with erratic glucose control. The first step in management of patients with neuropathy should be to aim for stable and optimal glycemic control.
Hypoglycemia of nondiabetic origin has been defined as a clinical syndrome with diverse causes in which low levels of plasma glucose eventually lead to neuroglycopenia (Service, 1995). Hypoglycemia literally means low (hypo) blood glucose (glycemia). Normally the body is remarkably adept at maintaining fairly steady blood glucose levels—usually between 60 and 100 mg/dl (3.3 to 5.6 mmol/L), despite the intermittent ingestion of food. Under physiologic conditions the brain depends almost exclusively on glucose for its energy needs. Even with hunger, either because it is many hours since food was eaten or because the last meal was small, blood glucose levels remain fairly consistent.

Pathophysiology

However, in a small number of people blood glucose levels become too low. Symptoms of hypoglycemia are often felt when blood glucose is below 65 mg/dl (3.6 mmol/L). If the brain and nervous system are deprived of the glucose they need to function, symptoms such as sweating, shaking, weakness, hunger, headaches, and irritability can develop. Hypoglycemia can be difficult to diagnose because these typical symptoms can be caused by many different health problems besides hypoglycemia. For example, adrenaline (epinephrine) released as a result of anxiety and stress can trigger the symptoms of hypoglycemia.

The only way to determine whether hypoglycemia is causing these symptoms is to measure blood glucose levels while an individual is experiencing the symptoms (Brun et al., 2000). Therefore hypoglycemia can best be defined by the presence of three features known as Whipple’s triad: (1) a low plasma or blood glucose level; (2) symptoms of hypoglycemia at the same time as the low blood glucose values; and (3) amelioration of the symptoms by correction of the hypoglycemia (Prince, 1997).

A fairly steady blood glucose level is maintained by the interaction of several mechanisms. After eating, food is broken down into glucose and enters the bloodstream. As blood glucose levels rise, the pancreas responds by releasing the hormone insulin, which allows glucose to leave the bloodstream and enter various body cells, where it fuels the body’s activities. Glucose is also taken up by the liver and stored as glycogen for later use. When glucose concentrations from the last meal decline, the body goes from a “fed” to a “fasting” state. Insulin levels decrease, which keeps the blood glucose levels from falling too low. In addition, stored glucose is released from the liver back into the bloodstream with the help of glucagon, a hormone that is also released from the pancreas. Normally the body’s ability to balance glucose, insulin, and glucagon (and other counterregulatory hormones) keeps glucose levels within the normal range. Glucagon provides the primary defense against hypoglycemia; without it full recovery does not occur. Epinephrine has an important role. Symptoms of hypoglycemia have been recognized at plasma glucose levels of about 60 mg/dl, and impaired brain function has occurred at levels of about 50 mg/dl (Cryer, 2003).

Types of Hypoglycemia

If blood glucose levels fall below normal limits within 2 to 5 hours after eating, this is often referred to as reactive hypoglycemia (named because the body is reacting to food) or postprandial (reactive) hypoglycemia. Postprandial hypoglycemia can be caused by an exaggerated or late insulin response caused by either insulin resistance or elevated GLP-1; alimentary hyperinsulinism; renal glycosuria; defects in glucagon response; high insulin sensitivity; rare syndromes such as hereditary fructose intolerance, galactosemia, leucine sensitivity; or a rare β-cell pancreatic tumor (insulinoma), causing blood glucose levels to drop too low (Brun et al., 2000).

Alimentary hyperinsulinism is the most common type of documented postprandial hypoglycemia and is seen in patients who have undergone gastric surgery or some other type of gastric surgery (Gebhard et al., 2001) (see Chapter 26). These procedures are associated with rapid delivery of food to the small intestine, rapid absorption of glucose, and exaggerated insulin response. These patients respond best to multiple, frequent feedings (Prince, 1997). α-Glucosidase inhibitors such as acarbose may also be helpful because they decrease the absorption of carbohydrates (Hasler, 2002).

The ingestion of alcohol after a prolonged fast or the ingestion of large amounts of alcohol and carbohydrates on an empty stomach (“gin-and-tonic” syndrome) may also cause hypoglycemia within 3 to 4 hours in some healthy persons.

Idiopathic reactive hypoglycemia is characterized by normal insulin secretion but increased insulin sensitivity and, to some extent, reduced response of glucagon to acute hypoglycemia symptoms (Brun et al., 2000). The increase in insulin sensitivity associated with a deficiency of glucagon secretion leads to hypoglycemia late postprandially (Leonetti et al., 1996). Idiopathic reactive hypoglycemia has been inappropriately overdiagnosed by both physicians and patients, to the point that some physicians doubt its existence. Although rare, it does exist but can be documented only in
persons with hypoglycemia that occurs spontaneously and who meet the criteria of Whipple’s triad.

**Fasting (food-deprived) hypoglycemia** may occur in response to having gone without food for 8 hours or longer and can be caused by certain conditions that upset the body’s ability to balance blood glucose. These include eating disorders and other serious underlying medical conditions, including hormone deficiency states (e.g., hypopituitarism, adrenal insufficiency, catecholamine or glucagon deficiency), acquired liver disease, renal disease, certain drugs (e.g., alcohol, propranolol, salicylate), insulinoma (of which most are benign, but 6% to 10% can be malignant), and other nonpancreatic tumors. Taking high doses of aspirin may also lead to fasting hypoglycemia. Factitious hypoglycemia, or self-administration of insulin or sulfonylurea in persons who do not have diabetes, is a common cause as well (Prince, 1997). Symptoms related to fasting hypoglycemia tend to be particularly severe and can include a loss of mental acuity, seizures, and unconsciousness. If the underlying problem can be resolved, hypoglycemia is no longer a problem.

**Diagnostic Criteria**

One of the criteria used to confirm the presence of hypoglycemia is a blood glucose level of less than 50 mg/dl (<2.8 mmol/L). Previously the OGT test was the standard test for this condition; however, this test is not helpful because it involves a nonphysiologic stimulus and because results show little correlation with persons who later are documented to have hypoglycemia. Recording finger stick blood glucose measurements during spontaneously occurring symptomatic episodes at home is a method that is often used to establish the diagnosis. An alternative method is to perform a glucose test in a medical office setting, in which case the patient is given a typical meal that has been documented in the past to lead to symptomatic episodes; Whipple’s triad can be confirmed if symptoms occur. If blood glucose levels are low during the symptomatic period and if the symptoms disappear on eating, hypoglycemia is probably responsible. It is essential to make a correct diagnosis in patients with fasting hypoglycemia because the implications for therapy are serious.

**Management of Hypoglycemia**

The management of hypoglycemic disorders involves two distinct components: (1) relief of neuroglycopenic symptoms by restoring blood glucose concentrations to the normal range, and (2) correction of the underlying cause. The immediate treatment is to eat foods or beverages containing carbohydrates. As the glucose from the breakdown of carbohydrates is absorbed into the bloodstream, it increases the level of glucose in the blood and relieves the symptoms. If an underlying problem is causing hypoglycemia, appropriate treatment of this disease or disorder is essential.

Almost no research has been done to determine what type of food-related treatment is best for the prevention of hypoglycemia. Traditional advice has been to avoid foods containing sugars and to eat protein- and fat-containing foods. Recent research on the GI and sugars has raised questions about the appropriateness of restricting only sugars because these foods have been reported to have a lower GI than many of the starches that were encouraged in the past. Restriction of sugars may contribute to a decreased intake in total carbohydrates, which may be more important than the source of the carbohydrates.

Guidelines for the prevention of hypoglycemia have been published (International Diabetes Center, 2004). The goal of treatment is to adopt eating habits that will keep blood glucose levels as static as possible. To stay symptom free, it is important for individuals to eat five to six small meals or snacks per day. Doing this provides manageable amounts of glucose to the body. Spreading carbohydrates throughout the day; eating consistent amounts of carbohydrates, particularly high-fiber carbohydrates, at meals and snacks from day to day; and avoiding skipping meals can also be helpful. Recommended guidelines are listed in Box 30-9.

### Guidelines for Preventing Hypoglycemic Symptoms

1. Eat small meals, with snacks interspersed between meals and at bedtime. This means eating five to six small meals rather than two to three large meals to steady the release of glucose into the bloodstream.

2. Spread the intake of carbohydrate foods throughout the day. Eating large amounts of carbohydrates at one time produces increased amounts of glucose and stimulates the release of increased amounts of insulin, which can cause blood glucose levels to drop. Most individuals can eat two to four servings of carbohydrate foods at each meal and one to two servings at each snack. If carbohydrates are removed from the diet completely, the body loses its ability to handle carbohydrates properly, so this is not recommended. Carbohydrate foods include starches, fruits and fruit juices, milk and yogurt, and foods containing sugar.

3. Avoid foods that contain large amounts of carbohydrates. Examples of these foods are regular soft drinks, syrups, candy, fruit juices, regular fruit-yogurts, pies, and cakes.

4. Avoid beverages and foods containing caffeine. Caffeine can cause the same symptoms as hypoglycemia and make the individual feel worse.

5. Limit or avoid alcoholic beverages. Drinking alcohol on an empty stomach and without food can lower blood glucose levels by interfering with the liver’s ability to release stored glucose (gluconeogenesis). If an individual chooses to drink alcohol, it should be done in moderation (one or two drinks no more than twice a week), and food should always be eaten along with the alcoholic beverage.

Patients with hypoglycemia may also benefit from learning carbohydrate counting and, to prevent hypoglycemia, eating three to four carbohydrate servings (15 g of carbohydrate per serving) at meals and one to two for snacks (see Appendix 34). Foods containing protein that are also low in saturated fat can be eaten at meals or with snacks. These foods would be expected to have minimum effect on blood glucose levels and can add extra food for satiety and calories. However, because both protein and carbohydrate stimulate insulin release, a moderate intake may be advisable.

**FOCAL POINTS**

- Nutrition therapy is a challenging but essential aspect of the management of diabetes and hypoglycemia of nondiabetic origin.
- Attention to nutrition and food and meal-planning principles is essential for metabolic (glucose, lipids, and blood pressure) control and overall good health.
- An RD who is knowledgeable and skilled in implementing current nutrition principles and making recommendations for diabetes or hypoglycemia of nondiabetic origin is the medical team member who should plan, implement, and evaluate MNT and the nutrition care process.
- Effective education and counseling of the person with diabetes will lead to his or her becoming a team player in management of his or her blood glucose.
- The effectiveness of nutrition interventions need to be continually monitored and documented to promote the best possible outcomes.

**CLINICAL SCENARIO 1**

**Type 1 Diabetes**

Ellen is a 15-year-old girl with newly diagnosed type 1 diabetes referred for diabetes nutrition education. She is 5 ft 2 in tall, weighs 115 lb, and is active in cheerleading and basketball in high school. Her physician will be regulating the dosage and timing of her insulin regimen. Her grandmother has diabetes and is supportive of Ellen’s need for education. Ellen’s parents are divorced, and she now lives with her grandmother.

**Nutrition Diagnosis:** Food- and nutrition-related knowledge deficit related to adjustment of meals and insulin as evidenced by new diagnosis of type 1 diabetes mellitus

1. What assessment information do you need to determine a nutrition diagnosis?
2. Write a nutrition diagnosis for Ellen.
3. What meal planning system would be helpful for Ellen?
4. What guidance should you offer regarding Ellen’s sports activities?
5. What signs and symptoms of lack of diabetes control must Ellen understand to manage her disease? Which problem is she more likely to experience—hyperglycemia or hypoglycemia?
6. What food- and meal-planning information needs to be shared with the health care team as insulin therapy is integrated into Ellen’s normal eating and exercise habits?
7. How will you monitor and evaluate Ellen’s progress?
**CHAPTER 30 | Medical Nutrition Therapy for Diabetes Mellitus and Hypoglycemia of Nondiabetic Origin**

John is a moderately obese (BMI = 29) 49-year-old man who complains of increased thirst, polyuria, and fatigue referred for nutrition counseling. He has not had a medical check-up for 2 years. She returns at this time with a primary complaint of chronic fatigue. Her laboratory test results show the following: A1C 8.3%; serum cholesterol 214 mg/dl; triglycerides 275 mg/dl. Her current weight is 175 lb, and her height is 64 in (BMI = 30). She states she hasn’t returned for any follow-up visits because the only advice she gets is to lose weight and not to eat sugar, neither of which she is able to do.

**CLINICAL SCENARIO 3**

Debra is a 45-year-old woman with a known diagnosis of type 2 diabetes for 3 years referred for nutrition counseling. She has not had a medical check-up for 2 years. She returns at this time with a primary complaint of chronic fatigue. Her laboratory test results show the following: A1C 8.3%; serum cholesterol 214 mg/dl; triglycerides 275 mg/dl. Her current weight is 175 lb, and her height is 64 in (BMI = 30). She states she hasn’t returned for any follow-up visits because the only advice she gets is to lose weight and not to eat sugar, neither of which she is able to do.

**CLINICAL SCENARIO 2**

Debra is a 45-year-old woman with a known diagnosis of type 2 diabetes for 3 years referred for nutrition counseling. She has not had a medical check-up for 2 years. She returns at this time with a primary complaint of chronic fatigue. Her laboratory test results show the following: A1C 8.3%; serum cholesterol 214 mg/dl; triglycerides 275 mg/dl. Her current weight is 175 lb, and her height is 64 in (BMI = 30). She states she hasn’t returned for any follow-up visits because the only advice she gets is to lose weight and not to eat sugar, neither of which she is able to do.

**Nutrition Diagnosis 1:** Food- and nutrition-related knowledge deficit related to appropriate diet for management of type 2 diabetes mellitus and moderate obesity as evidenced by self-reports of “constant hunger”

**Nutrition Diagnosis 2:** Physical inactivity related to perceived lack of time to exercise as evidenced by patient reports

1. What assessment data do you need to determine a nutrition diagnosis?
2. Write a nutrition diagnosis for John.
3. What type of diabetes does John likely have? Is it likely to be controlled by nutrition therapy alone?
4. Given his degree of hyperglycemia, is it more likely that an oral agent or insulin therapy will be recommended?
5. What meal-planning approach would be helpful for John?
6. What other lifestyle strategies will be helpful?
7. How will you monitor and evaluate John’s progress?
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