Chapter 48
Diabetes
Dominique Noë Long, Carol Probst, David E. Kelley and Emily L. Germain-Lee

CLASSIFICATION AND DIAGNOSIS OF DIABETES MELLITUS

In 2003, the American Diabetes Association (ADA) modified the diagnostic criteria for the classification of impaired fasting glucose (IFG) and diabetes. There are four clinical classes of diabetes including type 1, type 2, other specific types of diabetes (genetic defects in β-cell function or insulin action, disease of exocrine pancreas, drug or chemically induced diabetes) and gestational diabetes mellitus (GDM). An elevated fasting glucose is one of several risk factors that are known to increase an individual’s risk of developing heart disease, stroke and diabetes. These risk factors, grouped together, are called the metabolic syndrome or ‘syndrome X’ and will be discussed later in this chapter (see under Medical treatment). For the purposes of this chapter, discussion will focus on type 1 and type 2 diabetes (see Table 48.1).

There are three ways to diagnose diabetes, each of which must be confirmed on a subsequent day unless there are definitive symptoms of hyperglycemia, such as excess thirst and urination (polydipsia and polyuria), and unexplained weight loss accompanied by increased or normal food intake. The criteria for the diagnosis of diabetes include the following: (i) symptoms of diabetes and a random plasma glucose ≥200 mg/dl (11.1 mmol/l); (ii) fasting plasma glucose (FPG) ≥126 mg/dl (7.0 mmol/l) (fasting is defined as no

<table>
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<tr>
<th>Table 48.1 Comparison of type 1 and type 2 diabetes</th>
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<td><strong>Type 1 diabetes</strong></td>
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<tr>
<td>No. of diabetes (%)</td>
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<tr>
<td>Onset of disease</td>
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<td>Age of onset</td>
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<td>Symptoms at onset</td>
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<tr>
<td>Requiring insulin</td>
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<td>Risk for ketoacidosis</td>
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<td>Body type</td>
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<td>Suspected cause</td>
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<td>Genetic predisposition</td>
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Calyceal infection (for at least 8h) and (iii) 24-h plasma glucose ≥200 mg/dL (11.1 mmol/L) during an oral glucose tolerance test (OGTT) with 75 g of glucose. FPG is the preferred test for diagnosing diabetes in nonpregnant adults. Only ≥2 of the above criteria of diabetes are classified as poor wounding, fasting, gastric variceal infections and blunted vision (American Diabetes Association 2005).

Hypoglycemia that is not sufficient to meet the diagnostic criteria for diabetes is categorized as either IG or IGT. IG is defined as an FPG between 100 mg/dL (5.6 mmol/L) and 125 mg/dL (6.9 mmol/L). IG is defined as a 2-h plasma glucose between 130 mg/dL (7.2 mmol/L) and 199 mg/dL (10.8 mmol/L). IG and IGT are also called "pre-diabetes" (American Diabetes Association 2005).

**TYPES OF DIABETES MELLITUS**

**Type 1**

Type 1 diabetes is caused by autoimmune destruction of the insulin-producing β-cells of the pancreatic islet, as a result, these patients have an absolute need for insulin therapy. The age of onset of type 1 diabetes is commonly during childhood or young adulthood, although it can begin at any age. In the absence of insulin replacement, patients with type 1 diabetes develop severe hyperglycemia and metabolic acidosis, eventually leading to the cessation of ketosis, a by-product of fat breakdown in the absence of insulin. Diabetic ketoacidosis (DKA) is a medical emergency.

**Type 2**

Of all individuals with diabetes, 90-95% have type 2 diabetes. This is most commonly a disease of adults and its incidence increases with each decade of aging. However, type 2 diabetes is increasingly being diagnosed in children and adolescents. Type 2 diabetes is associated with obesity, a family history of diabetes, a previous history of gestational diabetes, IGT, physical inactivity and the physical finding of acanthosis nigricans. Other factors associated with type 2 diabetes are race/ethnicity with African-Americans, Hispanic/Latino Americans, native Americans, and other Pacific Islanders being at particularly high risk. Type 2 diabetes is regarded as being a metabolic disorder that is linked to a modern lifestyle involving stress, excess calorie intake (particularly fat) and inactivity. It is a metabolic process, and these patients generally have the twin defects of sluggish secretion of insulin following meals (feeding to poor overall insulin production with long duration) and insulin resistance (reduced cellular uptake and utilization of insulin).

**THERAPEUTIC INTERVENTION**

Newly diagnosed diabetes

Patients newly diagnosed with diabetes mellitus have a special need for comprehensive education. Diabetes self-management education is an integral component of medical care. The onset of diabetes can be precipitated by physical and emotional stress and other illnesses and, usually, the diabetic state persists. In addition, certain medications, such as corticosteroids or psychoactive drugs, can trigger the onset of diabetes mellitus or upset metabolic control in a previously diagnosed patient.

**Medical treatment**

Diet and exercise are the cornerstones of the treatment of type 2 diabetes mellitus and many individuals with diabetes can control their blood glucose by following a careful diet and exercise program, losing weight and reducing intake of alcohol, smoking, and medications. The diet should include high-fiber, low-sodium foods. Physical activity including walking or jogging should be encouraged by people with type 2 diabetes, although this is often unnecessary.

Among adults with diabetes diagnosed, about 12% take both insulin and oral medications, 19% take insulin only, 53% take oral medications only, and 20% take neither insulin nor oral medications.

Glycemic control in patients with type 1 and 2 diabetes is most often measured using levels of blood glycosylated hemoglobin, or hemoglobin A₁c. In addition to self-monitoring of blood glucose, the HbA1c level reflects the mean blood glucose concentration over the previous 2-12 weeks. The ADA's current glycemic goal for nonpregnant adults is a level of <7.0% (compared with a normal non-diabetic range of 4.4-6.0%).

As many as one in six Americans over the age of 50 may have the "metabolic syndrome," a pathologic condition that increases the risk of heart disease, stroke, and diabetes. The criteria for metabolic syndrome are met by having any three of the following risk factors, which have been recently defined by the American Heart Association (AHA):

- An elevated waist circumference (abdominal obesity),
- An elevated triglyceride level of ≥150 mg/dL (4.1 mmol/L),
- A reduced high-density lipoprotein (HDL) cholesterol level of <40 mg/dL (1.03 mmol/L).

Although it is clear that each of the above risk factors does increase the individual's risk of cardiovascular disease, it has been strongly stated that the metabolic syndrome has been vaguely defined and should not be designated as a syndrome until more research is completed (American Diabetes Association 2005). The AHA recommends that blood pressure in patients with diabetes should be ≤130/80 mmHg. Lipid goals for patients with diabetes include a low-density lipoprotein (LDL) cholesterol level of ≤100 mg/dL (<2.6 mmol/L), triglyceride levels ≤150 mg/dL (<1.7 mmol/L) and HDL cholesterol level ≥60 mg/dL (>1.6 mmol/L) (American Diabetes Association 2005).

**Insulin therapy for type 1 diabetes**

Therapy for individuals with type 1 diabetes always includes insulin. Insulin is given by subcutaneous injection or with an insulin pump, which delivers insulin subcutaneously. Insulin is produced by the insulinoma cells, which must be destroyed by an autoimmune process. Therefore, in type 2 diabetes patients generally have the twin defects of sluggish secretion of insulin following meals (feeding to poor overall insulin production with long duration) and insulin resistance (reduced cellular uptake and utilization of insulin).

**Diabetes Control and Complications Trial (DCCT)** demonstrated that the risk of progression of diabetic microvascular disease (neuropathy, retinopathy and nephropathy) and possibly the occurrence of other cardiovascular disease (CVD) diseases are increased in patients with type 1 diabetes can be significantly reduced with improved glycemic control.

**Treatment of type 2 diabetes**

Treatment options for patients with type 2 diabetes are diverse. Control can be achieved with diet and exercise therapy, especially if weight loss is achieved in an overweight patient. However, most type 2 patients also require some pharmacological treatment, either oral hypoglycemic medication or insulin. Oral medications include the sulfonylureas (e.g. glyburide, glipizide, and acarbose) and meglitinides, which increase insulin release; thiazolidinediones (troglitazone, pioglitazone), which increase target tissue sensitivity to insulin; metformin, which increases glucose utilization and decreases glucose production by the liver; acarbose, which slows down the absorption of carbohydrates through the intestine; and prandial glucagon-like peptide-1 (GLP-1), which are taken with meals and help to increase insulin release. These medications can be used alone or in combination.

The United Kingdom Prospective Diabetes Study (UKPDS) showed that good glycemic control in patients with type 2 diabetes results in a reduction in the risk of microvascular disease. Specifically, a 1% fall in HbA1c was associated with a 56% reduction in microvascular complications (retinopathy, nephropathy and neuropathy). The risk reduction of macrovascular disease was less clear. Based on the results of the UKPDS, normoglycemia is now the goal for most patients with type 2 diabetes. Although insulin may be considered for initial therapy in type 2 diabetes, especially if the patient presents with a very high HbA1c level, it is most often used when hypoglycemia is present despite the use of oral hypoglycemic agents. The dose of insulin needed to control glucose levels in obese patients with type 2 diabetes can be extremely large.

**Hypoglycemia**

The main adverse effect of insulin or oral therapy is hypoglycemia (low blood glucose). In patients with type 2 diabetes, symptoms of hypoglycemia generally have a rapid onset and occur when blood glucose is less than 70-80 mg/dL. If a severe reaction occurs to below 60 mg/dL, a patient may complain of shakiness and sweating or other symptoms caused by increased epinephrine (adrenaline) release, such as tachycardia and anxiety. Deprivation of glucose in the central nervous system causes blurred vision, weakness, confusion, altered speech and, potentially, somnolence and coma, with permanent neurological damage. Symptoms of hypoglycemia may be blunted in a patient with long-standing diabetes, especially in the elderly and in patients with diabetes with a history of prior episodes of hypoglycemia. An initial symptom in patients with long-standing diabetes mellitus may be confusion.

In a diabetic patient, hypoglycemia occurs because of too much insulin (oral medications), insufficient food intake (relative to

Table 48.2: Comparison of diabetic complications

<table>
<thead>
<tr>
<th>Complication</th>
<th>Hyperglycemia with diabetes (DKA)</th>
<th>Hyperglycemia, hyperosmolality, nonketotic illness</th>
<th>Hypoglycemia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preexisting factors</td>
<td>Absence of insulin</td>
<td>Illnesses, infections, steroid use, burns</td>
<td>Excessive exogenous insulin, decreased oral intake, stress</td>
</tr>
<tr>
<td>Onset</td>
<td>Gradual</td>
<td>Gradual</td>
<td>Abnormal</td>
</tr>
<tr>
<td>Initial effect</td>
<td>Lethargy</td>
<td>Lethargy</td>
<td>Agitation, shakiness</td>
</tr>
<tr>
<td>Skin</td>
<td>Hot, dry</td>
<td>Damp, diaphoretic</td>
<td>Unchanged</td>
</tr>
<tr>
<td>Serum glucose level</td>
<td>&gt;300 mg/dL</td>
<td>&gt;300 mg/dL</td>
<td>&lt;70 mg/dL</td>
</tr>
<tr>
<td>Hydration</td>
<td>Increased thirst, polyuria, dehydration</td>
<td>Rapid volume depletion with increased initial thirst, polyuria progressing to decreased urine output</td>
<td>Unchanged</td>
</tr>
</tbody>
</table>

**Early CNS symptoms**

- Headache
- Visual symptoms

**Late CNS symptoms**

- Confusion, coma, death

**Metabolic acidosis**

- Elevated serum bicarbonate
- Bicarbonate levels in urine, fruity breath

**GI symptoms**

- Abdominal pain

**Intervention required**

- Insulin, fluid and sodium bicarbonate replacement

**4 oz [120 mL] juice, half a nonfat soda, two glucose tablets or two or four hard candies**

**CNS, central nervous system; DL, diagnostic lumbar puncture**
Diabetes

Types of Diabetes Mellitus

Type 1
Type 1 diabetes is caused by autoimmune destruction of the insulin-producing β-cells of the pancreatic islet, as a result, these patients have an absolute need for insulin therapy. The age of onset of type 1 diabetes is commonly during childhood or young adulthood, although it can begin at any age. In the absence of insulin replacement, patients with type 1 diabetes develop severe hyperglycemia and metabolic acidosis, which leads to the classic presentation of ketosis, a by-product of fat breakdown in the absence of insulin. Diabetic ketoacidosis (DKA) is a medical emergency.

Type 2
All individuals with diabetes, 90-95% have type 2 diabetes. This is more common in a family of diabetics and its incidence increases with each decade of aging. However, type 2 diabetes is increasingly being diagnosed in children and adolescents. Type 2 diabetes is associated with obesity, a family history of diabetes, a previous history of gestational diabetes, IGT, physical inactivity and the physical finding of ascertainment anergics. Other factors associated with type 2 diabetes are race/ethnicity with African-Americans, Latin-American, native Americas and some Asian-Americans and other Pacific Islanders being at particularly high risk. Type 2 diabetes is regarded as being a metabolic disorder that is linked to a modern lifestyle involving stress, excess caloric intake (particularly fat) and insufficient physical activity. From a metabolic perspective, these patients generally have the twin defects of sluggish secretion of insulin following meals (leading to poor overall insulin production with long duration) and insulin resistance (reduced cellular uptake and utilization of insulin).

Therapeutic Intervention

Newly diagnosed diabetes

Patients newly diagnosed with diabetes mellitus have a special need for comprehensive education. Diabetes self-management education is an integral component of medical care. The onset of diabetes can be precipitated by physical and emotional stress and other illnesses and, usually, the diabetic state persists. In addition, certain medications, mouth or oral bacterial overgrowth can trigger the onset of diabetes mellitus or upset metabolic control in a previously diagnosed patient.

Medical treatment

Diet and exercise are the cornerstones of the treatment of type 2 diabetes mellitus and many individuals with diabetes can control their blood glucose by following a careful diet and exercise program, losing weight and managing their weight-reducing agents (medications that lower plasma glucose levels). Generally, it is not necessary to increase food intake before exercise of short duration or low intensity. Exercise increases insulin sensitivity (e.g., 11% of insulin) may be produced by consuming 10-15g of carbohydrate, although this is often unnecessary.

Among adults with diabetes diagnosed, about 12% take only insulin or oral medications, 19% take insulin only, 53% take oral medications only and 5% take combination with neither insulin nor oral medications.

Glucogenic metabolism in patients with type 1 and 2 diabetes is most often measured using levels of blood glycolated hemoglobin or glycated albumin (HbA1c), in addition to self-monitoring of blood glucose. The HbA1c level reflects the mean blood glucose concentration over the previous 6-12 weeks. The ADA's current glycemic goal for nonpregnant adults is a value of <7.0% (compared with a normal non-diabetic range of 4-6%).

As many as one in six Americans over the age of 50 may have the "metabolic syndrome," a pathological condition that increases the risk of heart disease, stroke and diabetes. The criteria for metabolic syndrome are met by having any three of the following risk factors, which have been recently defined by the American Heart Association (AHA): (i) an elevated waist circumference (abdominal obesity), (ii) an elevated triglyceride level of ≥150 mg/dl, (iii) a reduced high-density lipoprotein (HDL) cholesterol level of <40 mg/dl, (iv) high blood pressure, defined as systolic blood pressure of ≥130 mmHg or diastolic blood pressure of ≥85 mmHg for women, (v) an elevated blood pressure of 130/85 mmHg or higher; and (vi) an elevated fasting glucose of >100 mg/dl.

Although it is clear that each of the above risk factors does increase an individual's risk of diabetes, a recent study in Framingham, MA, clearly stated that the metabolic syndrome has been vaguely defined and should not be diagnosed as syndrome until more research is completed (American Diabetes Association Statement 2005). The ADA recommends that blood pressure in patients with diabetes should be <130/80 mmHg. Lipid goals for patients with diabetes include a low-density lipoprotein (LDL) level of <100 mg/dl, diastolic pressure <80 mg/dl, triglyceride level <150 mg/dl and HDL level ≥40 mg/dl. 

Insulin therapy for type 1 diabetes

Therapy for individuals with type 1 diabetes always includes insulin. Insulin is given by subcutaneous injection or with an insulin pump, which also delivers insulin subcutaneously. Conventional types of rapid-acting, short, intermediate- or long-acting insulin are used, such as Humalog, Regular, NPH and glulisine. In most cases, patients with type 1 diabetes are treated with two or three doses per day of rapid- or short-acting insulin combined with intermediate- or long-acting insulin. However, many patients are required to increase insulin injections to optimize glycemic control. The method of using long-acting insulin (glulisine) combined with rapid-acting insulin (Humalog) given before meals and snacks, provides greater flexibility but requires a knowledge of carbohydrate counting and the use of an insulin-carbohydrate ratio. Because blood glucose can fluctuate widely in patients with type 1 diabetes, it is essential to determine the times at which to eat meals and snacks, and insulin doses adjusted accordingly.

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Hypoglycemia

The main adverse effect of insulin or oral therapy is hypoglycemia (low blood glucose). In a patient with diabetes, symptoms of hypoglycemia generally have a rapid onset and occur when blood glucose is less than 70-80 mg/dl (Table 48.2). A severe reaction can occur below 40 mg/dl. A patient may complain of shakiness and sweating or other symptoms caused by increased epinephrine (adrenaline) release, such as tachycardia and anxiety. Deprivation of glucose in the central nervous system causes blurred vision, weakness, confusion, altered speech, irritability, seizures, coma, with permanent neurological damage. Symptoms of hypoglycemia may be blunted in a patient with long-standing diabetes, especially the early warning signs of hypoglycemia. The initial symptom in patients with long-standing diabetes mellitus may be confusion. In a diabetic patient, hypoglycemia occurs because of too much insulin (or oral medications), insuficient food intake (relative to the amount of insulin).

<table>
<thead>
<tr>
<th>Table 48.2: Comparison of diabetic complications</th>
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<tr>
<td><strong>Complication</strong></td>
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<tr>
<td>Precipitating factors</td>
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<tr>
<td>Diet</td>
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<tr>
<td>Oral</td>
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<td>Initial effect</td>
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<td>Skin</td>
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<td>Glucose levels</td>
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<td>Glucose levels</td>
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<tr>
<td>Hydration</td>
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<tr>
<td>Cardiopulmonary symptoms</td>
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<tr>
<td>Early CNS symptoms</td>
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<td>Late CNS symptoms</td>
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<tr>
<td>Metabolic acidosis</td>
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<tr>
<td>GI symptoms</td>
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<tr>
<td>Intervention required</td>
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<tr>
<td>4 or 120 mg/L 1 hour, half</td>
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<tr>
<td>2 glucose tablets or two four</td>
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<td>hard candies</td>
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</table>

CNS, central nervous system; DL, daily.
Exercising and diabetes

Individuals without diabetes can maintain stable blood glucose levels during exercise. However, physical activity can have a marked effect on blood glucose in a person with diabetes. Exercise increases glucose use by muscles and may reduce insulin sensitivity to insulin. A regular program of exercise may lessen the requirements for insulin or oral medication. These are desirable effects but it should be recognized that exercise can increase the risk of hypoglycemia. About 30 min of interval or continuous exercise can decrease blood glucose regardless of fitness level.

Glucose control does not improve with exercise, so the effect must be evaluated for each patient. Patients should increase their blood glucose self-monitoring during exercise. This is especially important in patients on insulin or oral medication. At the beginning of an exercise program, particularly with type 1 diabetic patients, blood glucose levels should be checked before exercise, every 15-30 min during exercise and after stopping exercise. Blood glucose should continue to be checked frequently, as lows can continue for up to 2 h after stopping exercise. Blood glucose self-monitoring data can be used to assess a patient's response to physical activity and improve performance.

Hypoglycemia

In type 1 diabetes, exercising during insulin insufficiency can promote a hyperglycemic response in the individual at risk for metabolic acidosis. Additional insulin may have to be administered and exercise deferred if the glucose level is higher than 250 mg/dL and ketonuria is present. Exercise should be avoided if blood glucose is >300 mg/dL and no ketosis is present. Patients with type 1 diabetes should ingest additional carbohydrate if glucose levels are below 100 mg/dL. The value for deferring exercise is higher (300 mg/dL) because ketosis if far less common and is unlikely to be provoked by exercise. Occasionally, especially in elderly type 1 individuals, a medical crisis of severe hypoglycemia and cellular dehydration may occur, often in response to the physiologic stress of infection, burns or illness. These individuals may progress to a hyperglycemic, hyperosmolar, nonketotic coma. Because of the absence of ketosis, the diagnosis may be overlooked and, in this population, treatment may not be seeked. (See Table 48.2)

Proper hydration during exercise is essential. If exercise substantially lowers blood glucose, particularly if it drops into the range where hypoglycemia is a risk, then some of the following strategies should be considered. The most fundamental options are to either reduce insulin (or the oral medication dose) on exercise days or to take a supplemental snack before exercise (Table 48.3). One approach is to reduce the insulin dose by approximately 20%; the glucose response to exercise will provide additional information when making this decision. If weight loss is a goal, it is desirable to avoid supplemental caloric intake. It is also important to consider the timing of exercise with respect to the timing of insulin or oral medication administration and meals. Exercise should be done at least 1-2 h after meals and vigorous exercise should be undertaken when insulin levels are near the lower range. This might mean moving activity to the morning or switching from snacks closely linked to regular meals. Also, consideration should be given to the site of the insulin injection. Inulin injected over an exercising muscle is less likely to be absorbed than if injected subcutaneously re- lowing terents. Of this, exercising within 30 min of injec tion, a patient should be advised to use the abdomen, not the arm or thigh, for the administration of insulin. (Table 48.4). Exercise should include a standard warm-up and cool-down period as in nonendurance individuals. It is common for a patient initially referred for rehabilitation to have a relatively low fitness level that requires a cautious and grad uated introduction to exercise. Before increasing the usual patients of physical activity or starting an exercise program, patients with diabetes should undertake a detailed medical evaluation and, if indic ated, appropriate diagnostic studies such as an echocardiography, graded exercise test or radionuclide stress testing. The presence of micro- and macrovascular complications should be screened for at some may be worsened by exercise the program. Identification of areas of concern will allow the formulation of an individualized exercise program that can minimize the patient's risk.

Dietary complications

Diabetes is a systemic disease and the function of every organ system in the body can be affected. (Table 48.5). The following complications emphasize the diabetic complications that have particular relevance to rehabilitation (see Table 48.6). As mentioned previously, several recent trials, including the DCCT and the UKPADS, have shown that improved glycemic control in patients with type 1 and type 2 diabetes mellitus significantly reduces the risk of development or slows the progression of the microvascular complications of diabetes (retinopathy, nephropathy and neuropathy). The risk of microvascular complications is highest if the HbA1c is above 12% but that is increased at all values above the non-diabetic range. The data on the effect of glycemic control on the development of macrovascular disease in patients with type 2 diabetes are less clear. However, a recent meta-analysis of 13 prospective cohort studies showed that, for every 1 percentage point increase in HbA1c, the relative risk for any cardiovascular event is 1.18 (Selvin et al 2004).
Exercise and diabetes

Individuals without diabetes can maintain stable blood glucose levels during exercise. However, physical activity can have a marked effect on blood glucose in a person with diabetes. Exercise increases glucose use by muscles and reduces the need for insulin. A regular program of exercise may lower the requirements for insulin or oral medication. These are desirable effects, but it should be recognized that exercise can increase the risk of hypoglycemia. About 30 minutes or continuous exercise can decrease blood glucose regardless of fitness level.

Choice of exercise does not always improve exercise, so the effect must be evaluated for each patient. Patients should increase their blood glucose self-monitoring during exercise. This is especially important on weekends and vacation periods. At the beginning of an exercise program, particularly with type 1 diabetic patients, blood glucose levels should be checked before exercise, every 15-30 min during exercise and after stopping exercise. Blood glucose should continue to be checked frequently, as levels can continue to fall for up to 24 hr after exercise. Blood glucose self-monitoring data can be used to assess a patient’s response to physical activity and improve performance.

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Proper hydration during exercise is essential. If exercise substantially lowers blood glucose, particularly if it drops into the range where hypoglycemia is a risk, then some of the following strategies should be considered. The most fundamental options are either to reduce insulin (or the oral medication dose) on exercise days or to take a supplemental snack before exercise (Table 48.3). One approach is to reduce the insulin dose before exercise by approximately 20%; the glucose response to exercise will provide additional information when making this decision. If weight loss is a goal, it is desirable to avoid supplemental caloric intake. It is also important to consider the timing of exercise with respect to the timing of insulin or oral medication administration and meals. Exercise should be done at least 1-2 hr after meals and vigorous exercise should be undertaken when insulin levels are near the lower range. This might require adjusting the basal insulin dose in the morning or taking more insulin 1-2 hr before exercise. This approach has been shown to be effective in reducing the incidence of regular insulin. Also, consideration should be given to the site of the insulin injection. Insulin injected over an exercising muscle is metabolized more rapidly than when injected into a nonexercising muscle. Exercise-induced defects in capillary blood flow and oxygen delivery decrease the metabolic rate and lead to increased oxygen availability that may increase substrate uptake. Exercise may reduce insulin absorption and prolong insulin activity, which may increase the risk of hypoglycemia. Exercise after an insulin injection may decrease the glucose-lowering effects of this injection. Exercise also reduces the capacity of adipose tissue to store glycogen.

The data on the effect of glycemic control on the development of microvascular disease in patients with type 2 diabetes are less clear. However, a recent meta-analysis of 13 prospective cohort studies showed that, for every 1% point increase in HbA1c, the relative risk for any cardiovascular event is 1.18 (Selvin et al 2004).
Diabetes 2003

NEW DIABETES TREATMENTS

Diabetes is a chronic condition that affects millions of people worldwide. It is characterized by high levels of blood sugar (glucose) due to a deficiency or resistance to insulin. Insulin is a hormone produced by the pancreas that helps glucose enter cells to provide energy. Diabetes can be categorized into two main types: type 1 diabetes and type 2 diabetes. Type 1 diabetes is an autoimmune condition where the immune system attacks and destroys the beta cells in the pancreas that produce insulin. Type 2 diabetes is primarily caused by insulin resistance or a combination of insulin resistance and reduced insulin production, often linked to obesity and lifestyle factors.

This article discusses the various aspects of diabetes management, including lifestyle modifications, medication options, and emerging treatments. It highlights the importance of early diagnosis and the role of healthcare providers in monitoring and managing diabetes to prevent complications.

**Diabetes Management Overview**

Effective diabetes management involves a comprehensive approach that includes dietary changes, regular physical activity, weight management, and medication as needed. Regular monitoring of blood glucose levels helps in adjusting treatments to maintain optimal control. Early detection through screening and adequate education are crucial for successful diabetes management.

**Complications and Management Strategies**

Diabetes can lead to severe complications if not managed appropriately. Managing blood glucose levels helps prevent or delay the onset of these complications. Common diabetes complications include nerve damage (neuropathy), eye problems (retinopathy), kidney disease, and circulation issues affecting the legs and feet.

**Conclusion**

Diabetes management requires a collaborative effort between healthcare providers and patients. Regular monitoring, adherence to treatment plans, and lifestyle modifications are key to preventing complications and maintaining a good quality of life. With advancements in diabetes research, new treatments and prevention strategies continue to emerge, offering hope for improved outcomes.

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**References**


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**New Treatments for Diabetes**

New treatments for diabetes continue to be developed. These include oral medications, insulin therapy, and emerging therapies like glucose-dependent insulinotropic polypeptide (GIP) analogs, which may offer improved control over blood sugar levels.
Diabetes

Vascular complications

Vascular complications are the leading cause of death among individuals with diabetes. As they may result from cardiovascular, renal, or neural complications. Diabetic foot ulcers are a principal cause of the high rate of lower-extremity amputations in diabetics, which is 1-3 times higher than in nondiabetic individuals. Prevention of foot ulcers is the best therapy, and prevention starts with a careful foot and lower-extremity examination along with an aggressive program of patient education. Patients must be taught careful foot care, closely follow treatment and other potential damage to their feet, both before and after exercise. Proper footwear is important, especially for patients with peripheral neuropathy. The use of silicone gel or air mitigation, as well as polyester or blend socks to prevent blisters and keep feet dry, may minimize trauma to feet during exercise (Larson et al 2003, American Diabetes Association 2003).

Neuropathy

Neuropathy is found in approximately 60-70% of individuals with diabetes, with sensory loss being more prevalent than motor loss (see Chapters 34 and 35) (National Diabetes Fact Sheet 2003). Sensory loss typically presents in a stocking/glove pattern. Patients who are unable to perceive the touch of a Semmes-Weinstein 5.07 monofilament on the plantar surface of the foot are at high risk for ulceration. Decreased proprioceptive input may cause balance and motor deficits that typically affect the smaller intrinsic muscles of the feet, thus altering foot pressure and pronation. Daily foot care with insensitive feet (see Chapter 31) is at increased risk for callus or blister formation and this can be the trigger event that leads to rot- to-neck complications (see Chapter 35) and loss of limb or life (see Chapter 69). The education of patients should include recommendations against walking barefoot and suggestion that water temperatures be tested with the elbow and daily foot inspections be made. Although walking is the form of exercise that many older people prefer, with the considerable advantage of being low in intensity and not exacting distance on individuals with a marked neuropathy or foot deformity may be exposed to an increased risk of foot ulceration. A simple form of exercise is to walk or do a low-impact exercise such as calisthenics or swimming. Prescription of footwear with orthotics may allay some of the risk. On a more general level, foot care for diabetics is selective and specialized footwear for diabetic individuals. When a transmural ulceration does occur, 60% of diabetic patients lose the remaining leg within 5 years. Smoking significantly compounds the problem (American Diabetes Association 2004).

Autonomic neuropathy

In total, 60-70% of individuals with diabetes have mild to severe nervous system damage (National Diabetes Fact Sheet 2003). Autonomic neuropathy develops in the sympathetic and parasympathetic nervous systems in 20-40% of those with long-term diabetes. Exercise programs for diabetic patients with autonomic neuropathy should proceed cautiously. Autonomic neuropathy can result in distal anesthesia, leading to poor blood circulation as a result of the decreased sensibility for the feet. Patients with this symptom should avoid overheating when exercising. Consolatory autonomic dysfunction leads to impaired sweating and risk of urinary retention, which sometimes congest the bowels and bladder. Some individuals with autonomic involvement may present with significant cardiac autonomic neuropathy. These individuals do not perceive anginal pain and may be at risk for "silent" myocardial infarction. Cardiac arrhythmias are not uncommon. Cardiac denervation syndrome (also referred to as cardiac autonomic autonomy), a result of autonomic dysfunction, produces a bradycardia that is typically around 60-90 beats per minute and is unresponsive to atropine levels, beta-blockers, or other medications. Aarrhythmia of the atria or interlocking of the heart may produce transient ischemic attacks. Whenever cardiac autonomic changes are present, monitoring vital signs to assess exercise tolerance may not always produce accurate information. Individuals in this state should have thorough cardiac workups before increasing activity levels, including stress or resting thallium myocard to look for the occurrence and extent of macrovascular coronary heart disease. If cardiac neuropathy is present, during exercise, emphasis should be placed on perceived exertion rates, dyspnea and other observed symptoms of distress and not simply on pulse and blood pressure. Exercise warm-ups and cool-downs should be stressed. Patients prone to ortho- static changes may benefit from minimizing changes in position during rehabilitation, wearing compressive stockings and ensuring an adequate fluid intake (American Diabetes Association 2004).

Retinopathy

Retinopathy is a frequent complication of diabetes. About 80% of type 1 diabetes will have some diabetic retinopathy 15 years of disease, and 60% of patients with type 2 diabetes will develop some degree of retinopathy after 20 years. Further, 20% of type 2 diabetics have some degree of retinopathy at diagnosis (Larson et al 2003). Although most cases of retinopathy of the nonproliferative variety (with only mild background changes in vision), some patients progress to proliferative retinopathy, which is the leading cause of blindness in adults aged 20 to 74. Using the Joslin Clinic experience, the degree of diabetic retinopathy has been used to stratify the risk of potential therapy and to discuss certain activities based on this stratification. For example, in patients with active proliferative diabetic retinopathy (PDR), severe activity may lead to vitreous hemorrhage or tractional retinal detachment. Patients with active PDR should avoid physical activity that involves straining, jarring, jiggling, highimpact athletic or Valsalva-like maneuvers. Patients with moderate to severe nonproliferative diabetic retinopathy (NPDR) should also limit activities such as heavy lifting, Valsalva maneuvers, and highly competitive sports. Systemic blood pressure should be kept below 170/110 during exercise (American Diabetes Association 2005).

Nephropathy

Diabetic nephropathy occurs in 20-40% of patients with diabetes and is the leading cause of end-stage renal disease, accounting for 40% of new cases (National Diabetes Fact Sheet 2003). The earliest sign of diabetic nephropathy in type 1 diabetes is proteinuria albuminuria in the range of 30-200 mg over 24h (microalbuminuria). Microalbuminuria is also a marker for the development of nephropathy in type 2 diabe- tes, as well as a marker for increased cardiovascular disease risk. Controlling blood pressure has been shown to reduce the development of nephropathy; blood pressure should be carefully monitored during exercise. The ADA has not developed specific physical activity recommendations for patients with microalbuminuria or overt nephropathy. Patients with nephropathy may have a reduced capac- ity for physical activity leading to self-limitation of activity level. High intensity and strenuous physical activity should probably be discouraged in these individuals unless blood pressure is carefully monitored (Larson et al 2003, American Diabetes Association 2005).

In 2001, a total of 129,198 people with diabetes underwent dialysis or kidney transplantation (National Diabetes Fact Sheet 2003). For patients on dialysis therapy, fluid replacement is a crucial that must influence the scheduling of exercise and rehabilitation. In addi- tion, patients are given heparin during infusions and any wound care that is performed within 24h of dialysis should minimize surgical debridement. Exercise programs should incorporate anti- coagulant precautions, such as guarding against skin trauma caused by weights, hand placement or jerking, especially at intravenous sites, and there should be renewed vigilance against falling.

CONCLUSION

Diabetes is a common and chronic disease that includes multisystem involvement in diabetes mellitus that medical and rehabilitation care because of complications resulting from the dia- betes or from other illness. It is important that the healthcare provider be aware of the significant influence that diabetes has on rehabilitation.