Diabetes mellitus

With diabetes mellitus, carbohydrate, fat, and protein metabolism are impaired because of a deficient response to insulin. There are two forms of diabetes mellitus:

• **Type I diabetes mellitus,** also called insulin-dependent diabetes mellitus (IDDM), is caused by impaired secretion of insulin.

• **Type II diabetes mellitus,** also called non–insulindependent diabetes mellitus (NIDDM), is caused by resistance to the metabolic effects of insulin in target tissues.

Type I Diabetes Is Caused by Impaired Secretion of Insulin by the Beta Cells of the Pancreas. Often, type I diabetes is a result of autoimmune destruction of beta cells, but it can also arise from the loss of beta cells resulting from viral infections. Because the usual onset of type I diabetes occurs during childhood, it is referred to as juvenile diabetes. Most of the pathophysiological features of type I diabetes can be attributed to the following major effects of insulin deficiency:

• Hyperglycemia as a result of impaired glucose uptake into tissues and increased glucose production by the liver (increased gluconeogenesis)

• Depletion of proteins resulting from decreased synthesis and increased catabolism

• Depletion of fat stores and increased ketogenesis

As a result of these fundamental derangements, the following occur:

• Glucosuria, osmotic diuresis, hypovolemia, and hypotension

• Hyperosmolality of the blood, dehydration, and polydipsia

• Hyperphagia but weight loss; lack of energy

• Acidosis progressing to diabetic coma; rapid and deep breathing

• Hypercholesterolemia and atherosclerotic vascular disease

Insulin Resistance Is the Hallmark of Type II Diabetes Mellitus. Type II diabetes is far more common than type I diabetes (accounting for approximately 90% of all cases of diabetes) and is usually associated with obesity. This form of diabetes is characterized by impaired ability of target tissues to respond to the metabolic effects of insulin, which is referred to as insulin resistance. In contrast to type I diabetes, pancreatic beta cell morphology is normal throughout much of the disease, and there is an elevated rate of insulin secretion. Type II diabetes usually develops in adults and therefore is also called adult-onset diabetes.

Although hyperglycemia is a prominent feature of type II diabetes, accelerated lipolysis and ketogenesis usually do not occur. Caloric restriction and weight reduction usually improve insulin resistance in target tissues; but in the late stages of the disease when insulin secretion is impaired, insulin administration is required.