IF YOU ARE READING THIS COLUMN, you are probably a member of at least 1 of 3 categories: individuals with diabetes, personal trainers who work with or plan to work with diabetic clients, or those who want to know more about this topic. Depending on the category or categories you fall into, your understanding of diabetes may be very good or very limited. For those of you who are already familiar with the disease, I suggest that you skip to the glycemic control section. For the rest of you, a basic understanding of diabetes is necessary before we discuss glycemic control. Diabetes mellitus is a disease that affects 16 million people in the United States, and that rate is increasing by 800,000 new cases each year. The disease represents a variety of clinical conditions but can be broken down into 2 basic types. Insulin-dependent diabetes mellitus (IDDM), or type 1 diabetes, is a rapid-onset form that usually develops at a young age. The symptoms appear after an infectious illness and can be attributed to an autoimmune destruction of the insulin-producing cells of the pancreas. Basically, the body fails to recognize the beta cells of the pancreas and destroys them. Insulin is essential for the control of glucose uptake by the muscle, and its loss allows glucose to become dangerously high. To maintain normal levels of glucose, IDDM patients must establish a daily insulin replacement routine. The second form of diabetes, called non–insulin-dependent diabetes mellitus (NIDDM), or type 2 diabetes, is more prevalent than type 1 and usually does not appear until later in life. Although the exact cause of NIDDM is still undetermined, its appearance is gradual and can be directly linked to obesity and a sedentary lifestyle. The condition is characterized by a consistent loss of insulin responsiveness and leads to a condition known as insulin resistance. As the effectiveness of insulin decreases, plasma glucose goes up, and the pancreas attempts to correct the problem by making more insulin. The higher insulin levels work for a short time, but eventually the new insulin also loses its effectiveness, and the cycle continues. This results in a steady increase in insulin and glucose, which eventually becomes a clinical problem. The differences between these 2 types of diabetes are quite clear and must be taken into account in prescribing an exercise routine.

Glycemic Control

The maintenance of normal blood glucose (glycemic control) in non-diabetic subjects is characterized by a unique hormonal system that regulates both glucose uptake and hepatic glucose output. The primary metabolic function of insulin is to remove glucose from the blood, and it accomplishes this by regulating 3 areas of glycemic control. The first of these is glucose usage by muscle and other tissues. The movement of glucose across the cell membrane is dependent on the presence of glucose transporter proteins. These proteins are termed GLUT 4 in the muscle and are stored within it. If insulin is released from the pancreas, it binds to its cellular receptors and stimulates the GLUT 4 to move from the storage site to the cell membrane. The incorporation of GLUT 4 into the membrane significantly enhances the muscle’s ability to take up plasma glucose. Once glucose enters the cell, insulin exerts its second glycemic control by stimulating the enzymatic machinery that converts glucose into glycogen, triglycerides, or both, thus promoting glucose storage. The final area of control results from insulin’s ability to suppress hepatic glucose production. Insulin acts alone to lower blood glucose, but raising it once levels drop too low requires the release of a variety of glucoreg-
ulatory hormones. Two of these hormones, cortisol from the adrenal cortex and glucagon from the pancreas, specifically target the liver and stimulate the breakdown of hepatic glycogen and the conversion of noncarbohydrates into glucose. The resultant hepatic glucose production brings plasma glucose levels back to normal. The glycemic control mechanism maintains one’s plasma glucose at a fairly constant level by responding to the changes made by food intake, increased activity, or both.

**Exercise Effects**

During aerobic forms of exercise, such as walking or running, the muscles utilize glucose at a constant rate without the aid of insulin. The stimulatory effects of insulin on glucose storage make its presence detrimental to exercise performance; therefore, its release is suppressed during exercise by the action of epinephrine. Fortunately, the muscle contains a calcium-mediated mechanism that becomes active during muscle contraction and that activates the movement of GLUT 4 in the absence of insulin. Several investigators have shown that this exercise-induced enhancement of glucose uptake and the insulin responsiveness associated with it can persist for up to 18–24 hours following exercise. This has not been tested in IDDM subjects, but data from our laboratory have shown that the residual effects of exercise can last for up to 18 hours in NIDDM or elderly subjects who are insulin resistant. Aerobic exercises are steady state in nature and range from low to moderate intensity, and they have been successfully prescribed for both IDDM and NIDDM subjects. In the case of the IDDM subjects, they have been shown to lower insulin replacement needs. With NIDDM subjects, this form of exercise gives them better glycemic control and decreases their risk factors for cardiovascular diseases. With strenuous forms of exercise, such as weightlifting, the energy demands of the muscle are very high, and the muscle depends on both cellular (glycogen) and plasma glucose. Although the calcium-mediated mechanism for glucose uptake is fully operational in the muscle, data from a recently completed study from my laboratory suggests that intense exercise may create a transient insulin resistance after exercise. That study examined the immediate postexercise insulin responses of nondiabetic untrained males (age range, 24 ± 1 years) following a single bout of running or weightlifting. The exercise intensity was set at 70% for both exercise trials and was estimated from the subject’s maximal aerobic capacity or 1 repetition maximum (1RM). The 2 forms of exercise produced similar plasma insulin responses in the absence of glucose but gave dramatically different responses if glucose was consumed 45 minutes after completion of the exercise trial. The presence of glucose had no effect following the run (the insulin response was the same as that in the nonexercise trial) but significantly elevated insulin if the subjects had lifted weights. The work of several different investigators has shown that eccentric forms of muscle contraction can damage the muscle’s cell membrane. This damage can raise plasma levels of a muscle enzyme called creatine kinase, and there is evidence that its presence following exercise may impair insulin action and GLUT 4 activity. Weightlifting has a higher eccentric component than running and can produce a considerable amount of cellular disruption in the muscle. Therefore, this form of training might represent a potential problem for diabetics unless they closely monitor their blood glucose levels, insulin dosage, or both.

**Training Recommendations**

The exercise guidelines I am suggesting for diabetic subjects are essentially the same as those used for nondiabetic subjects and can be used by individuals or personal trainers. However, prior to beginning an exercise program, the subject’s diabetic condition must be assessed. There is inadequate room to give the details of the assessment here, so I refer the reader to the American Diabetes Association’s Guidelines for Diabetes Mellitus and Exercise. If the individual has received a physician’s clearance but is physically out of shape, the program should start with light to moderate aerobic exercises to establish an aerobic base. Once the individual’s fitness level is acceptable, the trainer can establish a program with light weights and 6–8 lifting stations that train the major upper- and lower-body muscle groups. Concentrate your efforts on teaching good lifting techniques, and use this time (1 week) to establish a 10 repetition maximum (10RM) for each lift. Using the 10RM data and a 1RM estimation table (2), the trainer should then determine the subject’s 1RM for each lift and should calculate from that the 50, 60, and 70% workloads. Then set up a 10-repetition progressive-lifting program that employs a 50% workload for the first set, a 60% load for the second set, and a 70% load for the last set. If the workloads are accurate, the subject initially should not be able to complete 10 repetitions on the last set. When the subject can complete 12–14 repetitions on the last set, increase the weight to reduce the amount of repetitions back to 8–10 for the last set. These guidelines are
very general, but I hope they will give you an adequate starting point. The exact program you establish will depend on the subject’s initial physical condition, form of diabetes, and clinical status. If your pre-assessment is good and you monitor blood glucose and insulin replacement (type 1) on a regular basis, the rest should be easy. ▲

References