ATHLETIC TRAINERS and therapists in a traditional setting are used to working with a young, healthy population. With the expansion of certified athletic trainers and therapists into clinics and industrial settings, the opportunity arises to work with older athletes with a variety of medical conditions. Occasionally, athletic trainers and therapists might work with a diabetic athlete. A common condition associated with long-standing diabetes is peripheral vascular disease (PVD). The incidence of PVD and atherosclerosis is widespread in diabetics, and virtually all will suffer premature atherosclerosis (Campaigne & Lampman, 1994). PVD is characterized by reduced blood flow to skeletal muscle and consequently impaired exercise tolerance. Often seen in the lower leg, PVD can cause ischemia from reduced blood flow to muscles. Ischemia is most often associated with muscle weakness, impaired nerve function, decreased nerve-conduction velocity, and decreased maximal contraction (Styf, 1989). These symptoms are also closely associated in the lower leg with exertional compartment syndrome. Anterior compartment syndrome is a condition characterized by exercise-induced pain over the anterolateral aspect of the lower leg (see Figure 1) accompanied by swelling and impaired muscle and nerve function (Beckham, Grana, Buckley, Breazile, & Claypool, 1993).

The anterior compartment of the lower leg is surrounded by a relatively inelastic fascial covering and contains the tibialis anterior, extensor digitorum longus, peroneus tertius, and extensor hallucis longus. The inelastic fascia creates the possibility for increasing pressures in the compartment when the muscles, primarily the tibialis anterior, are used extensively during running (Friden, Seger, Sjostrom, & Ekblom, 1983). During running, eccentric contractions of the foot dorsiflexors can cause disturbances in the myofibrils, resulting in exercise-induced muscle damage and increased compartment pressures. The symptoms of anterior compartment syndrome are pain and impaired muscle and nerve function, caused by muscle ischemia as a result of decreased blood flow resulting from pressure in the compartment (Styf, 1989). It is a fairly common condition in runners, and its symptoms are generally relieved by rest. This report presents a case of chronic anterolateral exertional compartment syndrome of long-standing duration in which diagnosis was complicated because of the lack of pain in the athlete and the assumption that he had PVD because of his Type 1 diabetes.

**Case Report**

A 55-year-old runner presented with recurrent anterolateral leg tightness induced by exercise. The athlete had a history of lower anterior leg tightness extending back to high school. He had been diagnosed with Type 1 diabetes at age 12. No complications or sequelae of diabetes had developed since the diagnosis. His diabetes was well controlled, as demonstrated by recent HbA1c values averaging about 6.0% (upper normal range). He checked his blood glucose five or six times daily, and his insulin regimen included long-acting insulin for basal metabolic needs and fast-acting insulin before meals and snacks.

The anterior lateral leg tightness continued periodically throughout his 40 years of distance running. Arch supports had alleviated the symptoms until several years ago, at which point he began using pre-
scription orthotics to control overpronation. For years, the athlete had used dorsiflexor-strengthening exercises to reduce symptoms.

During running, the athlete’s dorsiflexors typically tightened in the initial 5–10 min, forcing him to walk. The tightness disappeared after several minutes of walking, and he could then run for several minutes until tightness reoccurred. After some 30 min of alternate running and walking, symptoms decreased and allowed the latter stage of a run to be completed unimpeded.

The athlete denied any anterior lower leg pain or point tenderness at any time and indicated that symptoms included only lower leg “tightness” without visible swelling. He assumed that the condition was “shin splints.” The history of anterolateral lower leg tightness with running was suggestive of exertional anterior compartment syndrome, although the runner did not exhibit most of the symptoms normally associated with that condition (i.e., swelling, pain, and impaired muscle or nerve function). The physical examination was unremarkable, without visible signs of swelling or muscle atrophy. The athlete had normal neurological findings of the lower extremity, and his strength was within normal limits. The anterolateral aspect of his leg was not point tender. The athlete had recently undergone routine Doppler assessment recommended by the physician supervising his diabetes because of weak pulses in his ankle and foot. Results were normal, which ruled out PVD as a possible cause of the leg tightness. The athlete was then referred to an orthopedic surgeon for further diagnosis.

Diagnosis was made by measuring bilateral anterior intracompartmental pressures (ICP) using a handheld electronic monitor. Initial resting pressure of both legs was 28 mmHg; normal pressure in the anterior tibial compartment at rest should be about 5 mmHg (Styf, 1989). Pressure can increase to 15–25 mmHg during exercise and returns to preexercise levels within 5–10 min in a normal leg (Friden, Seger, et al., 1983). The athlete ran for about 10 min until the anterior compartments were tight enough to force cessation of running. Immediate postexercise ICP was measured at 48 and 44 mmHg in the right and left legs, respectively. There was palpable tension in both anterior compartments but no pain. No deficits in strength or sensation were noted, and no muscle herniations or fascial defects were evident. Five minutes postexercise, ICPs were measured at 36 and 34 mmHg in the right and left legs, respectively. Increased pressure at rest and after exercise and prolonged time for normalization of the increased pressure are the most commonly used parameters in diagnosing chronic anterior compartment syndrome (Styf, 1989). Nonoperative treatment of this condition does not relieve the symptoms (Styf, 1986), but treatment by fasciotomy has proven successful in 60–100% of patients (Friden, Sjostrom, & Ekblom, 1983; Styf, 1989). Bilateral anterior and lateral fasciotomies were performed under general anesthesia in both legs of this athlete. Healthy muscle tissue was found in all compartments, but the superficial peroneal nerve was compressed against the anterior compartment fascia by adjacent muscle.

Swelling after surgery was minimized by elevating the legs at night and for as much time as possible during the first 3 days. Short walks (about 15–20 min) began on the fourth day in addition to cycling. Two weeks postsurgery, the athlete resumed running for 20 min on alternate days and progressed 5–10 min with each run. He resumed his normal running regimen within 5 weeks postsurgery.