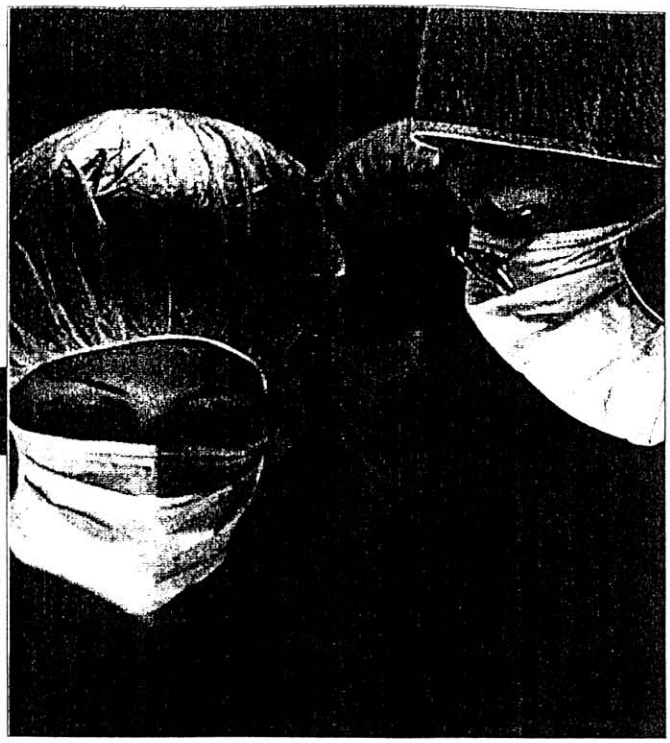
What A RELIEF

By Lawrence A. DiDomenico, DPM, FACFAS, and H. Ryan Hall, DPM

When conservative measures, fail surgical pressure relief can reduce the chances of further ulceration



With one-fourth of all diabetic hospital admissions the result of lower extremity complications, foot and ankle surgeons play an integral role in treating these conditions. One of the most frequent complications is ulceration of the lower extremity. Studies have implicated an equinus deformity in diabetic patients contributing to the development of ulcerations—particularly those of the forefoot.

Biomechanical Base

The calcaneal tendon is a strong plantarflexor of the foot. Its proper function depends upon appropriate length. Clinically, this is manifested as an early heel lift or "toe walking" during biomechanical gait analysis. The gastroc and soleous muscles begin to contract during the end of the contact portion of the stance phase of gait. They contract through midstance and the initial part of the propulsive period.

Because of its origin, only the gastroc is involved in knee flexion but acts together with the soleus in heel lift. The amount of ankle joint dorsiflexion required for normal locomotion is approximately 20° of plantarflexion and 10° of dorsiflexion. The dorsiflexion is necessary once the knee extends just prior to heel lift in the stance phase of gait. During this phase, the sub talar joint (STJ) will return to an approximate neutral position. As such, the ankle will need at least 10° of dorsiflexion when the knee is extended and the STJ is in its neutral position. Also consider the midtarsal joint—with its two axes of motion (long and oblique) that allow triplanar motion. This is due to the orientation of the joint axes from all three body planes. So, if the amount of dorsiflexion is less than adequate, compensation may occur at the STJ and midtarsal joint—with excess motion leading to an overall compensatory pronation of the foot.[1] This abnormal pronation leads to hypermobility of the joints in the foot with microtrauma to the soft tissues and joints.[2]

Testing for Deformity

When testing for an equinus deformity, proper technique is imperative. First, place the patient in the supine position on the examination table. Next, simulate the weight bearing that occurs during stance phase at the point where maximum dorsiflexion is required—knee extension and STJ-neutral position. Direct attention to the subtalar and midtarsal joints. If the STJ is not in the neutral position, a false measurement may be obtained. Ensure that there is no pronation of the STJ since this will also increase motion at the midtarsal joint. Likewise, the midtarsal joint alone will allow motion and it should be locked during the equinus examination to eliminate motion with the sagittal plane movement of the ankle.

To prevent this motion, perform the test by placing the STJ in its neutral position and holding in place using one hand to firmly grasp the patient's posterior heel. The midtarsal joint is then locked into place by using the opposite hand to align the midfoot to its locked position. Again, this eliminates any motion of the talonavicular and calcaneocuboid joints—providing a more correct result. In addition, the person performing the test should ensure that the patient is relaxed. It is particularly important that the extensor tendons (especially the tibialis anterior) are relaxed since many patients will inadvertently contract this muscle during examination.

The examiner then tests the range of motion past the perpendicular of the angles formed by bisecting the foot and leg. Different authors suggest varying minimum amounts of necessary dorsiflexion. Hansen states that at least 5° to 10° are needed. Root suggests 10° as a minimal amount, and Hibbs gives a range of 10° to 20°.[1,3,4]

When inadequate motion is observed, you must determine the etiology of either bone or soft tissue muscle. The differentiation of muscular equinus is assessed as either gastroc soleal or just gastroc equinus using the Silfverskiold test. By using this test you are in essence able to remove the gastrocnemius component of the complex with flexion of the knee. This is due to the fact that the gastroc muscle crosses the knee joint and the soleus does not. The proper positioning and execution of this test is paramount to accurately diagnosis equinus.

Occurrence in Diabetics

Recently, researchers described pathological changes to the structural components of the Achilles tendons in diabetics causing increased plantar pressures secondary to a shortened tendo-achilles.[5] This causes an affect ranging from a limitation in dorsiflexory ROM to a marked plantarflexed deformity of the ambulating foot.

Other researchers performed a study in which patients were divided into four groups: acute Charcot osteoarthropathy, neuropathy with ulcerations, neuropathy with no ulcerations, and no neuropathy with no ulcerations. The results showed that patients with the highest peak plantar pressures were those with acute Charcot osteoarthropathy. The next highest group was patients who had neuropathy with ulcerations, followed by those with neuropathy/no ulcerations. The patients who had the lowest pressures were individuals with no neuropathy/no ulceration.[6] The significance of this finding has prompted many foot and ankle surgeons to do adjunctive Achilles complex lengthening procedures or posterior muscle group releases when operating on individuals with acute Charcot or neuropathic ulcerations.[7]

Pathology

The probability of structural changes to the tendon is supported by Grant's study examining microscopic specimens of achilles tendons taken from diabetic patients with overt pathology (Charcot foot, diabetic ulcerations). The results indicated the presence of histopathological changes in tendon fiber organization for all diabetic patients. Under electron and light microscopy, changes were seen in the microarchitecture including foci of collagen disruption and disorganization, dense packing of collagen fibrils, adhesions, and alterations in fibrillar shape. In addition, there was breakdown of other soft tissue and bony structures.[4]

Obviously, microtrauma, inflammation, and excessive strain on the tendons will go primarily unnoticed in the neuropathic changes. This will lead to further breakdown and ultimate tissue destruction. The end result is changes to the overall structure of the foot as well as collapsing and breakdown of bones, arches, and joints. This causes a change in the overall biomechanical function of the foot.[5]

Currently, it is not known whether diabetes causes the microscopic changes within the tendon or if they are the result of neuropathic changes to the body. However, once they occur, or if equinus is already present, the likelihood of developing ulcerations is significant. If these changes contribute to causing ulcer formation or if they are inhibiting the healing process, they should immediately be addressed.

Consider conservative therapy when appropiate. However, the situation must be tailored to the individual. Often times, in the case of diabetics with equinus deformity, conservative treatment may be futile. If the ulcerations are severe and are clearly the result of a severe restriction of ankle joint dorsiflexion, surgical therapy should be an initial treatment. In less severe cases of equinus, evaluate the patient's age, activity level, and mental status to determine the appropriate treatment.

The aim of conservative therapy is to make accommodations to dissipate the forefoot pressure. If conservative measures such as footwear, orthoses, and pads fail, initiate surgical treatment.

Surgical treatment for this problem involves procedures which have an overall lengthening effect on the posterior muscle group or tendon.[7] Theoretically, any procedure that releases the posterior muscle groups and/or lengthens the tendon would be beneficial in decreasing these focal forefoot/midfoot pressures. When selecting any surgical procedure for a diabetic, consider the patient's ability to heal. Use minimally-invasive procedures whenever possible.

Achilles lengthening procedures would be indicated in the condition of diabetic equinus deformity that is gastroc soleal in nature. Perhaps one of the simplest ways to do this is through the percutaneous method: The patient is placed in a prone position on the table and the tendon is lengthened by way of midline stab incisions to the distal tendon. The blade is placed into the center of the tendon in a parallel orientation to the tendon and is rotated—transecting the fibers. Other procedures including the Z-plasty (either the sagittal or frontal plane) the White, Hoke, Hauser, and Conrad and Frost—are all effective but require a large, open incision.

Endoscopic gastroc recession is a procedure indicated for treatment of gastrocnemius equinus. For this procedure, place the patient in a supine position on the operating table. Make one incision medially approximately 10 cm to 15 cm proximal to the tendon insertion site. This is done by palpating the medial aspect of the gastroc tendon, and the small stab incision is made through the skin and subcutaneous tissue. Blunt dissection is carried out using a blunt fascial elevator to create a channel posteri-

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or to the gastroc aponeurosis, ensuring that the elevator is deep to the sural nerve. Next, insert an obturator into the probe and place in the channel. Remove the obturator and insert into a slotted canula. The scope is then inserted with the slotted

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groove facing anteriorly. At this point, dorsiflexion and plantarflexion of the ankle joint are performed, allowing the surgeon to see the gastroc aponeurosis fibers crossing the slotted canula. Next, the tissues are inspected to ensure that the sural nerve is clear. The slotted canula can be rotated 180° viewing the posterior subcutaneous tissue to look for the sural nerve.

Once the surgeon is certain that the sural nerve is not within the deep tissue of the aponeurosis, the gastrocnemius aponeurosis is cut. Often times, after removing the scope, a 15 blade can be inserted into the incision site to cut the plantaris tendon. This procedure can be performed using either a one or two portal technique. The correction can be immediately observed. This procedure is minimally invasive with only 1 or 2 small stab incisions to heal—thereby decreasing the likelihood of dehiscence and infection. The amount of soft tissue destruction is minimal. In addition, it is performed with the patient in a supine position facilitating any other concomitant procedures needing to be performed. Other gastroc recession procedures will require a large, open incision.

The treatment of this condition depends on the proper diagnosis of the deformity. In our institution, we include equinus testing in all diabetic foot exams. Early recognition aids prevention and provides a more favorable outcome. Prompt surgical intervention in patients will help decrease healing time and minimize the chance of infection and subsequent ulcers. ▮

Lawrence A. DiDomenico, DPM, FACFAS, is the director of podiatric medical education at Northside Medical Center, Youngstown, Ohio.

H. Ryan Hall, DPM, is a first-year resident at Northside Medical Center, Youngstown, Ohio.

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