COACTION PUBLISHING

Recurrence of diabetic pedal ulcerations following tendo-Achilles lengthening

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Foot and ankle surgeons are frequently challenged by the devastating systemic consequences of diabetes mellitus manifested through neuropathy, integumentary and joint breakdown, delayed healing, decreased ability to fight infection, and fragile tendon/ligaments. Diabetic neuropathic pedal ulcerations lead to amputations at an alarming rate and also carry a high mortality rate. This article will discuss causes of diabetic pedal ulcerations that persist or recur after tendo-Achilles lengthening and will highlight areas that need to be addressed by the practitioner such as infection, vascular and nutritional status, glucose control, off-loading, biomechanics, and patient compliance.

Keywords: *diabetic foot*; *tendo-Achilles lengthening*; *ulcer*; *neuropathy*; *equinus*

Diabetic neuropathic pedal ulcerations lead to amputations at an alarming rate and carry a 45%, 5-year mortality rate according to Moulik et al. (1). The ability to efficiently and expeditiously heal a diabetic pedal ulcer will effectively decrease the rate of lower extremity amputations and associated morbidity. Equinus is a well-documented biomechanical cause of increased pedal pressures contributing to the breakdown of friable diabetic integument (2–13). The treatment of choice for gastrocnemius-soleal equinus is often a percutaneous tendo-Achilles lengthening (TAL). The combination of appropriate care and patient compliance will lead to the resolution of most diabetic ulcerations. Unfortunately, there is a small subset of patients that despite appropriate care have pedal ulcerations that recur or fail to resolve.

Physicians should consider previously undiagnosed soft tissue or bone infection as the etiology of recurrent pedal ulcerations in diabetic patients. Ulcerations of any etiology can be complicated by skeletal, connective tissue, or integumentary infection. Diabetic patients have an increased risk of infection secondary to a systemic lack of chemotaxis, phagocytosis, and intracellular bacterial killing (5, 14). Local infection, whether associated with soft tissue or bone, will require surgical intervention to decrease bacterial load and decompress the area. A meta-analysis was conducted by Dinh et al. (15) comparing the usefulness of imaging modalities and the physical examination in accurate diagnosis of osteomyelitis. This review included only studies that confirmed osteomyelitis with histopathologic or microbiologic results via bone biopsy. Bone biopsy remains the gold standard for diagnosis of osteomyelitis. The 'probe to bone test' was the physical exam technique most suggestive of osteomyelitis with a sensitivity of .60 and specificity of .91 (15). Among imaging modalities studied, magnetic resonance imaging (MRI) was shown to be the most accurate with a sensitivity of .90 and specificity of .79. The Indium-111 (IN-111) scan had sensitivity of .74 and specificity of .68. The triphasic bone scan carried a sensitivity of .81 and specificity of .28. From a clinical standpoint and experience, the MRI examination offers little to no definitive evidence in differentiating the origin of bone marrow edema as infection or neuroarthropathy (16). An original article compared fluorodeoxyglucose positive emission tomography (FDG PET) to MRI in diagnosing neuroarthropathy with or without associated infection and found the sensitivity and accuracy to be 100 and 93.8% in FDG PET compared to 76.9 and 75% for MRI, respectively (17). The decrease in sensitivity and accuracy of MRI in the setting of neuroarthopathy and suspected bone infection is secondary to an increase in bone marrow

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signal on T2 and STIR sequences in both acute and subacute phases of neuroarthropathy and pedal osteomyelitis (17). Some authors have recommended adding IN-111 labeled leukocyte scintigraphy to the three-phase bone scan because of low specificity (17, 18). Studies have shown that labeled leukocytes do accumulate in the uninfected neurotrophic joint due in part to the hematopoetically active marrow. The addition of the IN-111 study has increased the specificity in detecting osteomyelitis (18, 19). Combined IN-111 and sulfur colloid scintigraphy is a reliable way to differentiate between hematopoetically active marrow and infection as the cause of leukocyte accumulation in a neuropathic joint. This combination has been shown to be superior to three-phase bone scintigraphy and combined IN-111/bone scintigraphy (18, 19). While the combined IN-111 and sulfur colloid has an accuracy of 95% in diagnosing neuropathic joint complicated by infection, the three-phase bone scan offers the advantage of increased anatomic resolution (18).

The senior author recommends the three-phase bone scan in combination with IN-111 scintigraphy (4 and 24 hr images) as well as sulfur colloid scintigraphy in certain situations. The three-phase bone scan permits for anatomic resolution that the other two nuclear scans lack, thus allowing for the greatest accuracy and highest success. Though imaging modalities are useful, bone biopsies are suggested in recurrent ulcerations where clinical and diagnostic testing were not diagnostic for infection. It is our experience that bone biopsy often reveals the presence of infection in cases of recurrent ulcerations where all possible etiologies have been addressed. Literature has shown that a bone biopsy sent to microbiology or histopathology carries the same sensitivity for diagnosis of osteomyelitis (20). Specimens sent to microbiology have the advantage of providing helpful information regarding antibiotic selection. It is the authors' current practice to take two bone biopsies from the area in question and send one specimen to microbiology and one specimen to histopathology.

Diabetic patients with pedal ulcerations are often found to have an associated ankle equinus deformity. Ankle equinus was defined by Root et al. (21) as <10 degrees of dorsiflexion at the level of the ankle with the knee extended and subtalar joint in neutral position . The etiology of diabetic tendo-Achilles equinus is usually related to a decrease in elasticity of diabetic tendons secondary to collagen cross-linking (22–24). Tensile strength is also compromised because of collagen cross-linking and predisposes diabetic patients to tendon failure. Practitioners should take into consideration this process of continued tendon glycosylation in patients with recurrent ulcerations following an appropriate TAL. Collagen and the non-enzymatic glycosylation of collagen are topics of interest when discussing diabetes and associated end-organ dysfunction. The process of non-enzymatic reactions resulting in the formation of complex pigments and protein–protein cross-linking is called glycation (22). The formation of advanced glycated end products (AGE) is responsible for the cross-linking of collagen. This has deleterious effects on skin, joint mobility, wound healing, arterial elasticity, bone formation and composition, lung expansion volume, and within the cornea (22, 23). Over time AGEs accumulate and cause functional impairment of affected tissues as the structural properties of collagen are altered. Glycation-induced collagen cross-linking is strongly associated with alterations in the biomechanical properties of tendinous structures. These alterations lead to the loss of flexibility, elasticity, and increased brittleness (22). Electron microscopic examination of the Achilles tendon harvested from neuropathic diabetic patients affected by neuroarthropathy revealed abnormal collagen fibril morphology, increased packing density of collagen fibrils, and decreased fibril diameter (22, 23). This was in direct comparison to the Achilles tendons harvested from diabetics not suffering from pedal neuroarthropathy. Tendo-Achilles equinus is either secondary to collagen cross-linking or a result of anterior leg paresis secondary to motor neuropathy, giving a mechanical advantage to the posterior musculature (22–24). A talo-tibial exostosis can be a cause of ankle joint pseudo-equinus and should be ruled out in diabetic patients with a non-healing or recurrent pedal ulceration. Gastrocnemius and gastrocsoleal equinus are the main perpetrators of increased pedal pressures during static and dynamic phases of gait. The Silfverskiold test is utilized once a posterior or ankle joint equinus has been identified and osseous ankle equinus has been ruled out with radiological examination (25). The TAL is often performed as an isolated or an adjunctive procedure in the treatment of pedal ulcerations associated with gastrocnemius-soleal equinus. Those patients that are found to have isolated gastrocnemius equinus may benefit from a gastrocnemius recession alone.

Mueller et al. (9, 10) described primary and secondary benefits to a TAL procedure when <5 degrees of dorsiflexion is quantitatively measured in a neuropathic diabetic patient with recurrent ulceration. The primary benefit of the TAL with total contact casting (TCC) compared to TCC alone is a 75% reduction in recurrent ulceration at 8 months and 53% at 2 years (10). This study also concluded that an acute 27% reduction in forefoot pressures during ambulation and an obvious increase in ankle joint dorsiflexion were two additional secondary benefits gleaned from a TAL. This coincides with other studies that show TCC is effective in aiding the healing of a plantar diabetic ulceration, but fairly ineffective in reducing the rate of recurrent ulceration (9, 10). Lin et al. (26) described 14 diabetic neuropathic

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patients who reported no recurrent ulcerations 17 months following a TAL. A TAL reduces total plantarflexor torque by 37% and shifts the peak torque toward dorsiflexion. Plantarflexor torque normalizes around 8 months after the patient is no longer immobilized without an associated reduction in peak torque toward dorsiflexion. This was the first study to evaluate the effects of a TAL prospectively and, as mentioned above, showed there is only a temporary decrease in active and passive plantar flexor muscle performance (9, 10). The first reported percutaneous TAL was performed by Delpech in 1816. In 1931 Hoke described a triple hemisection of the Achilles tendon through one 2-cm incision in the frontal plane with the most proximal and distal cuts exiting posteriorly and the central cut exiting anteriorly (27). Today the percutaneous triple hemisection is most often performed in the transverse plane, however not without risks. Those risks most readily identified are weak plantarflexion, neurovascular injury, musculotendious injury, Achilles tendon rupture, and Achilles tendon over lengthening or under lengthening (9, 28, 29). The anatomic relationships that were found to be the most at risk were associated with cut number two (7.9 mm to sural nerve) and three (5.8 mm to FHL and 8.3 mm to tibial nerve) (29). A study of percutaneous TAL performed by Salamon et al. (28) on 15 cadavers, measured the accuracy of the three cuts. Overall surgeon accuracy was relatively high. The widths of the tendon at the level of cuts one, two, and three from distal to proximal were found to average 61, 50, and 55%, respectively. This success was challenged by Hoefnagels et al. (29). They found that one-third of their hemisections resulted in failure to lengthen or in incomplete transection. Salamon performed the TAL in a prone position, while most surgeons today perform this procedure as Hoefnagels did with the patient in a supine position and leg elevated. Patients who have ulcers that fail to heal after a TAL need to be assessed for a recurrent equinus deformity secondary to either incomplete triple hemisection or continued Achilles tendon glycation. Performing another percutaneous triple hemisection may not be the most beneficial in these types of patients. After a percutaneous TAL, the tendon often fibroses and with continued glycosylation of the tendon this creates an environment not likely to allow for sliding within the tendon complex. The open frontal plane z-technique for Achilles tendon lengthening will allow for a visualized controlled lengthening of the gastrocnemius-soleal complex (Fig. 1).

Nutritional status of a patient is a vital component of wound healing that is often overlooked in the multidisciplinary, multifaceted approach to healing diabetic pedal ulcerations. Malnutrition in hospitalized patients is well documented throughout literature with rates as high as 50%. Jensen et al. (30) showed an average incidence of clinical and subclinical malnutrition of 42.4% in patients undergoing orthopedic procedures. This is especially true in surgical patients where increased stress causes protein and energy requirements to go through a hypermetabolic catabolic state (30). Malnutrition causes immune system dysfunction by impairing complement activation and production, thus decreasing the function of lymphocytes, macrophages, and neutrophils. This impairment of humoral and cell-mediated immunity makes the patient

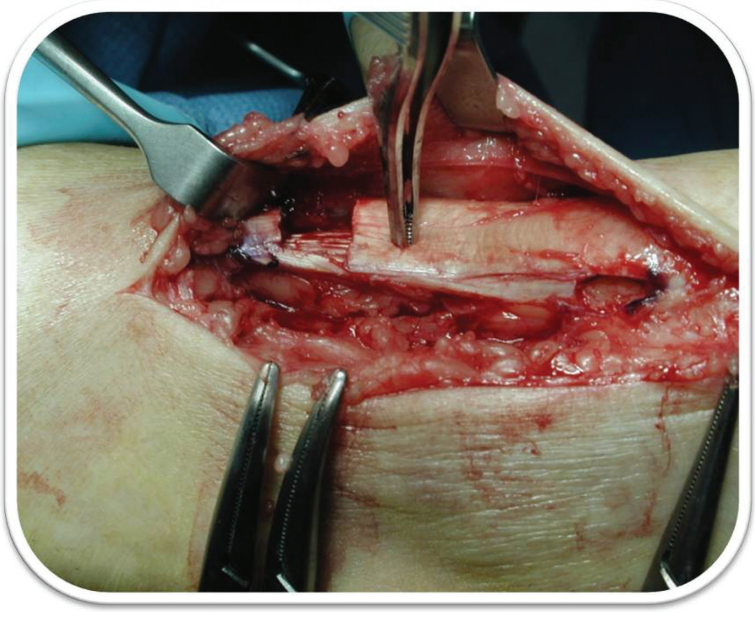

Fig. 1. This picture demonstrates an open, frontal plane, Z-lengthening of the Achilles tendon. The most distal cut, to the right of the picture, is directed anterior while the proximal cut, to the left, is directed posterior.

more susceptible to infection (3, 14). Laboratory analysis of malnutrition is as important as your general physical examination. Visceral-protein depletion can be identified by evaluating levels of serum albumin, transferrin, or prealbumin (5, 16). These three serum markers of protein status have different half-lives; thus, each with their own benefit. Serum albumin has the longest half-life of 18–20 days. Hypoalbuminemia of <2.2 g/dl is a marker of a negative catabolic state and a predictor of poor outcome. Serum transferrin has a half-life of 8–9 days and assesses protein status over the past 2–4 weeks (16). This lab value is an accurate evaluation of nutritional status only in lieu of normal serum iron levels. The third screening test, prealbumin, has the shortest half-life of 2–3 days. Prealbumin is the least helpful in assessing overall nutritional status. Hypoalbuminemia may adversely alter wound healing by affecting intravascular oncotic pressure and amino acid transport from the liver. Albumin plays the role of an amino acid donor for extra-hepatic tissue synthesis and a zinc transporter. Zinc plays a vital role in collagen cross-linking (14, 20). A useful adjunct to malnutrition screening is the total number of lymphocytes per cubic millimeter, and this speaks to a patient's immunocompetence (14). An 'instant nutritional assessment' can be performed with evaluation of albumin and lymphocytes (14). The diagnosis of hypoalbuminemia and lymphopenia identifies malnourished patients with poor potential for wound healing, impaired cellular defense, and increased susceptibility to infection (30). These authors found that when low serum albumin was noted in conjunction with lymphopenia a fourfold increase in complications and 20-fold increase in death was seen. A mortality rate of 62% was noted in those whose albumin levels fell to 2.0 g/dl (30). Recommendations made by Jensen and colleagues are that elective cases should be delayed if serum albumin is <3.4 g/dl or the total lymphocyte count is $<1{,}500$ cells/mm^3 (30, 31). Though not strongly supported through literature, some vitamins and minerals such as iron, copper, zinc, vitamin A, vitamin C, and

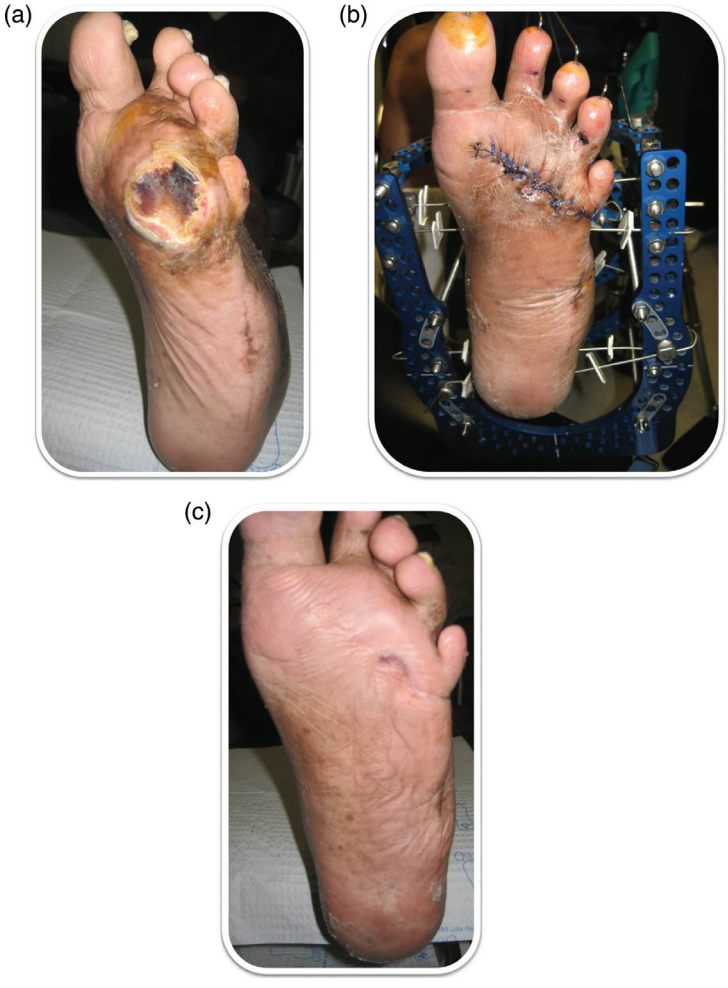

Fig. 2. A pre-operative clinical picture (a) showing the left foot with a large plantar ulceration after a failed tendo-Achilles lengthening and metatarsal resection and ulcer debridement. A post-operative picture (b) showing the external fixation device and local flap closure to address the skeletal deformity and large open wound. Final post-operative picture at 2-year follow-up (c).

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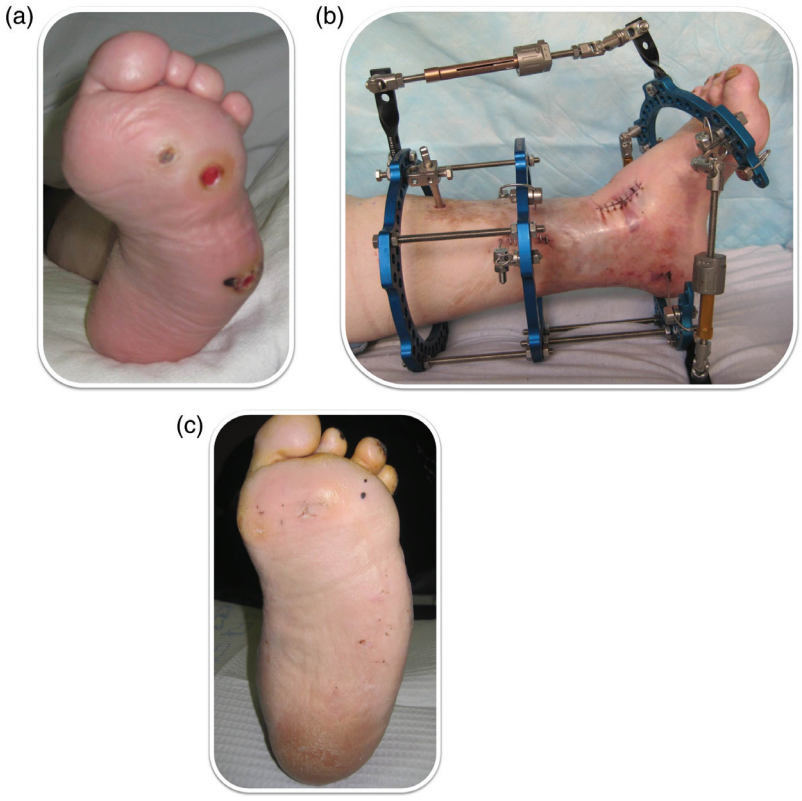

Fig. 3. A pre-operative clinical picture (a) showing the left foot with a multiple plantar ulcerations after a failed tendo-Achilles lengthening and ray amputation. Post-operative picture (b) showing the external fixation device and soft tissue realignment procedures to address the recurrent ulcerations. Final post-operative picture at approximately 2-year follow-up (c).

vitamin B complexes are thought to play a role in collagen synthesis and aid in wound healing (5). It is the authors recommendation to assess nutritional status via albumin, as other screening tests have many variables. Nutritional replacement therapy can be monitored with prealbumin as this will show level of compliance and response to treatment.

Vascular disease in the diabetic patient is thought to result from abnormalities of cellular signal transduction, cell membrane fluidity, and changes in oxidative stress (5). Nitric oxide is an important cell mediator that interferes with monocyte and leukocyte adhesion to the endothelium, platelet vessel wall interaction, smooth muscle proliferation, and vascular tone. These processes are paramount in the development of atherosclerosis. Diabetic patients often have an intrinsic dysfunction of nitric oxide (5). Hyperglycemia impacts diabetic circulation by decreasing sympathetic activity. This causes a decrease in precapillary resistance and an increase in capillary flow and pressure, resulting in capillary basement membrane thickening (5). These changes have a deleterious effect on autoregulation causing decrease blood flow, thus microangiopathy. Decreased oxygen tension will cause wound bed necrosis. Dying cells release endotoxins that prevent fibroblasts and keratinocytes from reaching the wound site. It should be common practice to perform a thorough physical examinations on all diabetics and further assess vascular status with non-invasive means if non-palpable pedal pulses are encountered.

Assessments such as arterial duplex, ankle brachial indexes, and diagnostic angiography may be warranted in patients suspected of macrovascular compromise. Practitioners should consider microvascular disease as an etiology of recurrent ulcerations in diabetics who do not have documented macrovascular disease. If palpable pedal pulses are present or no macrovascular disease is found, then further microvascular analysis should be performed through transcutaneous partial pressure of oxygen testing. This will assess whether or not hyperbaric therapy will be beneficial. Practitioners may also consider rheologic therapy to help aid in treatment of microvascular disease.

Many clinical factors can be aggressively controlled. However, the inappropriate or inadequate treatment a patient receives at home or at a nursing facility can be overlooked. Patient non-compliance must also be considered. Therapies that are appropriate for a clinical presentation may lead to failure if a patient or caregiver is unwilling or incapable of performing the prescribed therapy. Evaluation of a patient's home environment and quality of care is important to appropriately assess physical inability or patient/care giver unwillingness to

provide the prescribed care. If the patient is already in a nursing facility, evaluation of the facility compliance to prescribed care may be warranted.

Conclusion

Diabetic patients who have recurrence of their pedal ulceration following TAL need to be reassessed. Several studies have noted that the strongest predictor of complete healing of a diabetic ulceration is the 4-week percentage change in wound area (3, 5, 8, 11). Local wound care must be combined with adjuvant therapies to achieve this 4-week goal. Persistent ulcerations after surgical intervention need to be assessed for multiplanar deformities, and if noted need to be surgically addressed. (Figs. 2 and 3). The value of strict glucose control can never be underestimated and should not be an understated aspect of our day-to-day interactions with diabetic patients. Foot and ankle surgeons should be advocating strict glucose control through direct communication with patient's primary care physician or endocrinologist. In our experience, a patient with glycosylated hemoglobin of $>8\%$ and an average daily glucose of >250 mg/dl will experience prolonged wound healing. The practitioner should suspect previously undiagnosed or recurrent bone or soft tissue infection, vascular compromise, noncompliance, hypoalbuminemia, hyperglycemia, boney prominence, and recurrence of equinus contracture when dealing with recurrent diabetic pedal ulcerations.

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References

1. Moulik DK, Mtonga R, Gill GU. Amputation and mortality in new-onset diabetic foot ulcers stratified by etiology. Diabetes Care 2003; 26: 491–4.
2. Batista F, Nery C, Pinzur M, Monteiro AC, de Souza EF, Felippe FH, et al. Campos RS. Achilles tendinopathy in diabetes mellitus. Foot Ankle Int 2008; 29: 498–501.
3. La Fontaine J, Brown D, Adams M, VanPelt M. New and recurrent ulcerations after percutaneous Achilles tendon lengthening in transmetatarsal amputation. J Foot Ankle Surg 2008; 47: 225–9.
4. Greenhagen RM, Johnson AR, Peterson MC, Rogers LC, Bevilacqua NJ. Gastrocnemius recession as an alternative to tendoachillis lengthening for relief of forefoot pressure in a patient with peripheral neuropathy: a case report and description of a technical modification. J Foot Anke Surg 2010; 49: 159.e9–13.
5. Guyton GP, Saltzman CL. The diabetic foot: basic mechanisms of disease. Instr Course Lect 2002; 51: 169–81.
6. Mueller MJ, Sinacore DR, Hastings MK, Strube MJ, Johnson JE. Effects of a tendo-Achilles lengthening procedure on muscle function and gait characteristics in a patient with diabetes mellitus. J Orthop Sports Phys Ther 2000; 30: 85–90.
7. Salsich GB, Mueller MJ, Hastings MK, Strube MJ, Johnson JE. Effect of Achilles tendon lengthening on ankle muscle performance in people with diabetes mellitus and a neuropathic plantar ulcer. Phys Ther 2005; 85: 34–43.
8. Laborde JM. Neuropathic toe ulcers treated with toe flexor tenotomies. Foot Ankle Int 2007; 28: 1161–3.
9. Mueller MJ, Sinacor DR, Hastings MK, Lott DJ, Strube MJ, Johnson JE. Impact of Achilles tendon lengthening on functional limitations and perceived disability in people with a neuropathic plantar ulcer. Diabetes Care 2004; 27: 1559–64.
10. Mueller MJ, Sinacore DR, Hastings MK, Strube MJ, Johnson JE. Effect of Achilles tendon lengthening on neuropathic plantar ulcers: a randomized clinical trial. J Bone Joint Surg Am 2003; 85: 1436–45.
11. Sheehan P, Jones P, Caselli A, Giurini JM, Veves A. Percent change in wound area of diabetic foot ulcers over a four week period is a robust predictor of complete healing in a 12-week prospective trial. Diabetes Care 2003; 26: 1879–82.
12. Willrich A, Angirasa AK, Sage RA. Percutaneous tendo-Achilles lengthening to promote healing of diabetic plantar foot ulceration. J Am Podiatr Med Assoc 2005; 95: 281–4.
13. Wukich DK. Current concepts review: diabetic foot ulcers. Foot Ankle Int 2010; 31: 460–7.
14. Dickhaut SC, DeLee JC, Page CP. Nutritional Status: importance in predicting wound-healing after amputation. J Bone Joint Surg 1984; 66: 71–5.
15. Dinh MT, Abad CL, Safdar N. Diagnostic accuracy of the physical examination and imaging tests for osteomyelitis underlying diabetic foot ulcers: meta-analysis. Clin Infect Dis 2008; 47: 520–7.
16. Tess A, Lipman TO, Collins KA. Nutritional issues in the surgical patient. UpToDate. September 2010; 1–13.
17. Basu S, Chryssikos T, Houseni M, Malay S, Shah J, Zhuang H, et al. Potential role of FDG PET in the setting of diabetic neuro-osteoarthropathy: can it differentiate uncomplicated Charcot's neuroarthropathy from osteomyelitis and soft-tissue infection? Nucl Med Commun 2007; 28: 465–72.
18. Palestro CJ, Mehta HH, Patel M, Freeman SJ, Harrington WN, Tomas MB, et al. Marrow versus infection in the Charcot joint: indium-111 leukocyte and technetium-99m sulfur colloid scintigraphy. J Nucl Med 1998; 39: 346–50.
19. Schauwecker DS, Park HM, Mock BH, Burt RW, Kernick CB, Ruoff AC, et al. Evaluation of complicating osteomyelitis with Tc-99m MDP, In-111 granulocytes, and Ga-67 citrate. J Nucl Med 1984; 25: 849–53.
20. Weiner RD, Viselli SJ, Fulkert KA, Accetta P. Histology versus microbiology for accuracy in identification of osteomyelitis in the diabetic foot. J Foot Ankle Surg 2011; 50: 197–200.
21. Root ML, Orien WP, Weed JH. Clinical biomechanics: normal and abnormal function of the foot, vol. 2. Los Angeles, CA: Clinical Biomechanics Corp; 1977, pp. 37–41.
22. Reddy GK. Cross-linking in collagen by nonenzymatic glycation increases the matrix stiffness in rabbit Achilles tendon. Exp Diabesity Res 2004; 5: 143–53.
23. Murugan P. Tanner's Cassia (*Cassia auriculata* L.) extract prevents hemoglobin glycation and tail tendon collagen properties in experimental diabetic rats. Cell Tissue Res 2010; 10: 2109–14.
24. Grant WP, Foreman EJ, Wilson AS, Jacobus DA, Kukla RM. Evaluation of Young's modulus in Achilles tendons with diabetic neuroarthropathy. J Am Podiatr Med Assoc 2005; 95: 242–46.
25. Silferskiold N. Reduction of the uncrossed two-joints muscles of the leg to one-joint muscles on spastic conditions. Acta Chir Scand 1924; 56: 315–30.
26. Lin SS, Lee TH, Wapner KL. Plantar forefoot ulceration with equinus deformity of the ankle in diabetic patients: the effect of

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tendo-Achilles lengthening and total contact casting Orthopedics 1996; 19: 465–75.
27. Hoke M. An operation for the correction of extremely relaxed flatfeet. J Bone Joint Surg 1931; 13: 773–83.
28. Salamon ML, Pinney SJ, Van Bergeyk A, Hazelwood S. Surgical anatomy and accuracy of percutaneous Achilles tendon lengthening. Foot Ankle Int 2006; 27: 411–13.
29. Hoefnagels EM, Waites MD, Belkoff SM, Swierstra BA. Percutaneous Achilles tendon lengthening: a cadaver-based study of failure of the triple hemisection technique. Acta Orthop 2007; 78: 808–12.
30. Jensen JE, Jensen TG, Smith TK, Johnston DA, Dudrick SJ. Nutrition in orthopaedic surgery. J Bone Joint Surg Am 1982; 64: 1263–72.
31. Gray D, Cooper P. Nutrition and wound healing: what is the link? J Wound Care 2001; 10: 86–9.

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