Complications of the Diabetic Foot

Paul J. Kim, DPM, MS, John S. Steinberg, DPM*

INTRODUCTION

The foot is a complex structure that requires delicate and deliberate orchestration for normal weight bearing and ambulation. The foot and ankle serve the dual role of being able to adapt to the ground to absorb shock as well as becoming a rigid lever necessary for forward propulsion. The result of altered biomechanics is disability and deformity. The diabetic foot is particularly at risk for complications because of its inability to tolerate stress. Prolonged uncontrolled blood glucose imparts deleterious effects on all structures related to the foot and ankle, including the skin and subcutaneous tissue, nerve, blood vessels, fascia, ligaments, tendons, muscle, and bone. The diabetic foot follows a common pathway that begins with a small ulcer or surgical wound and

Disclosures: None.
Department of Plastic Surgery, Georgetown School of Medicine, Center for Wound Healing & Hyperbaric Medicine, MedStar Georgetown University Hospital, 3800 Reservoir Road Northwest, Washington, DC 20007, USA
* Corresponding author.
E-mail address: JSS5@gunet.georgetown.edu

KEYWORDS
- Diabetes
- Ulcer
- Wound
- Amputation
- Infection
- Multidisciplinary

KEY POINTS
- A discussion should be had between the physician and the patient regarding the relationship between glucose control and complications encountered in the foot and ankle.
- Peripheral neuropathy and peripheral vascular disease create an environment that will lead to ulceration and possible amputation.
- Calluses are a sign of impending ulceration and should be debrided; the underlying cause should be addressed surgically or through offloading in a brace or shoe.
- Infection should be treated aggressively with culture-sensitive oral or parenteral antibiotic therapy, topical antimicrobials, and/or surgical intervention.
- Residual foot function should be considered when amputation is considered to prevent new ulcer formation or ulcer recurrence.
- A multidisciplinary team approach is vital to the prevention and treatment of the diabetic foot.

Introduction

The foot is a complex structure that requires delicate and deliberate orchestration for normal weight bearing and ambulation. The foot and ankle serve the dual role of being able to adapt to the ground to absorb shock as well as becoming a rigid lever necessary for forward propulsion. The result of altered biomechanics is disability and deformity. The diabetic foot is particularly at risk for complications because of its inability to tolerate stress. Prolonged uncontrolled blood glucose imparts deleterious effects on all structures related to the foot and ankle, including the skin and subcutaneous tissue, nerve, blood vessels, fascia, ligaments, tendons, muscle, and bone. The diabetic foot follows a common pathway that begins with a small ulcer or surgical wound and
terminates in limb loss. This article discusses key complications in the diabetic foot and ankle caused by peripheral neuropathy, peripheral vascular disease, and soft-tissue and bone deformity that contribute to ulceration, infection, and amputation (Fig. 1). Treatment options are mentioned but are not discussed in great detail, the principal focus being the recognition and understanding of the complications unique to the diabetic foot.

RISK FACTORS

Peripheral Neuropathy

A spectrum of peripheral neuropathy encountered in the lower extremity affects up to 66% of patients with diabetes.1–3 The type of peripheral neuropathy experienced in the diabetic patient encompasses both large and small nerve fibers, with size describing the relative degree of myelination. Small nerve fiber disease is classically related to the sensation of pain experienced in the diabetic patient. Furthermore, small nerve fiber disease disrupts temperature discrimination and autonomic function. In the early stages of peripheral neuropathy, patients may present with burning, tingling, radiating pain beginning at the toes and progressing proximally on the foot and leg. The pain may increase during periods of elevated blood glucose and decrease with better control of blood glucose.4,5 Although anticonvulsant medications (gabapentin, pregabalin), vitamin supplementation (B, folic acid, thiamine), and surgical decompression have been used for the treatment of peripheral neuropathy, none have consistently shown an ability to restore sensory loss. As peripheral neuropathy advances, the patient will become insensate with a noted loss of protective sensation distally in the extremities. This problem is irreversible, and can significantly elevate the risk for limb loss in the patient with diabetes. The patient is unable to detect trauma

Fig. 1. Plantar neuropathic ulceration in a patient with diabetes. Note the fibrotic white base with lack of granulation tissue.
to the foot and thus does not respond by protecting or treating the area. Hence, even a smaller blister can progress to a chronic ulcer because of the delay in care. The peripheral neuropathy experienced by the diabetic patient encompasses other types of nerve dysfunction, including the loss of proprioception (position sense) and motor control. The decrease in proprioception can lead to falls and difficulties in ambulation. Peripheral motor neuropathy can also lead to ambulation difficulties via muscle atrophy and lack of muscle coordination needed for steady ambulation. All of these challenges can contribute to increased and uneven weight-bearing pressure on the foot, which results in a heightened risk for nonhealing wounds and infections (Fig. 2).

**Peripheral Vascular Disease**

Adequate perfusion is fundamental to tissue repair and regeneration. Diabetes-induced peripheral vascular disease affects both small and large vessels in the lower extremities. The 3 large vessels that deliver arterial blood to the foot are the posterior tibial artery, anterior tibial artery, and the peroneal artery. Specific areas of the foot (angiosomes) correspond to 1 of these 3 arteries. With advancing diabetic disease, 1 or all of the arteries may be compromised. In the person with diabetes and peripheral vascular disease there is often focal or long-segment stenosis or occlusion, as seen on angiography of the lower extremity. There are redundant arterial pathways through perforator vessels that connect arteries to arteries. Furthermore, collateral vessels are formed to bypass areas of significant disease. However, even partial compromise of 1 of the major vessels can lead to chronic ulcerations with poor healing potential and tissue loss. A hand-held Doppler examination is the first means of detecting perfusion problems. If signals are any less than triphasic, assessment of segmental pressures (ankle brachial index [ABI]) should be conducted, followed by a vascular consultation. An ABI of less than 0.50 in a chronic ulcer environment has

**Fig. 2.** Posterior neuropathic heel ulceration with ischemia caused by pressure and poor shoe gear.
a high likelihood of amputation. An angiogram may reveal significant macrovascular disease requiring intervention, including angioplasty or open bypass. Microvascular disease may also develop in diabetic patients with altered local blood flow and dysregulation of vascular tone. Hence, successful macrovascular intervention does not necessarily correlate with adequate perfusion to the tissue.

It is important to identify early signs of a vascular compromised limb before ulcer formation. The foot may appear atrophic, evidenced by the lack of hair growth, cool temperature of the limb, and thin atrophic and shiny skin. Patients may also complain of pain in the limb as the ischemia progresses, caused by oxygen and nutrient deprivation of the tissue. This environment places the foot and ankle at risk for ulceration. Once an ulcer forms it is important to determine the degree of perfusion loss to that area of the foot or ankle. Indirect blood flow to the area of ulceration may be sufficient to heal the ulcer but may take a long time to do so, placing it at risk for infection or increasing depth. Rapid recognition of compromised blood flow and optimization of perfusion to the affected limb will support ulcer healing (Fig. 3).

**Soft-Tissue and Bone Deformity**

Inherent changes in the quality of soft tissue predispose the diabetic foot to a destructive pathway. It is important to consider 2 important forces experienced by the foot

Fig. 3. (A) Forefoot gangrene with osteomyelitis and abscess in patient with diabetes and prior partial forefoot amputation. (B) Intraoperative view showing deep tissue necrosis and abscess extending to plantar tissues. (C) Status post open midfoot amputation.
during ambulation. The first is direct sagittal plane force experienced on the plantar aspect of the foot during heel strike and forefoot push-off. The force experienced is in a relatively confined surface area of the plantar foot. There is a direct correlation between peak sagittal plantar forces and ulcer location.\textsuperscript{15,16} The second is a lesser understood force called shear, which is either a transverse force (eg, side to side or front to back) or frontal-plane force (rotational).\textsuperscript{17,18} These shear forces are experienced between the foot and the ground or foot and the inside of the shoe. This type of force produces blister formation whereby the epidermis separates from the dermis. In the healthy foot both the peak plantar pressure and shear forces are quickly dissipated, which the soft tissue is able to tolerate. The environment of the diabetic foot is profoundly different. The epidermis is often dry, making it susceptible to tears and fissuring. Furthermore, there is atrophy of subcutaneous tissue, which negatively affects its ability to absorb shock during ambulation.\textsuperscript{19,20}

The tendons and ligaments become stiff and lose their elasticity.\textsuperscript{21,22} Tendon contracture produces deformities such as hammertoe, which increase the likelihood of distal toe ulcers.\textsuperscript{23} Equinus deformity caused by Achilles tendon contracture can increase plantar forefoot pressures, creating wounds on the plantar forefoot.\textsuperscript{16} Biomechanical surgical correction of tendon contractures can alleviate many of these weight-bearing pressures. Flexor tenotomies of the toes and tendo-Achilles lengthenings can correct these abnormalities.\textsuperscript{24,25} Thus the combination of compromised skin-related structures and tendon contractures places the foot at high risk for ulceration (Fig. 4).

Bony deformity is also encountered in the diabetic foot. Standard weight-bearing radiographs are sufficient to appreciate many of these changes. Any bony prominences

\textbf{Fig. 4.} Severe forefoot contracture with joint and tendon deformity causing plantar pressure and ulceration in a patient with diabetes and neuropathy.
cause increased pressure against the shoe or ground, which may result in ulcer formation or chronicity.26,27 These bony prominences can be the result of instability of joints in the foot or frank subluxations/dislocations. Repetitive minor trauma or a discrete traumatic event may be the cause. Patients with later-stage peripheral neuropathy may not notice the change in foot structure and continue to ambulate, causing further subluxation/dislocation. These deformities require conservative offloading or surgical reconstruction. Conservative offloading involves the use of offloading camwalkers, total-contact casts, or CROW (Charcot Restraint Orthotic Walker) devices during the early stages of the deformity. Once the deformity has stabilized and no longer progresses, multidensity inserts in customized shoes or braces that offload and realign the foot and ankle are necessary. Surgical reconstruction involves exostectomies (removal of prominences) or realignment and arthrodesis of joints using screws, plates, rods, or external fixation. If the joint deformity is not manageable using shoes or braces, the surgical option should be considered.

A limb-threatening example of joint deformity is Charcot neuroarthropathy. Overt or subtle trauma may trigger a cascade of events that culminates in fractures, subluxations, and dislocations of the bones in the foot or ankle. Charcot foot is characterized by edema, erythema, and calor. During the acute stages of Charcot neuroarthropathy there is gross instability of the affected joints. Immediate non-weight bearing is critical to prevent further collapse. If left untreated, these fracture dislocations will proceed rapidly to ulceration and bone infection. After the Charcot neuroarthropathy deformity has consolidated, bony prominences develop, causing areas of high pressure resulting in ulcerations. Surgical reconstruction is not necessary if the deformity is shoeable or braceable (Fig. 5).

Fig. 5. Plantar midfoot ulceration with osteomyelitis caused by Charcot neuropathic osteoarthropathy.
Peripheral neuropathy, peripheral vascular disease, and bony deformity set the stage for ulceration. Approximately 15% of diabetic patients will develop ulceration over their lifetime. The diabetic foot ulcer is typically located on the plantar aspect of the foot. Other common areas include the medial first metatarsal phalangeal joint, lateral aspect of the fifth metatarsal phalangeal joint, and the posterior calcaneus. All these locations are areas of higher pressure. Other chronic ulcerations related to diabetes may result from elective or nonelective surgical incisions. The diabetic foot ulcer typically evolves over time. Hyperkeratosis (callus) is the precursor to ulceration, increasing the relative risk 11-fold. The skin reinforces areas of pressure or shear with layers of dense stratum corneum. Hyperkeratosis is counterproductive because these areas experience even greater pressure. Hence, frequent debridement is needed to remove this tissue and to uncover underlying hidden ulcers or infection. Multidensity inserts with extra-depth, wide-toebox shoes are necessary to more evenly distribute pressure and reduce shear forces.

The classic definition of a diabetic foot ulcer implies some level of peripheral neuropathy. Patients continue to ambulate on the ulcer site because they lack the ability to perceive pain. Thus the ulcer continues to enlarge and begins to penetrate to deeper layers of tissue. However, diabetic foot ulcers can also be classified as ischemic, neuropathic, or decubitus wounds. For example, a diabetic patient may develop a posterior heel ulcer from prolonged pressure while being confined to bed. This patient may also have peripheral neuropathy and peripheral vascular disease. In this case the ulcer is most appropriately described as a decubitus ulcer. However, it can also be described as a diabetic foot ulcer, ischemic ulcer, or a neuropathic ulcer. Furthermore, there are wound-classification schemes that help describe the wound in relationship to depth of the ulcer, presence of infection, and ischemia.

The duration and size of the ulcer relates directly to healing potential. Full-thickness or deeper wounds of longer than 2 months’ duration are 79% less likely to heal. A chronic ulcer is defined as a wound that does not decrease in size by 50% in 1 month. Measurements (length × width × depth) of the wound should be taken every 1 to 2 weeks to track changes in wound size. Photographs should also be taken at every visit to remind the clinician of the appearance of the wound on prior visits. The wound should be fully explored, including all areas of undermining, and tunneling should be explored for any pus pockets or communication to bone or hardware (eg, internal screws or plates). The wound edges may have a rolled appearance indicating a chronic ulcer state. Senescent cells are found in the base and perimeter of the wound, preventing active wound healing and repair. Nonviable tissue (necrotic and fibrotic) may be evident in the wound bed, delaying wound healing. The wound with healing potential contains predominantly granulation tissue (bed of capillaries). The quality of the surrounding tissue should also be evaluated. Typically all diabetic foot ulcers have some level of serous drainage unless there is an active infection, in which case purulence may be present. Maceration about the surrounding tissue indicates drainage causing the tissue to be friable, which impedes wound healing.

Treatment of a diabetic foot ulcer involves a multimodal approach that includes conservative and surgical interventions. Paramount to ulcer healing is glucose control, which includes medical management, dietary/nutritional control, and exercise. Regarding the direct care of the diabetic ulcer, there are 4 fundamental treatment principles: optimization of perfusion (see previous discussion), biofilm/infection control,
debridement, and offloading. Biofilm/infection control is discussed in detail in the next section. This section focuses on debridement and offloading.

Numerous purported effective therapies for diabetic foot ulcers exist. There are numerous claims that dressings, ointments, solutions, cellular and/or tissue-based products, and other topical therapies heal diabetic foot wounds. However, there is a paucity of evidence to support that any of these therapies is superior to another.36 There is a role for some of these therapies, but none assist in healing wounds without addressing the 4 aforementioned treatment principles. For example, dressings can be an effective adjunctive treatment by facilitating the removal of drainage from the wound site. Furthermore, wound-healing ointments (antimicrobial, growth-factor impregnated) and solutions (cleansers, antiseptics) can promote a healthy wound environment. Bioengineered alternative tissues can include either xenografts or allografts that are impregnated with living cells or are acellular matrices.37 Cell-impregnated allografts potentiate wound healing by introducing cells that secrete growth factors that activate native cells. Acellular matrices serve as scaffolds for organized migration of native cells. Although bioengineered alternative tissues promote wound healing, they should not be viewed as the sole agent for wound healing.

Debridement serves multiple functions that promote wound healing.38,39 First, debridement removes detritus, foreign material, and nonviable tissue, and activates senescent cells by creating acute trauma to the wound bed and perimeter. Second, it removes infectious material associated with planktonic bacteria or biofilm. Debridement includes enzymatic debridement using collagenases, mechanical debridement using wet-to-dry dressing changes or whirlpool therapy, and biological debridement via maggot therapy. These techniques require a long duration of treatment and are not efficient in isolation. The preferred method is sharp debridement, which includes clinic-based debridement and surgery-based excisional debridement. Clinic-based debridement involves the use of scalpels, scissors, and curettes. Clinic-based debridement is constrained by the inability to aggressively debride tissue, owing to pain and the inability to control excessive bleeding. Furthermore, the clinic is not a sterile environment, with cross-contamination a real possibility. Surgery-based excisional debridement is performed in the sterile environment of an operating room, where more aggressive removal of infectious material and nonviable tissue can be conducted. However, there is expense related to surgery as well as the risks inherent with anesthesia. A combination of debridement strategies that includes all of the modalities described here should be used. Wounds should generally be debrided in the clinic with every visit. Enzymatic and mechanical debridement can be performed between clinic visits. Once the wound is sufficiently prepared in the clinic, the patient can be taken to the operating room for excisional debridement, after which the wound can be closed or covered with a split-thickness skin graft. Large soft-tissue defects require local or free tissue flaps for closure and/or coverage of deep structures and bone.

Offloading is of critical importance to successful ulcer healing, and is also perhaps the most difficult aspect of wound healing. As discussed earlier, diabetic foot ulcers are typically located on the plantar aspect of the foot. Hence, the patient with an insensitive foot is likely to continue to bear weight on the affected limb. Wheelchairs, crutches, and wheeled single-limb offloading platforms are some options that may be used to completely offload a limb. Although the ideal situation is that the patient remains completely non-weight bearing, this is an unrealistic expectation. Patients will continue to bear weight on the ulcer area. Therefore, devices are needed to allow the patient to bear weight on the affected extremity while still offloading the ulcer. The use of “donut-shaped” cutouts from felt or foam is discouraged. These offloading pads can create an “edge” effect, causing increased pressure to the wound margins.
and resulting in the wound becoming larger. Surgical shoes and removable cast walkers with multidensity inserts assist in offloading the ulcer by more evenly distributing pressure to varying degrees. The multidensity aspect of these inserts allows for a gradual decline in durometry (hardness) from the most outer layer of the insert to the layer that is in contact with the dressing on the foot. Some of these devices contain hexagonal plugs that can be removed. The removal of these plugs is not encouraged because this will again produce an edge effect. The advantage of the plugs is that they function to independently contour to the plantar aspect of the foot. Compliance with many of these removable offloading devices may be an issue, with patients removing the device once they are at home.

Total-contact casts are plaster or fiberglass constructs that cannot be removed and evenly distribute pressure on the plantar aspect of the foot. Total-contact casts need to be replaced every 1 or 2 weeks. There is good evidence to suggest that total-contact casts are the most effective modality of offloading, with healing rates of almost 90%. However, there is an ulcer recurrence rate of 59% 7 months after the total-contact cast has been removed. Although compliance can be maintained, the wound cannot be monitored on a daily basis. Furthermore, heavily draining wounds are not amenable to this type of offloading because the plaster or fiberglass will become saturated with fluid. Moreover, the patient may refuse a total-contact cast because of its restrictive nature.

Surgical intervention may be the most effective way to offload an ulcer. As briefly described earlier, tendon lengthening/rebalancing, exostectomies, and bone/joint reconstruction can reduce the deforming forces that create a diabetic foot ulcer and contribute to its chronicity. The key in deciding between soft-tissue tendon lengthening/rebalancing and bone/joint reconstruction is the reducibility of the deformity. If the deformity is reducible, tendon lengthening and rebalancing may be effective. If the deformity is rigid, bone/joint reconstruction is necessary. A combined tendon and bone reconstruction may also be necessary.

Infection

The diabetic foot ulcer can have an active and/or passive (biofilm) infection. Active infection includes the classic signs of ascending erythema, edema, purulence, increased drainage, and malodor. However, the diabetic patient is not able to mount a robust immune response and can present without these signs; this is particularly the case when end-stage renal disease is superimposed with diabetes. Therefore, there may not be obvious signs of infection. Furthermore, nonelevated laboratory values (eg, white blood cell count) may not reflect an active infection, although elevated blood glucose levels may. The degree of soft-tissue infection and depth of infection (eg, to the level of muscle or bone) will dictate the course of treatment. Superficial soft-tissue infection can be managed with oral or parenteral (via peripherally inserted central catheter) antibiotics, debridement, and topical antimicrobials. Deeper soft-tissue or bone infections may require hospital admission with parenteral antibiotics and serial surgical debridement/decompression. Soft-tissue infections of the diabetic foot are often polymicrobial with gram-positive species as well as gram-negative bacteria, whereas bone infections are typically monomicrobial; this includes staphylococcal and streptococcal species as well as Pseudomonas and Escherichia coli. Antibiotic therapy requires broad-spectrum coverage, based on sensitivities from deep cultures, for an extended duration until resolution of the infection (Fig. 6).

Biofilm consists of bacterial colonies that form on the surface of chronic wounds, and certainly plays a detrimental role in ulcer healing. Biofilm is present in 60% of
chronic wounds but in only 6% of acute wounds. These bacterial colonies are often multispecies and differ from planktonic bacteria. These bacteria have low metabolic activity and are encased with a glycocalyx matrix, making them resistant to oral, parenteral, and topical antibiotics. Biofilm will reform within 10 hours of debridement. A comprehensive multimodal strategy is needed that includes sharp excision of the wound to disrupt the biofilm, with immediate antimicrobial therapy to prevent its rapid reformation.

**Amputation**

The unfortunate sequela of peripheral neuropathy, peripheral vascular disease, soft tissue/bony deformity, ulceration, and infection is often amputation. An amputation may be the result of a diabetic foot ulceration that progresses in depth, causing bone infection that is not readily amenable to antibiotic therapy and requires bone resection. Amputation may also be the result of an ischemic process whereby there is tissue necrosis and no revascularization option. The diabetic patient has a 20 to 30 times higher risk of amputation than a nondiabetic patient, with high probability of an amputation after developing a diabetic foot ulcer. Amputations at any level of the foot affects the ambulatory capability of the patient, with higher-level amputations having a greater cardiovascular impact. Furthermore, the survival rate diminishes with ascending amputation level. For example, in particularly high-risk type 2 diabetes populations the 5-year mortality rate for a forefoot amputation is 39%, compared with 67% for above-knee amputations. Others report cumulative mortality rates after the first proximal amputation has been reported to be high as 45.8% at 5 years and 70.4% at 10 years for patients with diabetes. Hence, every attempt should be made to preserve as much length as possible.

**Fig. 6.** (A) Dorsal forefoot abscess resulting from web-space tinea pedis in a patient with diabetes. (B) Resolved abscess and cellulitis status post incision, drainage, and intravenous antibiotics.
Amputations are generally performed at the level of the joints in the foot or along the rays of the foot (eg, hallux and first metatarsal). However, a commonly performed amputation is across the diaphysis of the metatarsals (ie, transmetatarsal amputation [TMA]). Multiple factors need to be assessed when amputation is considered. After serial operative debridements are performed to clear the infected tissue, the level of viable tissue needs to be assessed. The patient may require vascular intervention to maximize perfusion to the tissue before a decision is made regarding the amputation level. If the patient does not have triphasic Doppler signals, angiography should be performed to evaluate where the flow terminates. Furthermore, a qualitative assessment of tissue viability should be made, evaluating for necrosis and bleeding wound edges. Attention should then be turned to biomechanical concerns. Soft-tissue coverage is certainly important; however, the preservation of function should also play an important role in the selection of amputation level. For example, the TMA preserves the weight-bearing parabola that allows for postamputation function. This consideration is important, because an unbalanced amputation of any part of the foot can lead to ulceration in a new area as a result of changes in pressure distribution. TMA should be selected if more than 1 ray has been wholly or partially amputated (Fig. 7).

It is vitally important to properly shoe or brace all feet that have had an amputation to prevent new ulcer formation. Furthermore, it is important to recognize that there is a significant alteration in gait after ulceration, even with the use of a prosthesis. The use of toe and forefoot fillers is important as part of the custom insert, to prevent excessive motion of the residual foot in the shoe for amputations distal to the tarsometatarsal joint. For more proximal amputations, a brace that spans across the ankle may be necessary to facilitate ambulation. Rocker-bottom configuration of the shoe also allows for fluid transfer from heel to forefoot loading.

An amputation should not necessarily be viewed as a complication. An elective amputation may be the best option for some patients. For example, if a patient presents with gangrene of the toes and minimal perfusion to the foot that is not amenable to vascular intervention, a below-knee amputation may be considered at the first visit. Such a decision may save the patient countless operations attempting to salvage the foot as well as prolonged hospital stays and clinic visits. However, the challenge lies in trying to identify which patients would be better served by having a proximal leg amputation rather than aggressive limb salvage.

![Fig. 7. Open transmetatarsal amputation as part of the treatment of diabetic foot ulceration with infection.](image)
FUTURE CONSIDERATIONS AND SUMMARY

The diabetic foot is a complex structure that is at risk for multiple complications. The weight-bearing demands required for ambulation place the diabetic foot at particular risk. Uncontrolled glucose levels lead to peripheral neuropathy and peripheral vascular disease, which can potentiate the breakdown of soft tissue and lead to ulceration, infection, and possible amputation. Although glucose control and ulcer prevention are the key to stopping this progression, diligent monitoring and aggressive treatment are necessary after a diabetic ulcer has occurred. New devices, biologics, drugs, and therapies will assist in healing wounds. However, fundamental principles of frequent debridement, microbial control, and offloading are still crucial. Most importantly, a multidisciplinary approach is needed to prevent and treat the diabetic foot.67 A multidisciplinary team approach can reduce amputation rates and wound-related complications by greater than 50%.68–71 Such an approach involves both medical and surgical fields lending their expertise through intensive communication and interaction.

REFERENCES

64. Armstrong DG, Lavery LA. Plantar pressures are higher in diabetic patients following partial foot amputation. Ostomy Wound Manage 1998;44:30–2, 34, 36 passim.