



Treating the Diabetic Ulcer: Practical Approach and General Concepts

Eran Tamir MD

Department of Orthopedic Rehabilitation, Sheba Medical Center, Tel Hashomer, Israel

Key words: diabetic foot, ulceration, off-loading, surgical treatment, prevention

Abstract

Neuropathic foot disease is commonly seen in clinical practice and is related primarily to the high and increasing prevalence of diabetes mellitus in the western world. The treatment of diabetic ulcer can be divided into three stages: evaluation, healing, prevention. Based on the evaluation, foot ulceration in diabetic patients can be categorized as non-infected and infected. Non-infected and non-ischemic ulcers heal when the area is off-loaded, constituting the cornerstone of treatment. Off-loading is combined with wound care. Surgical curative procedures are performed when conservative means fail and include internal off-loading, i.e., correction of the foot deformity and wound closure procedures. Off-loading is also important for infected ulcers but controlling the infection is essential. The infection is controlled by antibiotics, pus drainage, debridement of necrotic tissues including surgical debridement, and partial foot amputations. Diabetic ulcer is an expression of a complex systemic disease and requires treatment by multidisciplinary means, combining medical and paramedical personnel. The orthopedic surgeon plays a major role since a prominent part of the treatment involves foot surgery.

IMAJ 2007;9:610-615

Neuropathic foot disease is commonly seen in clinical practice, due primarily to the high and increasing prevalence of diabetes mellitus in the western world. For example, diabetes affects 6% of the American population [1], and foot pathology is found in 15% of diabetics. Over half of the non-traumatic amputations are performed in diabetic patients, 85% of which are precipitated by a foot ulcer [2-6].

The treatment of diabetic foot complications can be divided into three stages: evaluation, healing, and prevention and education. The diabetic foot is an expression of a complex systemic disease and requires treatment by multidisciplinary means, combining medical and paramedical personnel [7]. Orthopedic knowledge and operative skills are utilized in all three stages of treatment [8]. We present a practical approach to the diabetic foot, with special attention given to the orthopedic aspect. A systematic approach to the diabetic foot and an understanding of the multidisciplinary approach is essential for dealing with this common but difficult problem.

Evaluation

Medical co-morbidities

The general health status and co-morbidities of the patient have a direct effect on the treatment and prognosis of diabetic foot disease. Systemic complications of diabetes, such as peripheral vascular disease, renal and cardiac disease, other diseases such as liver disease, and the concomitant administration of steroid and immunosuppressive medications, all significantly affect the course of infection, sepsis and wound healing [9]. Peripheral vascular disease is the single most important factor affecting wound healing. A patient with a history of severe and non-reconstructible peripheral vascular disease is at high risk for amputation and should be treated vigorously for even minor foot complications [10].

Glycemic control

The level of glycemic control is represented by HbA1c and this should be assessed at the start of any treatment. Although not yet proven, glycemic control may be important for wound healing since hyperglycemia is associated with impaired collagen formation and tensile strength and decreased chemotaxis and phagocytosis [11].

Systemic signs of infection

The presence of fever, tachycardia, tachypnea, leukocytosis and hyperglycemia is an indication for admission, intravenous antibiotics and possible immediate surgical debridement. However, fever, chills and leukocytosis are absent in 66% of diabetic patients with a limb-threatening infection, complicating treatment decisions [12].

Peripheral vascular disease

Palpably normal dorsalis pedis and posterior tibial artery pulses are a good prognostic sign. However, the presence of foot edema (accompanying infection) does not allow reliable palpation, and therefore other physical findings of peripheral vascular disease such as loss of hair, cold skin temperature and skin atrophy should also be evaluated. Any patient with a wound on a foot without palpable dorsalis pedis and posterior tibial arteries should be sent for further vascular studies such as Doppler, pulse

volume recording, toe pressure, partial oxygen tension and laser Doppler skin perfusion pressure. The ankle brachial index, which is most commonly used, may be inaccurate in diabetic patients due to non-compressible arteries and other tests may therefore be needed.

Significant peripheral vascular disease is present in only 20% of diabetics with foot ulceration, but when present, a prompt vascular surgical consultation with regard to vascular reconstruction should be considered [13,14].

Evaluation of the ulcer

The ulcer should be carefully debrided and surgically unroofed and any necrotic tissue and detached skin should be removed. The callus surrounding the ulcer should also be removed. The depth of the ulcer and involvement of deep structures such as tendon, joint capsule and bone should be assessed with a probe. The grade and size of the ulcer should be recorded for follow-up.

The University of Texas Wound Classification system classifies diabetic ulcers by stage and grade: Stage A – no infection or ischemia, stage B – infection present, stage C – ischemia present, stage D – infection and ischemia present. Grade 0 – epithelialized wound, grade 1 – superficial wound, grade 2 – wound penetrating to tendon or capsule, grade 3 – wound penetrating to bone or joint. This classification can predict the risk for amputation [10].

Off-loading is also important for infected ulcers but controlling the infection is essential. The infection is controlled by antibiotics, pus drainage, debridement of necrotic tissues including surgical debridement, and partial foot amputations

Evaluation of infection

Infection can be both limb and life threatening and must be treated aggressively. Signs of soft tissue infection include malodorous pus secretion, edema, erythema and acute tissue necrosis. Some patients with severe neuropathy complain of pain in the presence of infection, although this sensation can be significantly blunted by the neuropathy itself. Cyanotic toe and swelling of the forefoot in the presence of a plantar ulcer of the corresponding metatarsal head signify acute deep infection with thrombosis of the digital arteries. Linear erythema starting at a plantar forefoot ulcer and spreading proximally indicates abscess formation of the long flexor tendon. Plantar forefoot ulcer accompanied by adjacent dorsal tissue necrosis indicates full-thickness soft tissue involvement of the forefoot. Gas in the soft tissues, extensive edema and metastatic foci of infection indicate severe and rapidly progressive infection. The borders of the erythema should be marked for follow-up, and if abscess

formation is suspected aspiration using a large caliber needle must be attempted. Another technique is compression of the soft tissues around the ulcer for the presence of pus. A positive probe to bone is strongly correlated with osteomyelitis, while a positive probe into bone (penetrating into the spongy bone) signifies destruction of cortical bone and osteomyelitis, even in the presence of a normal radiograph. While radiography is an effective method for evaluating the presence of osteomyelitis of the forefoot, it may be negative in the acute infection period. Radiography of the midfoot and hindfoot may also yield false negative results, and other imaging methods such as computed tomography and magnetic resonance imaging scans or white blood cell scans should be considered. Finally, aerobic and anaerobic cultures should be taken. Tissue or pus cultures are preferable to swab culture, although recent research suggests that the difference between swab and tissue specimens is not as marked as previously thought [15]. Whenever systemic involvement is suspected blood cultures should be taken [16].

Evaluation of neuropathy and the presence of pain

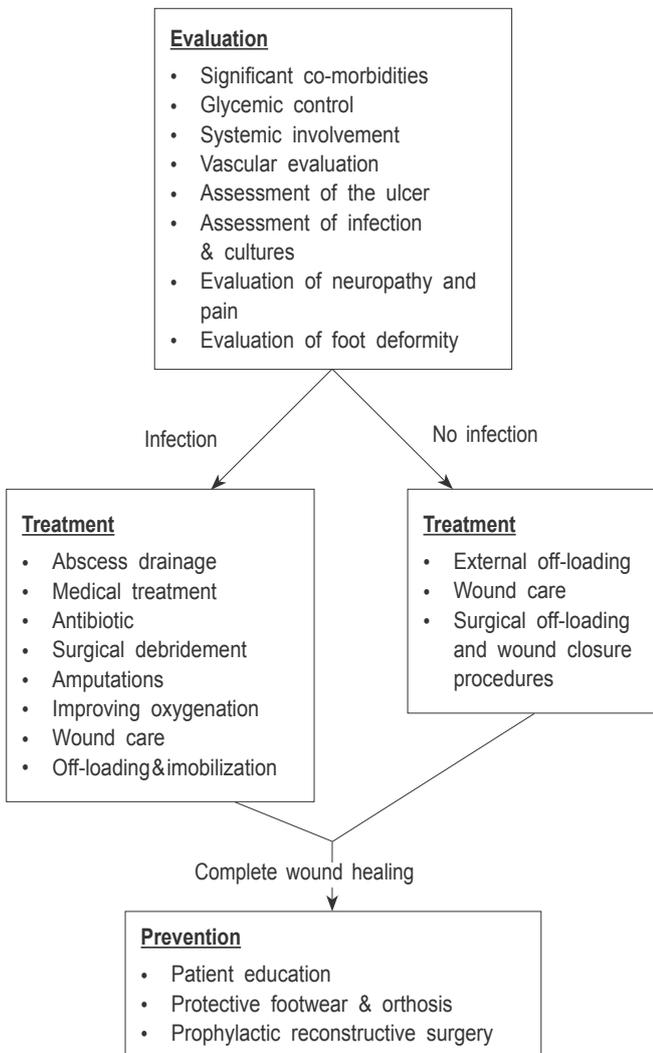
Sensory neuropathy and corresponding lack of protective sensation is a major risk factor for diabetic foot complications. The presence of painless neuropathic ulcer signifies significant sensory neuropathy and assessment with the monofilament test [17] is not necessary. The presence of pain in a neuropathic foot should raise the suspicion of infection or neuroarthropathy.

Evaluation of foot deformity

The most significant factors contributing to diabetic foot ulceration are neuropathy and foot deformity. Foot deformity may result from motor neuropathy and muscle imbalance, or Charcot foot. Careful evaluation of the foot deformity and its biomechanical role in ulcer formation has a direct therapeutic application.

Motor neuropathy leads to the development of a cavus foot with a mild equinus contracture at the ankle, claw toes, and increased pressure to the forefoot (metatarsal heads). Claw toe results from imbalance between the intrinsic muscles (lumbricals and interossei muscles that flex the metatarsal phalangeal joint and extend the proximal interphalangeal and distal interphalangeal joints) and the long extensor and flexor tendons. Intrinsic muscle dysfunction causes the long tendons to dominate, such that the extensor digitorum longus extends the metatarsophalangeal joint and the flexor digitorum longus flexes the proximal interphalangeal and distal interphalangeal joints. The combination of this deformity with sensory neuropathy may result in ulceration in three distinctive sites. The most common site for foot ulceration is below the metatarsal heads. This results from extension of the MTP joint and distal migration of the soft tissue at the plantar aspect of the metatarsal head. Limited dorsiflexion and Achilles tendon tension increase forefoot plantar pressure and contribute to ulcer formation. The dorsal aspect of the PIP joint may ulcerate from rubbing against the toe box of the

MTP = metatarsophalangeal
PIP = proximal interphalangeal



shoe, and the tip of the toe may ulcerate as it is being pushed against the sole. Abnormalities of the first ray and hallux may also contribute to ulcer formation. Stiffness of the first MTP joint promotes ulcer formation at the plantar aspect of the hallux. A hyperflexed first metatarsal contributes to ulcer formation below the first metatarsal head and hallux valgus deformity contributes to ulcer formation at the bunion.

As a general rule, midfoot and hindfoot ulceration (excluding decubitus heel ulcers) result from Charcot foot arthropathy (neuropathic osteoarthropathy). The most common joint complex involved is the midtarsal joints, with collapse leading to a rocker-bottom deformity: the medial arch is lost and the midtarsal bones protrude plantarly, with the resulting bony configuration resembling the legs of a rocking chair. This produces increased pressure at midfoot bony prominence and, if the pressure exceeds the resilience of the soft tissues, a neuropathic ulcer develops. Charcot arthropathy affecting the hindfoot may result in deformity and instability of the ankle and subtalar joints, with increased pressure and ulceration over the malleoli.

Healing

Based on the evaluation, foot ulceration in diabetic patients can be divided into two categories: non-infected and infected, with possible shifting from one category to the other during the course of treatment.

Treatment of non-infected ulceration

Non-infected ulceration can be successfully treated by combining off-loading and wound care (debridement and dressing). The major factor preventing wound healing of non-infected ulcers is physical pressure. Ischemia, when present, is also important, and every patient without distal pulses should undergo vascular assessment and, if indicated, a surgical vascular consultation. Glycemic control may also play a role, and a diabetologist should be consulted if satisfactory control is lacking.

Non-infected and non-ischemic ulcers usually heal when the area is off-loaded, constituting the cornerstone of treatment. This includes complete bed-rest, and the constant use of walking aids, although such measures may be impractical for many patients. Unfortunately, even brief periods of weight bearing (such as walking to the toilet at night) may delay or prevent healing [18].

Off-loading methods can be broadly categorized as external (avoidance of weight bearing, casting, braces, orthotics, and shoes) or internal (surgical correction of the deformity).

The total-contact cast is widely considered to be the most effective external technique for off-loading plantar ulcers, with a reported healing rate of 70–100% within an average of 36 days [19,20]. Although effective, it may involve some complications, as in the case of excessive movement of the foot or leg inside a loosely fitting cast, causing new abrasion wounds and ulcers [21]. The total-contact cast should be changed weekly and requires significant casting expertise as well as time. Alternative off-loading methods less frequently used include pneumatic bracing, healing shoes and walking splints. Pneumatic bracing has been reported to be effective as a total-contact cast and is now a commonly used alternative [22].

Regarding wound care, during the external off-loading treatment, the wound should be routinely evaluated for healing and complications at least every 10 days, debridement should be performed if necessary and dressings, which keep the wound moist and promote healing (such as alginates, hydrocolloids, etc.), should be applied.

Deep ulcers with an exposed tendon or joint capsule are a contraindication for total-contact casting. In such cases, a removable off-loading device, such as pneumatic bracing, should be fitted to enable daily wound care.

Neuropathic ulcers that are not plantar, such as at a bunion, may result from trauma or abrasion of the skin against the shoe and simple measures such as therapeutic shoes and wound care usually suffice.

Surgical procedures for non-infected ulcers include internal off-loading, i.e., correction of the foot deformity, and wound closure procedures. In general, surgery is indicated only when conservative means fail.

- *Internal off-loading.* Tip of the toe ulcer in a flexible claw toe can be treated by percutaneous flexor tenotomy. This simple and effective procedure off-loads the ulcer by reducing the degree of the deformity. This again promotes not only wound healing but also prevents recurrence by correcting the deformity. Tip of the toe ulcer or ulcer at the dorsal aspect of the PIP joint in a rigid claw toe can be treated by claw toe correction.

An ulcer below the metatarsal heads with tight Achilles tendon and limited dorsiflexion can be treated by percutaneous Achilles tenotomy. This procedure promotes healing and prevents recurrence by off-loading the forefoot [23]. The procedure is combined with off-loading by the total-contact cast, which also keeps the ankle in a dorsiflexed position such that the desired Achilles tendon lengthening is maintained while the ulcer is healing.

An ulcer below the first metatarsal head on a foot with flexible hyperflexed first ray can be treated by peroneus longus to brevis tendon transfer. If the hyperflexed first ray is rigid, an elevation of the first ray by osteotomy can be performed.

An ulcer below the head of one of the lesser metatarsal heads can be treated by metatarsal head excision, although this may transfer more load to the remaining metatarsal heads. Ulceration below two or more metatarsal heads can be treated by forefoot reconstruction: excision of metatarsal heads 2 to 5 with claw toe correction and arthrodesis of the first toe.

Midfoot ulceration in a Charcot foot can be treated by exostectomy of a bony prominence, or exostectomy with osteotomy and reconstruction of the medial arch. Ulceration at the medial or lateral malleolus in a Charcot foot can be treated by hindfoot realignment and fusion [24-26].

- *Wound closure procedures.* Wound closure can be performed either when conservative off-loading and wound care treatments fail, or in order to shorten the healing time. Split-thickness skin grafting is efficient but requires that the wound bed already be covered with red granulation tissue in order to accept the graft. Primary wound closure can be performed by excising the ulcer to clean surgical margins and closing the wound. Extensive bone resection is usually required to enable the soft tissue flaps to be apposed without tension and also off-load the ulcer and prevent recurrence. Unfortunately, the complication rate for these procedures is high and they must be used judiciously. Finally, local and free flaps can be effectively used to achieve ulcer closure in selected cases [27].

Treatment of infected ulceration

Off-loading is also important for infected ulcers but controlling the infection is the mainstay of treatment. The infection is controlled by antibiotics, pus drainage, debridement of necrotic tissues including surgical debridement, and partial foot amputations. After the infection is controlled, the wound is treated

by off-loading and wound care. Surgical procedures for wound closure such as skin grafting are commonly used.

The clinical picture of infected diabetic ulcers ranges from acute rapidly progressing limb and life-threatening infection to chronic osteomyelitis that may persist for many months. Since any acute foot infection in a diabetic patient is potentially limb and life threatening, prompt referral to the emergency room should be considered. A minor infection presenting as cellulitis of the toe extending to the dorsum of the foot, with no systemic involvement, may respond to broad-spectrum antibiotic treatment. The borders of the cellulitis should be marked and the patient should be followed closely.

Non-infected and non-ischemic ulcers heal when the area is off-loaded, which is the cornerstone of treatment. Surgical curative procedures are performed when conservative means fail

Any patient with more severe acute foot infection – for example, an infection accompanied by systemic signs of fever and leukocytosis, tissue necrosis and foul odor of the wound – and acute foot edema should be hospitalized. High risk patients, such as those with known peripheral vascular disease, chronic renal failure, or receiving immune suppressive medications following kidney transplantation, should be hospitalized for any foot infection.

- *Medical treatment.* After routine blood analysis and obtaining tissue and blood cultures, broad-spectrum antibiotic treatment should be initiated. In addition, fluids and electrolyte balance should be maintained, blood sugar level should be controlled, cardiac output and renal function should be made optimal, and nutritional support should be provided.
- *Surgical procedures.* If an abscess is diagnosed (never hesitate to puncture the suspected site), an emergency drainage should be performed; this is a simple bedside procedure that does not require specialized surgical skills. Early abscess drainage can dramatically improve the patient's condition; postponing the procedure may prove deleterious. Subsequently, further debridement may be performed in the operating room as required. Linear plantar skin erythema extending from a plantar ulcer proximally along the flexor tendons route signifies a deep abscess of the long flexor sheath; this requires prompt surgical intervention, with the flexor sheath opened and debrided [28]. Pus emanating from an ulcer also indicates a deep infection and the need for surgical intervention. The ulcer should be probed for sinus tracts and incision and drainage should never stop distal to the proximal extension of the sinus tract.

Major limb amputation should be performed either as a life-saving procedure in case of severe life-threatening sepsis, or if the foot is deemed non-salvageable. The factors involved in such an important decision include the patient's age, co-morbidities and level of function, the degree of systemic involvement, the blood supply to the foot and the ability to perform successful vascular reconstruction, and the degree of damage to the foot tissues. Partial foot amputations, which include toe and ray resection, trans-metatarsal, midfoot and hindfoot amputations, are undertaken as part of the debridement protocol in the presence of osteomyelitis and bone destruction. The expected residual function of the foot should be considered when performing partial foot amputations, with the goal being the preservation of function rather than of tissue. For example, a trans-metatarsal amputation compared to combined resection of the first, second and third rays results in superior function despite greater tissue loss.

Patients with chronic ulcer and osteomyelitis but no signs of acute infection can be treated less aggressively. Osteomyelitis of the toe may be treated successfully with antibiotic treatment alone.

Diabetic ulcer is an expression of a complex systemic disease and requires multidisciplinary treatment by both medical and paramedical personnel

Osteomyelitis of the metatarsal head may be treated either with local debridement and antibiotic treatment or by ray resection depending on the extent of bone destruction. It is important to note that not all tissue necrosis requires surgical debridement. In the case of a patient with arterial insufficiency who develops dry gangrene with a sharp border between the black necrotic tissue and the surrounding viable tissues, it is recommended not to debride the necrotic tissues but to allow natural healing to occur, resulting in auto-amputation.

- *Improving oxygenation.* When the blood supply to the foot is compromised, successful surgical revascularization can dramatically improve the prognosis. Other means of improving tissue oxygenation include reduction of edema, administration of hyperbaric oxygen [29], and medications that can improve the circulation such as ileomedin. The efficacy of many adjunctive modalities, such as hyperbaric oxygen, has not been established by large-scale randomized clinical trials.
- *Off-loading and immobilization.* Successful treatment of the infected diabetic foot involves off-loading, preferably by bed-rest in the case of the hospitalized patient, avoidance of weight bearing on the affected foot, and the use of a wheelchair. If a surgical procedure has been performed, the

off-loading should continue until satisfactory wound healing has occurred. Whenever excessive motion compromises the wound healing, the foot should be splinted.

- *Wound care.* Open wounds may require a long time to heal. Wound-bed preparation and proper dressing should be used to promote wound healing, and more recent techniques such as negative pressure wound therapy can be considered if available [30].

Prevention and education

As soon as healing has occurred following treatment, the prevention stage begins and continues throughout the patient's life. This consists of patient education and the use of protective shoes and orthoses. Reconstructive surgery is indicated only when such conservative means have failed.

Protective footwear and orthoses

Partial foot amputation creates a new foot deformity, and therefore previous protective footwear should be replaced. All old shoes must be carefully examined and are often discarded as they may increase the likelihood of ulcer recurrence.

In the presence of neuropathy without significant foot deformity, ready-made protective footwear can be used. The protective features include a high toe box to accommodate a thicker insole and claw toes, soft leather with no seams in the toe box, and a stiffer sole with a rocker to reduce the forefoot pressure [31]. A custom-made full-contact insole can be used to reduce pressure at specific areas, while a more severe foot deformity requires custom-made protective shoes. An ankle-foot orthosis is used in cases of significant midfoot or hindfoot deformity or instability.

Patient education

This includes information on the nature of the patient's specific foot problem and how to reduce complications by daily self-inspection of feet, maintenance of strict glycemic control, proper skin and nail care, and use of protective footwear. The patient is also instructed on how to modify lifestyle and activities according to the severity of the foot problem [32].

Reconstructive surgery

Reconstructive surgery is indicated when conservative measures fail to prevent recurrent ulceration. The goals of the surgery are to decrease the degree of deformity, off-load areas at risk (internal off-loading) and decrease the risk of re-ulceration. All surgical procedures should be performed only on a foot with good blood supply and no signs of infection.

When reconstructive surgery is performed on a foot with an ulcer it is called curative and when surgery is performed on a foot without an ulcer it is called prophylactic. It is preferable to try and heal the ulcer prior to surgery to decrease the chance for postoperative infection.

Summary

The diabetic foot is an increasingly common and severely debilitating disease that requires intensive treatment using all

of several different modalities. The orthopedic surgeon plays a major role since a prominent part of the treatment involves foot surgery. Other medical team members are the vascular surgeon for performing bypass surgeries and endovascular procedures in cases where the blood supply is compromised, the plastic surgeon for performing wound closure procedures, the diabetologist for controlling sugar levels, and the infectious diseases specialist. A nurse specializing in wound care and a podiatrist are also essential for providing proper care of the wound, skin and nails, as well as patient education. The ortho-technician is responsible for total-contact casting or other off-loading devices, and finally the shoemaker and the orthotist supply protective footwear and orthotic braces. The treatment of the diabetic foot is complex but feasible, and clearly requires a multidisciplinary approach rather than being the domain of any specific profession.

References

1. Brodsky JW. The diabetic foot. In: Mann RA, Coughlin MJ, eds. *Surgery of the Foot and Ankle*. 6th edn. St. Louis, MO: Mosby, 1993:877-958.
2. Mayfield JA, Reiber GE, Sanders LJ, Janisse D, Pogach LM. Preventive foot care in people with diabetes. *Diabetes Care* 1998;21:2161-77.
3. Palumbo PJ, Melton LJ III. Peripheral vascular disease and diabetes. In: *Diabetes in America: Diabetes data compiled 1984*. Washington, DC: Government Printing Office, August 1985:XV-1- XV-21. (NIH publication no. 85-1468)
4. Consensus Development Conference on Diabetic Foot Wound Care: 7-8 April 1999, Boston, MA. *Diabetes Care* 1999;22:1354-60.
5. Reiber GE, Vileikyte L, Boyko EJ, et al. Causal pathways for incident lower-extremity ulcers in patients with diabetes from two settings. *Diabetes Care* 1999;22:157-62.
6. Most RS, Sinnock P. The epidemiology of lower extremity amputations in diabetic individuals. *Diabetes Care* 1983;6:87-91.
7. Aksoy DY, Gurlek A, Cetinkaya Y, et al. Change in the amputation profile in diabetic foot in a tertiary reference center: efficacy of team working. *Exp Clin Endocrinol Diabetes* 2004;112:526-30.
8. Strauss MB. The orthopedic surgeon's role in the treatment and prevention of diabetic foot wounds. *Foot Ankle Int* 2005;26:5-14.
9. Falanga V. Wound healing and its impairment in the diabetic foot [Review]. *Lancet* 2005;366:1736-43.
10. Armstrong DG, Lavery LA, Harkless LB. Validation of a diabetic wound classification system. The contribution of depth, infection, and ischemia to risk of amputation. *Diabetes Care* 1998;21:855-9.
11. McMurry JF Jr. Wound healing with diabetes mellitus. Better glucose control for better healing in diabetes. *Surg Clin North Am* 1984;64:769-78.
12. Myerson MS, Henderson MR, Saxby T, Short KW. Management of midfoot diabetic neuroarthropathy. *Foot Ankle Int* 1994;15:233-41.
13. Lepantalo M, Biancary F, Tukiainen E. Never amputate without consultation of a vascular surgeon [Review]. *Diabetes Metab Res Rev* 2000;16(Suppl 1):S27-32.
14. Sumpio BE, Lee T, Blume PA. Vascular evaluation and arterial reconstruction of the diabetic foot [Review]. *Clin Podiatr Med Surg* 2003;20:689-708.
15. Slater RA, Lazarovitch T, Boldur I, et al. Swab cultures accurately identify bacterial pathogens in diabetic foot wounds not involving bone. *Diabet Med* 2004;21:705-9.
16. Jude EB, Unsworth PF. Optimal treatment of infected diabetic foot ulcers [Review]. *Drugs Aging* 2004;21:833-50.
17. Saltzman CL, Rashid R, Hayes A, et al. 4.5-gram monofilament sensation beneath both first metatarsal heads indicates protective foot sensation in diabetic patients. *J Bone Joint Surg Am* 2004;86-A:717-23.
18. Cavanagh PR, Ulbrecht JS, Caputo GM. Biomechanical aspects of diabetic foot disease: aetiology, treatment, and prevention. *Diabet Med* 1996;13(Suppl 1):17-22.
19. Armstrong DG, Nguyen HC, Lavery LA, van Schie CH, Boulton AJ, Harkless LB. Off-loading the diabetic foot wound: a randomized clinical trial. *Diabetes Care* 2001;24:1019-22.
20. Myerson M, Papa J, Eaton K, Wilson K. The total contact cast for management of neuropathic ulceration of the foot. *J Bone Joint Surg Am* 1992;74:261-9.
21. Guyton GP. An analysis of iatrogenic complications from the total contact cast. *Foot Ankle Int* 2005;26:903-7.
22. Hartsell HD, Fellner C, Saltzman CL. Pneumatic bracing and total contact casting have equivocal effects on plantar pressure relief. *Foot Ankle Int* 2001;22:502-6.
23. 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-A:1436-45.
24. Early JS, Hansen ST. Surgical reconstruction of the diabetic foot: a salvage approach for midfoot collapse. *Foot Ankle Int* 1996;17:325-30.
25. Bono JV, Roger DJ, Jacobs RL. Surgical arthrodesis of the neuropathic foot. A salvage procedure. *Clin Orthop Relat Res* 1993;296:14-20.
26. Schon LC, Marks RM. The management of neuroarthropathic fracture-dislocations in the diabetic patient. *Orthop Clin North Am* 1995;26:375-92.
27. Ozkan O, Coskunfirat OK, Ozgentas HE. Reliability of free-flap coverage in diabetic foot ulcers. *Microsurgery* 2005;25:107-12.
28. Gavrilov M, Petkov R, Gavrilov N, et al. Surgical treatment of creeping infections of the flexor foot tendons in diabetic patients. *Khirurgiiia (Sofia)* 2004;60:49-51.
29. Strauss MB. Hyperbaric oxygen as an intervention for managing wound hypoxia: its role and usefulness in diabetic foot wounds. *Foot Ankle Int* 2005;26:15-18.
30. Armstrong DG, Lavery LA. Negative pressure wound therapy after partial diabetic foot amputation: a multicentre, randomised controlled trial. *Lancet* 2005;366:1704-10.
31. Uccioli L, Faglia E, Monticone G, et al. Manufactured shoes in the prevention of diabetic foot ulcers. *Diabetes Care* 1995;18:1376-8.
32. Pinzur MS, Slovenkai MP, Trepman E, Shields NN. Guidelines for diabetic foot care: recommendations endorsed by the Diabetes Committee of the American Orthopedic Foot and Ankle Society. *Foot Ankle Int* 2005;26:113-19.

Correspondence: Dr. E. Tamir, 39 Tidhar Street, Ramat Gan 52243, Israel.
 Phone: (972-3) 678-3246
 Fax: (972-3) 678-3246
 email: eranetta@012.net.il

It's impossible to be loyal to your family, your friends, your country, and your principles, all at the same time

Mignon McLaughlin (1915-1983), American journalist and author