Primary Care

FOOT ULCERS

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ULCERS result from breaks in the dermal barrier, with subsequent erosion of underlying subcutaneous tissue. In severe cases, the breach may extend to muscle and bone. Primary care providers are often the first to evaluate nonhealing pedal ulcers. Early recognition of the cause and prompt management of ulcers are essential for a good functional outcome. In many cases, successful salvage of an extremity depends on a multidisciplinary team of specialists, and timely consultation is warranted.

CAUSES

Walking consists of a complex series of biomechanical events involving triplanar movements of the foot and ankle (Fig. 1A).[2] A variety of external and internal forces can affect foot function (Fig. 1B and 1C). Shear force is the major cause of tissue breakdown in the insensate foot.[1]

Although simple cutaneous breakdown is not infrequent, healing is the rule unless the wound-repair mechanisms are suboptimal because of impaired perfusion, infection, or repeated or continuous traumatic insults (Table 1). Lack of sensation allows the damage to progress to ulceration. The progression to ulceration can be attributed to an impaired arterial supply, neuropathy, musculoskeletal deformities, or a combination of these factors.[3]

Arterial Insufficiency

Lack of perfusion decreases tissue resilience, leads to rapid death of tissue, and impedes wound healing. Wound healing and tissue regeneration depend on an adequate blood supply to the region. Ischemia due to vascular disease impedes healing by reducing the supply of oxygen, nutrients, and soluble mediators that are involved in the repair process.[4] Reduction in blood flow to the feet is due primarily to atherosclerotic obstruction of the major conduit vessels. Although peripheral arterial disease alone infrequently precipitates ulceration, the disease has a major role in delayed wound healing and gangrene.

It is often a surprise to the primary care provider when limb-threatening foot ulcers develop in a patient with relatively mild symptoms of arterial insufficiency. This can happen because the blood supply needed to allow healing of an ulcer, once one is present, is greater than that needed to maintain intact skin. The ulcer will progress to a chronic wound or gangrene unless the blood supply is improved.

Neuropathy

Neuropathy is a frequent risk factor for foot ulcers and can involve both somatic and autonomic fibers. Myelinated type A sensory fibers are associated mainly with proprioception; sensation of light touch, pressure, and vibration; and motor innervation of the muscle spindles. The clinical sequelae of neuropathy of these fibers are an ataxic gait and intrinsic weakness of the foot muscles. Type C sensory fibers are associated with free nerve endings that appear to help detect noxious, painful, and thermal stimuli. The clinical result of neuropathy of these fibers is loss of protective sensation. High loads are undetected because of loss of the pain threshold, which results in prolonged and increased shearing forces and repeated trauma to the foot.[5,6] Loss of protective sensation due to peripheral neuropathy is the most common cause of ulceration (Fig. 2A and 2B).[7]

Motor neuropathy is associated with demyelinization and motor end-plate damage, which contribute to conduction defects. The distal motor nerves are the most commonly affected, resulting in atrophy of the small intrinsic muscles of the foot. Wasting of the lumbrical and interosseous muscles of the foot results in collapse of the arch and loss of stability of the metatarsal–phalangeal joints during mid-stance of the gait (Fig. 1B).[2] Overpowering by extrinsic muscles can lead to depression of the metatarsal heads, digital contractures, and cocked-up toes (Fig. 2A); equinus deformities of the ankle; or a varus hind foot.[8] These changes result in abnormal pressure points, increased shearing, and greater friction on the foot.

Autonomic-nerve involvement results in diminished sweating of the feet and dry, brittle skin that is at risk for cracking and the development of fissures within calluses.[9] Any break of the skin barrier is a potential portal of entry for bacteria, leading to infection.

Musculoskeletal Abnormalities

Altered foot biomechanics, limited joint mobility, and bony deformities have been associated with an increased risk of ulceration and amputation.[3] Abnormalities in foot biomechanics result in a dysfunctional gait and can lead to more damaging structural changes in the foot. Limitations of the range of motion of any of the foot joints will disrupt gait mechanics. Bony deformities, such as deformities of the metatarsal heads and the forefoot, are areas of excessive focal pressure during the midstance and heel-rise parts of the gait cycle. A decrease in the surface area

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of the tissue below a metatarsal head with rigid deformities leads to increased forces or pressure on the sole of the foot. The plantar aspect of the foot is protected by fat pads that dissipate weight-bearing forces in all directions. Dislocation or atrophy of these pads results in high pressures under them. If the magnitude of these forces in a given area is large enough, either skin loss or hypertrophy of the stratum corneum (callus) develops (Fig. 1C). This increases the risk of ulceration by two orders of magnitude.[7]

The risk of ulceration is proportional to the number of risk factors.[10] The risk is increased by 1.7 in persons with isolated peripheral neuropathy, by 12 in those with peripheral neuropathy and foot deformity, and by 36 in those with peripheral neuropathy, deformity, and previous amputation, as compared with persons without risk factors.

Diabetes Mellitus

Persons with diabetes are particularly prone to foot ulcers.[11,12] The American Diabetes Association consensus group found that among persons with diabetes, the risk of foot ulceration was increased among men, patients who had had diabetes for more than 10 years, and patients with poor glucose control or with cardiovascular, retinal, or renal complications.[3] Foot ulcers in patients with diabetes usually have mixed ischemic and neuropathic components (Fig. 2C).[13]

Atherosclerosis occurs at a younger age in persons with diabetes than in other persons, and its hallmark is the involvement of the tibioperoneal vessels with sparing of the pedal vessels.[14] The relative absence of atherosclerosis in the feet often facilitates bypass to the

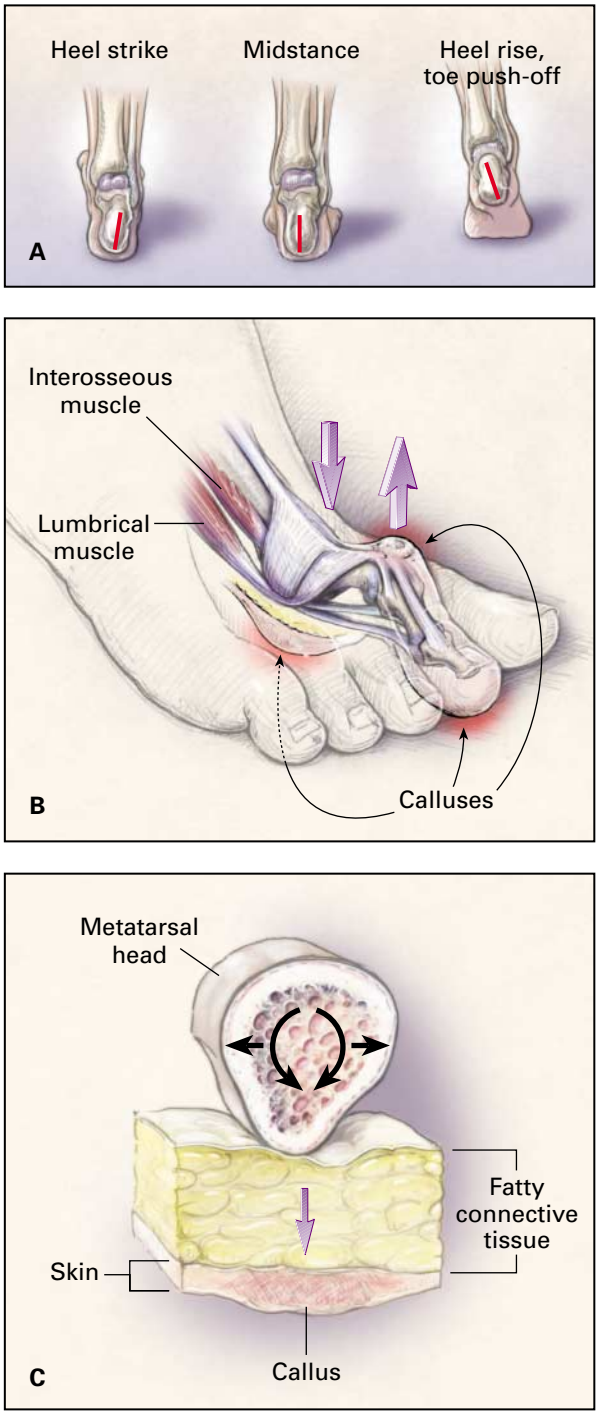

Figure 1. Biomechanics of Foot Ulcers.

Panel A shows the biomechanics of gait. The normal mechanics of the foot and ankle result from the combined effects of muscle, tendon, ligament, and bone function. Gait is classically broken into four segments. The first segment is heel strike, when the lateral calcaneus makes contact with the ground and the muscles, tendons, and ligaments relax, providing for optimal energy absorption. The second is midstance, when the foot is flat and is able to adapt to uneven terrain, maintain equilibrium, and absorb the shock of touchdown. The calcaneus is just below the ankle, keeping the front and back of the foot aligned for optimal weight bearing. The third is heel rise, when the calcaneus lifts off the ground, the foot pronates, the muscles, tendons, and ligaments tighten, and the foot regains its arch. This segment is followed by the fourth, toe push-off.

Panel B shows the forces on the foot. Friction and compressive forces are produced by the pushing down of the body weight and the pushing up of the ground reactive forces. Friction and pressure combine as a shear force during dynamic walking as the bones of the foot slide past each other in a direction parallel to their plane of contact during pronation and supination. Wasting of the intrinsic muscles of the foot results in an imbalance of the forces acting on the bony structures. This can lead to toe deformities, prominent metatarsal heads, equinus deformity, varus position of the hind foot, and proximal malalignment.

Panel C shows the consequences of callus formation. Inadequate distribution of the forces of weight bearing or the presence of foot deformities can lead to abnormal movement, which produces excessive stress and results in the breakdown of connective tissue and muscle. Adapted from Habershaw and Chzran[1] with the permission of the publisher.

TABLE 1. RISK FACTORS FOR THE DEVELOPMENT AND LACK OF HEALING OF FOOT ULCERS.

Vascular
Arterial insufficiency (atherosclerosis, vasculitis)
Transcutaneous oxygen pressure ≤30 mm Hg
Ankle pressure ≤40 mm Hg, toe pressure ≤30 mm Hg
Venous hypertension
Neurologic
Sensory neuropathy (insensate to Semmes–Weinstein 5.07 monofilament)
Motor neuropathy
Autonomic neuropathy
Foot-related
Altered biomechanics, limited joint mobility, bony deformity (Charcot's foot, claw toes, prominent metatarsal heads, other plantar bony prominences), severe pathologic changes in nails
Infections
Trauma
Diabetes mellitus

pedal arteries. Neuropathy is present in 42 percent of diabetic patients after 20 years[15] and is usually a distal symmetric sensorimotor polyneuropathy. Diabetic patients are especially prone to the development of a neuro-osteoarthropathy, Charcot's foot (Fig. 2D). This condition is thought to involve autonomic-nerve dysfunction resulting in abnormal perfusion to foot bones, which leads to bony fragmentation and arch collapse. The resulting "rocker-bottom foot" is prone to tissue breakdown and ulceration.[16]

ASSESSMENT AND MANAGEMENT

Management of a foot ulcer consists of recognition and correction of the underlying cause of the ulcer, appropriate wound care, and prevention of recurrence. The causes of most foot ulcers can be ascertained quite accurately by a careful, problem-focused history taking and physical examination.

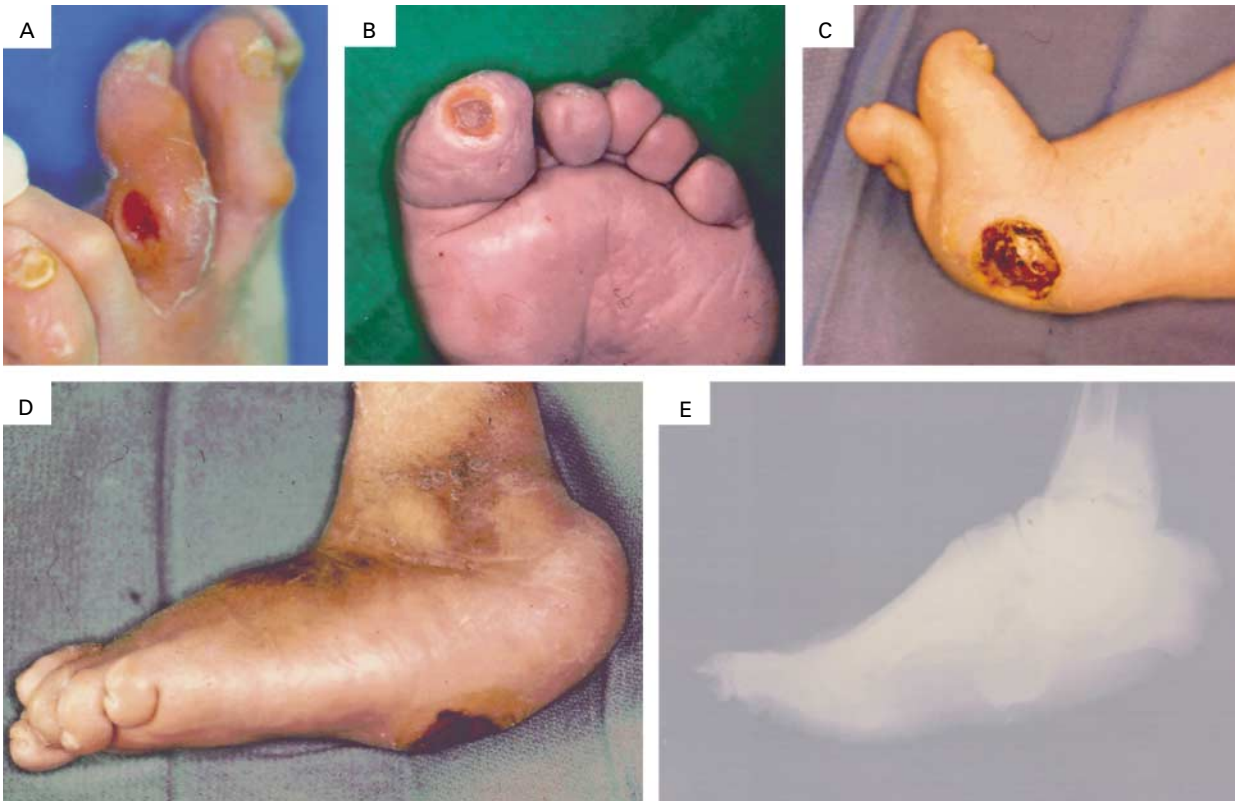

Figure 2. Examples of Foot Lesions.

Panel A shows digital ulceration with underlying osteomyelitis in a diabetic patient with neuropathy but with normal vascular perfusion. Digital contractures with underlying hypertrophic bone (hammer toes) and onychomycoses are visible. Panel B shows a neuropathic toe ulcer caused by rigid contracture of the interphalangeal joint. Panel C shows a combined ischemic and neuropathic ulcer. The exposed first metatarsal head with necrotic flexure tendon results in an extended toe. Panel D shows Charcot's foot with resulting ulcer. Panel E shows the "rocker-bottom" bony deformity of Charcot's foot on a plain film.

Vascular Causes

Arterial insufficiency is suggested by a history of underlying cardiac or cerebrovascular disease, leg claudication, impotence, or pain in the distal foot when the patient is supine (the "rest pain" syndrome). Findings of diminished or absent pulses, pallor on elevation, redness of the foot on lowering of the leg (dependent rubor), sluggish refilling of toe capillaries, and thickened nails or absence of toe hair are consistent with impaired arterial perfusion to the foot.

Ischemic foot lesions are characterized by the absence of bleeding, pain, and a precipitating trauma or underlying deformity. These ulcers often develop on the plantar surface of the foot and over the first and fifth metatarsal heads (Fig. 2C). Ischemic ulcers are uncommon on the dorsum, because the pressure is usually less sustained and perfusion is better.

In the majority of patients, the presence of a palpable pulse in the affected foot is a good indication of an adequate vascular supply. However, in patients with diabetes or chronic renal failure, the arteries may be calcified, making the results of the pulse examination less reliable. It is important that these patients, as well as those with an absent or diminished pulse, be referred to a vascular specialist for further evaluation. The adequacy of perfusion and the healing potential of the wound can be predicted from measurements of segmental limb pressures, pulse-volume wave form, and transcutaneous oxygen.[17] The latter two measurements are useful for evaluating perfusion in the diabetic foot because they are not affected by vessel calcification.

If the results of noninvasive tests are abnormal, there is a high likelihood that wound healing is being impeded by arterial insufficiency, and revascularization must be considered. Intravenous contrast angiography continues to be the gold standard for imaging of the blood vessels and is used for anatomical assessment before a planned intervention.

There are basic guiding principles in the management of vascular insufficiency. It is imperative that flow-limiting arterial lesions be evaluated and reconstructed or bypassed. Prosthetic vascular grafts, such as those made of polytetrafluoroethylene or Dacron, have outstanding 10-year patency rates when they are used as conduits in large-caliber, high-flow arterial systems such as the aorta and iliac arteries. For replacing or bypassing diseased small-caliber vessels below the inguinal ligament, autologous material, usually vein, is preferred. Autologous vein continues to be the most durable for distal bypass, with reported patency rates of 63 to 80 percent over five years, even in patients with diabetes.[18-20] Refinements in vascular technique and technology have allowed revascularization to the pedal vessels, with remarkably durable long-term results,[21] giving more impetus to an aggressive approach to limb salvage, even in patients with diabetes.

Neurologic and Musculoskeletal Causes

The gait of the patient should be observed to detect any gross asymmetry or unsteadiness. Deformities of the foot, range of motion of the joints, and joint mobility should be systematically ascertained, since they are indicative of the potential for ulceration. Neurologic assessment includes testing for motor strength, deep-tendon reflexes, and vibratory, proprioceptive, and protective sensation. Muscle strength should be assessed with a graded manual muscular test.[22] Symmetry should be noted. Patellar and ankle reflexes should be routinely checked. Testing for vibratory sensation with a 128-Hz tuning fork, pain perception with a pin, and touch perception with a cotton swab should be performed with the patient's eyes closed.

Measurement of cutaneous pressure perception with the use of Semmes–Weinstein monofilaments has been widely considered an ideal method of screening for neuropathy and ulceration potential because of its simplicity, sensitivity, and low cost.[10,23,24] Persons with normal foot sensation can usually feel the 4.17 monofilament (whose effect is equivalent to 1 g of linear pressure). Those who cannot feel the 5.07 monofilament (equivalent to 10 g of linear pressure) before it buckles are considered to have lost protective sensation.[25] An increase in the risk of ulceration by seven times in persons insensitive to the 5.07 monofilament has been reported.[26] However, up to 10 percent of persons who can feel the monofilament before it buckles may still have skin breakdown.

Once the biomechanical basis of the ulceration is understood, treatment can be instituted according to biomechanical principles. Measures to eliminate or minimize foot pressure should be instituted so that healing can occur. Strict bed rest is the simplest and most straightforward method, but it has the disadvantage of restricting the patient's activities and therefore frequently resulting in noncompliance. Specially designed shoes, accommodative inserts, or orthoses can help transfer forces away from sensitive or threatened areas, reduce friction and shear, accommodate or correct deformities, and modify weight transfers involved in gait. The use of such methods has been shown to reduce plantar foot pressures and the risk of recurrence of ulcers.[27-29]

Reconstructive foot surgery may prevent recurrent ulceration and decrease the risk of major amputation.[30-32] Again, the rationale for each procedure is based on the underlying biomechanical disorder. Removal of pieces of bone or cartilage may reduce internal foot pressures. Joint arthrodesis or the transfer, release, or lengthening of tendons may be warranted on the basis of knowledge of how the specific tendons influence motion.

Wound

Assessment of the ulcer consists of a determination of the location, size, and depth of the wound and

inspection of the surrounding area for signs of infection or gangrene. The absence of systemic manifestations, such as fever, chills, or leukocytosis, is an unreliable indicator of the absence of infection in soft tissue or bone. In general, it is beyond the scope of primary care to perform a costly, exhaustive evaluation of the foot wound. Referral to a multidisciplinary team of specialists is desirable to provide a concise, cost-effective, yet comprehensive assessment.

A number of imaging techniques can be used to diagnose osteomyelitis. Plain films of the foot are relatively inexpensive and can show soft-tissue swelling, disruption of the bone cortex, and periosteal elevation. However, these radiographic changes frequently lag behind actual bone destruction by up to two weeks, and often nearly 50 percent of the bone is destroyed before the changes are evident. Nuclear scanning methods rely on the use of specific isotopes, either alone or linked to white cells, to localize areas of infection. The disadvantages of these methods are their cost, the delay in obtaining the results, and their low specificity for detecting infections.[33] Magnetic resonance imaging can provide details of pathologic anatomical features and has a high sensitivity for detecting inflammation in both medullary bone and soft tissue.[34] Its sensitivity is further enhanced by the use of gadolinium. However, the test is costly and may not be readily available in all hospitals.

A prospective trial found that probing of sinuses and deep ulcers was a highly sensitive method of detecting bone infection.[35] The finding of palpable bone at the base of an ulcer, with no intervening soft tissue, has a higher positive predictive value than imaging findings and correlates strongly with the presence of underlying osteomyelitis. The results of superficial swab cultures of an open wound are unreliable. If cultures are warranted, percutaneous needle biopsy, deep aspiration, or open débridement and tissue biopsy should be performed by a consultant in order to detect the relevant bacteria.[16,36,37]

The role of a multidisciplinary group of consultants in the management of these wounds cannot be overemphasized. Aggressive mechanical débridement, systemic antibiotic therapy, and strict measures to reduce weight bearing are the cornerstones of effective wound care. Sharp débridement, usually in the operating room, allows for thorough removal of all necrotic material and diminishes the bacterial load, and thus promotes healing.[37] All necrotic bone, plus a small portion of the uninvolved bone, soft tissue, and devascularized structures, should be excised, and the degree of penetration of the infection should be established. Curettage of any exposed or remaining cartilage is important to prevent this avascular structure from becoming a nidus of infection. Foot soaks and whirlpool therapy are rarely effective and may lead to further skin maceration or wound breakdown.

The use of moist dressings on clean, granulating wounds improves the wound environment.[4,38] The dressings not only provide protection against further bacterial contamination but also maintain moisture balance, optimize the wound pH, absorb fibrinous fluids, and reduce local pain. A variety of dressings are currently available that can be targeted to specific characteristics of the wound.[39] However, moist normal-saline dressings are probably sufficient for the majority of wounds.[38] These inexpensive dressings are highly absorptive of exudative drainage and maintain the moist environment.

With gross wound infections and rampant cellulitis, use of a topical antimicrobial agent such as sulfadiazine or mupirocin may be necessary initially to reduce the bacterial load and serve as a chemical barrier to exogenous pathogens. Caution in the use of such medications is warranted to avoid selecting resistant organisms and preventing wound drainage. Oral antimicrobial therapy should be instituted on the basis of the suspected pathogen (usually streptococci or staphylococci) and clinical findings. Severe infections should be treated with intravenous antimicrobial agents with a spectrum broad enough to cover gram-negative and gram-positive aerobes and anaerobes.[36,40]

Plastic reconstructive techniques may be necessary to expedite closure of foot wounds. After bacterial contamination has been controlled, small ulcers can usually be excised and closed immediately. Large, open wounds, however, are treated with a staged approach, with frequent débridement and establishment of a granulation base. The clean wounds can then be closed with healthy tissue, with the use of local or free-flap coverage and soft-tissue repair. Meticulous surgical reconstruction of these wounds can help avert the production of inelastic scar tissue over weight-bearing surfaces. Any remaining extrinsic or intrinsic pressures can be reduced with the postoperative use of orthoses. Surgical correction of biomechanical defects, plastic and soft-tissue reconstruction, and appropriate measures to minimize foot pressure are all essential to enable the patient to walk again effectively.

In general, the optimal strategy is to perform the vascular-bypass procedure, if indicated, as soon as possible. Closure of the ulcer by primary healing or secondary reconstructive surgery will then be expedited. If revascularization of an ischemic ulcer is not possible for medical or technical reasons, amputation of the foot or limb will probably be required.

SUMMARY

The role of the primary care physician in the evaluation, diagnosis, and management of pedal wounds is critical (Table 2). Patient education is essential for the reduction of risk factors and for the early recognition of foot complications.[41] Careful assessment of vascular disease (leading to bypass surgery when indicated), evaluation and management of biomechanical

TABLE 2. THE SIX PRINCIPLES OF PREVENTION OF FOOT ULCERS.*

Podiatric care
Regular visits, examinations, and foot care
Risk assessment
Early detection and aggressive treatment of new lesions
Pulse examination
Evaluation for claudication and pain at rest
Assessment of foot pulses; noninvasive vascular testing when indicated
Protective shoes
Adequate room to protect feet from injury, well-cushioned
Walking sneakers, shoes with extra depth and width, custom-molded shoes
Special modifications as necessary
Pressure reduction
Pressure measurements
Cushioned insoles, custom orthoses, padded hosiery
Prophylactic surgery
Correction of structural deformities: hammer toes, bunions, Charcot's foot
Prevention of recurrent ulcers over deformities
Intervention at opportune time
Preventive education
Patient education: need for daily inspection and early intervention
Physician education: importance of foot lesions, importance of regular foot examination, and current concepts of foot management in patients with diabetes

*Adapted from Frykberg.[41]

abnormalities, and aggressive treatment of any infections are also required.[42] Optimal management can reduce the number of major amputations, prevent infection, decrease the probability of ulceration, maintain skin integrity, and improve function. The multidisciplinary approach will provide a comprehensive treatment protocol that will increase the long-term viability of the foot.

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