Chapter 25
The Diabetic Foot

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Introduction

The importance of the physician's role in examining and assessing the diabetic foot is hard to overstate,[1] yet studies have shown that primary care physicians are rarely performing foot examinations on their diabetic patients during routine visits.[2,3]

Uncontrolled diabetes is the cause of 60% of the 67,000 non-traumatic amputations encountered annually in the United States. The majority of these amputations are preceded by a foot ulcer and are therefore preventable.[4,5]

In 2006, the projected lifetime health-care cost for each patient having undergone a below-knee amputation was $509,275 and the annual cost of these amputations was $600 million. Lost wages and morbidity were estimated at $1 billion, annually.[6]

The United States National Diabetes Advisory Board stated that "the early detection, monitoring and treatment of the risk factors will lead to an 85 percent reduction in lower extremity amputation."[7] Physicians treating diabetic individuals must obtain a foot history and perform a foot examination in the course of office visits. By offering advice and instruction during routine visits, primary care physicians can assist diabetic patients in developing good foot care habits. They must also know when to refer patients to the appropriate specialist for preventive and curative care or to the emergency room for admission and possible urgent surgery. The foot history and exam will enable the physician to classify each patient according to the Relative Risk Factor (RRF) for Lower Extremity Amputation Scale (explained later in the chapter). If the RRF rating is high, a consultation with a podiatrist is in order. It has been found that the preventive care, diagnosis, and treatment of the existing risk factors by a podiatrist are important in determining the health, quality of life, and longevity of diabetic patients' feet and that podiatry is an integral part of the team approach to diabetes.[8]

The Lower Extremity Amputation Prevention Program (LEAPP)[9] consists of five relatively simple activities: annual foot screening, patient education, daily self-inspection of the foot, appropriate footwear selection, and management of simple foot problems (Table 25.1). In addition, it is becoming clear that more aggressive measures are appropriate in order to reduce or remove risk factors for ulcers and amputations. For example, if there is an extremely prominent metatarsal bone that would serve as the site of an eventual neurotrophic ulcer, a foot insert (orthotic) or even a surgical elevation or other mechanical correction should be considered as preventive care before the ulcer or a recurrence develops.[10]

Biomechanics of the Foot

Understanding the inherited biomechanics of feet is important in the evaluation of the diabetic foot. Functional Lower Extremity Biomechanics (FLEB) is the field of knowledge which focuses on the human body from the low

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Table 25.1 Lower Extremity Amputation Prevention (LEAP) Program

1. Annual foot screening
2. Patient education
3. Daily self-inspection of the foot
4. Appropriate footwear selection
5. Management of simple foot problems

back down, when in c*losed chain,* e.g., standing [stance] or active [gait] and weightbearing [upon the ground]. Classical medicine studies subjects in o*pen chain* (on an examining table and not weightbearing).

The etiological forces that must be overcome in managing a patient are the hard unyielding ground, hard unyielding shoeboxes, and the deforming force of the earth's gravity.

Compensating pathological forces from the weightbearing surface into the foot, balancing the posture, and providing functional and safe footwear for the diabetic foot are the biomechanical keys to preventing and treating foot ulcers and gait and balance problems in addition to maintaining quality of life. The use of straps, pads, foot orthotics, therapeutic footwear, and bracing reduces foot ulcers, foot infections, amputations, and hospitalizations in any diabetic population.

Functional Anatomy of the Foot

Biomechanically, the foot is divided into two arched longitudinal segments, the medial dynamic arch, and the lateral dynamic arch. Together, they form the Vault of the Foot (Fig. 25.1).[9]

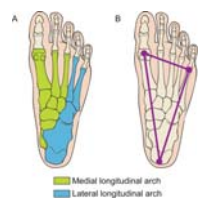

Fig. 25.1 The medial and lateral dynamic arches and the vault of the foot

The Centering Theory of FLEB

If one utilizes architectural engineering to develop a paradigm to diagnose and treat the foot as a supporting and functional entity, then, unlike the architectural arch which has equal pillars, a centered keystone, and symmetrical bases of support, the foot has a short rearfoot pillar, a long forefoot pillar, a keystone that is off-center proximally and has unequal bases of support. In summary, feet lack the centering inherent in architectural arches (Fig. 25.2).

The Use of Centrings in Architecture and the Foot

In architecture, if you want to build an arch, you first build a form in the shape of the arch called a centring. You lay the stones on the centering and set the central keystone and after cementing and drying, you remove the centring and the arch stands forever (Fig. 25.3).

The foot, on the other hand, in order to function as a mobile rigid lever and a functional adaptor to move us about and perform tasks fails as a supporting unit over time and therefore is destined to collapse, deform, degenerate, and perform poorly unless it is supported with a centring underneath (Fig. 25.4).

In summary, in architecture you use a centring to build arches and then remove it while in the foot, you add a centring to support inherited weakness in the arches to prevent collapse and improve performance.

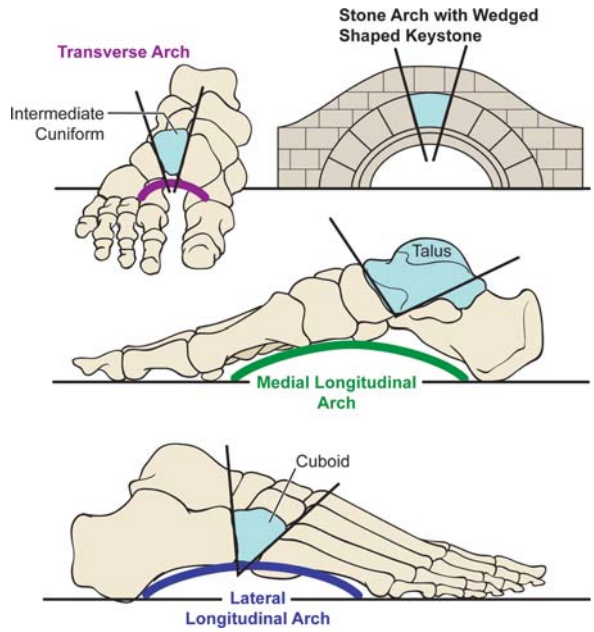

Fig. 25.2 Architectural vs. foot arches

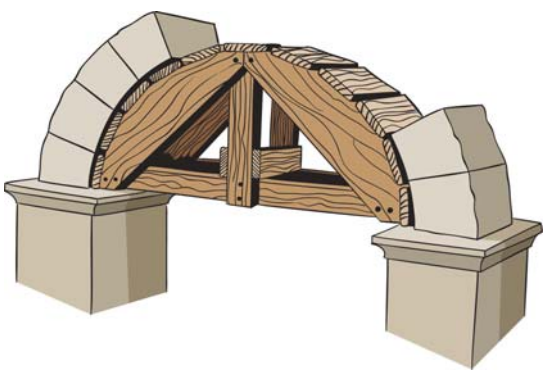

Fig. 25.3 Centring construction of an arch

Fig. 25.4 A foot centring orthotic providing arch support

Functional Foot Types® (FFTs)

Some feet have a rigid rearfoot pillar, some a flexible rearfoot pillar, and others fall in between. In addition, some feet have a rigid forefoot pillar, some a flexible forefoot pillar, and others fall in between. Utilizing two rearfoot tests called rearfoot SERM and rearfoot PERM (Serm = Supinatory End Range of Motion and PERM = Pronatory End Range of Motion) and two forefoot tests called forefoot SERM and forefoot PERM, one can classify the feet into four rearfoot types (rigid, stable, flexible, and flat) and four forefoot types (rigid, stable, flexible, and flat) forming 16 possible Functional Foot Types or FFTs.[11] Although there is a matrix of 16 possible FFTs, to date, 9 pure FFTs have been identified. Each Functional Foot Type® has its own characteristic open- and closed-chain presentations, X-ray results, lesion pattern, shoe wear, and foot and postural strengths and weaknesses (Table 25.2).

Table 25.2 The pure Functional Foot Types®

Foot type	Features/callus pattern	Associated problems
Flat RF/flat FF	Flat footed when sitting or standing,	Functional weakness, unable to perform daily tasks
Flexible RF/flexible FF	Flat foot, early bunion callus 2–3 mets, hallux IP jt	Early poor posture, fatigue tired feet, wide feet
Flexible Rf/flat FF	Flat foot, bunions, callus second met, hallux I-P jt	Poor posture, fatigue, second and fifth toe hammertoes
Flexible Rf/rigid Ff	Flat foot, first, fifth callus, hammertoes	
Stable Rf/flexible Ff	Normal arch sitting, flat foot standing	Bunions, heel spurs postural breakdown and problems
Stable FF/stable FF	No callus, normal arch	No problems
Rigid Rf/flexible Ff	High arch sitting, low arch standing callus, second and fifth met, hammertoes	Bunions, thick toenails heel and arch pain, postural pain and problems
Rigid RF/Stable FF	High arch sitting, normal arch standing fifth met callus	Low back pain
Rigid RF/rigid FF	High arch, callus sub 1st met/5th met, 1–5 hammertoes, met-cun exostosis	Low back and shock problems, heel and arch pain

Once a subject's FFT has been determined, foot type-specific care can be rendered. Foot type-specific locations of future deformity, foot and postural breakdown, infections, and ulcerations can be predicted, prevented, and controlled by treating FFTs, resulting in fewer amputations and reduced morbidity in any diabetic population.[12] In addition, a foot type-specific cast and prescription can produce a foot centering orthotic (Fig. 25.5)

Fig. 25.5 A pair of custom foot centering orthotics

that compensates off-center pathology allowing for improved support and quality of life and preventing ulcers, Charcot feet, and amputations.

The Inclined Posture (TIP)

Since 60% or more of all people have one leg at least 5 mm shorter,[12–14] the balancing of this biomechanical variation is fundamental to treating any unilateral (asymmetrical) foot problem and establishing balanced function, especially in the feet of those suffering from peripheral neuropathy, such as in the diabetic patient.

By testing for asymmetry in ankle dorsiflexion (functional equinus) and subtalar joint inversion (functional varus) between limbs, short limbs can be exposed. This Functional Equinovarus of the Joints of the Ankle or FEJA Test determines if one leg is functionally short.[14] Practitioners can then balance one leg to the other with lifts or physical therapy effectively eliminating a source of biomechanical pathology.

The use of heel lifts or platforms placed on the inside or outside of the short side shoe or incorporated into the short side foot orthotic compensates for TIP if physical therapy fails to stretch soft tissue contractures that may have caused one of the limbs to shorten.

Diabetic Neuropathy

Historically, peripheral arterial disease has been considered the most common complication observed in diabetic lower extremities. However, it is now accepted that the distal symmetric sensory, autonomic, and motor polyneuropathy occurs in up to 60% of patients with long-standing disease.[23] Furthermore, insensitivity coexists with diabetic foot wounds more than 80% of the time.[21] Peripheral nerve dysfunction is significantly associated with both impaired balance and lower extremity impairments.

The Phases of Diabetic Sensory Neuropathy

Diabetic neuropathy may precede other classical signs of diabetes. Its sensory component can be divided into five phases which are often progressive.

Phase I is a tingling sensation in the plantar aspect (bottom) of the foot that may manifest occasionally as a feeling of “bugs crawling” or “bees stinging,” and is referred to as fornication.

Phase II has the same symptoms that occur more frequently and are more intense.

Phase III is characterized by a constant burning of the feet that causes disruptions in sleep. This phase usually requires medication for pain control.

Phase IV has increased burning with anesthesia, giving a false sense of improvement from phase III.

Phase V is complete loss of sensation (LOPS).

Corn, Callus, and Poroma Formation

Biomechanical pathology and unhealthy and poorly fitting shoes cause pressure and friction in areas of the foot that are not meant to tolerate such loads. As a result, compensatory protective hyperkeratoses in the form of corns, callus, and porokeratotic (poroma) lesions develop in foot type-specific location. In the diabetic foot, continued pressure causes breakdown of these protective lesions in the form of pressure ulcers and wounds that can become infected. Monitoring and biomechanically controlling compensatory hyperkeratoses and wearing healthy and well-fit shoes are a vital part of preventive diabetic foot care.

The Biomechanics of Charcot Foot

Charcot joint disease or Charcot foot involves a devastating collapse of one of three specific areas of the foot. Since an inherited biomechanical weakness can exist at the midtarsal joint, the tarsometatarsal joint, or the first metatarsal phalangeal joint, it is these areas of the foot that are often affected (as well as the ankle joint). Diabetes

is the most commonly underlying disease associated with Charcot foot, end-stage syphilis and leprosy being a distant second and third. Once a Charcot foot develops, morbidity of the foot is permanent and progressive.[13] The patient's lifestyle and his/her ability to walk, work, and wear normal shoes are reduced.

Charcot foot develops in subjects with patent circulation and is composed of a quartet of symptoms:

(1) Patent circulation
(2) Loss of protective sensation (LOPS)
(3) A structurally weak foot type
(4) An active lifestyle, personality, and/or obesity

An individual with intact pain sensation will experience pain and swelling in the area of potential collapse when this area is stressed. Secondary to pain, the patient would likely reduce activity, lose weight, or introduce a biomechanical support, such as a custom foot orthotic, and therefore prevent the potential collapse of the foot. The patient with LOPS in this triad remains pain free and thus does not adjust his or her active lifestyle. Eventually, the weakest link in the biomechanical chain collapses, producing a Charcot foot (Fig. 25.6).

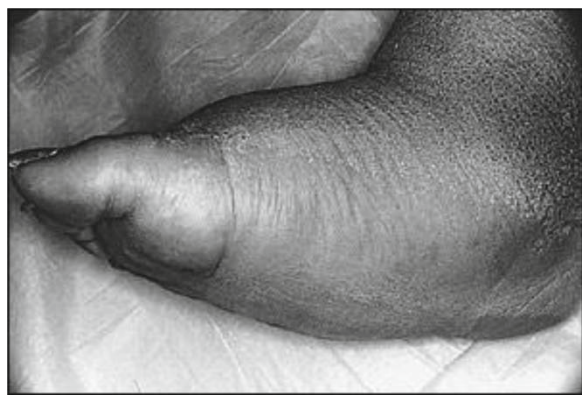

Fig. 25.6 The rocker bottom deformity of a Charcot foot. (Adapted from Sommer,[16] with permission)

There is no successful way to reestablish a normal lifestyle and biomechanics once a Charcot foot occurs. Therefore, it is essential for the physician to detect the impending signs of this quartet and to consider a biomechanical evaluation with a podiatrist for all patients with LOPS before a Charcot foot develops.[12]

The Physician Foot Evaluation

Physicians treating diabetic individuals should obtain a baseline history when it comes to examining the feet of diabetic patients. He or she should then monitor the feet in order to prevent ulcers and amputations and to maintain quality of life. A history of foot and shoe fit problems and quality of life issues should be reviewed. The pedal physical exam should include vascular, neurological, and orthopedic evaluation so that a preventive treatment plan can be developed and monitored. The treatment plan may involve annual examination (if there are no abnormal findings) or more rapid follow-up and consultation with a specialist, such as a podiatrist, if abnormalities are found.

History and Chief Complaint

The patient should be questioned regarding foot and postural problems, including the location and severity of corns, calluses, thick or ingrown toenails, infection and ulceration, as well as ankle, knee, hip, and lower

back complaints. Orthopedic deformities, such as bunions and hammertoes, should be noted. Problems with mechanics and posture, such as flat feet or high arches, should be noted, as should a family history of foot and postural problems. Shoe sizing and fit problems must be discussed along with a discussion of lifestyle and activity level. The patient should be asked if he or she is a "slow wound healer," has poor circulation or pedal numbness, burning, tingling, or anesthesia.

The Diabetic Foot Examination

All tests should be performed bilaterally, with asymmetry of complaints, systems, and deformities strongly noted.

Neurological. Touch, cold, and vibratory sense, as well as joint position sense must be tested and the deep tendon reflexes recorded for Achilles and patella. The vibratory sensation should be recorded and, in addition, the time it takes for the sensation of vibration to disappear should be equal in the hands and feet (vibratory dampening). Feet that dampen before the hands should be considered to have reduced proprioception. The Semmes–Weinstein monofilament test (Fig. 25.7)[15] should be taken at least eight sites to determine insensitivity (refer to section on loss of protective sensation).[17]

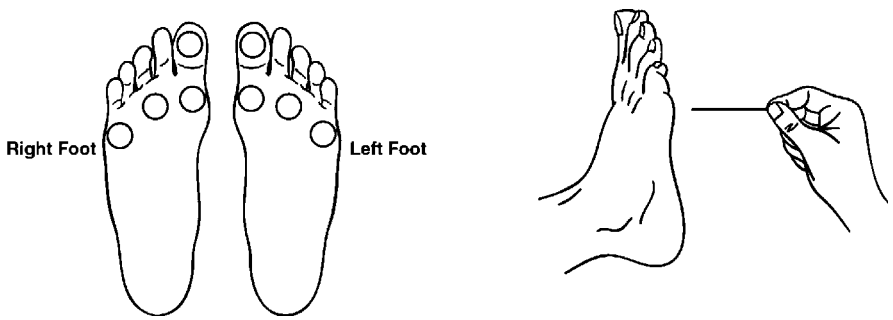

Fig. 25.7 The monofilament sensation test and common test sites. Adapted from LEAPP website www.bphc.hrsa.dhhs.gov/leap/def.htm, with permission

Vascular. Dorsalis pedis and posterior tibial pulses should be recorded. Pedal hair growth, temperature, and skin texture should be examined. Capillary return time/venous filling tests should be performed. The lower extremity should be checked for spider veins, varicosities, and edema. Cuts, abrasions, and wounds should be checked for healing time.

Dermatological. Toenails should be checked for dystrophy, ingrowing, microtrauma, and fungus infection.[18] Skin texture, dryness, and fissures should be appreciated. Skin rashes or even tinea pedis should be noted. Location and severity of corns and callus should be noted. The location and depth of ulcers, wounds, and infections should be determined. Associated skin findings such as yellowish plaques indicative of necrobiosis lipoidica, brown pretibial macules characteristic of diabetic dermopathy, and skin atrophy associated with microvascular compromise should be noted.

Orthopedic. Pedal and digital deformities, such as bunions, hammer toes, and prominent metatarsal heads, should be located and graded. The foot should be examined for intrinsic muscle wasting. The Functional Foot Type® and the existence of the inclined posture should be determined. Shoes should be checked for wear and fit. Gait and postural abnormalities should be noted.

The Diabetic Foot Ulcer Prevention Plan

Risk factors for ulcer development should be determined and utilizing a classification system, a plan of preventive care should be instituted.[19] This type of plan is capable of preventing not only ulceration, but also infection, hospitalization, and amputation.

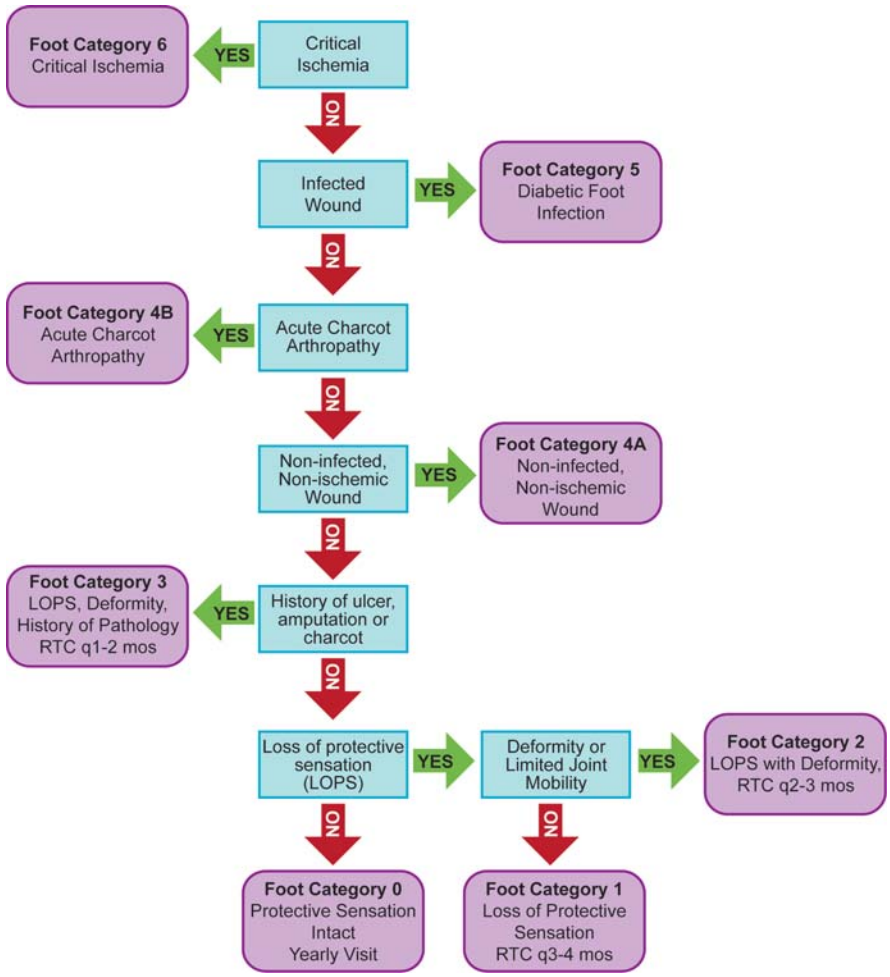

Fig. 25.8 The UT risk classification flowchart (Adapted from Armstrong DG et al.,[18] with permission)

Risk factors include the level of peripheral neuropathy and vascular compromise, the degree of foot deformity and joint mobility, and the existence of current or previous ulceration, infection, or Charcot foot. The University of Texas Risk Classification System (Fig. 25.8) utilizes all of these risk factors to classify diabetic foot ulcer risk. Once the patient is classified, the corresponding level of foot care needed in order to prevent ulceration is instituted. For example, if a patient has a loss of protective sensation and a previous history of ulceration of the foot, he or she would be rated as belonging to the UT foot category 3 and require foot care every 1–2 months. In this manner, an appropriate plan of foot care and monitoring can be established for each patient.

The Team Approach to Diabetic Foot Care

Successful management of the diabetic foot involves the concept of a team approach. The team consists of medical specialists, each focusing on specific risk factors, and commonly includes an endocrinologist, vascular surgeon, neurologist, podiatrist, diabetic nurse educator, and nutritionist.[19]

In successful models, the "captains" of the team include the treating endocrinologist, vascular surgeon, or podiatrist. New patients undergo a diabetic foot history and physical examination and have an initial consultation with the team members. Each specialist provides a baseline report including diagnosis, recommended immediate care, and long term follow-up to the captain who then reports to the patient's primary care physician.

Risk Factors for Amputation

The risk factors for lower extremity amputation are classified into primary and secondary.

The ***primary risk factors*** include peripheral neuropathy, peripheral vascular disease, structural and functional foot deformities, infection, and ulceration.

The ***secondary risk factors*** include obesity, impaired vision, improper footwear, lack of a home-based support system, and apparent noncompliance on the patient's part (Table 25.3).

Table 25.3 Risk factors for diabetic foot ulceration and amputation

Primary risk factors
1. Loss of protective sensation (LOPS)
2. Autonomic neuropathy (dryness and fissuring of the skin)
3. Peripheral vascular disease
4. Structural and biomechanical deformities
5. Prior infection
6. Prior ulceration
Secondary risk factors
1. Obesity
2. Impaired vision and retinopathy
3. Nephropathy
4. Poor control of diabetes
5. Poor footwear selection
6. At home noncompliance
7. Lack of adequate home support system

Primary Risk Factors

Peripheral Neuropathy

Peripheral neuropathy is the clinical manifestation of any of a number of potential defects in the physiologic function of the peripheral nervous system. The classic pattern of peripheral neuropathy development is distal to proximal with regard to anatomic location and small to large with regard to the size of the nerves that are involved. In other words, peripheral neuropathy typically begins in the distal lower extremity in a stocking distribution and then progresses proximally. In most cases, the initial nerves that are involved are the smallest and most terminal branches of the peripheral nerves within the epidermis. These myelinated (A-delta) and unmyelinated (c) fibers become diminished in number thereby leading to positive symptoms (pain and paresthesias) and/or negative findings (numbness and coolness). Patients may also experience autonomic deficits (hyperhidrosis, hyperperfusion); however, these rarely are "chief presenting complaints." This early stage of peripheral neuropathy has been designated as "small fiber" peripheral neuropathy. As more proximally located larger nerves become involved the neuropathy becomes "mixed."

Not withstanding the fact that most cases of diabetic peripheral neuropathy begin in the aforementioned distal to proximal pattern, such is not always the case. Patients may also be afflicted by primary large nerve peripheral neuropathy, whether involving single large peripheral nerves or multiple large or medium-sized nerves. The hallmark of large nerve peripheral neuropathy is diminished proprioception, vibratory sensation, and/or conduction velocity.

Loss of Protection Sensation

Insensitivity coexists with diabetic foot wounds more than 80% of the time.[20] The combination of structural foot deformities, biomechanical abnormalities, and poor fitting shoes with a lack of protective sensation in diabetic

feet dictates the need for frequent foot examination. Repetitive friction or trauma that would ordinarily cause no more than a painful blister can fester into a lower extremity amputation when LOPS is concomitant.[21]

When a 5.07-mm nylon monofilament (a 10-g force) is pressed against the skin to the point of buckling (Fig. 25.6), patients who cannot feel the filament are at risk for ulceration and require special care. Testing with the monofilament is the most effective screening device and is cost-effective, quick, and easy to perform. Other more time-consuming tests of vibration and thermal sensation are available for identifying patients prone to ulceration.[22] In addition, nylon monofilament testing can potentially register small fiber involvement whereby testing for vibratory sensation is only indicative of large fiber involvement. All patients with diabetes should be tested frequently with this inexpensive, rapidly performed test.

Autonomic Neuropathy

The autonomic component of the diabetic neuropathy produces reduced sweating and fissuring of the skin of the heels and soles and increased sweating in the toe web spaces, making them prone to infection and ulceration. In addition, there is a potential for osseous hyperemia that can be involved in the development of a Charcot foot.

Peripheral Arterial Disease (PAD)

Occlusive arterial disease of the posterior tibial and common peroneal arteries is four times more prevalent in diabetic patients.[23] Reduced pedal pulses, pedal hair loss, claudication, rest and night pain in the arch and calf, cool feet, indurated or shiny skin, dependent rubor, clubbed digits, and thickened toenails, as well as poor healing of cuts and wounds indicate the existence of PAD.[24]

Structural and Biomechanical Deformity

Structural deformities, such as bunions and rigid hammertoes, as well as normal anatomical prominences, such as the fifth metatarsal head and the base of the fifth metatarsal, serve as predictable locations for ulceration in the diabetic patient. It is important to document where these deformities exist for each patient and to instruct the patient to observe these areas carefully for change.

Orthopedic deformities are preceded by underlying biomechanical pathology that reduces weightbearing under one anatomical site thereby producing a compensatory increase in weightbearing in predictable pedal locations. This increased weightbearing creates repetitive injury, callus formation, and ulceration. This is the common mechanism in the creation of plantar metatarsal and great toe ulcers (malum perforans) in the diabetic patient and can be prevented by the use of well-selected and fit shoes and orthotics.

Infection

Diabetic patients tend to have cuts, contusions, and superficial tineal infections that are slow to respond to care. These otherwise minor injuries tend to get infected. Because of concomitant risk factors, multiple aerobic bacteria, yeast, and anaerobic organisms become pathogens in these wounds, making them difficult to control and heal. In addition, because the deep structures in the foot (such as the bone) are actually quite close to the surface, osteomyelitis is more common.[25]

Ulceration

Repetitive microtrauma, repetitive friction, and continuous pressure lead to corn and callus formation which, if left unattended in the insensate foot, leads to sublesional hemorrhage (intracorneal exsanguination) within the keratoses, with eventual ulceration, infection, and amputation.

Ulceration usually occurs in areas of bony prominence that are being irritated by shoe, weightbearing pressure, or excess activity. Without the reflexive repositioning that is necessary to prevent ischemia related to compressive forces over bony prominences, the affected skin is stressed secondary to localized ischemia and is therefore susceptible to ulceration. Thus, ulcerations occur over bony prominences that are irritated by shoe gear or excessive weightbearing pressure.

Secondary Risk Factors

Obesity

Obesity plays an important role in initiating and maintaining type 2 diabetes. It also plays a role in lower extremity amputation since, with obesity, weightbearing increases for all foot structures. The presence of obesity magnifies biomechanical pathology. For this reason, among others, weight reduction must be considered a critical goal in overweight diabetic patients.

Impaired Vision

The demographic characteristics for lower extremity amputation are skewed toward senior citizens with an age greater than 60. This population suffers from age-related vision problems such as cataracts and glaucoma. In addition, these patients may suffer from diabetic retinopathy. Impaired vision prevents patients from self-examination and self-care of the feet. It also impairs the ability of patients to observe their surroundings and plan movements safely.

Improper Footwear

Irritation and pressure from poor sizing and selection of footwear play a critical role in the development of ulcers and infections. Since insensitivity also includes reduced proprioception, patients with diabetes often cannot tell if their shoes are well fit or creating irritation. Therefore, diabetic patients need skilled shoe fitters and ongoing monitoring of their shoes.

Tight shoes, high heels, low toe boxes, and improper fitting (either too small or too large) may press shoes onto bony prominences and contribute to the formation of ulcers. Shoes should be selected and sized with sufficient toe box, width, closing systems, and depth in order to accommodate all existing deformities without being too large. Selecting a larger size, if in doubt, should reduce errors, but it should be noted that as a shoe becomes too large for a patient's foot, balance and gait problems become exposed.

The Congress has tried to address the needs of diabetic patients by initiating The Medicare Therapeutic Shoe Bill in 1996.[26] Under this bill, a physician must certify that a patient has diabetes, is under a treatment plan for diabetes, and has a related foot problem. A professional with shoe prescribing knowledge, such as a podiatrist, may then prescribe a pair of shoes with protective insoles. Medicare will pay for one pair of shoes and three protective insoles or a molded shoe annually.

Shoe Noncompliance at Home

Patients with diabetes and LOPS often do not wear their protective shoes when at home.[27] Since this may be where they spend most of their day, slippers should be dispensed with protective foot inserts (orthotics) and the

use of diabetic socks should be considered. If problems with the feet continue, outdoor shoes should be worn at all weightbearing moments, even when at home.

Lack of a Home-Based Support System

The ability to observe and care for a wound and the additional needs of a diabetic patient are enhanced by the support system. Without adequate support from a family member or visiting professional, such as a wound care nurse, wounds that would otherwise heal uneventfully can lead to a lower extremity amputation in a matter of days. The physician must coordinate home support or change the patient's environment in order to prevent such tragedies.

Apparent Noncompliance on the Patient's Part

When working with diabetic feet that are ulcerated or infected, a physician may experience a false sense that the patient is noncompliant. Since the lack of pain sensation can interfere with the patient's compliance, it is the responsibility of the physician to alert all parties involved in the patient's care, including the patient himself or herself, to this problem and to assume adequate monitoring.

Plantar Offloading of the Diabetic Foot

The feet are the foundation of the posture and must accept a lifetime of weightbearing stress. Biomechanics, body weight, and activity level determine the location and timing of areas of potential breakdown. It is necessary to disperse the plantar weightbearing forces away from high stress areas in order to prevent ulceration or to heal an existing wound.

Thermography and computerized pressure scanning can be used to predict sites that will ulcerate on the plantar surface of the diabetic foot. Plantar off-loading of the diabetic foot encompasses the use of pads, inserts (foot orthotics), shoe modifications, pressure-distributing boots, and prophylactic foot surgery to remove or redistribute stress away from areas under extreme pressure.[28]

Principles of Padding

The use of $^{1}/_{4}''$ adhesive felt and other pads to relocate pressure proximally to a problematic area can be a key element of wound care. Given the human gait cycle, pads that are horseshoe or rectangular in shape and placed just proximal to a callus (or ulcer) will reduce pressure under the callused area (or ulceration) without being too bulky. This will prevent the breakdown of a callus (or heal the ulcer). These pads can be adhered directly to the foot or incorporated into the footbed of a shoe. It should be noted that pads placed directly on a pressure area would actually add to the pressure.

Foot Orthotics

Foot orthotics can be *prefabricated* (over-the-counter), *customized prefabricated* (over-the-counter with custom modifications), or *custom* (taken from a cast or scan of the foot). They are made from materials that vary from soft and accommodating to rigid and supportive. Orthotics may be soft, hard, or mixed in nature, depending on their purpose. A rigid device can support and control the arches to prevent collapse. An accommodative device

can cushion and give comfort and protection to a weak or diabetic foot that is beyond salvage. A mixed material orthotic, when custom casted, can support the arch while removing pressure from specific overweighted areas.

Since an orthotic can be utilized to improve function and quality of life, as well as to reduce pressure in desired locations, the diabetic foot, especially the insensitive diabetic foot, deserves a custom orthotic shoe bed for safe and maximum performance.

Shoe Modifications

Because of cosmetics, shoe modifications should be a last resort. Today, there is over-the-counter footwear that fills the need for almost all diabetic patients. Depth inlay shoes, therapeutic shoes, Velcro closure shoes, wound healing shoes, walking shoes, and comfort shoes have largely replaced the molded shoe from a cast. Custom modifications, such as rocker bars, lifts, cutouts, and heel and sole wedge, can then be added to overcome specific problems.

Pressure-Distributing Casts and Boots

Non-healing wounds (older than 6 months) can often be healed with total contact casting (TCC). Weekly application of a pressure-distributing cast reduces pressure underneath the wound, yet allows for weightbearing function. This gold standard is slowly being replaced by a new generation of healing boots that reduce pressure under wounds yet, unlike TCC, allow for their removal for inspection, physical therapy, and unencumbered bed rest. While these removable devices are generally better accepted than fixed casting, they do present the added variable of concern over patient compliance.

Prophylactic Foot Surgery

Podiatrists and orthopedic surgeons perform osteotomies, soft tissue balancing procedures, corrective digital procedures, and bony spur excisions on diabetic feet in order to eliminate the occurrence or the recurrence of ulcers and infections. Utilizing the information on foot typing, weightbearing X-rays, thermography, and pressure mat scanning, a surgeon can predict the precise locations that may ulcerate and become infected in the future. In well-selected cases, foot surgeons can prevent a future problem at a time when the vascular system is adequate to allow healing. The same surgical procedures, if performed at a later date, in the face of peripheral vascular disease, may be contraindicated. For example, if a diabetic patient with an insensate foot but adequate circulation has a plantarflexed second metatarsal that is rapidly forming thick callus, a prophylactic dorsiflectory osteotomy of the second metatarsal will prevent a future malum perforans at this site.

Diabetic Neuropathy Testing

Most testing methods that are used in the assessment of peripheral neuropathy have at least one of three important limitations. Foremost is subjectivity that is incorporated into almost all tests involved in the testing of small fiber neuropathy. Secondly, some tests, such as sural nerve biopsies and nerve conduction studies, characterize only large fiber neuropathies and fail to assess the small fiber component. Finally, many available testing methods are performed only at major academic centers.

Epidermal nerves fiber density (EDNF) testing can be used to diagnose peripheral neuropathy. Epidermal nerve fibers are the terminal branches of peripheral nerves which pass into the epidermis as unmyelinated C-fibers or myelinated A-delta fibers. The presence of epidermal nerve fibers has been hypothesized for over a century; however, their existence was not confirmed until the advent of electron microscopy. In persons with small fiber peripheral neuropathy, the number of epidermal nerve fibers is characteristically diminished per area

unit. Since the average density of epidermal nerve fibers is consistent for each anatomic location (independent of age or gender), a "normal" range can be determined. [29] An epidermal nerve fiber density below this reference range is consistent with small fiber peripheral neuropathy.

Epidermal nerve fiber density is usually measured within a standard 3-mm cutaneous punch biopsy. By convention samples are obtained from the lower leg and foot, 10 cm proximal to the lateral malleolus and the heel or great toe so that the epidermal nerve fibers within the skin sample may be quantified.

Immunoperoxidase stain PGP 9.5 is employed.[30] This stain uses antibodies that are neuron specific and once applied, it highlights epidermal nerve fibers thereby allowing them to be quantified under light microscopy (Fig. 25.9)

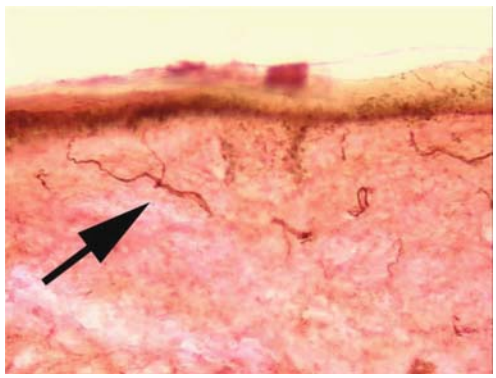

Fig. 25.9 Epidermal nerve fiber test, note epidermal nerve fibers, Dr. Bradley Bakotic

This technique has numerous advantages over others; foremost among them is the fact that it is a wholly objective way to detect peripheral neuropathy. This makes ENFD testing an excellent option for establishing a baseline from which to assess improvement following therapy. Other important advantages are its ease, sensitivity, and specificity. Epidermal nerve fiber density is significantly more sensitive than sural nerve biopsy.[29] Overall, the specificity of ENFD testing is an impressive 97% at the fifth percentile cutoff value, while the specificity is roughly 45%. Maintaining the fifth percentile cutoff value for the ENFD will correctly classify 88% of all tested neuropathy cases.[29] In addition, studies have shown that ENFD can reveal small nerve neuropathy in the precursor of diabetes known as impaired glucose tolerance (IGT) making it a valuable tool in monitoring "borderline" or prediabetic patients.[31] The use of long- and short-acting local anesthetics and cyanocobalamine as common peroneal and posterior tibial nerve "chemical sympathectomies" has been shown to be effective in the treatment of diabetic peripheral neuropathy.[32]

Alternative Treatment

One alternative therapy is based on the evidence of insufficient dietary intake of gamma linoleic acid (GLA) as a possible cause of the diabetic peripheral neuropathy.[30] Normal subjects can convert linoleic acid (LA), which is readily available in our diet, into GLA. However, some diabetic patients have a reduced capacity for this conversion. Evening primrose oil (EPO) seeds, when crushed, are a safe source of GLA. A total of 450 mg, given orally, twice a day, may reverse the signs and symptoms of diabetic neuropathy in 10–14 days in a diabetic individual.[32]

Alpha lipoic acid and L-arginine are two other supplements that may be helpful in some cases. Topical capsaicin cream in low concentration (0.025%) applied sparingly to affected areas may be of some use in subjects that cannot tolerate other treatment.

The use of long- and short-acting local anesthetics and cyanocobalamine as common peroneal and posterior tibial nerve "chemical sympathectomies" has been shown to be effective in the treatment of diabetic peripheral neuropathy.[32] Exercises are generally effective in remobilizing the hammered digits. Toe fists (15X), toe pickups

(picking up cotton balls or pencils) (15X), extending the toes over the binding of a book (20X) and toe creeping (crawling with the toes on the ground) (2 min) should be repeated 1–2 times/day.

Surgically, a decompression of the areas where swollen nerves become entrapped is becoming more accepted in relieving late-phase sensory neuropathy.[33]

Cellulitis and Osteomyelitis

Cellulitis and osteomyelitis are still major risk factors for amputation in the diabetic foot. These risks are somewhat reduced due to advances in diagnostic techniques, oral and intravenous antibiotics, and the team approach to care. The bone scan and MRI studies have replaced X-rays as the standard techniques for early diagnosis and monitoring followed by bone biopsy, if necessary. In a vascularized limb, with appropriate antibiotic therapy, cellulitis resolves in a matter of days[31] and 6- to 8-week course of antibiotics will heal osteomyelitic bone. If this protocol fails, the insertion of antibiotic beads can be considered.[32] Since early diagnosis is critical, the physician must react quickly and aggressively when the signs and symptoms of cellulitis and osteomyelitis are apparent.[33]

Non-healing Wound Care

With the evolution of modern antibiotics, diagnostic tools such as MRI, home wound care, and educational programs that allow the physician to make an earlier diagnosis and provide more effective treatment than in the past, most diabetic foot wounds heal uneventfully.[34] However, wounds that have not significantly healed after 3–6 weeks of care can be considered non-healing. They have sparse angiogenesis potential, reduced growth factor release, and are more likely to become infected. These wounds can, however, be converted into acute, healing wounds, with proper management.

After evaluation of the status of the foot and of the wound a clinician can classify a wound according to its depth, size, and healing ability. A multifaceted program of care can then be instituted and monitored.

Clinical attention to many factors involved in healing a wound, rather than focus on a single risk factor, can lead a non-healing wound to improve at a time when it seems to be stagnant. If the status of a particular system or risk is uncertain, then immediate consultation with a specialist (s) is in order.

Evaluation of the Status of the Wound

The wound should be measured and photographed.[35] The wound should be classified according to depth as follows:

1. pre- or post-ulceration, completely epithelialized;
2. superficial and not involving tendon, capsule, or bone;
3. penetrating to tendon or capsule; and
4. penetrating to bone.

The location of the wound is critical and will determine the importance for off-loading. The margins should be examined for redness, heat, swelling. The level of pain should be determined, but since insensitivity of wounds in diabetic patients is the rule, painless wounds should be considered high risk. The epithelial borders of the wound should be assessed. The quality of the base of the wound should be determined. A fresh, granular base is desirable and necrosis and detritus are to be avoided. The presence of bleeding and/or exudate should be recorded. Microbial culture and sensitivity should be obtained and an antibiotic should be started if the wound is showing signs of drainage, infection, or poor healing. It should be noted that even the smallest amount of

healing every week will lead to a healed wound but a wound that remains stagnant for more than 2 weeks needs immediate reassessment. Some changes in the treatment regimen or consultation are necessary at this critical time.

Evaluation of the Status of Complications

Wounds should be classified according to the vascular status and the presence of infection:
(1) absence of vascular complications and infection;
(2) presence of infection;
(3) presence of vascular complications; and
(4) presence of both infection and vascular complications.

A vascular and/or infectious disease consultation becomes critical when these complications are present.

Classification of Diabetic Foot Wounds

Once the clinician determines the status of the wound and the foot, it is necessary to utilize this information to classify the wound and establish a treatment protocol. The UT wound classification[36] gives a wound status number (0–3) and a foot status letter (A–D) which gives a final description of the wound. For example, a UT wound classification of C-3 would be a wound penetrating to bone involving an avascular foot. Figure 25.10 shows the University of Texas Wound Classification Flowchart which incorporates both the status of the wound and complications.

Meticulous Debridement

Meticulous debridement is the most important service that a chronic, non-healing wound deserves since wounds that are not aggressively debrided form a callus that masks the clinician's ability to evaluate, treat, and monitor a wound. In a poorly healing wound, the epithelial edges seal and stop growing inward, preventing closure of the wound.

The base of the wound becomes necrotic and infected and growth factors cease to be released (Fig. 25.11). Meticulous debridement utilizes aggressive sterile surgery to remove peripheral callus and make the epithelial edges raw. It eliminates necrotic tissue and detritus as well as free bleeding at the base and periphery of the wound.

Debridement stimulates the release and production of the multiple growth factors that contribute to wound healing.[38] It denudes circumferential epithelial margins so that the skin margins can continue to close instead of "sealing off" at the wound edge. It stimulates angiogenesis and the vascular cascade. Wounds should be measured weekly and debrided using a sterile field and instruments, as they heal. Debridement can be carried out on a weekly basis in the outpatient office or wound healing center setting. In contrast, a deep wound with need for more aggressive tissue dissection and instrumentation should be performed in the operating room.[40,41]

Off-loading of the Non-healing Wound

The use of $\frac{1}{4}''$ adhesive sponge horseshoe-shaped pads just proximal to non-healing wounds reduces plantar pressure on these wounds dramatically. Wound healing shoes and boots with custom insoles to reduce weightbearing pressure are invaluable. If, in spite of these measures, a wound remains resistant to healing, a wheelchair, contact casting, or bed rest must be considered.

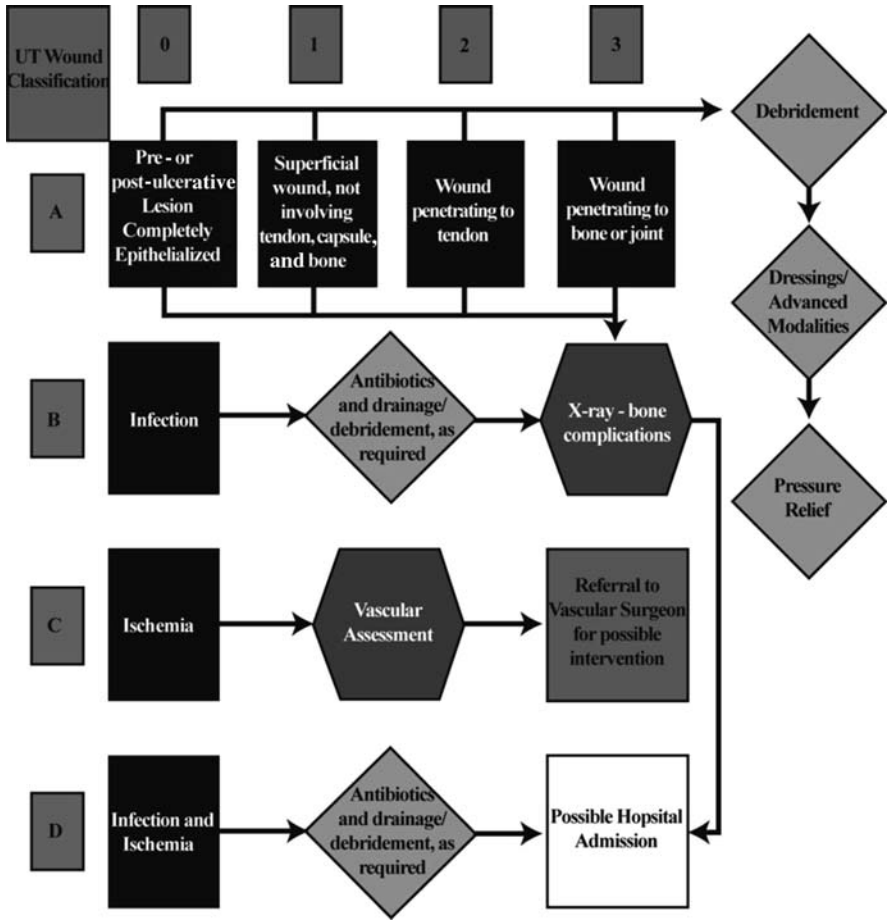

Fig. 25.10 The UT wound classification flowchart. (Adapted from Armstrong DM et al.,[37(p150)] with permission)

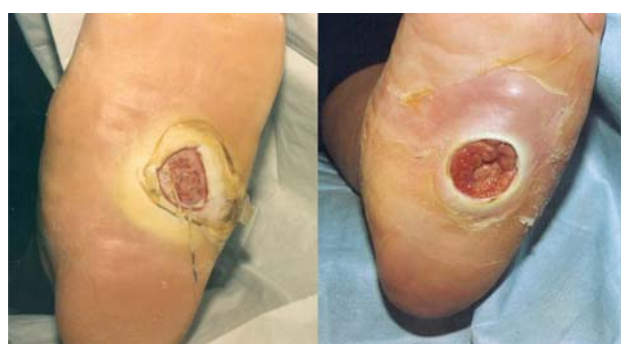

Fig. 25.11 A diabetic ulcer under the cuboid of a Charcot foot, before and after meticulous debridement. (Adapted from Brenner,[1] with permission.)

Advances in Wound Healing

The three most utilized advances in wound care are hyperbaric oxygen treatment,[39] recombinant growth factors,[42] and bioengineered alternative tissues.[43] These modalities are adjunctive to meticulous debridement and

off-loading and, although costly, when used in properly selected cases, can reduce the healing time of wounds as well as the overall cost of care.

Hyperbaric oxygen treatment (HBO) uses pressurized 100% oxygen, delivered in a full body chamber. If the PO_2 of the wound surface is low, then HBO may be of benefit. This is of particular benefit in wounds that are complicated by microvascular disease.[44]

Platelet-derived growth factor (PDGF) has been successfully produced in a gel form and, when applied to large and deep noninfected wounds, it can accelerate the healing process.[45] Other growth factors are under investigation and additional products are on the horizon, including vascular endothelial growth factor (VEGF).

Bioengineered alternative tissues (BAT) are a highly specialized group of products that include both living and nonliving applications which can serve as wound healing adjuncts. The most sophisticated products to date include living human keratinocytes and living human dermal fibroblasts derived from neonatal foreskin and propagated in culture.[46,47] These products are easy to apply and, since BAT serves as a substrate for the patient's own skin repair, the end result is a plantar wound covered by plantar tissue.

Vacuum-assisted closure (VAC) is a new dynamic adjunct to the wound healing armamentarium.[48] Also called *negative pressure wound therapy*, this technology is best suited for large, deep wound defects or post-partial foot amputation wounds. Negative pressure wound therapy utilizes an electrically powered suction pump and occlusive dressing system to create a subatmospheric pressure at the wound base. This serves to stimulate angiogenesis, reduce bacterial burden, and efficiently remove exudates from the wound site.

The past decade has brought an enormous number of new topical wound care technologies to this field. Two categories are seeing wider acceptance in wound healing circles. The first is the utilization of slow release topical silver into the wound base for the reduction of bacterial burden and biofilm contaminant that often retard healing or cause clinical wound infection.[49] The second is the use of active honey tested for its antibacterial component topically found only in honey produced from *Leptospermum* (Manuka) rated with a Unique Manuka Factor (UMF) of 10+, even when diluted and in a non-sterile state.[50,51]

Conclusions

Diabetes affects the feet, with regularity as the disease progresses. Complications of the foot are the leading cause for hospital admission in patients with diabetes.

By implementing a foot care program that involves risk classification, biomechanical evaluation, the team approach, and close monitoring, the physician will be able to keep his/her patients active, functional, ulcer and infection free and will reduce the risk of limb loss and maintain a high quality of patient's life.

References

1. Brenner MA, Ed. *Management of the Diabetic Foot (Chapter 6)*. Williams and Wilkins, New York, 1987.
2. Mayfield J. Foot examinations reduce the risk of diabetic amputations. *J Fam Practice*. 1998;49:499–504.
3. American Diabetes Association. Foot care in patients with diabetes mellitus. *Diabetes Care*. 1998;21:54–58.
4. Reiber GE, Raugi GJ. A model for foot care in people with diabetes. *Lancet*. 2005;366:1676–1677, 1719–1724.
5. Boulton AJ, Vileikyte L, Apelqvist J. The global burden of diabetic foot disease. *Lancet*. 2005;366:1719–1724.
6. Einhorn DA. The state of diabetes complications in America. The American Association of Clinical Endocrinologists 16th Annual Meeting and Clinical Congress; May 2007; Seattle, Washington.
7. Ragnarson TG, Apelqvist J. Health and economic consequences of diabetic foot lesions. *Clin Infect Dis*. 2004;39(Suppl 2):S132–S139.
8. Todd WF, Armstrong DG, Liswood PJ. Evaluation and treatment of the infected foot in a community teaching hospital. *J Am Podiatr Med Assoc*. 1996;86:421–426.
9. Lower Extremity Amputation Prevention Project. *Diabetes Care*. 1998;21:23–25.
10. Armstrong DG, Lavery LA, Stern S, et al. Is prophylactic diabetes foot surgery dangerous? *J Foot Ankle Surg*. 1996;35:585–590.
11. Shavelson D. A closer look at neoteric biomechanics. *Podiatry Today*. Sept 2007;9:234–241.
12. Root ML, Orien WP, Weed JH. Normal and abnormal function of the foot. In: *Clinical Biomechanics*, Vol 2, Clinical Biomechanics Corp, Los Angeles, CA, 1977.
13. Inman VT. *The Joints of the Ankle*. Mann RA and Inman VT, Eds. Williams and Wilkins, Baltimore, MD 1976.

14. Shavelson D. The unequal limb syndrome: biomechanical considerations. *J Am Acad Pod Sport Med*. 1983;1:18–23.
15. Caputo GM. The nylon monofilament test for sensation. *New Eng J Med*. 1994;331:854–859.
16. Sommer, TA. Charcot foot, the diagnostic dilemma. Amer Fam Prac 11: 109, 1995.
17. Frykberg R, Ed. *The High Risk Foot in Diabetes*. Churchill Livingstone, New York, 1991.
18. Armstrong DG, Lavery LA, Harkless LB. Who is at risk for diabetic foot ulceration?. *Clin Podiatr Med Surg*. 1998;15:11–19.
19. Frykberg RG. Team approach toward lower extremity amputation prevention in diabetes. *J Am Podiatr Med Assoc*. 1997;87: 5–12.
20. Selby JV, Hang D. Risk factors for lower extremity amputation in persons with diabetes. *Diabetes Care*. 1995;18:509–516.
21. Day MR, Harkless LB. Factors associated with pedal ulceration in patients with diabetes mellitus. *J Am Podiatr Med Assoc*. 1997;87:365–369.
22. Young MJ, Breddy JL, Veves A, et al. The prediction of diabetic neuropathic foot ulceration using vibration perception thresholds. *Diabetes Care*. 1994;17:557–562.
23. McNeely MJ, Boyko EJ, Ahroni JH, et al. The independent contributions of diabetic neuropathy and vasculopathy in foot ulceration. *Diabetes Care*. 1995;18:216–219.
24. Sykes MT, Godsey JB. Vascular evaluation of the problem diabetic foot. *Clin Podiatr Med Surg*. 1998;15:49–83.
25. Apelqvist J. Wound healing in diabetes: outcome and costs. *Clin Podiatr Med Surg*. 1998;15:21–39.
26. The Medicare Therapeutic Shoe Bill. Department of Health and Human Services, Medical Carriers Manual, Section 2134, p2-85.1-2-86. US Gov't Printing Office, Washington, DC, 1994.
27. Armstrong DG, et al. Continuous activity monitoring in persons at high risk for diabetes-related lower extremity amputation. *J Am Podiatr Med Assoc*. 2001;91:451–455.
28. Lavery LA, Vela SA, Lavery DC, et al. Reducing dynamic foot pressures in high-risk diabetic subjects with foot ulcerations. *Diabetes Care*. 1999;19:818–821.
29. McArthur JC, Stocks EA, Hauer P, Cornblath DR, Griffin JW. Epidermal nerve fiber density. Normal reference range and diagnostic efficiency. *Arch Neurol*. 1998;55:1513–1520.
30. Chien HF, Tseng TJ, Lin WM, et al. Quantitative pathology of cutaneous nerve terminal degeneration in the human skin. *Acta Neuropathol*. 2000;102:455–461.
31. Shavelson D Local anesthetics and injectable cortisone. In: T Delauro, Ed. *Clinics in Pod Med & Surg*, Chapter 8, W.B. Saunders, Philadelphia, 1993
32. Jamal GA. The use of gamma linoleic acid in the prevention and treatment of diabetic neuropathy. *Diabetes Medicine*. 1994;11:145–149.
33. Hounsom L, et al. GLA is effective against multiple indices of experimental diabetic neuropathy. *Diabetologia*. 1998;41: 839–843.
34. Mayo Clinic Reports. Alpha Lipoic Acid (ALA) improves symptoms of diabetic neuropathy. *Diabetes Care*. March 2003;26(3): 456–459.
35. Head KA. Peripheral neuropathy: pathogenic mechanisms and alternative therapies. *Altern Med Rev*. Dec 2006;11(4):32–37.
36. Resneck HE, et al. Independent effects of peripheral nerve dysfunction on lower-extremity physical function in old age. *Diabetes Care*. 2000;23:132–138.
37. Armstrong DG, Harkless LB, et al. Validation of a diabetic wound classification system: the importance of depth, infection and ischaemia. *J Am Podiatr Med Assoc*. 1998;79:144–153.
38. Marcinko DE. Antibiotic-impregnated polymethyl methacrylate beads. In: *Infections of the Foot: Diagnosis and Management*. Habershaw GM Ed. Mosby-Year Book Pub, St. Louis, MO, 174–176, 1998.
39. Gibbons GW, Habershaw GM. Diabetic foot infections: anatomy and surgery. *Infect Dis Clin North Am*. 1995;9:131–142.
40. Frykberg RG. Diabetic foot ulcers: current concepts. *J Foot Ankle Surg*. 1998;37:440–446.
41. Sage RA. The management of foot ulcers. In: *Advances in Podiatric Medicine and Surgery*. W Joseph, Ed. Mosby-Year Book Pub, St. Louis, MO 139–153, 1995.
42. Sommer TA. Charcot foot: the diagnostic dilemma. *J Fam Pract*. 2001;11:1591–1599.
43. Poretsky LY, Shavelson DE, et al. The pre-charcot foot. A new clinical entity to prevent Charcot Joint Disease. *Clin Diabetol*. Sept 2006;25(3):24–28.
44. Faglia E, Favales F, Aldeghi A, et al. Adjunctive systemic hyperbaric oxygen therapy in treatment of severe prevalently ischemic diabetic foot ulcer: a randomized study. *Diabetes Care*. 1996;19:1338–1342.
45. Wieman TJ, Smiell JM, Su Y. Efficacy and safety of a topical gel formulation of recombinant human platelet-derived growth factor-BB (becaplermin) in patients with chronic neuropathic diabetic ulcers: a phase III randomized placebo-controlled double-blind study. *Diabetes Care*. 1998;21:822–829.
46. Snyder RJ, Simonson DA. Cadaveric allograft as adjunct therapy for nonhealing wounds. *J Foot Ankle Surg*. 1999;38:93–101.
47. Falanga V. How to use Apligraf to treat venous ulcers. *Skin and Aging*. 1999;2:30–36.
48. Webb LX. New techniques in wound management: vacuum assisted wound closure. *J Am Acad Orthop Surg*. September/October 2002;10(5):303–311.
49. Lansdown ABG. Silver: toxicity in mammals and how its products aid wound repair. *J Wound Care*. 2002;11(5):173–177.
50. Cutting K. Antibacterial honey: in vitro activity against clinical isolates of MRSA, VRE and Pseudomonas aeruginosa. *Wounds*. 2007;19(9):231–236.
51. Molan PC. The evidence supporting the use of honey as a wound dressing. *Int J Low Extrem Wounds*. 2006;5:40–42.