

# ProCare Training Manual

## Chapter 8

### Diabetic Foot Ulcers

Diabetes has been characterized as a modern day epidemic. In the United States, approximately 6% of the population has diabetes, which is roughly 16 million people! Every year, 798,000 new cases of diabetes are diagnosed. 15% of all patients with diabetes develop diabetic foot ulcers and of these 14%-20% of these will require an amputation. Diabetes is the seventh leading cause of death in the United States and is more prevalent in African Americans, Hispanics, and Native American cultures and the risk increases from middle- age to the older adult population.

Diabetes Mellitus is a metabolic disorder characterized by hyperglycemia as a result of lack of insulin or the lack of the effect the insulin has on the body. The role of insulin in the body is as a vehicle. Insulin transports glucose into the cells where it is used as fuel or stored as glycogen. Insulin also stimulates protein synthesis and storage of free fatty acids in fat deposits. The lack of useable insulin or the inability of the body to accept and use this insulin available, compromises these important functions. Diabetes can begin in early childhood, or develop slowly or rapidly throughout the life cycle. The onset of diabetes usually indicates the type of diabetes each patient has. In most cases, childhood onset is often referred to as Type I and adult onset is often referred to as Type II.

### Causes of Diabetic Foot Ulcers

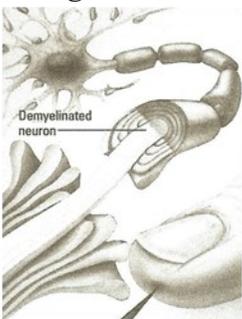
Diabetic neuropathy, poor circulation, pressure and other mechanical forces can cause foot ulcers in patients with diabetes. Uncontrolled diabetes commonly results in a trineuropathy (three concurrent neuropathies) that significantly increase the patient's risk for diabetic foot ulceration.

### Neuropathy

Peripheral neuropathy is the primary cause of the development of diabetic foot ulcers. Neuropathy is a nerve disorder that results in impaired or lost function in the tissues served by the affected nerve fibers. There are two main causes for the development of neuropathy in the diabetic patient. The first being ischemia which is caused by the thickening of tiny blood vessels that supply the nerve and the second by nerve demyelination. Nerve demyelination is the destruction of protective myelin sheath surrounding a nerve, this slows the conduction of the nerve impulse, or sensation.

Polyneuropathy, or damage to multiple types of nerves, is the most common form of neuropathy in patients with diabetes. In the foot, loss of sensation, loss of motor function and loss of autonomic functions combined forms what is known as trineuropathy.

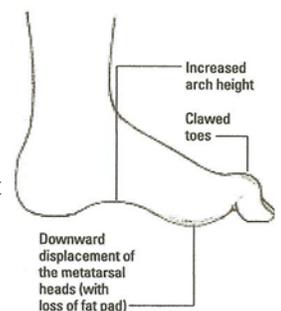
Figure 8-A



**Sensory Neuropathy-** Ischemia or demyelination causes nerve death or deterioration. This causes the inability of the patient to feel painful stimuli and therefore, the inability to respond to appropriately. (Figure 8-A)

**Motor Neuropathy-** Intrinsic muscles deep in the plantar surface of the foot atrophy. This results in an increased arch height and clawed toes. In addition, the fat pad that normally covers the metatarsal heads migrates toward the toes, exposing the metatarsals to increased pressure. This increases the risk of ulceration to the upper surfaces of the clawed toes, especially if the patient has ill-fitting shoes. (Figure 8-B)

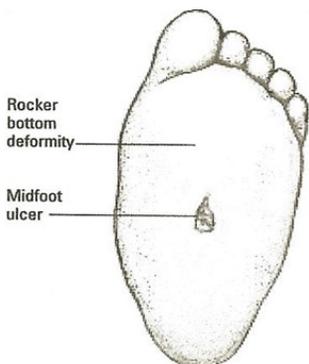
Figure 8-B



**Autonomic Neuropathy-** Inhibits or destroys the sympathetic component

of the autonomic nervous system, which controls vasoconstriction in peripheral blood vessels. This in turn may cause osteopenia, a reduction in bone volume in the foot and ankle bones. In Charcot disease (pronounced Shar-Co) disease, the bones weakened by osteopenia suffer fractures that the patient doesn't feel due to sensory neuropathy. Overtime, this process causes bony dissolution that culminates with the collapse of the midfoot into a rocker-bottom deformity. Midfoot ulcers that are the result of a rocker-bottom foot heal more slowly than ulcers of the forefoot.

As sensory nerves degenerate and die, the patient experiences a burning or "pins and needles" sensation. This sensation is usually worse at night. Medications, such as Neurontin, can be prescribed by the physician to help alleviate the symptoms of neuropathy, but it will not reverse or decrease the effects of the neuropathy. Some patients have also reported a



decrease in symptoms by moisturizing the feet and sleeping in cotton, seamless, socks over night. As the sensation declines, anything from stepping on something sharp to wearing ill-fitting shoes can result in foot injury because the patient can not feel the pain.

As motor nerves degenerate and die, muscles in the limbs atrophy, especially the intrinsic muscles of the feet, which causes foot drop and structural deformities. These changes increase the patients risk for stumbling or falling and further increases the damage to the foot.

As autonomic nerves degenerate and die, sweat and sebaceous glands malfunction and skin on the patient's feet becomes very dry and cracks. If fissures (deep cracks in the skin, usually on the heels) develop, the risk of infection rises.

## **Poor Circulation**

In Peripheral Vascular Disease (PVD), atherosclerosis narrows the peripheral arteries, slowly reducing the flow of blood to the limbs. In diabetes, the arterial damage caused by atherosclerosis reduces the blood flow to the lower limbs as well as to the nerves that innervate them. In addition to promoting ulcer development, poor perfusion slows the healing process for existing ulcers and impedes circulation of systemic antibiotics to the infected areas. As perfusion drops, the risks of ischemia and tissue necrosis increases.

## **Pressure and Other Mechanical Forces**

The mechanical forces involved in diabetic foot ulceration are the same as those responsible for pressure ulcer formation: pressure, friction and shear. Due to the impaired sensation from sensory neuropathy, the patient is not able to feel the increased discomfort from increased or prolonged pressure. The most common areas for diabetic ulcers are those over bony prominences, the metatarsal heads, great toe and heel.

Although pressure is the main culprit in mechanical force injuries, it is not the only one. A few examples of friction are when a loose shoe rubs against the foot or the foot sliding across a bed sheet. Either of these repetitively can cause blister formation. In shearing injuries, the skin of a sweating foot can cling to a shoe while the underlying tissues slide beneath the skin.

Being knowledgeable of the causes of diabetic foot ulcers is imperative for healing as well as prevention. Instruct your patients in the underlying causes and repercussions of uncontrolled diabetes. It is especially important to instruct patients suffering from sensory neuropathy to always wear shoes and inspect their feet daily for injury. Diabetics should be instructed to wear white cotton, seamless socks. This allows the foot to "breathe" as well as the patient to easily assess for blood secondary to unknown trauma. Synthetic socks increase the risk of friction and shear due to the foot's increased or decreased sweating mechanisms secondary to motor neuropathy.

Shoes should fit well and be replaced often. Diabetics with foot anomalies, such as bone deformities (Charcot, etc), should be custom fitted for diabetic shoes. These are made by orthotists from a mold or a mapping taken of the patient's foot. If the diabetic foot is free from foot malformations presently, instruct them to wear shoes made of breathable material (cotton or leather) with a wide toe box. An example of shoes with wide toe boxes is New Balance tennis shoes. Proper foot ware will be discussed more extensively later on in this chapter.

## **Assessment of Diabetic Foot Ulcer Patient**

Assessment of diabetic foot ulcers includes a thorough patient history, a physical exam and special testing of the lower extremities. In addition to the basic information elicited during a patient history, ask the patient about the following:

- Date of onset of diabetes
- Management measures
- Medications
- Other diagnosed problems
- Status and history of neuropathy
- Allergies, including skin reactions
- Tobacco and alcohol use
- Recent changes in activity level
- Date and location of previous ulcers
- Onset of current ulcer
- Cause of current ulcer, if known
- Pain associated with ulcer

Use a holistic approach when performing a physician examination, keeping in mind that overall physical health and state of mind affect wound healing. Assess the musculoskeletal system, including posture, gait, strength,

flexibility, endurance and range of motion. The neurological exam should include balance, reflexes and sensory functions. To assess the vascular system, it is important to assess the strength of posterior tibial and dorsalis pedis pulses as well as the ankle-brachial index (ABI). Finally, assess the skin, including the texture, temperature, color, presence or absence of hair, sweat glands and nails.

When assessing the feet, pay special attention to the high risk areas for existing or impending ulcers such as calluses. These can be found on the plantar surfaces (soles) of the toes, tips of toes, between the toes, and the lateral aspect of the foot's plantar surface. The periwound (skin surrounding the wound) can include calluses (considered pre wounds in a diabetic), blood blisters, erythema, induration, skin fissures and dry scaly skin. The clinical characteristics of a diabetic foot ulcer, usually depends on where the ulcer is located. The table below lists the typical wound characteristics, but is not all inclusive of diabetic foot ulcers. See table 8-D

**Table 8-D**

Ulcer Location	Clinical Features
Plantar Surface	Even wound margins
Great Toe	Deep wound bed
Metatarsal head	Dry or low to moderate exudates
Heel	Low to moderate exudates
Tip of top of toe	Pale granulation with ischemia or bright red, friable granulation tissue with infection.

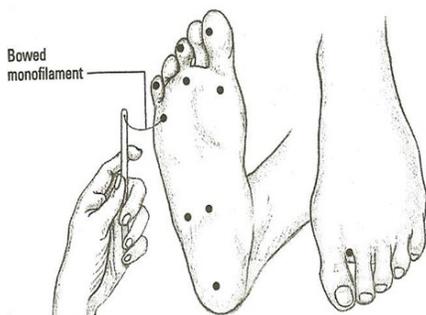
## Testing

Special tests provide a clearer picture of the health of the lower leg and foot. These tests evaluate pressure, neurological function, and perfusion. The results of these tests provide insight into the cause of injury, condition of the wound bed and the surrounding tissues, prognosis for healing and the required treatment interventions.

Harris mat prints and computerized pressure mapping are special musculoskeletal tests that provide information about the plantar pressures of the foot. High pressure areas usually correlate with calluses (prewounds) or existing wounds. The results help guide the choice of special off-loading devices which help relieve pressure when the patient stands or walks. These tests are typically performed by the orthotist.

Neurological tests for the lower extremities include deep tendon reflex testing, vibration perception testing with a tuning fork, Semmes-Weinstein monofilament testing for the presence of protective sensation. Decreases in deep tendon reflexes correlate with muscular atrophy, usually the intrinsic muscles of the foot are affected in the diabetic patient. The tuning fork is used to assess peripheral nerve function and help identify and quantify the neuropathy. The tuning fork is struck against a solid surface to make it "hum" or vibrate. The handle end of the tuning fork is then held against a bony prominence in the foot, the patient verbalizes the vibration sensation and when it stops. The second step is to repeat the above procedure except place the tuning fork on a different bony prominence that is unaffected by neuropathy such as the patella or elbow or shoulder. The amount of time it takes for the vibration sensation to cease is compared to the amount of time it took for the sensation to cease in the suspected neuropathic area. The Semmes-Weinstein test helps determine the level of protective sensation the feet have. Using a monofilament exert enough pressure to the 10 testing points. (See figure 8-E) The amount of pressure is indicated when the

**Figure 8-E Semmes-Weinstein test**



monofilament bows. The patient, with his eyes closed, must inform the examiner when and where the monofilament is being placed. Assess both feet in all diabetics, even if no ulcers are present. As protective sensation decreases, plantar pressures tend to rise as does the patient's risk for ulcer formation.

Vascular tests such as pulse palpation, ABI, toe pressures, and transcutaneous oxygen levels (TCPO<sub>2</sub>) help assess the circulation in the lower extremities. Pulses should be palpated at the dorsalis pedis, posterior tibial, popliteal, and femoral pulses. If you are unable to locate the pulse due to edema, deformity or other hidden vascular process, assess with a Doppler for an audible signal before determining that the pulse is absent. A palpable dorsalis pedis pulse is roughly equivalent to 80 mm HG, which is usually

adequate for healing most diabetic ulcers. After the pulse is located by palpation, it is beneficial to document the amplitude of the pulse using the guidelines below.

**Table 8-F**

Rating	Pulse Characteristic
0	No palpable pulse
1+	Weak or thready pulse, difficult to feel or easily obliterated by slight finger pressure
2+	Normal pulse, easily palpated, obliterated by strong finger pressure
3+	Bounding pulse, readily palpated and forceful, not easily obliterated with finger pressure

Ankle Brachial Index (ABI), is used in conjunction with other vascular test to determine and monitor the patient's risk of ischemia in the area of the ankle. Poor perfusion increases the patient's likelihood of developing ulcers and reduces the speed with which existing ulcers heal. This is a twofold complication to diabetics. The ABI is a ratio of systolic blood pressure in the brachial artery of the arm to the systolic blood pressure of the dorsalis pedis artery in the ankle. Obtaining ABI's is further discussed in Chapter 6, vascular ulcers. Table 8-G contains the standard interpretation of the ABI.

**Table 8-G**

ABI Ratio	Interpretation of Risk
> 1.1 (diabetic patients)	Skewed reading due to calcified or non compressible ankle arterial vessels
0.9 – 1.1	Normal arterial perfusion
0.7 – 0.9	Adequate perfusion
0.5 – 0.7	Arterial occlusive disease, claudication
< 0.5	Severe arterial occlusive disease, resting ischemic pain
< 0.2	Risk of gangrene

One must be careful when interpreting the ABI test results of a diabetic patient. Localized edema and significant vascular disease along with autonomic changes can negatively skew the ABI results. Toes pressures are a better interpretation of vascular status in the diabetic.

Toe pressures are a more sensitive indicator of changes in the vascular integrity in the distal areas of the foot. They are obtained by utilizing a special cuff, similar to those used on premature infants. Due to the tiny arteries in the digits, the corresponding arterial pressures are lower than those measured in the arm or leg. Therefore, typical toe pressures are about 70% of the systolic values obtained in the arm. The toe pressure allows you to gauge the patient's risk for ischemia. Generally a pressure of 45mm Hg or higher is needed for proper wound healing. See Table 8-H.

**Table 8-H**

Toe Pressure	Ischemia Risk
> 55 mmHg	Low risk
< 40 mmHg	High risk
< 20 mmHg	Severe risk

Transcutaneous oxygen levels (TCPO<sub>2</sub>) reflect the oxygen saturation of the tissues. Measurements are taken near the ulcer site with a measurement from the left upper chest used as a baseline reading for comparison. There are many challenges that can be done during a TCPO<sub>2</sub> testing session that can lead the technician to various treatment options. A few of these are elevating the leg for 20 minutes, this challenges the arterial flow to the extremity to assess the circulation under stress. An oxygen challenge is used to determine the body's response to inhaled oxygen in relation to tissue perfusion. This can be used as an indicator as to the response a patient will have if administered hyperbaric oxygen therapy (100% oxygen at 2.5 ATA- increased barometric pressure). The results of the TCPO<sub>2</sub> test have also been used to determine the level of amputation in the ischemic limb, in order to compliment healing.

**Table 8-1**

<b>TCPO2 level</b>	<b>Interpretation (degree of hypoxia)</b>
> 40 mmHg	Normal tissue perfusion
20 – 40 mmHg	Moderate Hypoxia
< 20 mmHg	Severe hypoxia

## Classification

Diabetic foot ulcers are classified according to depth, presence of ischemia, and presence of infection depending on the classification system. The Wagner Ulcer Grade Classification System and the University of Texas Wound Classification System are two commonly used scales used to classify diabetic ulcers. The Wagner scale considers the depth of the ulcer but does not allow for assessment of infection at all tissue levels. A modified version, however, does account for levels of infection. The University of Texas scale does take infection and ischemia into account and provides a more detailed breakdown of classifications than the Wagner scale.

At ProCare Healing Centers, we will utilize the classification system recommended by wound expert, our data base for tracking healing outcomes. Classification systems are discussed in Chapter 3 Wound Assessment. See Table 3-0 for a comparison of the different grades, stages and wound classifications.

## Complications

The most common complications that impede the healing of diabetic ulcers are :

- Multiple comorbidities- including PVD, increase the risk of ulceration and slow down the healing process
- Uncontrolled hyperglycemia- commonly signals infection and inhibits the immune system, especially the scavenging function of the neutrophils
- Psychosocial problems- depression and poverty profoundly affect the patient's nutritional status, which in turn affects the body's ability to prevent ulcers and heal existing ulcers
- Infection- An infection in the wound or elsewhere consumes proteins needed for wound healing and interferes directly by damaging the wound bed. A 4-5 degree increase in temperature is a local sign of infection. The wound bed of an infected wound commonly has friable (easy to bleed), bright red granulation tissue.
- Osteomyelitis– Bone infection is common in deep wounds. A palpable bone that has been exposed greater than 30 days usually indicates osteomyelitis. However, osteomyelitis may be difficult to distinguish from acute Charcot neuropathic osteoarthropathy. The best way to differentiate between the two is to culture a bone fragment from the wound bed. MRI's, CT scans and x-rays usually will not differentiate between the two. The only way to rid the body of osteomyelitis is with 6-8 weeks of antibiotic treatment and bone resection. Hyperbaric oxygen therapy, along with antibiotics and surgery is considered the gold standard treatment for the treatment of osteomyelitis.

## Diabetic Foot Ulcer Care

Successful healing of the diabetic foot ulcers depends on a multitude of factors. Some of these are: proper cleansing of the wound, including debridement, appropriate dressings and offloading of pressure, topical antimicrobials, biotherapies, adjunctive hyperbaric oxygen therapy and corrective surgery.

Wound cleansing is a fundamental step in the healing process. Necrotic tissue can act as a reservoir for bacteria and inhibits wound healing. Using commercial wound cleansers can be toxic to the cells, therefore, flushing the wound with normal saline is the best method to clean the diabetic foot ulcer.

Wound dressings must maintain a moist wound environment while avoiding maceration. Wound dressings include transparent films, foams, hydrocolloids, hydrogels, alginates, collagen based dressings and composites (combination dressings). Diabetic foot ulcers tend to produce low to moderate exudates (drainage). Therefore, care should be used when choosing a dressing to balance the need for exudates absorbency vs. adequate hydration levels of the wound bed.

Off-loading- relieving the pressure- from the plantar tissues is imperative in the treatment of diabetic neuropathy as well in the prevention for those patients at risk for ulcer development. Off-loading seeks to control, limit or remove all intrinsic and extrinsic factors that affect plantar pressures. Examples of intrinsic factors are the presence of bony prominences. Extrinsic factors include trauma, ill-fitting shoes, or maintaining a position too long allowing the damaging effects of pressure to build up as seen in sensory neuropathy. Off-loading is especially important in the neuropathic patient as the preventative measures utilized to reduce the pressure reduces or limits the kind of tissue damage that causes ulcers to form.

Off-loading can be accomplished using nonsurgical or surgical interventions or a combination of both. Nonsurgical interventions include therapeutic foot wear, custom orthotics, or walking casts. When using any of these devices, keep in mind that the patient may be more prone to falling until they have adjusted to the off-loading device and should therefore be instructed on fall precautions.

## Shoes

For a patient with no loss of protective sensation, a comfortable, a well fitting pair of tennis shoes may be sufficient. On the other hand, a patient with loss of sensation, foot deformities or a history of recurrent ulcers may need a custom made shoe or orthotic (insert). Custom orthotics relieve pressure, reduce shearing, cushion the foot, and accommodate deformities. In either case, these characteristics should be kept in mind when choosing appropriate foot ware:

- Soft, breathable leather that conforms to foot deformities
- High tops for ankle stability
- Rocker soles and bottoms for pressure and pain relief across the plantar metatarsal heads
- A toe box with extra depth and width to accommodate deformities such as claw toes and hallux valgus (displacement of the great toe towards the other toes)
- Flared lateral soles for stability



**Figure 8-L Diabetic foot ulcers caused from secondary pressure of an ill fitting shoe. Notice the callus formation around the edges from continued walking on the ulcer.**

## Walking Casts

Walking casts range from total contact cast to splints to walking boots. A total contact cast (TCC) is the treatment of choice for the non-infected diabetic foot ulcer in need of off-loading. TCC are custom made by the physician or orthotist with padding fitted over the bony areas of the foot, ankle, and leg. A plaster shell reinforced with plaster splints are covered with fiberglass to make the cast rigid and durable. A walking heel is utilized for ambulation.

The cast is molded to fit snugly and prevent the foot from sliding inside the cast, thus reducing the shearing forces over the plantar surface. Careful attention should be paid to the patient's history of inflammation, edema and neuropathy as the patient may not be able to discern if the cast is too tight. It is recommended that the cast be reassessed in 24-48 hours and changed weekly. TCC is not recommended for use over infected ulcers as the wound cannot be easily accessed for assessment and or treatment. The occlusive nature of the cast also provides a good environment for bacteria and anaerobes to flourish.



**Figure 8-K DH walker. The foam hexagon shaped inserts can be removed for pressure reduction at the ulcer site.**

## Surgical Interventions

Resection (surgical removal) of the bony deformity and pathologic tissue reduces peak plantar pressure. Examples of this type of surgery are: exostectomy, digital arthroplasty, bone and joint resections, and partial calcaneotomies.

**Figure 8-J Total Contact**



Splints and walking boots have cushioned inserts with an outer shell of fiberglass or copolymer. There are several varieties available on the market. The Cam-walker has the capability of removing small 1x1 cm pieces of foam to assist in off-loading an ulcer or bony deformity. Splints and walkers have an advantage to the TCC in that they can be removed easily to inspect and/or provide treatment to the wound. On that same note, the non-compliant patient will be more likely to remove a splint than a TCC.

## Topical Antimicrobials

Routine wound cleansing handles much of the surface microbial population. However, applying a topical antimicrobial directly to the wound bed can help control the microorganisms and improve healing. Commonly used topical antimicrobials include:

- Bacitracin (Baciguent)
- Metronidazole (MetroGel)
- Mupirocin (Bactroban)
- Silver Sulfadiazine (Silvadene)
- Cadexomer Iodine (Iodosorb gel or Iodoflex pads) \*\*
- Silcryst Nanocrystals (Silver impregnated dressings- Acticoat, Argalase, etc..) \*\*

\*\* These new products contain iodine or silver with kills or inhibits microbes. The active ingredients are released slowly into the wound be at concentrations that are toxic to microbes but not to the important cells, such as the fibroblasts. As an added benefit, research has shown that the microbes are not as likely to develop a resistance to this new generation of products.

Keep in mind that some microorganisms are resistant to certain topical agents. Culture and sensitivity results will assist you in selecting the appropriate treatment. Recent studies have also found that products containing neomycin, such as Neosporin, can cause allergic reactions and are therefore no longer recommended for the treatment of ulcers.

## Biotherapies

Growth factors and living skin equivalents are two forms of biotherapy that may be included in the care of a patient with a diabetic foot ulcers. Simply stated, growth factors orchestrate healing in the wound bed. These are available naturally in the body or have been engineered to stimulate the body's own growth factors. Platelet derived growth factors (PDGF) play a central role by stimulating chemotaxis and the proliferation of neutrophils, fibroblasts and monocytes. The most common growth factors available on the market are Regranex 1% gel (Becaplermin) and Autogel. Regranex is a product of Smith & Nephew, whereas Autogel is a product marketed by Cytomedix in that the process incorporates taking the patient's blood and spinning it down to produce a by-product of platelets and growth factors. Each of these products have shown to decrease healing time, but they are very costly. Some insurance, including Medicare and Medicaid, will not pay for these treatments, therefore the reimbursement and financial abilities of the patient must be taken into consideration. With this in mind, also remember that the therapy relies on adequate vascular supply, no underlying infection, and proper wound bed preparation.

Living Skin Equivalents are products composed of living cells and matrix, or scaffolding that serves as the extracellular medium. These products act as interactive wound coverings, providing growth factors and other needed molecules. Dermagraft and Apligraf are examples of a living skin equivalent. They have viable fibroblasts that enhance wound healing rates in diabetic ulcers. As with the products derived from growth factors, these preparations are quite costly and the patient's insurance coverage should be investigated for coverage prior to application.

## Prevention

Teaching your patient how to eliminate or minimize the risk factors associated with diabetic foot ulcers is the best forms of prevention. The American College of Foot and Ankle Surgeons have compiled the following list of risk factors for diabetic ulcers.

- Structural foot deformity (claw toes, rocker bottom, hallux vagus)
- Trauma and improperly fitting shoes
- Prior history of ulceration and/or amputation
- Prolonged, elevated pressure on tissue
- Limited joint mobility
- Uncontrolled hyperglycemia
- Prolonged history of diabetes
- Blindness or impaired vision
- Chronic renal disease
- Advanced age

Research has indicated that tight glycemic control reduces the frequency and severity of peripheral neuropathy and vascular damage in individuals with diabetes. With proper skin and foot care, patients can decrease their risk of ulceration. Below is information that should be taught to all diabetic patients. Please see Table 8-M

**Table 8-M**

Assessment	Action
Foot Care	<ul style="list-style-type: none"> <li>• Assess daily for injury</li> <li>• Wash feet with mild soap and water</li> <li>• Dry thoroughly between toes.</li> <li>• Check bath water with elbow (insensate feet are easily burned)</li> <li>• Apply a moisturizing cream to prevent drying and cracking (Vaseline is inexpensive and effective)</li> <li>• Do not apply moisturizer between toes</li> <li>• Cut toe nails off squarely or have a podiatrist clip nails that are thickened and/or deformed</li> <li>• DO NOT go barefooted</li> </ul>
Socks	<ul style="list-style-type: none"> <li>• Use silver-Ion lined socks for fungus control</li> <li>• Wear socks that are seamless</li> <li>• Wear socks that are white or light colored so that bleeding from trauma is easily detected</li> <li>• Wear cotton blended socks to wick away moisture and allow air to circulate around the foot. Natural fibers breathe better than synthetic fibers.</li> <li>• Use diabetic padded socks for shear and friction control</li> </ul>
Shoes	<ul style="list-style-type: none"> <li>• Wear well-fitting shoes</li> <li>• Wear shoes that breathe to reduce maceration and fungal infections</li> <li>• Wear new shoes for 1 hour at a time initially until your feet adjust and the shoes are “broke in”</li> <li>• If deformities are present or you have a history of ulceration, wear professionally fitted shoes (orthotics)</li> <li>• Wash shoes, if possible, to destroy microorganisms</li> <li>• Check shoes before putting them on to make sure nothing has fallen into the shoe that could harm your foot</li> </ul>

Successful prevention programs for diabetic ulcers begin with health promotion. Your patient is at the center of the health care team. The patient should play an active role in setting personal health care goals. Most patients with diabetes have many health problems requiring involvement from a multitude of practitioners within the health care system. These include, but are not limited to, doctors, nurses, physical therapists, occupational therapists, nutritionists, podiatrists, endocrinologists, psychologists, diabetes educators, prosthetists or orthotists, and social workers to name a few. It is imperative that the patient is agreeable and compliant with the treatment plan to increase wound healing.