DIABETIC FOOT DISORDERS
A Clinical Practice Guideline

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DIABETIC FOOT DISORDERS
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For the American College of Foot and Ankle Surgeons and the American College of Foot and Ankle Orthopedics and Medicine
ABSTRACT

Foot ulcerations, infections, and Charcot neuropathic osteoarthropathy are three serious foot complications of diabetes mellitus that can too frequently lead to gangrene and lower limb amputation. Consequently, foot disorders are one of the leading causes of hospitalization for persons with diabetes and can account for expenditures in the billions of dollars annually in the U.S. alone. Although not all foot complications can be prevented, dramatic reductions in their frequency have been obtained through the implementation of a multidisciplinary team approach to patient management. Using this concept, the authors present a Clinical Practice Guideline for diabetic foot disorders based on currently available evidence. The underlying pathophysiology and treatment of diabetic foot ulcers, infections, and the diabetic Charcot foot are thoroughly reviewed. Although these guidelines cannot and should not dictate the standard of care for all affected patients, they are intended to provide evidence-based guidance for general patterns of practice. The goal of a major reduction in diabetic limb amputations is certainly possible if these concepts are embraced and incorporated into patient management protocols.

INTRODUCTION

Foot disorders are a major source of morbidity and a leading cause of hospitalization for persons with diabetes mellitus. Ulceration, infection, gangrene, and amputation are significant complications of the disease. Costs of management are estimated at several billion dollars annually. Another serious complication of long-standing diabetes, neuropathic osteoarthropathy (Charcot foot), can lead to the development of other limb-threatening disorders. Although the underlying pathophysiology of diabetic foot complications has been elucidated to a great extent, much research is yet needed to determine which of our treatments are most effective. Furthermore, we must determine how to more effectively prevent those ulcerations which are now known to be leading precursors to lower extremity amputation in patients with diabetes.

Although not all diabetic foot disorders can be prevented, it is possible to effect dramatic reductions in their incidence and morbidity through appropriate evidence-based prevention and management protocols. Utilizing a multidisciplinary approach, consistent improvement has been noted in rates of limb salvage from centers around the world. With this premise as our central theme, the authors present this Clinical Practice Guideline to diabetic foot disorders based on currently available evidence. Three major pedal complications of diabetes are thoroughly reviewed: diabetic foot ulcers, diabetic foot infections, and the diabetic Charcot foot. These guidelines are intended to provide evidence-based guidance for general patterns of practice and not to necessarily dictate the care of a particular patient. Although our intent is to be as comprehensive as possible, we realize that this work is, in fact, a

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work in progress and will require future modification as new knowledge becomes available.

Entities

Diabetic foot complications are considered under ICD-9-CM classifications:

250.0x Diabetes without Complications
250.4x Diabetes with Renal Complications

Note: You are also required to use one of these additional codes to identify the manifestation, as:

Diabetic:
- nephropathy NOS (583.81)
- nephrosis (581.81)
- intercapillary glomerulosclerosis (581.81)
- Kimmelstiel-Wilson syndrome (581.81)

250.5x Diabetic Retinopathy

Note: You are also required to use one of these additional codes to identify the manifestation, as:

Diabetic:
- background diabetic retinopathy (362.01)
- proliferative diabetic retinopathy (362.02)

250.6x Diabetes with Neurologic Manifestations

Note: You are also required to use one of these additional codes to identify the manifestation, as:

Diabetic:
- amyotrophy (358.1)
- mononeuropathy (354.0-355.9)
- neurogenic arthropathy (713.5)
- peripheral autonomic neuropathy (337.1)
- polyneuropathy (357.2)

250.7x Diabetes with Peripheral Vascular Disease

Note: You are also required to use one of these additional codes to identify the manifestation, as:

Diabetic:
- gangrene (785.4)
- peripheral angiopathy (443.81)

250.8x Diabetes with Other Special Manifestations

Note: You are also required to use one of these additional codes to identify the manifestation, as:

Diabetic:
- diabetic bone changes (731.8)

All codes in category 250 have to be reported as five-digit subclassification codes, as follows:

0 — Type 2: non-insulin-dependent (NIDDM) or (adult-onset) or unspecified type, not stated as uncontrolled. Fifth-digit 0 is for use with type 2, adult-onset diabetic patients, even if the patient requires insulin.
1 — Type 1: insulin-dependent (IDDM) or juvenile-onset, not stated as controlled.
2 — Type 2: non-insulin-dependent (NIDDM) or adult-onset or unspecified type, uncontrolled. Fifth-digit 2 is for use with type 2, adult-onset diabetic patients, even if the patient requires insulin.

3 — Type 1: insulin-dependent (IDDM) or juvenile-onset, uncontrolled.

When applicable, the following diagnosis codes also should be reported:

<table>
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<tr>
<th>Code</th>
<th>Diagnosis</th>
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<tbody>
<tr>
<td>040.0</td>
<td>Gas gangrene</td>
</tr>
<tr>
<td>357.2</td>
<td>Polyneuropathy in Diabetes</td>
</tr>
<tr>
<td></td>
<td>(Report code 357.2 as secondary to 250.6x)</td>
</tr>
<tr>
<td>440.23</td>
<td>Atherosclerosis of the Extremities with Ulceration</td>
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<td>(Report code 440.23 as a secondary code to 250.7x)</td>
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<td>681.11</td>
<td>Cellulitis/Abscess, Toe</td>
</tr>
<tr>
<td>682.6</td>
<td>Cellulitis/Abscess, Leg (including ankle)</td>
</tr>
<tr>
<td>682.7</td>
<td>Cellulitis/Abscess, Foot (except toes)</td>
</tr>
<tr>
<td>707.1</td>
<td>Ulcer of lower limbs, except Decubitus</td>
</tr>
<tr>
<td></td>
<td>(Both codes 707.1 and 785.4 should be used to report an ulcer with gangrene)</td>
</tr>
<tr>
<td>713.5</td>
<td>Arthropathy Associated with Neurological Disorders (Charcot’s)</td>
</tr>
<tr>
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<td>(Report code 713.5 as a secondary code to 250.6x)</td>
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<tr>
<td>730.06</td>
<td>Osteomyelitis/Acute/Lower Leg</td>
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<tr>
<td>730.07</td>
<td>Osteomyelitis/Acute/Ankle and Foot</td>
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<tr>
<td>730.16</td>
<td>Osteomyelitis/Chronic/Lower Leg</td>
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<td>730.17</td>
<td>Osteomyelitis/Chronic/Ankle and Foot</td>
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<tr>
<td></td>
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</tr>
<tr>
<td>785.4</td>
<td>Gangrene (any site)</td>
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<td>V49.71</td>
<td>Status Post Amputation, Great Toe</td>
</tr>
<tr>
<td>V49.72</td>
<td>Status Post Amputation, Other Toe(s)</td>
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<tr>
<td>V49.73</td>
<td>Status Post Amputation, Foot</td>
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<tr>
<td>V49.74</td>
<td>Status Post Amputation, Ankle</td>
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<tr>
<td>V49.75</td>
<td>Status Post Amputation, Below Knee</td>
</tr>
<tr>
<td>V49.76</td>
<td>Status Post Amputation, Above Knee</td>
</tr>
</tbody>
</table>

**Definitions**

**Amputation:** The complete or partial removal of a limb or body appendage by surgical or traumatic means.

**Charcot Foot:** (arthropathy, osteoarthropathy, neuroarthropathy): Noninfectious destruction of bone and joint associated with neuropathy.

**Diabetic Foot:** The foot of a diabetic patient that has the potential risk of pathologic consequences, including infection, ulceration, and/or destruction of deep tissues associated with neuropologic abnormalities, various degrees of peripheral vascular disease, and/or metabolic complications of diabetes in the lower limb. (Based upon the World Health Organization [WHO] definition.)

**Diabetes Type 1:** Formerly called insulin-dependent diabetes mellitus (IDDM), describes an autoimmune disease of younger patients with a lack of insulin production causing hyperglycemia and a tendency toward ketosis.

**Diabetes Type 2:** A metabolic disorder resulting from the body’s inability to produce enough or properly utilize insulin. Formerly called non-insulin-dependent diabetes mellitus (NIDDM), these patients also have hyperglycemia but are ketosis resistant.
Epidemiology: The study of occurrence and distribution of disease.
Gangrene: The death or necrosis of a part of the body secondary to injury, infection, and/or lack of blood supply. This indicates irreversible damage where healing cannot be anticipated without loss of some part of the extremity.
Incidence: The rate at which new cases of disease occur within a specified time period.
Infection: Invasion and multiplication within body tissues by organisms such as bacteria, fungi, or yeast, with or without the clinical manifestation of disease.
Intrinsic Minus Foot: A neuropathic foot with intrinsic muscle wasting and associated clawtoe deformities.
Ischemia: The impairment of blood flow secondary to an obstruction or constriction of arterial inflow.
LEAP: Lower Extremity Amputation Prevention program.
Limited Joint Mobility: The stiffness or restricted range of motion of a joint (chiaroarthropathy) due to protein glycosylation.
LOPS: Loss of Protective Sensation describes the progression of neuropathy in the diabetic foot to the point that the foot is at risk for ulceration.
Neuropathy: Nerve dysfunction affecting sensory, motor, and/or autonomic fibers with varying degrees of impairment, symptoms, and signs. Diabetic peripheral neuropathy is the presence of symptoms and/or signs of peripheral nerve dysfunction in people with diabetes after exclusion of other causes.
Prevalence: A measure of frequency describing the percent of persons in a given population with a stated disease or characteristic at a point in time.
Ulceration (Ulcer): A partial- or full-thickness defect in the skin that may extend to subcuticular tissue, tendon, muscle, bone, or joint.

Goals and Objectives of Diagnosis and Treatment

The objectives of diagnosis and treatment of diabetic foot sequelae center around maintaining the patient as an ambulatory, productive member of society or returning the patient to that state as quickly and safely as possible. This may at any time require the expertise of a number of different generalists and specialists on the diabetic foot care team.

- Primary Goals
  - Prevent limb loss
  - Maintain quality of life

- Objectives
  - Appropriate screening and examination
  - Patient and provider education
  - Prevention of ulceration and recurrence
  - Early recognition and treatment of diabetic foot complications

Provider

The podiatric physician, by virtue of his or her training, is uniquely suited to serve as a primary member of a multidisciplinary team for the management of diabetic foot disorders. The evaluation, diagnosis, and the conservative treatment of these disorders are skills attained at the professional degree level.

The podiatrist as part of a multidisciplinary team should be able to recognize impending diabetic foot complications, the need for advanced diagnostic studies, and the need for appropriate referral as indicated. Surgical management of these conditions should be undertaken only by those individuals who, by virtue of
specialized training and/or experience in foot and ankle surgery, are able to manage the perioperative and intraoperative treatment.

BACKGROUND

Epidemiology of Diabetic Foot

The incidence of diabetes in the United States is estimated at 798,000 new cases annually with an overall prevalence of approximately 6% of the population (1–3). An estimated 10.3 million persons are currently diagnosed with the disease, while an additional 5.4 million people who have diabetes remain undiagnosed. This represents a sixfold increase in the number of persons with diabetes over the past four decades (3). Furthermore, there is a higher incidence of diabetes among non-Hispanic blacks, Hispanic/Latino Americans, and Native Americans as compared to non-Hispanic whites (4). Diagnosed diabetes is most prevalent in the middle-aged and elderly populations, with rates estimated at 11% for those persons aged 65 years and older (5,6). The seventh leading cause of death (sixth-leading cause by disease) in the United States, diabetes contributes to more than 193,000 deaths per year (1,3).

Several types of diabetes are recognized: Type 1, formerly insulin-dependent diabetes mellitus (IDDM), is an autoimmune disease affecting the pancreas in which the individuals are ketosis prone and are unable to produce endogenous insulin. Type 2, formerly non-insulin-dependent diabetes mellitus (NIDDM), accounts for 90–95% of cases diagnosed. Persons with Type 2 diabetes mellitus are characterized by hyperglycemia in the presence of hyperinsulinemia due to peripheral insulin resistance. Gestational and other types such as drug or surgically induced are also recognized (5). Numerous complications of diabetes can be related to microvascular, macrovascular, and metabolic etiologies. These include cerebrovascular, cardiovascular, and peripheral vascular disease, retinopathy, neuropathy, and nephropathy. Currently, cardiovascular complications are the most common cause of premature death among patients with diabetes (1,4,6,7).

One of the most common complications of diabetes in the lower extremity is the diabetic foot ulcer. It is estimated that 15% of patients with diabetes will develop a lower extremity ulcer during the course of their disease (8–10). Several reports of population-based studies indicate an annual cumulative incidence for diabetic foot ulcers of 2–3% (11,12). In one British study of a large cohort of neuropathic patients, there was a 7% one year incidence of first foot ulcer (13). Reported foot ulcer prevalence in a variety of populations has ranged between 2% and 10% (9,11,13,14). The cumulative effects of neuropathy, deformity, high plantar pressure, poor glucose control, duration of diabetes, and gender are all contributory factors for foot ulceration that are fully discussed in the next section (15–17). While most ulcers can be successfully treated in the office or outpatient setting, infected and/or ischemic foot ulcers are a major cause for diabetes-related hospitalization (18,19). National hospital discharge data indicate that the average hospital length of stay in diabetic patients with ulcer diagnoses was 59% longer than in those diabetes discharges without them (9). While 14–20% of patients with foot ulcers will subsequently require an amputation, foot ulceration is the precursor to approximately 85% of lower extremity of amputations in persons with diabetes (20–22).

Diabetes continues to be the most common underlying cause of lower extremity amputation (LEA) in the United States and Europe. More than 50% of all nontraumatic LEAs in the United States occur in people with diabetes, averaging 56,000 per year (1,3,4,7,9,23). In 1994 there were 67,000 diabetes-related LEA discharges, accounting for 984,000 days of hospital stay with an average length of stay (LOS)
The age-adjusted rate of amputation for that year was 82 per 10,000 persons with diabetes. Generally, the rate of LEA in the diabetic population is 15–40 times higher than that found in nondiabetic individuals, with males having rates at least 50% greater than diabetic women. (1, 6, 23).

There are several striking differences in the frequency of diabetes-related amputations between ethnic groups in the United States and abroad. Mexican (Hispanic) Americans, Native Americans, and African Americans each have at least a 1.5- to 2-fold increased risk for diabetes-related amputation compared to age-matched diabetic Caucasians (4, 7, 9, 10, 24, 25). When risks for LEA are compared between diabetic and nondiabetic populations worldwide, it becomes clearly apparent that both diabetes and ethnicity have profound implications on rates of lower limb amputation (10).

Survival rates after diabetes-related lower extremity amputation are significantly lower than those in age-matched nondiabetic individuals as well as in persons with diabetes without history of amputation (9, 10, 21). The 3-year and 5-year survival rates are about 50% and 40%, respectively, with the major cause of death being cardiovascular disease (7). One study reported a 5-year mortality rate of 68% after lower limb amputation, with lower survival rates in those patients with higher levels of amputation (21). Following one lower extremity amputation, there is a 50% incidence of serious contralateral foot lesion and a 50% incidence of contralateral amputation within 2–5 years (9, 21).

The total costs for both direct and indirect health care for the persons with diabetes in 1997 has been estimated at $98 billion. Of this total, direct medical costs including hospitalization, medical care, and supplies, accounts for $44.1 billion (26). Costs for ulcer care in the United States have been estimated in the range of $4.595 per ulcer episode to nearly $28,000 for the 2 years after diagnosis (12, 27). One report estimates 800,000 prevalent ulcer cases in the United States with costs averaging $5,457 per year per patient or total national annual costs of $5 billion (28). Over the past few decades the length of hospitalizations for lower extremity amputations in the United States has decreased, but the overall direct costs have remained high (4, 9). Direct and indirect costs of LEA vary greatly by year, payer, level of amputation, length of stay, or attendant comorbidities and can range from $20,000 to $40,000 (9). If the lower figure is applied to the 67,000 amputations performed in 1994, the total direct and indirect costs of LEA might be estimated at greater than $1 billion annually. In addition to the costs for ulcer care that preceded these amputations, the estimated overall total costs in the United States for diabetic foot disease can approach or exceed $6 billion annually.

**Etiology/Risk Factors**

**Risk for Ulceration**

Foot ulceration is the most common single precursor to lower extremity amputations among persons with diabetes (20–22). Treatment of infected foot wounds accounts for up to one-quarter of all diabetic admissions in the United States and Britain. This staggering figure makes it the single most common reason for diabetes-related hospital admission in these nations (9, 18, 19, 29, 30). The multifactorial nature of diabetic foot ulceration has been elucidated by numerous observational studies (9, 13, 15, 17, 31–35). Risk factors identified include peripheral neuropathy, vascular disease, limited joint mobility, foot deformities, abnormal foot pressures, minor trauma, a history of ulceration or amputation, and impaired visual acuity. These and other putative causative factors are listed in Table 1.
Peripheral sensory neuropathy in the absence of perceived trauma is the primary factor leading to diabetic foot ulcerations (13,15–17). Approximately 45–60% of all diabetic ulcerations are purely neuropathic, while up to 45% have neuropathic and ischemic components (15,36). A recent prospective multicenter study of diabetic patients revealed that sensory neuropathy was the most frequent component cause in the causal sequence to ulceration (15).

Other forms of neuropathy may also play a role in foot ulcerations. Motor neuropathy resulting in anterior tibial muscle atrophy or intrinsic muscle wasting can lead to foot deformities such as foot drop, equinus, hammertoes, and prominent plantar metatarsal heads (16,29,30,37). Autonomic neuropathies may commonly result in dry skin with cracking and fissuring, thus creating a portal of entry for bacteria (29,38). Autonomic neuropathy with attendant sympathetic failure, arteriovenous shunting, and microvascular thermoregulatory dysfunction impairs normal tissue perfusion and microvascular responses to injury. These alterations can subsequently be implicated in the pathogenesis of ulceration (39–41).

Foot deformities resulting from neuropathy, abnormal biomechanics, congenital disorders, or prior surgical intervention may result in high focal foot pressures (15,16,34,42–44). This may lead to vulnerable areas on the foot predisposing to ulcerations. These areas are primarily located plantarly, although medial and dorsal ulcerations may occur from footwear irritation. Such deformities might include prior partial foot amputations, prominent metatarsal heads, hammertoes, Charcot arthropathy, or hallux valgus.

Trauma to the foot in the presence of peripheral sensory neuropathy is an important component cause of ulcerations (15). While trauma may include puncture wounds and blunt injury, a common injury leading to ulceration is moderate repetitive stress resulting from walking or day to day activity (45). This is often manifested by callus formation under the metatarsal heads (15,34,46,47). Shoe-related trauma has been identified as a frequent precursor to foot ulceration (15,20,36,48).

Peripheral vascular disease rarely leads to foot ulcerations directly. However, once an ulceration develops, arterial insufficiency will result in prolonged healing and imparts an elevated risk for amputation (20,49). Attempts to resolve any infection will be impaired due to lack of oxygenation and difficulty in delivering antibiotics to the site of infection. Early recognition and aggressive treatment of lower extremity ischemia is therefore vital to lower limb salvage (22,34,37,50–52).

Limited joint mobility has recently been described as a potential risk factor for ulcerations (34,53–56). Glycosylation of collagen as a result of long-standing diabetes may lead to stiffening of capsular structures and ligaments (cheiroarthropathy). The subsequent reduction in ankle, subtalar, and first metatarsophalangeal
(MTP) joint mobility has been shown to result in high focal plantar pressures with increased risk of ulceration (54).

Other factors often associated with heightened risk for ulceration include: nephropathy, poor diabetes control, blindness, advanced age, and poor nutrition (16,29,34,38,53).

**Mechanisms of Injury**

The multifactorial etiology of diabetic foot ulcers is evidenced by the numerous pathophysiological pathways which can potentially lead to this disorder (15,30,37,38). Notwithstanding, there are two common mechanisms by which foot deformity and neuropathy may bring about skin breakdown in persons with diabetes: injuries due to continuous low pressure, typically from ill-fitting shoes, and injuries due to chronic repetitive trauma from walking (45).

The first mechanism of injury refers to prolonged low pressure over a bony prominence (i.e., bunion or hammertoe deformity). This generally causes wounds over the medial, lateral, and dorsal aspects of the forefoot and is associated with tight or ill-fitting shoes. Studies have shown that shoe trauma, in concert with loss of protective sensation and concomitant foot deformity, are major precipitating events leading to foot ulceration in persons with diabetes (15,20,48).

Regions of high pedal pressure are directly associated with foot deformity (34,37,43,50,57). When an abnormal focus of pressure is coupled with lack of protective sensation, the result can be the development of a callus, blister, and ulcer. The other common mechanism of ulceration involves prolonged repetitive moderate stress (45). This normally occurs on the sole of the foot and is related to prominent metatarsal heads, atrophied or anteriorly displaced fat pads, structural deformity of the lower extremity, and prolonged walking. Rigid deformities such as hallux valgus, hallux rigidus, hammertoes, and limited range of motion of the ankle, subtalar, and metatarsophalangeal joints have been associated with the development of diabetic foot ulcers (55,56). Other biomechanical perturbations, including partial foot amputations, will have the same adverse effects (42,43,58). Sensory neuropathy is the predisposing factor which allows progression to ulceration in each of these mechanisms of injury.

Figure 1 summarizes the various pathways and contributing factors leading to diabetic foot ulceration.

**Risk for Amputation**

(See Color Plate 1 on page 30.) The reported risk of lower extremity amputations (LEA) in diabetic patients ranges from 2% to 16% depending on study design and the population(s) under investigation (10–12,59–61). Rates of LEA in persons with diabetes can be 15–40 times higher than those found in persons without diabetes (7,9,10,23–25). The same risk factors which predispose to ulceration can also generally be considered as contributing causes for amputation, albeit with several modifications (Table 2).

Whereas peripheral vascular disease (PVD) may not always be an independent risk factor for ulceration when controlling for neuropathy, it can be a significant risk factor for amputation (9,15–17,20,29,59). PVD affecting the feet and legs is present in 8% of adult diabetic patients at diagnosis and in 45% after 20 years (62,63). The incidence in diabetic men and women is four to seven times greater than their nondiabetic counterparts. Since this impairment of arterial perfusion can be an isolated cause for amputation as well as a predisposing factor leading to gangrene, arterial insufficiency must be diagnosed early and managed by revascularization procedures to avoid limb loss (22,50).
Infection is a significant risk factor in the causal pathway to amputation, while it is not often implicated in the pathway leading to ulceration (9,15). Lack of wound healing, systemic sepsis, or unresolved infection can lead to extensive tissue necrosis and gangrene requiring amputation to prevent more proximal limb loss. This includes soft-tissue infection with severe tissue destruction, deep space abscess, or osteomyelitis. Adequate debridement may require amputation at some level as a means of removing all infected material (16,22,50).

Another frequently described risk factor for amputation is chronic hyperglycemia. Results of the Diabetes Control and Complications Trial (DCCT) and the United Kingdom Prospective Diabetes Study (UKPDS) have supported the long-held
theory that chronically poor control is associated with a host of systemic complications (64,65). The association between degree of glucose control and incidence or progression of numerous diabetic complications has consequently been well established by these and other studies (7,66,67). Such complications include peripheral neuropathy, microangiopathy, impaired leukocyte phagocytosis, and glycosylation of tissue proteins. Each has adverse effects on the diabetic foot, can contribute to the etiology of foot ulceration, delay normal wound healing, and subsequently lead to amputation (34,38,53). Several studies have reported significant associations between elevated glucose and lower extremity amputation (11,60,68–73). Amputation has also been associated with other diabetes-related co-morbidities such as nephropathy, retinopathy, and cardiovascular disease (6,34,53). Aggressive glucose control, management of associated comorbidities, and appropriate lower extremity care coordinated in a team environment may indeed lower overall risk for amputation (9,22,34,37,51,74,75).

The best predictor of amputation is a history of previous amputation. A past history of a lower extremity ulceration or amputation increases the risk for further ulceration, infection, and subsequent amputation (6,21,31,59). It may also be inferred that patients with a past history of ulceration possess all the requisite risk factors necessary to produce another ulceration, having demonstrated that they already have the component elements in the causal pathway (15,20,35). These data are substantiated by the fact that up to 34% of patients develop another ulcer within 1 year after healing an index wound, while the 5-year rate of developing a new ulcer is 70% (75,76). The rate of recurrence is always higher in those patients who have previously undergone amputation. Recurrence of pedal ulceration is due to abnormal distribution of plantar pressures, and changed osseous architecture following amputation. The cumulative identified risks of neuropathy, deformity, high plantar pressure, poor glucose control, and male gender are all additive factors for pedal ulceration in these diabetic patients (15,17,20,31,35,59,60). Re-amputation can be attributed to progression of the disease process, nonhealing wounds, and the development of additional risk factors for limb loss that develop as a result of the first amputation. Tragically, the 5-year survival rate after a diabetes-related lower extremity amputation has been reported as low as 28% (77).

**Risk for Charcot Joint Disease**

It is estimated that 1 in 680 diabetic patients (0.15%) will develop Charcot joint disease (78). The data concerning the true incidence of osteoarthropathy in diabetes are limited by the small number of prospective or population-based studies currently available. Much of the data we rely upon is based upon retrospective studies of small single-center cohorts. Nonetheless, the incidence of Charcot cases reported is very likely an underestimation since many cases go undetected, especially in the early stages (79–81).

The primary risk factors for this potentially limb-threatening deformity are the presence of dense peripheral sensory neuropathy, normal circulation, and a history of preceding trauma, often minor in nature (81–84). Trauma is not limited to injuries such as sprains or contusions. Foot deformities, prior amputations, joint infections, or surgical trauma may result in sufficient stress that can lead to Charcot joint disease (80).

**Risk for Infection**

Infections in patients with diabetes are not only common but are often more severe than those found in nondiabetic persons. It is well documented that diabetic
foot infections are polymicrobial in nature (22,50,85–88). Hyperglycemia, impaired immunological responses, neuropathy, and peripheral vascular disease are the main predisposing factors leading to limb-threatening diabetic foot infections. Uncontrolled diabetes results in impaired ability of host leukocytes to fight bacterial pathogens, while ischemia will also affect the ability to fight infections since delivery of antibiotics to the site of infection will be impaired. Consequently, infections can develop and spread rapidly and produce significant and irreversible tissue damage (50). Even in the presence of adequate arterial perfusion, underlying peripheral sensory neuropathy will often allow the progression of infection through continued walking or delay in recognition (8).

**PROCESS OF CARE**

The pedal manifestations of diabetes are well documented and potentially limb-threatening when left untreated. Recognition of potential problems and treatment of foot disorders in a diabetic patient requires the skill of a specialized practitioner to diagnose, manage, treat, and counsel the patient. The integration of knowledge and experience, afforded by a multidisciplinary team, promotes more effective treatment thereby improving outcomes and limiting the risk of lower extremity amputation (22).

**Diagnosis and Evaluation**

The evaluation of the diabetic foot involves careful assimilation of the patient's historical and physical findings and the results of necessary diagnostic procedures. Screening tools may be valuable in patient evaluation and determining levels of risk (see Appendix 1).

**History**

A thorough medical and foot history should be obtained from the patient. The following chart provides guidelines of specific diabetic foot issues that should be addressed.

<table>
<thead>
<tr>
<th>Global History</th>
<th>Foot-Specific History</th>
<th>Wound/Ulcer History</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diabetes disease duration</td>
<td>General</td>
<td>Location</td>
</tr>
<tr>
<td>Glycemic management/control</td>
<td>Daily activity</td>
<td>Duration</td>
</tr>
<tr>
<td>Cardiovascular, renal, and</td>
<td>Footwear</td>
<td>Inciting event or trauma</td>
</tr>
<tr>
<td>ophthalmic evaluations</td>
<td></td>
<td>Recurrences</td>
</tr>
<tr>
<td>Other comorbidities</td>
<td>Chemical exposures</td>
<td>Infections</td>
</tr>
<tr>
<td>Current treating physicians</td>
<td>Callus formation</td>
<td>Hospitalizations</td>
</tr>
<tr>
<td>Social habits —</td>
<td>Deformities</td>
<td>Wound care/off-loading methods</td>
</tr>
<tr>
<td>Alcohol/tobacco</td>
<td>Previous foot surgery</td>
<td>Patient's compliance/wound response</td>
</tr>
<tr>
<td>Current medications</td>
<td>Neuropathy symptoms</td>
<td>Interference with wound care/</td>
</tr>
<tr>
<td>Allergies</td>
<td>Ischemic symptoms</td>
<td>family or social problems for patient</td>
</tr>
<tr>
<td>Previous hospitalizations/</td>
<td></td>
<td>Previous foot trauma or surgery</td>
</tr>
<tr>
<td>surgeries</td>
<td></td>
<td>Edema-unilateral vs. bilateral</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Previous or active Charcot joint treatment</td>
</tr>
<tr>
<td></td>
<td></td>
<td>to date</td>
</tr>
</tbody>
</table>

12 A Clinical Practice Guideline
Physical Examination

Recognizing important risk factors and making a logical, treatment-oriented assessment of the diabetic foot requires a consistent and thorough diagnostic approach using a common language. Without such a method, the practitioner is more likely to overlook vital information and to pay inordinate attention to less critical points in the evaluation. A useful examination will involve identification of key risk factors and assignment into an appropriate foot risk category. Only then can an effective treatment plan be designed and implemented.

Clinical Examination

All patients with diabetes presenting to any health care practitioner require a pedal inspection and should receive a thorough foot examination at least once each year (89). Patients with diabetic foot-related complaints will require detailed evaluations more frequently. The examination should be performed systematically so that important aspects are not overlooked. First, one should grossly evaluate the patient and his or her extremities. Any obvious problem can then receive closer scrutiny with examination. For clarity, the key components of the foot examination are presented below in a bulleted format. Each bulleted item represents an important component of the pedal examination or a significant finding to be noted based on evidence which indicates likely predictors for ulceration. Although not specifically mentioned in this section, it is assumed that a general medical assessment will be determined including measurements of vital signs.

Vascular Examination

- Palpation of pulses
  (dorsalis pedis, posterior tibial, popliteal, femoral)
- Subpapillary venous plexus filling time (normal ≤3 seconds)
- Venous filling time (normal ≤20 seconds)
- Color changes:
  Cyanosis
  Dependent rubor
  Erythema
- Presence of edema
- Temperature gradient
- Dermal thermometry
- Integumentary changes consistent with ischemia:
  Skin atrophy
  Nail atrophy
  Abnormal wrinkling
  Diminished pedal hair

Neurologic Examination

- Vibration perception:
  Tuning fork 128 cps
  Measurement of vibration perception threshold (Biothesiometer)
- Light pressure: Semmes–Weinstein 10-gram monofilament
- Light touch: cotton wool
- Two-point discrimination
- Pain: pinprick
- Temperature perception: hot and cold
- Deep tendon reflexes: ankle, knee
- Clonus testing
- Babinski test
- Romberg’s test

**Musculoskeletal Examination**
- Biomechanical abnormalities:
  - Orthopedic deformities
  - Hammertoes
  - Bunions (or Tailor’s bunions)
  - Flat or high-arched feet
  - Charcot deformities
  - Iatrogenic deformities (e.g., amputations)
- Limited joint mobility
- Tendo-Achilles contractures/equinus
- Gait evaluation
- Muscle group strength testing:
  - Passive and active, non-weightbearing and weightbearing
  - Foot drop
- Atrophy — intrinsic muscle atrophy
- Plantar pressure assessment:
  - Computerized devices
  - Harris ink mat

**Dermatologic Examination**
- Skin appearance:
  - Color, texture, turgor, quality
  - Dry skin
- Calluses: Discoloration/subcallus hemorrhage
- Fissures (especially posterior heels)
- Nail appearance:
  - Onychomycosis, dystrophic
  - Atrophy
  - Hypertrophy
  - Paronychia
- Presence of hair
- Ulceration, gangrene, infection (Note location, size, depth, infection status, etc.)
- Interdigital lesions
- Tinea pedis
- Markers of diabetes:
  - Shin spots — diabetic dermopathy
  - Necrobiosis lipoidica diabeticorum
  - Bullous diabeticorum
  - Granuloma annulare

**Footwear Examination**
- Type of shoe
- Fit
- Shoewear, patterns of wear
- Lining wear
- Foreign bodies
- Insoles, orthoses
Communicating and Classifying Cumulative Risk

Following a detailed diabetic foot examination, the patient may be classified according to a cumulative risk category. This enables the physician to design a treatment plan which may possibly reduce lower extremity amputations and reduce the patient from a high-risk category to the lowest risk level possible for that patient. Several risk stratification schemes have been proposed, assigning different weights to important risk factors for ulceration including peripheral neuropathy, arterial insufficiency, deformity, high plantar pressures, and prior history of ulceration or amputation (34,35,37,38,90–92). Although no one system has been universally adopted which can predict ulceration, the following simplified risk stratification has been accepted by the International Working Group (37):

<table>
<thead>
<tr>
<th>Category</th>
<th>Risk Profile</th>
<th>Evaluation Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No neuropathy</td>
<td>Annual</td>
</tr>
<tr>
<td>1</td>
<td>Neuropathy</td>
<td>Semi-annual</td>
</tr>
<tr>
<td>2</td>
<td>Neuropathy, PVD, and/or deformity</td>
<td>Quarterly</td>
</tr>
<tr>
<td>3</td>
<td>Previous ulcer or amputation</td>
<td>Monthly to quarterly</td>
</tr>
</tbody>
</table>

Diagnostic Procedures

Diagnostic procedures may be indicated in the assessment and care of the diabetic foot. Consideration should be given to the following tests in concert with members of the consulting team. It should be noted that many of the following tests lack the ability to give a definitive diagnosis and clinical correlation is required.

Laboratory Testing

Clinical laboratory tests that may be necessary in the appropriate clinical situations may include: fasting or random blood glucose, glycohemoglobin (HbA1C), complete blood count (CBC) with or without differential, erythrocyte sedimentation rate (ESR), serum chemistries, wound and blood cultures, and urinalysis. Caution must be exercised in the interpretation of laboratory tests in these patients, since several reports have documented the absence of leukocytosis or fever in the presence of severe foot infections (87,88,93–97). Frequently, the most prognostic sign of infection severity is recalcitrant hyperglycemia despite normal antihyperglycemic regimens.

Imaging Studies

The diabetic foot may be predisposed to developing both common and unusual infectious or noninfectious processes. This is due in part to the complex nature of the disease and its associated vascular and neuropathic complications. As a result, imaging presentations will vary due to lack of specificity in complex clinical circumstances (88,96,98). This will create a challenge in the interpretation of the imaging studies. Studies should only be conducted to establish or confirm a suspected diagnosis and/or direct patient management.

Plain radiographs should be the initial imaging study in diabetic patients with signs and symptoms of a diabetic foot disorder (96,99). X-ray findings in a diabetic foot infection, such as osteomyelitis, may not demonstrate any osseous changes on radiographs for up to 14 days. Plain radiographs may be indicated in the detection
of osteomyelitis, osteolysis, fractures, dislocations seen in neuropathic arthropathy, medial arterial calcification, and soft-tissue gas (100).

Computed tomography (CT) scans may be indicated in the assessment of suspected bone and joint pathology not evident on plain radiographs (96,100). This study offers high anatomic detail and resolution of bone with osseous fragmentation and joint subluxation being well visualized (101).

Technetium bone scans are often used in diabetic foot infections although this modality lacks specificity, especially in the neuropathic patient (102). Three-phase bone scans may be indicated in the early detection of osseous pathology such as osteomyelitis, fractures, and Charcot arthropathy. However, such imaging tests are best utilized to confirm clinical suspicion and have higher specificity when combined with other scintigraphic techniques such as white blood cell scans (98,103,104).

Gallium 67 citrate is another nuclear medicine technique that is not used as frequently today due to more accurate alternative imaging studies. This study can be used in concert with technetium bone scans to aid in the diagnosis of osteomyelitis and also may be of value in the presence of acute osteoarthropathy (98,100,104).

Indium-111 leukocyte scans, Tc-99m-labeled white-cell scan (HMPO), or other variations of white blood cell scintigraphy are useful in differentiating between osteomyelitis and neuropathic arthropathy due to their relatively high sensitivity and specificity. These tests are expensive and time consuming, but are available at most hospitals when early identification of bone infection is required (96,100,103,105–108).

Magnetic resonance imaging (MRI) is often used in evaluating soft-tissue and bone pathologies. This scan may be indicated to aid in the diagnosis of osteomyelitis, deep abscess, septic joint, and tendon rupture. It is a readily available modality which has a very high sensitivity for bone infection and can also be used for surgical planning (96,100,109). Despite its high cost, MRI has gained wide acceptance in the management of patients with diabetic foot infection.

**Vascular Procedures**

When the history and physical examination suggest ischemia or the presence of a nonhealing ulcer with absent pedal pulses, further noninvasive testing is warranted. Noninvasive arterial studies (NIAS) should be performed to determine lower extremity perfusion. Such studies may include Doppler segmental arterial pressures, and waveform analysis, ankle-brachial indices (ABI), toe pressures, and transcutaneous oxygen tension (TO2) (34,110,111). Ankle-brachial indices may be misleading since ankle pressures can be falsely elevated due to medial arterial calcinosis and noncompressibility of affected arteries (112,113). A growing body of evidence suggests that toe blood pressures may have a role in predicting those diabetic patients at risk for foot ulceration as well as in the prediction of successful wound healing (34,114–116). Transcutaneous oxygen tension measurements have received similar support in the literature (33,49,117). Although not consistently predictive of wound healing outcomes, these physiologic measures of tissue oxygenation are highly predictive of wound healing failure at levels below 25 mm Hg (49,117,118). Both of these tests can be performed distally on the foot, regardless of arterial calcification in the major pedal arteries, and are favorable at pressures in the range of 40 mm Hg (37,115).

Laser Doppler velocimetry and measurement of skin perfusion pressure (SPP) with this modality has been used primarily in research settings, but can accurately assess blood flow velocity in the superficial arterioles and capillaries of the skin (118–121). Several recent reports indicate that laser Doppler measurement of SPP can be highly predictive of critical limb ischemia and wound healing failure at levels less than 30 mm Hg (120,121).
Vascular consultation should be considered in the presence of abnormal noninvasive arterial studies and a nonhealing ulceration (22,50). Arteriography with clearly visualized distal runoff allows appropriate assessment for potential revascularization (122,123). Digital subtraction angiography (DSA) or magnetic resonance angiography (MRA) are alternatives for evaluation of distal arterial perfusion (122,124).

**Neurologic Procedures**

Peripheral sensory neuropathy is the major independent risk factor for diabetic foot ulcerations (15,17,31,33,53). The patient history and physical examination utilizing the 5.07 Semmes-Weinstein monofilament (10 g) wire is sufficient to identify those individuals at risk for ulceration (17,125–127). Vibration perception threshold assessment with the Biothesiometer is also useful in predicting those patients at high risk for ulceration (32,35). More sophisticated studies, such as nerve conduction studies, are rarely necessary to diagnose peripheral sensory neuropathy. Patients with neuropathic ulcerations will usually have such profound sensory neuropathy that these studies add little to the management of these patients.

**Plantar Foot Pressure Assessment**

High plantar foot pressures have been identified as a significant risk factor for ulcerations (15,17,32,35,127). Measurement of these foot pressures is possible utilizing a variety of modalities. Several computerized systems can provide quantitative measurement of plantar foot pressure (128–131). These measurements may be important in identifying areas of the foot at risk for ulceration and possibly in the evaluation of orthotic adjustments. Their primary usage, however, has been in the area of diabetic foot research. The Harris mat, while not as sophisticated, can provide a qualitative measurement of plantar foot pressures and can identify potentially vulnerable areas for ulceration (132).

**ASSESSMENT AND TREATMENT OF PATHOLOGIC ENTITIES (FOOT ULCER, INFECTION, AND CHARCOT)**

Effective management of diabetic foot disorders requires knowledge of the potential pathologies, the associated classification systems and the principal tenets of intervention. Ulceration, infection, and Charcot arthropathy, are the most significant of these pathologies and classification systems have been developed for each entity. While the conditions may be seen either as an isolated event or coexisting in the same extremity, each entity is discussed independently.

**Diabetic Foot Ulcers: Assessment**

(See Color Plate 2 on page 30.)

**Extremity Assessment**

The lower extremity must be assessed for vascular and neuropathic risk factors. The acceptable evaluation parameters are listed in Table 3. Although positive findings in the neurologic examination rarely require further evaluation, positive findings of vascular insufficiency may require further consultation. The indications for vascular consultation include an ankle brachial index of less than 0.7, toe blood pressures <40 mm Hg or TcPO2 levels of less than 30 mm Hg, since these measures of arterial perfusion are associated with impaired wound healing (33,37,49,115,117).
TABLE 3  Vascular and neurologic examination of the lower extremity

<table>
<thead>
<tr>
<th>Evaluation Parameters</th>
<th>Normal Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vascular</td>
<td></td>
</tr>
<tr>
<td>Palpation of pulses</td>
<td>Present</td>
</tr>
<tr>
<td>Dependent rubor</td>
<td>Absent</td>
</tr>
<tr>
<td>Venous filling time</td>
<td>&lt;20 s</td>
</tr>
<tr>
<td>Capillary refill</td>
<td>&lt;3 s</td>
</tr>
<tr>
<td>Arterial Doppler exam for ankle-brachial index (ABI)</td>
<td>1.1</td>
</tr>
<tr>
<td>Toe pressures</td>
<td>&gt;40 mm Hg</td>
</tr>
<tr>
<td>Transcutaneous oxygen tension (TcPO2)</td>
<td>&gt;40 mm Hg</td>
</tr>
<tr>
<td>Neurologic</td>
<td></td>
</tr>
<tr>
<td>Semmes-Weinstein 5.07 monofilament (10 g)</td>
<td>Detected</td>
</tr>
<tr>
<td>Biothesiometer (vibration perception threshold)</td>
<td>&lt;25 V</td>
</tr>
<tr>
<td>Vibration perception — 128 cps tuning fork</td>
<td>Detected</td>
</tr>
<tr>
<td>Deep tendon reflexes</td>
<td>Present</td>
</tr>
</tbody>
</table>

Ulcer Evaluation

Description of the ulcer characteristics on presentation is critical for the mapping of its progress during treatment (22,30). While some characteristics are more important than others, they all have a prognostic value during management. The presumed etiology of the ulcer needs to be determined (i.e., chemical vs. mechanical) as well as ascertaining whether the lesion is neuropathic, ischemic, or neuroischemic in character (37). The evaluation should include the size and depth of the ulcer, as well as a description of the margins, base, and geographic location on the extremity or foot. All but the most superficial ulcers should be examined with a blunt, sterile probe. The description should note whether or not the sterile probe detects sinus tract formation, undermining of the ulcer margins, or extension of the ulcer into tendon sheaths, bone, or joints. A positive probe to bone finding has a high predictive value for osteomyelitis (133). The existence of odor or exudate and the character of each should be noted. Cultures may be necessary when signs of inflammation are present. Current recommendations for culture and sensitivity include thorough surgical preparation of the wound site with curettage of the wound base for specimen or with aspiration of abscess material (22).

Classification of Ulcers

Appropriate classification of the foot wound is predicated upon its thorough assessment, should facilitate its treatment, and be generally predictive of expected outcomes (22). Several systems of ulcer classification are currently in use in this nation and abroad in an attempt to meaningfully describe these lesions and to communicate severity (16,22,37,38,134–137). Perhaps the easiest system is to simply classify the lesions as neuropathic, ischemic, or neuroischemic with descriptors of wound size, depth, and infection (37). Regardless of which system is ultimately used, the clinician must be able to easily categorize the wound and, once classified, the ensuing treatment should be directed by the underlying severity of pathology.

Although no single system has been universally adopted, the classification system most often used was described and popularized by Wagner (137). Since this system fails to consider the important roles of infection, ischemia and other comorbid factors, subsequent authors have modified the classification systems by including descriptors for these considerations (135,136).
In the Wagner classification system, foot lesions are divided into six grades based on the depth of the wound and the extent of tissue necrosis (137):

- **Grade 0** — Ulcer. No open lesions, skin intact; may have deformities, erythematous areas of pressure or hyperkeratoses.
- **Grade 1** — Superficial ulcer. Disruption of skin without penetration of the subcutaneous fat layer. Superficial infection with or without cellulitis may be present.
- **Grade 2** — Full-thickness ulcer. Penetrates through fat to tendon, or joint capsule without deep abscess or osteomyelitis.
- **Grade 3** — Deep ulcer which may or may not probe to bone, with abscess, osteomyelitis, or joint sepsis. Includes deep plantar space infections or abscesses, necrotizing fasciitis, and tendon sheath infections.
- **Grade 4** — Denotes gangrene of a geographical portion of the foot such as toes, forefoot or heel. The remainder of the foot is salvageable though it may be infected. (See Color Plate 3 on page 30.)
- **Grade 5** — Gangrene or necrosis to the extent that the foot is beyond salvage and will require a major limb- or life-sparing amputation.

Failure of the Wagner classification to specifically address infection and ischemia within each grade (30) has been recognized, and hybrid schemes have been developed to account for these important attributes of foot ulcers (16,134). A simplified system which only attaches modifiers for ischemia (A) and infection (B) to the well-known Wagner system is presented (Table 4), recognizing that grades 3 through 5 usually have some degree of infection inherent within these lesions.

Another hybrid method for classifying diabetic foot lesions has been popularized by the University of Texas and has been retrospectively validated within that center (135,136). This scheme employs four grades of depth with four associated stages based on ischemia, infection, or both (Table 5). This system is also generally

<table>
<thead>
<tr>
<th>Grade</th>
<th>Lesion</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No open lesions: may have deformity or cellulitis</td>
</tr>
<tr>
<td>A</td>
<td>Ischemic</td>
</tr>
<tr>
<td>B</td>
<td>Infected</td>
</tr>
<tr>
<td>1</td>
<td>Superficial ulcer</td>
</tr>
<tr>
<td>A</td>
<td>Ischemic</td>
</tr>
<tr>
<td>B</td>
<td>Infected</td>
</tr>
<tr>
<td>2</td>
<td>Deep ulcer to tendon, or joint capsule</td>
</tr>
<tr>
<td>A</td>
<td>Ischemic</td>
</tr>
<tr>
<td>B</td>
<td>Infected</td>
</tr>
<tr>
<td>3</td>
<td>Deep ulcer with abscess, osteomyelitis, or joint sepsis</td>
</tr>
<tr>
<td>A</td>
<td>Ischemic</td>
</tr>
<tr>
<td>B</td>
<td>Infected</td>
</tr>
<tr>
<td>4</td>
<td>Localized gangrene — forefoot or heel</td>
</tr>
<tr>
<td>A</td>
<td>Ischemic</td>
</tr>
<tr>
<td>B</td>
<td>Infected</td>
</tr>
<tr>
<td>5</td>
<td>Gangrene of entire foot</td>
</tr>
<tr>
<td>A</td>
<td>Ischemic</td>
</tr>
<tr>
<td>B</td>
<td>Infected</td>
</tr>
</tbody>
</table>
TABLE 5  University of Texas Wound Classification System

<table>
<thead>
<tr>
<th>Stage</th>
<th>Grade</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0</td>
</tr>
<tr>
<td>A</td>
<td>Pre- or postul-</td>
</tr>
<tr>
<td></td>
<td>cerative lesion</td>
</tr>
<tr>
<td></td>
<td>completely epithelialized</td>
</tr>
<tr>
<td>B</td>
<td>Infected</td>
</tr>
<tr>
<td>C</td>
<td>Ischemic</td>
</tr>
<tr>
<td>D</td>
<td>Infected and ischemic</td>
</tr>
</tbody>
</table>

predictive of outcome since increasing grade and stage of wounds are less likely to heal without revascularization or amputation (136).

Imaging studies play an important role in the assessment and evaluation of the diabetic foot ulcer (96,98,100). Plain X-rays are indicated based on the extent and nature of the ulcer. Clinical change in the appearance of the ulcer or failure to heal with appropriate treatment may dictate repeating the radiograph periodically to monitor for osseous involvement (22). Additional imaging modalities such as nuclear medicine scans, ultrasonography, MRI, and CT may be indicated predicated on the clinical picture. Recommendations for these modalities are discussed elsewhere.

Table 6 summarizes the important elements of the overall assessment of the patient with a diabetic foot ulcer based upon the underlying pathophysiology, possible causal factors, and important predictors of outcome (16,38,74).

TABLE 6  Assessment objectives for foot ulcerations

| Classification: grade, depth, site, clinical descriptors of wound |
| Etiology: mechanical, thermal, chemical trauma |
| Neuropathy: vibration perception, light touch (10-gram monofilament), deep tendon reflexes |
| Vascular: pulses, ankle-brachial index, toe pressures, TcPO₂ |
| Infection: cultures, radiographs, probe, scans, MRI |
| Deformity/High Pressure: callus, hammertoes, bunion, Charcot, amputation |

Diabetic Foot Ulcers: Treatment

Goals and General Principles of Treatment

The primary goal in the treatment of diabetic foot ulcers is to obtain wound closure as expeditiously as possible. The resolution of foot ulcers and decreasing the rate of recurrence can lower the probability of lower extremity amputation in the diabetic patient (16,30,50,51,76).

The essential therapeutic objectives include:

- Debridement
- Pressure relief (off-loading)
- Appropriate wound management
- Management of infection
- Management of ischemia
- Medical management of comorbidities
- Surgical management

Frequent re-evaluation with response-directed treatment is essential. Once healed, the management consists of decreasing the probability of recurrence.

**Debridement**

Debridement of necrotic tissue is an integral component in the treatment of chronic wounds since they will not heal in the presence of nonviable tissue and debris. Adequate debridement must always precede the application of topical wound healing agents, dressings, or wound closure procedures (22,138–140). Types of debridement include: autolytic, enzymatic, mechanical, and surgical. **Autolytic debridement** occurs naturally in a healthy, moist wound environment when arterial perfusion and venous drainage are maintained. The efficacy of **enzymatic debridement** (using topical, proteolytic enzymes) has been questioned in the literature and suffers from a lack of randomized, controlled clinical trials (22,38,141). However, it is commonly used as an adjunctive therapy in the management of chronic wounds. **Mechanical** debridement, including sharp debridement, wet-to-dry dressings, and high pressure irrigation or pulsed lavage, are well accepted therapeutic measures (18,22,38,141,142). The only method which has been proven efficacious in clinical trials is **surgical** debridement (140).

Surgical debridement is a key component and a cornerstone in the management of diabetic foot ulcers. Thorough sharp debridement of all nonviable soft tissue and bone from the open wound is accomplished primarily with a scalpel, tissue nippers, and/or curettes. Excision of necrotic tissue extends as deeply and proximally as necessary until healthy, bleeding soft tissue and bone are encountered. Any callus tissue surrounding the ulcer must also be removed. A diabetic ulcer associated with a deep abscess requires hospital admission and immediate incision and drainage. Joint resection or partial amputation of the foot is needed in the presence of osteomyelitis, joint infection, or gangrene (50,87,88,96).

Necrotic tissue removed on a regular basis can expedite the rate at which a wound heals and has been shown in a recent study to increase the probability of attaining full secondary closure (140,143). Less frequent surgical debridement can impact negatively on the rate of wound healing and secondarily increase the risk of infection. Surgical debridement is repeated as often as needed if new necrotic tissue continues to form. Weekly debridement is commonly required.

**Off-loading**

Reducing pressure to the diabetic foot ulcer is an essential component of treatment (22,34,50,128,144). Without proper off-loading and pressure reduction, ulcers will continually be traumatized to the point that they cannot heal. A study reported in 1999 supports the major role that minor trauma (repetitive stress, shoe pressure, etc.) plays in the causal pathway to ulceration (15).

The choice of off-loading modality should be determined by the patient’s physical characteristics and ability to comply with the treatment, as well as the location and severity of the ulcer. Various centers prefer specific initial modalities, but the clinician frequently must alternate treatments based upon clinical progress of the wound. It is not unusual to practice step-up therapy where increasingly effective modalities are used when little improvement is noted with initial therapy. Some centers prefer to apply total contact casts (TCC) initially and then step-down to less restrictive modalities when lesions have healed or are nearly healed.

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The following off-loading techniques have been found to be useful in the management of diabetic foot ulcers:

- Total nonweightbearing: crutches, bed, wheel chair
- Total contact casting (145–147)
- Foot casts or boots (148,149)
- Removable walking braces with rocker bottom soles (150)
- Total contact orthoses — custom walking braces (144)
- Patellar tendon-bearing braces (151)
- Half shoes or wedge shoes (152)
- Healing sandal — surgical shoe with molded plastizote insole (38,153)
- Accommodative dressings: felt, foam, felted-foam, etc, (38,154,155)
- Shoe cutouts (toe box, medial, lateral, or dorsal pressure points)
- Assistive devices: crutches, walker, cane, etc.

It is critically important to remove the patient from the shoes that caused the ulcer. In fact, the consensus of opinion is such that no patient with an active foot ulcer should be placed back into an unmodified shoe until complete healing has occurred (22,37,50,128).

**Wound Management**

Generally, a moist wound environment bandaged to protect it from trauma and local contamination has been shown to facilitate the healing process (141,156). The type of dressing selected depends upon such factors as size, depth, location, and the wound surface. Normal sterile saline or fractionalized sterile saline (such as 0.5% normal) are frequently used and are often considered as a standard for wound care. However, there is a conspicuous lack of formal clinical trials to support this practice. Many wound care products are available as viable alternatives to saline-moistened gauze dressings, although few of these agents have been subjected to comparative trials (157). These various agents are grouped into different categories and each has its own indications for usage. A brief listing of the dressings and topical agents available are presented in Table 7.

The length of time a wound must exist until it is considered chronic is not well defined in the literature. The Wound Healing Society defines a chronic wound as one which has failed to proceed through an orderly and timely repair process to produce anatomic and functional integrity (138). Skin ulcers, including diabetic foot ulcers, are included in the category of chronic wounds (22,141). Recent clinical trials for the treatment of such wounds have used a period of at least 8 weeks during which there have not been signs of active healing or attaining closure (143). The primary goal in treating the chronic ulcer is to convert it to an acute wound which will then possess the active matrix and cells needed for healing. Reassessment of the entire treatment program is the first step in establishing a new directed approach. The basic principles of treatment discussed for the acute ulcer apply here.

Chronic ulcers have demonstrated benefit from autologous platelet releasates or genetically engineered products, such as recombinant DNA platelet-derived growth factor (bepacrilmin) (143,158,159). These agents have been shown to stimulate chemotaxis and mitogenesis of neutrophils, fibroblasts, and monocytes as well as other components that form the cellular basis on which wound healing can develop (157,160). In one pivotal randomized placebo-controlled blinded trial in patients with full-thickness diabetic foot ulcers, recombinant human platelet-derived growth factor (bepacrilmin) demonstrated a 43% increase in complete closure versus placebo gel (50% vs. 35%) (159). In an economic analysis based upon a large clinical trial, 358 patients treated with bepacrilmin demonstrated a 5% reduction in episode
### TABLE 7  Wound care products

<table>
<thead>
<tr>
<th>Category</th>
<th>Indications</th>
<th>Contraindications</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Dressings</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Transparent films — polyurethane film</td>
<td>Dry to minimally draining</td>
<td>Infection; significant drainage; over prominence or friction</td>
</tr>
<tr>
<td>with adhesive layer; semipermeable</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hydrogels — gel, sheet, gauze; 95%</td>
<td>Dry to minimally draining</td>
<td>Moderate or heavy drainage</td>
</tr>
<tr>
<td>water or glycerin</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Foam — polyurethane foam; open cell</td>
<td>Moderate, large exudate clean wound surface</td>
<td>Dry wounds</td>
</tr>
<tr>
<td>absorbent</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hydrocolloids — wafer with adhesion</td>
<td>Low to moderate drainage</td>
<td>Heavy drainage; sinus tract or deep wound requires</td>
</tr>
<tr>
<td>carboxymethylcellulose; pectin gelatin</td>
<td></td>
<td>debridement, packing prior to hydrocolloid</td>
</tr>
<tr>
<td>impermeable to oxygen</td>
<td></td>
<td>Minimal drainage or dry wounds</td>
</tr>
<tr>
<td>Calcium alginites — pad made of fiber</td>
<td>Heavy exudative wounds</td>
<td></td>
</tr>
<tr>
<td>from seaweed</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gauze pads — Sterile cotton</td>
<td>Low to heavily draining wounds, surgical wounds</td>
<td>Undefined</td>
</tr>
<tr>
<td>Collagen dressings — composite pads</td>
<td>Low to heavily draining wounds</td>
<td>Dry wounds</td>
</tr>
<tr>
<td>with collagen component</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Antimicrobial dressings — contain</td>
<td>Infected or clean wounds to prevent infection</td>
<td>Allergies to components</td>
</tr>
<tr>
<td>silver or iodine in various preparations</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Topical therapies/agents</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Saline — amorphous hydrogels; skin</td>
<td>Clean or infected wounds</td>
<td>Undefined</td>
</tr>
<tr>
<td>cleansers</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Detergents/antiseptics — povidone-</td>
<td>Contaminated or infected wounds</td>
<td>Healthy, granulating wounds</td>
</tr>
<tr>
<td>iodine, etc.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Topical antibiotics — Silver sulfadia-</td>
<td>Contaminated or infected wounds</td>
<td>Healthy, granulating wounds</td>
</tr>
<tr>
<td>zine, Bacitracin, Mupiricin, etc.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Enzymes — collagenase, papain,</td>
<td>Necrotic or escharotic wounds</td>
<td>Healthy or infected wounds</td>
</tr>
<tr>
<td>urea, etc.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Growth factors — Becaplermin gel</td>
<td>Neuropathic diabetic foot ulcers</td>
<td>Infected wounds, necrotic wounds</td>
</tr>
<tr>
<td>(Regranex®); Autologous platelets</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dermal/skin substitutes — Apligraf®;</td>
<td>Venous stasis ulcers; diabetic foot Ulcers</td>
<td></td>
</tr>
<tr>
<td>Dermagraft® (not available in U.S.)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Costs when compared to good wound care alone. Savings were largely due to a 21% lower incidence of hospitalization. However, such improvements in outcomes are also dependent on the presence or restoration of adequate vascular supply in conjunction with good wound care regimens and frequent debridement (161). Becaplermin gel (Regranex®) has received an indication from the FDA for the treatment of neuropathic diabetic foot ulcers.

Tissue-engineered human dermal replacement (Dermagraft®) and human skin equivalent (Apligraf®) containing the characteristics of dermis or both dermis and epidermis, respectively, have also shown promise in preliminary clinical trials (141,162–164). These bioengineered tissue therapy products do not require the donor site needed for conventional split or full-thickness skin grafts. They function not only as biological dressings, but also as delivery systems for growth factors and extracellular matrix components through the activity of live human
TABLE 8 Alternative technologies for wound management

<table>
<thead>
<tr>
<th>Modality</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hyperbaric oxygen therapy (HBO)</td>
<td>165-168</td>
</tr>
<tr>
<td>Vacuum assisted closure (VAC)</td>
<td>170</td>
</tr>
<tr>
<td>Heat therapy</td>
<td>157</td>
</tr>
<tr>
<td>Laser therapy</td>
<td>171</td>
</tr>
<tr>
<td>Mechanical constant tension</td>
<td>172</td>
</tr>
<tr>
<td>Larval/maggot therapy (Biodebridement)</td>
<td>173</td>
</tr>
<tr>
<td>Electrical stimulation (ESTR)</td>
<td>174,175</td>
</tr>
</tbody>
</table>

fibroblasts contained in their dermal elements (141,157). Apligraf® has recently received approval for use on diabetic foot ulcers in the United States.

Hyperbaric oxygen therapy (HBO), might be of benefit although this has not been proven conclusively in prospective clinical trials (22). Several reviews and retrospective studies on this modality purport efficacy in difficult wounds where there might be nonreconstructible occlusive vascular disease or limb-threatening infection (165–168). Local, topically applied oxygen has not been proven effective in clinical trials and cannot be advocated for use (169). HBO and other alternative or unproven technologies which are occasionally used in the management of diabetic foot wounds are listed in Table 8 (157,165–168,170–175).

A variety of other modalities have also been advocated for the chronic wound, although most lack supportive clinical trials. Efficacy for many of these has not clearly been demonstrated and studies supporting their use in diabetic foot ulcers are still needed (176,177).

Management of Infection

Pedal ulcerations provide a portal for pathogen entry and therefore can often lead to the secondary development of infection (88). Rarely does infection directly cause ulceration in the diabetic foot (15). However, when unaddressed this process can threaten both limb and life. Its presence must be determined and identified as either local (soft tissue or osseous), ascending and/or systemic. Treatment requires early incision and drainage with broad-spectrum empirical antimicrobial therapy (22,50).

Debridement of all necrotic tissue including bone and joint resection when these structures are involved must also be performed followed by culture-directed antibiotic therapy. In cases involving gangrene or extensive tissue loss, early amputation at the appropriate level should be considered to remove the focus of infection and to attain viable tissue margins (50,85,87). The necessity for culturing and antimicrobial treatment of clinically uninfected wounds is still under investigation (22). A thorough discussion of the management of infected wounds is presented later in this document.

Vascular Insufficiency

Arterial perfusion is a vital component for healing and must be assessed in the ulcerated patient. Vascular reconstructive surgery of the occluded limb improves prognosis and may be required prior to debridement, foot-sparing surgery, and/or partial amputation (50,178,179).

Management of Comorbidities

Diabetes is a multiorgan systemic disease, and comorbidities must be assessed and managed via a multidisciplinary team approach for optimal outcomes. Patient
TABLE 9  Ulcer Treatment Guidelines

- Debridement of necrotic tissue: surgical, mechanical, autolytic, enzymatic
- Pressure reduction: crutches, healing sandal, contact cast, walking brace, foot cast, felt aperture padding, etc.
- Wound care: topical saline gauze dressings, antiseptics, special dressings, growth factors (beclomethasone), bioengineered tissues, HBO, etc.
- Infection: incision and drainage, empiric and culture directed antibiotics, soft tissue/bone/joint resection, amputations
- Vascular: pedal or proximal bypass, endovascular procedures
- Medical management: hyperglycemia, hypertension, nutritional status, renal status
- Reduce the risk of recurrence:
  - Regular podiatric care and evaluation
  - Patient preventative education
  - Protective footwear
  - Pressure reduction
  - Surgery to reduce bony prominence / chronic pressure points

Compliance has been identified as a significant factor in the expected prognosis and the prevalence of both ulceration and limb loss (74, 75, 136, 180).

Surgical Management

Surgical intervention can generally be classified as curative, ablative, or elective (181). Curative surgery, as the name implies, is performed to effect healing of a nonhealing ulcer or a chronically recurring one when off-loading and standard wound care techniques are not effective (181). Multiple surgical procedures are aimed at removing areas of chronically increased peak pressure. Curative procedures include those used to resect infected bone and/or joints as an alternative to partial foot amputation (22). Operations frequently performed in this regard include exostectomy, digital arthroplasty, sesamoidectomy, single or multiple metatarsal head resections, joint resections, or partial calcanectomy (38, 153, 181–189).

Ablative surgery, often synonymous with amputation, is a common sequela to gangrene and ulcers associated with osteomyelitis. Ablative surgical intervention requires removal of all infected and necrotic tissue to the level of viable soft tissue and bone. When possible, it is also performed in a manner to allow for the maximum function from the remaining portion of the limb (50).

(See Color Plate 4 on page 31.) Wounds may be closed primarily if the surgeon is confident no infection or ischemic tissue remains and enough soft tissue is available. Other wounds may be packed open initially, requiring well controlled and frequently assessed wound care with delayed primary closure or closure by secondary intention. Mechanical assistance using a variety of skin stretching devices are the surgeon's option and may help to attain delayed primary closure for some wounds (172). Plastic surgical techniques utilizing split- and full-thickness skin grafts and a variety of flaps are other options that may be utilized (190, 191). All patients must be assessed on an individual basis for the selection of the surgical management that best meets their needs. Secondary wound healing with or without adjunctive wound therapies may still be the best choice for some patients.

Elective surgery will be discussed in the following section. The basic guidelines or tenets for the management of diabetic foot ulcers are summarized in Table 9.

Diabetic Foot Disorders 25
Preventing Ulcer Recurrence

Prevention is considered a key element in avoiding ulcer recidivism and diabetic lower extremity amputation (16,22,30,34,51,92). This is best accomplished with a multidisciplinary approach consisting of a team of dedicated professionals committed to this ideal (36,74,75,192-196). Typical team members might include the following specialists: podiatrist or pediatric surgeon, internist, endocrinologist, infectious disease physician, cardiologist, nephrologist, neurologist, vascular surgeon, orthopedic surgeon, teaching nurse, and pedorthist (Fig. 2). Patient education assumes a primary role in this scheme and encompasses instruction in foot hygiene, the need for daily inspection, proper footwear, and the necessity for prompt treatment of new lesions (16,74,197-200). Regular podiatric visits, including debridement of calluses and ingrown toenails, provide an opportunity to reinforce appropriate self-care behavior as well as allowing early detection of new or impending foot problems (201). Therapeutic shoes with pressure-relieving insoles and high toe box which protect the high-risk foot are an essential element of the prevention program and have been associated with significant reductions in ulcer development (36,202-204). Walking/athletic style footwear or commercially available orthopedic shoes can be accommodated with various types of foot orthoses to effectively relieve high plantar pressures. Custom-molded shoes are sometimes necessary for severely deformed feet which cannot be adequately protected by standard footwear.
Prophylactic or elective surgical correction of structural deformities that cannot be accommodated by therapeutic footwear in the carefully selected patient, serve to reduce high-pressure areas and ultimately prevent ulcer recurrence (38,50,181,183,188,189,205,206). Many of the procedures previously mentioned in the discussion on curative surgery would also be indicated in the elective reconstruction of the nonulcerated foot. Common operations performed in this regard include the correction of hammertoes, bunions, and various exostoses of the foot. Tendo-Achilles lengthening procedures are often performed as ancillary procedures to reduce forefoot pressures which contribute to recurrent ulcerations (207). Since patients with healed ulcers are at high risk for future ulceration, these prevention efforts must be incorporated into a life-long surveillance and treatment program (16,22,34,37,51,74,92).

A Practice Pathway (algorithm) which summarizes the important parameters for both assessment and treatment of foot ulcers is illustrated in Figure 3.

Diabetic Foot Infections

Foot infections are major causes for hospitalization of patients with diabetes and are also important causal factors leading to lower limb amputation (88). There may be various different presentations of diabetic foot infections as well as several ways to classify these entities (208). They may be described in terms of severity, extent of involvement, clinical appearance, location, and etiology. Any such system for categorizing these infections should also serve to facilitate their management and predict outcomes. Recent literature suggests that a well-accepted method of classifying such entities is to simply categorize them into non-limb-threatening or limb-threatening infections (18,22,50,88,94). This scheme does indeed imply severity of infection and, accordingly, directs subsequent management while portending a general prognosis for outcome.

Non-Limb-Threatening Infections

Clinically, non-limb-threatening infections are usually seen with an ulceration which is typically superficial. Significant ischemia is not present, and the wound should not probe to bone or joint. Ulceration, however, does not need to be present since non-limb-threatening infections can result from small puncture wounds, scratches, or simple fissures. Cellulitis is 2 cm or less from the ulceration or portal of entry. These patients are medically stable and usually do not present with signs and symptoms of systemic involvement. This relatively mild to moderate infection can usually be managed on an outpatient basis with close supervision from the practitioner (18,22,50).

Limb-Threatening Infections

(See Color Plate 5 on page 31.) Diabetic foot infections in this category have cellulitis that extends greater than 2 cm (18). Additional clinical features may include fever, edema, lymphangitis, hyperglycemia, leukocytosis, and/or ischemia. If an ulcer is present, it may probe to bone or joint, which is highly predictive of osteomyelitis (133). Because the diabetic patient with a relatively severe infection may not necessarily present with these signs and symptoms, it is important to review the entire clinical assessment to guide the practitioner to the proper course of treatment (95). Gangrene, abscesses, osteomyelitis, and necrotizing fasciitis may also be present. Hospitalization is required in order to treat the infection as well as the systemic sequelae. Patients with poor vascular status and ischemia have an
FIGURE 3 Ulcer: A Clinical Practice Pathway.
Assessment of Diabetic Foot Infections

When evaluating the patient, a problem-directed history and physical examination should be obtained. A systematic approach to the complete assessment of these patients is required since there is evidence that they are often inadequately evaluated even when hospitalized (99). The past medical history should assess the neurologic, cardiovascular, renal, and dermatological status of the diabetic patient. Medications that the patient is currently taking, as well as prior antibiotic use, may interfere with planned treatments or indicate that standard treatments are likely to be ineffective. Pain should be considered an unreliable symptom in persons with peripheral neuropathy. The patient should be questioned regarding previous ulcerations, infections, trauma, and surgeries at the present site or any other past location of infection. Constitutional symptoms such as nausea, malaise, fatigue, vomiting, fever, or chills are important clinical clues when presented with an infected diabetic foot. Severe infection or sepsis may be present and must be considered. In approximately 50% of diabetic patients presenting with significant infection, however, systemic signs (fever and leukocytosis) are absent (95). Frequently, the only indication of infection is unexplained or recalcitrant hyperglycemia (18). Laboratory testing might include CBC with or without differential, blood cultures, glycosylated hemoglobin, fasting blood sugar, sedimentation rate, and urinalysis. Other tests should be performed as indicated by the patient’s condition or comorbidities.

The history of the wound or infection should include the onset, duration, and appearance before infection of the area. Depth or size of the ulcer, amount of drainage, swelling, color, odor, and extent of infection should be evaluated. The infection or ulcer should be probed to determine the presence of bone or joint involvement, sinus tracts, or extension into tendon sheaths. The latter are common routes for the spread of infection both distally and proximally. If bone is exposed it is assumed that the patient has osteomyelitis until proven otherwise (86, 133). Both anaerobic and aerobic cultures should initially be obtained, since antibiotic therapy will be required. Reliable cultures should be obtained from pus or curettage of the ulcer base, since studies have shown good concordance with the true pathogen (208–210). For patients with clinically uninfected or noninflamed neuropathic ulcers, the role of antibiotic therapy is still in question (22). In these instances, therefore, wound culture is most likely unnecessary (209). If osteomyelitis is suspected, bone cultures are necessary to make the definitive diagnosis as well as to isolate the true pathogen (96). However, this must be balanced against the possibility of contaminating noninfected bone in the presence of an active soft-tissue infection.

Imaging studies are helpful in the overall assessment of diabetic foot infections, notwithstanding their shortcomings. Plain-film x-rays may indicate the presence of bony erosions and/or gas in the soft-tissues. It is important to note that the demonstration of osteomyelitis by plain radiographs lags the onset of bone involvement by 10–14 days (96, 100). Radionuclide bone scans such as Tc99 may demonstrate abnormal uptake of the radionuclide before changes are visible on radiographs (98). This finding may be less specific in patients with peripheral neuropathy or with any pre-existing osseous condition that causes increased bone turnover (e.g., surgery, fracture, neuropathic arthropathy) (102). A combination of scans such as the Tc99m and an indium-labeled leukocyte scan, or the Tc99m-HMPAO-labeled leukocyte scan may aid in differentiating between Charcot arthropathy and osteomyelitis with greater accuracy (104–106). MRI has generally supplanted the CT scan in the early diagnosis of osteomyelitis due to its higher tissue contrast and ability to detect
Plate 1  Below-knee amputation resulting from a foot ulcer.

Plate 2  Classic diabetic “malperforans” foot ulcer.

Plate 3  Ischemic gangrene of the forefoot.
Plate 4  Transmetatarsal amputation.

Plate 5  Limb-threatening infection with large midfoot ulceration.
Plate 6  Osteoarthropathy of the ankle showing severe osteolysis and talonavicular dislocation.

Plate 7  Classic appearance of the diabetic Charcot foot.
Plate 8 Radiographic appearance of Charcot foot shown in Plate 7.

Plate 9 Micfoot osteoarthropathy.
both soft-tissue and marrow inflammation (96, 107, 211). MRI can also be used to follow the resolution of infection or as an aid in surgical planning (109). None of the aforementioned imaging modalities are 100% sensitive and specific for diagnosing or ruling out the presence of bone infection. Furthermore, these tests may not be readily available and are quite expensive. Appropriate clinical assessment and diagnostic acumen should therefore remain the guiding principles to management.

Potential items for initial patient evaluation on hospital admission include the following:

- History and physical
- Radiographs
- Vascular testing
- Possible consultations — medicine/endocrine, infectious disease, vascular surgery, orthopaedic surgery, nutrition
- Cultures — reliable wound cultures, blood cultures
- Labs — CBC with differential, sedimentation rate, glucose, others

Treatment of Diabetic Foot Infections

Diabetic foot infections should be managed with a multidisciplinary team approach (88, 95, 192, 194). This should include obtaining the appropriate consultations as well as admitting the patient to a hospital setting in emergent cases or when the patient does not respond to a course of outpatient treatment. Hospitalization of limb-threatening infections should be considered mandatory. Diabetic foot infections, whether non-limb-threatening or limb-threatening, need to be monitored very closely (50). Equally important, especially in the outpatient management of foot infections, patient compliance and education must be addressed in order to provide the best possible outcome.

Non-Limb-Threatening Infections

Non-limb-threatening infections complicating foot ulcers may be initially treated in an outpatient setting (18, 22, 88, 212). Many of these mild or moderate infections are monomicrobial, with Staphylococcus aureus, Staphylococcus epidermidis, and streptococci being the most common infecting organisms (86, 88, 213). Cultures should be taken from a curettage of the ulcer base to obtain a reliable specimen (208). Antibiotic therapy should be initiated as soon as possible with an agent providing adequate gram-positive coverage, recognizing that gram-negative organisms might also be involved (22, 50, 86, 87) (Table 10). Antibiotic therapy should be adjusted according to culture results and the patient’s response to treatment. The wound should be assessed and cleansed thoroughly, using proper debridement as indicated. Of the several topical agents that can be used on the infected wound, no one agent or topical antibiotic has been proven superior. The wound itself should be managed according to principles discussed under “Wound Management.” Most importantly, the patient should be reassessed within 48–72 hours (18, 87). If no improvement is noted, hospitalization with intravenous antibiotics should be considered. Management of this type of infection should also include close monitoring of the patient’s hyperglycemia and general health status. Patient compliance as well as a reduction in the pressure of the infected limb must be considered early on in the treatment of any diabetic foot infection (50).

Limb-Threatening Infections

Limb-threatening infections may have life-threatening complications, especially when left untreated. Due to immunosuppression from diabetes, up to 50% of these
patients may present with no systemic symptoms or leukocytosis (95,97,214). Other patients, however, do present with evidence of systemic toxicity including fever, chills, loss of appetite, and malaise. Such findings in diabetic patients should alert the clinician to the potential severity of infection (209). Often present is an uncontrollable hyperglycemia, despite routine therapy and a loss of appetite (85,215).

Limb-threatening infections have one or more of the following findings: greater than 2 cm of cellulitis, lymphangitis, soft-tissue necrosis, fluctuance, odor, gangrene, and/or osteomyelitis. When such an infection is recognized, the patient requires emergent hospital admission for appropriate intervention (22). Upon admission, the patient requires a complete history and physical examination. The patient’s cardiovascular, renal, and neurologic risks should be evaluated to assess for secondary complications of diabetes and associated comorbidities. The foot requires a thorough evaluation to assess clinical extent of the infectious process. Vascular status must be assessed to ensure adequate arterial inflow is present. Ulceration, if present, must be probed for bone or joint involvement and subcutaneous sinus tracts, while also measuring for size and depth (50,133). Radiographs should be taken and evaluated for evidence of osteomyelitis or soft-tissue gas. If gas is identified in the ankle or hindfoot, radiographs of the lower leg should be obtained to assess the extent of the gas formation. Deep, reliable aerobic and anaerobic cultures should be obtained from the wound (87,210). Blood cultures should be obtained in the presence of high fever since such clinical findings are often indicative of septicemia (22). Other appropriate laboratory studies, including CBC with differential and sedimentation rate, should be obtained as appropriate. Glucose management must be initiated to optimize metabolic perturbations and to improve leukocyte function (87). The patient’s nutritional and metabolic status must be assessed and properly maintained since such relatively common impairments in these patients can have adverse effects on wound healing and resolution of infection (216,217).

Consultations are typically required in the management of these complex patients, and are indicated for risk assessment and medical management. Medical, endocrinology, cardiology, nephrology, and diabetic teaching nurse consultations are often routinely necessary to optimize patient care and for full assessment of surgical risks (88). Infectious disease and vascular surgery consultations are obtained when complex infections or significant ischemia are identified. A multidisciplinary approach to managing these patients has been shown to significantly improve outcomes (36,74,192–195).

Early surgical treatment of the affected site is typically necessary as an integral part of infection management (22,50,95,218). This may include simple debridement of the soft tissues, wide incision and drainage of the pedal compartments, or open amputation to eliminate extensive areas of infection (95,219,220). Aerobic and anaerobic tissue cultures should be obtained at the time of debridement, and should
be obtained from the depth of the wound to provide reliability (210). Although many initial drainage procedures can be done at the bedside for neuropathic patients, most will require thorough debridement in the operating room (22). Anesthesia may include local, regional, or general anesthetics. Spinal blocks are typically avoided in patients who may be septic. Even the sickest of patients should be considered for emergent incision, drainage, and debridement procedures since their illness is directly attributable to the severity of their infection. Life-threatening infections necessitate immediate surgical attention and such procedures should not be delayed while waiting for radiologic or medical workup of other comorbid conditions (22,50,85,218).

Polymicrobial infection should be anticipated, with a variety of gram-positive cocci, gram-negative rods, and anaerobic organisms predominating. (Table 11). Empirical antibiotic therapy typically includes broad-spectrum coverage for more common isolates from each of these three categories (Table 12). Fully comprehensive empiric coverage is usually unnecessary unless the infection is life threatening (86,214). Hospital therapies are usually initiated with intravenous medications, although most fluoroquinolones can be administered orally in conjunction with other parenteral therapy (86,209). Once wound culture results have been obtained, the initial antimicrobial therapy may require adjustment to provide more specific coverage or to provide therapy against resistant organisms that are causing persisting infection. Recent evidence also supports the efficacy of initial parenteral therapy followed by the appropriate oral agent (221). If the patient develops evidence of recurrent infection while under antibiotic therapy, repeat cultures should be obtained to assess for superinfection. Methicillin-resistant staphylococci have been emerging as important pathogens in chronically treated diabetic foot ulcer patients (222). These organisms must be detected early and treated appropriately to avoid further tissue loss or extension of infection.

The surgical wound may require repeated surgical debridements to completely eradicate infection and soft-tissue necrosis. Wound care is initiated on the first or 2nd postoperative day and may initially involve saline gauze dressing changes. Other dressings may be utilized to aid with healing and are listed elsewhere. If the wound fails to progress, the patient's vascularity, nutrition, infection control, and wound off-loading must be re-evaluated.

Once soft-tissue infection is under control and management of any osseous infection has been initiated, consideration may be given to wound closure or definitive amputation. Restoration and maintenance of function and independence is the ultimate goal for the patient (50). The residual extremity requires close follow-up, regular diabetic foot exams, periodic foot care, and appropriate footwear therapy (22,203,204).

**Osteomyelitis** and joint infection, when identified by clinical assessment or imaging studies, will require excision of bone for microbiological and histopathological evaluation (96). If the patient's soft-tissue infection is controlled, consideration may be given to stopping antibiotic therapy 24–48 hours preoperatively to improve culture accuracy. Both studies should have positive findings including necrosis, chronic inflammatory infiltrates, and positive isolation of bacteria to diagnose osteomyelitis (96,223). Resection of infected bone with or without local amputation and concurrent antimicrobial therapy is the optimal management for osteomyelitis (220,224). If the affected bone has been completely resected or amputated, the infection may be treated as a soft-tissue infection. However, if residual bone is present in the wound, the patient will likely require 4–8 weeks of antibiotic therapy based on the culture results (86,88,96). Intravenous or oral agents may be used depending on the microbial isolates and the infection severity (209,221). Antibiotic-impregnated bone cement has been advocated for treatment of osteomyelitis but
TABLE 11  Common pathogens in diabetic foot infections

Aerobes
   Gram +
      Staphylococcus aureus (methicillin-sensitive and resistant)
      Staphylococcus epidermidis (coagulase negative staphylococci)
      Streptococcus species
      Enterococcus (Strep. faecalis, Group D Streptococcus)
      Corynebacterium species (Diptheroids)
   Gram -
      Proteus mirabilis
      Proteus vulgaris
      Escherichia coli
      Klebsiella species
      Serratia species
      Enterobacter cloacae
      Pseudomonas aeruginosa
      Acinetobacter species

Anaerobes
   Peptococcus magnus
   Peptostreptococcus species
   Bacteroides fragilis
   Bacteroides species
   Clostridium perfringens
   Clostridium species

Other
   Candida albicans
   Candida species

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TABLE 12  Empirical antibiotic therapy: limb- or life-threatening infection

<table>
<thead>
<tr>
<th>Limb-Threatening</th>
<th>Life-Threatening</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ampicillin/Sulbactam</td>
<td>Ampicillin/Sulbactam + Aztreonam</td>
</tr>
<tr>
<td>Ticarcillin/Clavulanate</td>
<td>Piperacillin/Tazobactam + Vancomycin</td>
</tr>
<tr>
<td>Piperacillin / Tazobactam</td>
<td>Vancomycin + Metronidazole + Ceftazidime</td>
</tr>
<tr>
<td>Ceftazidime + Clindamycin</td>
<td>Imipenem/Cilastatin</td>
</tr>
<tr>
<td>Cefotaxime ± Clindamycin</td>
<td>Fluoroquinolone + Vancomycin + Metronidazole</td>
</tr>
<tr>
<td>Fluoroquinolone + Clindamycin</td>
<td>Vancomycin + Levofloxacin + Metronidazole</td>
</tr>
</tbody>
</table>

should only be utilized if the bone has been thoroughly debrided and the soft-tissue envelope is adequate for wound closure after antibiotic-impregnated bead placement (95,225). Typically, gentamycin, tobramycin, or vancomycin are the agents used in the beads. It is generally recommended that the antibiotic beads be removed 2 weeks or so after placement.

Figure 4. A Practice Pathway (algorithm) presents a comprehensive overview to the diagnosis and management of diabetic foot infections.
**Diabetic Foot Disorders INFECTION:** A Clinical Practice Pathway

**Diagnosis and Management**

**History**
- Bruises, Puncture Wound, Foreign Body
- Fever, Chills, Nausea, Malaise
- Ulcer? If so, Duration
- Drainage, Swelling, Erythema
- Pain / Sensation
- Diabetes Duration / Control

**Examination**

**LIMB-THREATENING INFECTION**
- >2 cm Cellulitis
- Lymphangitis
- Edema
- Fever +/-
- Occur from Wound
- Deep Ulcer
- Purulent Drainage
- Hypotension, Cardiac Arrhythmia (Systemic Toxemia)
- Ischemic Changes

**NON-LIMB-THREATENING INFECTION**
- ≤ 2 cm Cellulitis
- Superficial Ulcer
- Does Not Probe to Bone
- No Bone, Joint Involvement
- Mild Infection
- No Systemic Toxemia
- No Significant Ischemia

**Diagnostic Procedures**
- Cultures from Base of Ulcer / Wound (Tissue Specimen if possible)
- Diagnostic Imaging: X-Ray, MRI, Nuclear Bone / Leukocyte Scans, Arthrography
- Serologic Testing
- CBC with Differential
- Blood Cultures
- ESR
- Blood Glucose
- Renal and Hepatic Profile as Appropriate
- Oral Temperature

**Treatment**
- Debridement of All Necrotic Tissue and Callus
- Appropriate Off-Loading
- Wound Care / Dressing
- Empiric Antibiotic Coverage, Modified by Culture Findings
- Outpatient Management, with Follow-Up in 24-72 Hours
- Wound Care Continued - e.g., Pack, Dressings, Debridement as Needed
- Hospital Admission if infection Progresses or Systemic Signs / Symptoms Develop
- If Infection Improves but Ulcer remains - See Ulcer Pathway
- Refer to Podiatrist for Follow-Up Care, Patient Education, Special Shoes and Prostheses as Needed
- Foot Soaking Reconstructive Procedures

**FIGURE 4** Infection: A Clinical Practice Pathway.
Charcot Foot (Neuropathic Osteoarthropathy)

(See Color Plates 6–9 on pages 32–33.) Charcot foot (neuropathic osteoarthropathy) is a progressive condition characterized by joint dislocation, pathologic fractures, and severe destruction of the pedal architecture. Osteoarthropathy may therefore result in debilitating deformity or even amputation (79,80,226–228). The condition is associated with severe peripheral neuropathy and the most common etiology today is diabetes mellitus. The prevalence of this condition is variable, ranging from 0.15% of all diabetic patients to as high as 29% in a population of only neuropathic diabetic subjects (80). The frequency of diagnosis of the diabetic Charcot foot appears to be increasing as a result of increased awareness of its signs and symptoms (79,81,229).

Etiology of Neuropathic Osteoarthropathy

The etiology of Charcot neuropathic osteoarthropathy most likely is a combined effect of both the neurovascular and neurotraumatic theories (80,226–231). It is generally accepted that trauma superimposed on a severely neuropathic extremity can precipitate the development of an acute Charcot foot. With the development of autonomic neuropathy, there is an increased blood flow to the foot, resulting in osteopenia and a relative weakness of the bone (232). The presence of sensory neuropathy renders the patient unaware of the precipitating trauma and often profound osseous destruction taking place during ambulation. A vicious cycle ensues whereby the patient continues to walk on the injured foot, thereby allowing further damage to occur (80–82,229) (Figure 5).

Clinical Diagnosis of Acute Charcot Neuropathic Osteoarthropathy

The initial diagnosis of acute Charcot arthropathy is often clinical, based on profound unilateral swelling, increased skin temperature, erythema, joint effusion, and bone resorption in an insensitive foot (79,80,228,233). These characteristics, in the presence of intact skin, are often pathognomonic of acute Charcot arthropathy. In more than
75% of cases, the patient will present with some degree of pain in an otherwise insensate extremity (83,227). The diagnosis is complicated by the fact that in some cases, patients first present with a concomitant ulceration which raises questions of potential contiguous osteomyelitis (233).

When faced with a warm, edematous, erythematous, insensate foot, plain radiographs are invaluable in ascertaining the presence of osteoarthropathy (234). In most cases, no further imaging studies will be required to make the correct diagnosis. With a concomitant wound, it may initially be difficult to differentiate between acute Charcot arthropathy and osteomyelitis solely based on plain radiographs (79,228). Additional laboratory studies may prove useful in arriving at a correct diagnosis. The white blood cell count (WBC) with a left shift will often be elevated in acute osteomyelitis, although this can be blunted in persons with diabetes (97). While the erythrocyte sedimentation rate may also be elevated in the case of acute infection, it often responds similarly to any inflammatory process and is therefore nonspecific. As in the case with any ulcer, it should be probed to ascertain penetration to bone. A bone biopsy, when indicated, should be considered as the most specific method of distinguishing between osteomyelitis and osteoarthropathy in these circumstances. A biopsy consisting of multiple shards of bone and soft tissue embedded in the deep layers of synovium is pathognomonic for neuropathic osteoarthropathy (235).

Technetium bone scans are relatively expensive and generally nonspecific in assisting in the differentiation between osteomyelitis and acute Charcot arthropathy (98). Indium scanning, while still expensive, has been shown to be more specific (103). Additional studies utilized in differentiating Charcot arthropathy from osteomyelitis include bone scans utilizing white blood cells labeled with Tc-HMPAO and magnetic resonance imaging (106,211).

The Classification of Charcot Arthropathy

The most common classification system of Charcot arthropathy is based on radiographic appearance as well as physiologic stages of the process. The Eichenholz classification divides osteoarthropathy into developmental, coalescent, and reconstructive stages (235). The developmental stage is characterized by significant soft-tissue swelling, osteochondral fragmentation, or joint dislocation of varying degrees. The coalescent stage is marked by a reduction in soft-tissue swelling, bone callus proliferation, and consolidation of fractures. Finally, the reconstructive stage is denoted by bony ankylosis and hypertrophic proliferation. While the system radiologically is very descriptive and useful, its practical clinical applicability is less so. In clinical practice, the initial stage is considered active, while the coalescent and reconstructive stages are considered to be the quiescent or reparative stages. A more recent classification system has been described based upon anatomic sites of involvement but does not describe the activity of the disease (80) (Fig. 6).

Management of Acute Charcot Neuropathic Osteoarthropathy

Immobilization and reduction of stress are the mainstays of treatment for acute Charcot arthropathy (79—81,226—229). Many investigators advocate complete nonweightbearing through the use of crutches or other assistive modalities during the initial acute period. While this is an accepted form of treatment, three-point gait may, in fact, increase pressure to the contralateral limb, thereby predisposing it to repetitive stress and ulceration or neuropathic fracture (236). Following a period of off-loading,
a reduction in skin temperature and edema indicates the stage of quiescence at which point the patient progresses into the post-acute phase of treatment. Progression to protected weightbearing is permitted, usually with the aid of some type of assistive device. Through the use of appropriately applied total contact casts or other off-loading modalities (e.g., fixed ankle walker, bivalved casts, total contact prosthetic walkers, patellar tendon-bearing braces, etc.), most patients may safely ambulate while bony consolidation of fractures progresses (80,227,230) (Table 13). The mean time of rest and immobilization (casting followed by removable cast walker) prior to return to permanent footwear is approximately 4–6 months (80,83,226–228,234). There is recent interest in the adjunctive use of bisphosphonate therapy in acute Charcot arthropathy to help expedite the conversion of the acute process to the quiescent, reparative stage (229,237). Similarly, there is interest in managing acute cases with ancillary bone growth stimulation to promote rapid consolidation of fractures (238). Although promising in theory, neither of these adjunctive treatments to date have been conclusively proven effective through large prospective, randomized clinical trials.
Reconstructive surgery may be considered if a deformity or instability exists that cannot effectively be controlled or accommodated by prescription footwear or bracing (229,230,233,239–243). If the arthropathy is identified in its early stages and non-weightbearing is instituted, surgery is usually unnecessary. The consensus of opinion is such that surgery in the acute stage is generally not advisable due to the extreme hyperemia, osteopenia, and edema present (226–230,239,242,243). Surgical intervention during the acute phase, however, may be considered in the presence of acute subluxation without osteochondral fragmentation (236). One recent report, however, indicates successful arthrodesis rates with preserved foot function in patients with acute arthropathy of the midfoot (244). Notwithstanding, this small retrospective study needs confirmation through larger trials prior to adopting this approach in the routine management of the acute Charcot foot.

As few as 4% to as many as 51% of patients presenting to tertiary centers are reported to undergo surgical procedures for Charcot deformities (242,243). Such centers, however, are often receiving chronic patients from multiple referral sources with various degrees of deformity present. Their rate of operation on these patients does not reflect the true incidence or need for such treatment in the community. Large population-based studies are needed to assess the need for surgical intervention as well as to contrast the efficacy of various conservative therapies (229,234).

The goal of any surgery undertaken on the Charcot foot is to create a stable, plantigrade foot that may be appropriately accommodated (226–230,244). Surgery is generally undertaken only after radiographic, dermal thermometric and clinical signs of quiescence. Most operations on persons with Charcot feet consist of exostectomies for prominent plantar (“rocker-bottom”) deformities causing ulceration when the remainder of the foot is stable (226,227,239,245). However, more complex arthrodesis procedures are being utilized with increasing frequency and success rates (230,233,236,239–243,244,246). These include isolated or multiple midfoot fusions, triple arthrodeses, tibio-calcaneal, and ankle fusions. Following surgery, patients are immobilized until skin temperatures and postoperative edema normalize. As with those treated nonsurgically, following prolonged cast immobilization patients progress to a removable cast walker followed by permanent prescription footwear (227). Mean times from operation to the wearing of therapeutic shoes have been reported in the range of 27 weeks (7 months) (83,244). Careful patient selection and management is the rule with these complex diabetic patients since amputation can be an unwanted complication of failed surgical procedures (239,240,242,243).

Figure 7 illustrates a suggested Practice Pathway (algorithm) for the assessment and management of diabetic neuropathic osteoarthropathy.
Diabetic Foot Disorders CHARCOT FOOT: A Clinical Practice Pathway

**FIGURE 7**  Charcot Foot: A Clinical Practice Pathway.
SURGICAL PROTOCOLS

Site of Surgery

Surgical treatment of the complications of the diabetic foot may be performed in a variety of settings including the hospital, either as an inpatient or outpatient, an ambulatory surgical center or office based surgical facility. In today’s environment of managed care and Medicare requirements for hospital admission, often a combination of settings for the surgical treatment of the diabetic with ulcerations, infections and ablative or reconstructive surgery is the rule. The hospital is a good central core that allows the group of physicians and support personnel to interact and coordinate care in an efficient manner, whether care is provided as an in-patient, out-patient or through home health services.

Generally hospitalization is indicated, but not necessarily limited to the following:

- Coexisting medical conditions that generally require the more intensive management afforded by inpatient care, for example, diabetic control, cardiovascular disease, and peripheral vascular disease.
- The initial workup and management of a limb threatening diabetic foot infection.
- Necessity for parenteral medications when administration at home, outpatient or an extended care facility is inappropriate or impractical.
- High risk of perioperative complications.

Preoperative Laboratory Testing

Preoperative laboratory requirements for surgery in a diabetic patient are generally more extensive than those considered for surgery in healthy patients. For example, glucose control, renal function assessment, cardiovascular status and peripheral vascular analysis are among the most common medical concerns which impact the care rendered.

Anesthesia

In most cases, monitored anesthesia care is desirable due to the patient’s medical condition, even in cases where the patient may be insensate. General, regional or local field blocks are all appropriate in the surgical management of diabetic foot disorders. Local anesthesia usually is avoided with ascending infections, for example with an initial incision and drainage of a foot infection, but may be appropriate for later debridement or definitive reconstruction.

Prophylactic Antibiotics

Prophylactic antibiotics may be indicated in patients with diabetes undergoing elective surgery due to their immunocompromised status. As with non-diabetic patients, the chief concern is for staphylococci and streptococci infection. Therefore, standard prophylactic regimens should be utilized with appropriate recognition of the patient’s renal status.
Hemostasis

Hemostasis in diabetic patients undergoing surgery usually is in the form of judicious use of electrocautery. Generally, there is an attempt to avoid ligatures in infections and avoidance of tourniquets in patients with significant ischemia or after undergoing distal revascularization procedures. Tourniquets, however, may be used in well perfused neuropathic limbs. Often, with wounds that are left open in cases of initial management of infections, various packing materials or wound dressings may be used to control blood loss.

PREVENTION

Prevention is considered a pivotal element in avoiding ulceration and subsequent major amputation in the diabetic patient (16,22,30,34,37,74,92). This is best accomplished with a multidisciplinary approach consisting of a team of specialists and personnel providing a coordinated process of care. Team members typically include: podiatrist, podiatric surgeon internist, endocrinologist, infectious disease specialist, cardiologist, nephrologist, vascular surgeon, orthopedic surgeon, nurse educator, visiting home nurse, and pedorthist. Patient and family education assumes a primary role and encompasses instruction in glucose assessment and insulin administration, diet, daily foot inspection and care, proper footwear, and the necessity for prompt treatment of new lesions. Regularly scheduled podiatric visits, including debridement of calluses and toenails, provide an opportunity for frequent foot examination and patient education (74,201). Such visits can provide early warning of impending problems and subsequent modification of activity and care (22). Risk stratification based on the presence of predisposing causal risk factors, including prior history of ulceration, will also act as a guide to the necessary frequency of foot care visits (37). Identification of the high risk patient and tailoring a total foot care prevention program according to the level of risk assigned is considered effective in reducing the incidence of ulceration and lower extremity amputation (37,74,195,199–201).

Therapeutic shoes with pressure relieving insoles and a high toe box which protects the high risk foot are essential to reduce ulceration and ultimately the incidence of amputation (16,202–204). For severe foot deformities which cannot be accommodated by standard therapeutic footwear, custom molded shoes should be fabricated according to these same guidelines. Prophylactic correction of structural deformities that cannot be accommodated by therapeutic foot wear should be considered, but patients must be carefully selected. Elective surgery should be undertaken under optimal metabolic conditions and with the full understanding of rationale, goals, and attendant risks (181,189,206,247). Diabetes is a life-long problem and the incidence of diabetic foot complications increases with age and duration of the disease. Patients, particularly those at risk due to prior infection, ulceration, or amputation must be educated that a program of lifelong surveillance must be incorporated to prevent repeated episodes of these complications. Provider and physician education is equally important in this regard since not all health care providers are cognizant of important signs and risk factors for pedal complications (74,248). Furthermore, provider education is effective in reinforcing proper diabetes management and foot care practices with consequent reductions in ulceration and adverse lower extremity outcomes (89,199). Table 14 lists the important attributes of a diabetic foot prevention program undertaken within the framework of the multidisciplinary team.
TABLE 14 Diabetic foot prevention program

1. Podiatric Care
   Regular visits, examinations, and footcare
   Risk assessment
   Early detection and aggressive treatment of new lesions

2. Protective Shoes
   Adequate room to protect from injury; well cushioned
   walking sneakers, extra depth, custom-molded shoes
   special modifications as necessary

3. Pressure Reduction
   Cushioned insoles, custom orthoses, padded hosiery,
   pressure measurements — computerized or Harris mat

4. Prophylactic Surgery
   Correct structural deformities — hammertoes, bunions, Charcot
   Prevent recurrent ulcers over deformities
   Intervene at opportune time

5. Preventive Education
   Patient education — need for daily inspection and
   necessity for early intervention
   Physician education — significance of foot lesions,
   importance of regular foot examination, and
   current concepts of diabetic foot management

CONCLUSION

Ulceration, infection, gangrene and lower extremity amputation are complications
often encountered in patients with diabetes mellitus. These often result in extensive
morbidity, repeated hospitalizations and mortality to the patient. They take a
tremendous toll on the physical, mental and financial well-being of the patient as
well as potentially removing the patient from a country’s work force and financial
drain on our health care system.

All diabetic foot complications cannot be prevented but it is indeed possible to
dramatically reduce their incidence through appropriate management and prevention
programs. The multidisciplinary team approach to diabetic foot disorders has been
demonstrated as the optimal method to achieve favorable rates of limb salvage in the
high risk diabetic patient. Foot care programs emphasizing preventive management
can reduce the incidence of foot ulceration through modification of self care practices,
appropriate evaluation of risk factors, and the formulation of treatment protocols
aimed at early intervention, limb preservation, and the prevention of new lesions.
The podiatric surgeon should play an integral role in this scheme, providing ongoing
surveillance, education, and management of new or impending lesions. The goal of a
40–50% reduction in diabetic limb amputations is certainly attainable if we embrace
these principles and incorporate them into daily patient care.

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## Screening Form for Diabetes Foot Disease

**Name:** ____________________________  **Date:** ____________________________  **Type DM** 1 — — 2 —

**Age** — — **Age at Onset** — —  **Current Treatment:**  **Diet** — —  **Oral** — —  **Insulin** — —

### I. Medical History (check all that apply)
- ☐ Peripheral Neuropathy
- ☐ Nephropathy
- ☐ Retinopathy  ☐ Peripheral Vascular Disease
- ☐ Cardiovascular Disease

*(For Sections II & III, fill in the blanks with an "R" or "L" for right or left; "Dl" or "Dr" for positive findings on the right or left)*

### II. Current History
1. Any change in the foot since the last examination?
   - Y — —
   - N — —
2. Current ulcer or history of foot ulcer?
   - Y — —
   - N — —
3. Is there pain in the calf muscles when walking that is relieved by rest?
   - Y — —
   - N — —

### III. Foot Exam
1. Are the nails thick, too long, ingrown, infected with fungal disease?
   - Y — —
   - N — —
2. Note foot deformities
   - Bunions (Hallus Valgus)
   - Charcot foot
   - Foot drop
   - Prominent Metatarsal Heads
   - Amputation (Specify date, site and level)

### IV. Sensory Foot Exam
Label sensory level with a "*" in the seven circled areas of the foot if the patient can feel the 5.07 Semmes-Weinstein (10-gram) nylon filament and "*" if the patient cannot feel the filament.

### V. Risk Categorization (Check appropriate box)
- ☐ Low Risk Patient
- ☐ High Risk Patient
  - ☐ Intact protective sensation
  - ☐ Loss of protective sensation
  - ☐ Pedal pulses present
  - ☐ Absent pedal pulses
  - ☐ No severe deformity
  - ☐ Severe foot deformity
  - ☐ No prior foot ulcer
  - ☐ History of foot ulcer
  - ☐ No amputation
  - ☐ Prior amputation

### VI. Footwear Assessment (Fill in the blanks)
- Does the patient wear appropriate shoes?
  - Y — —
  - N — —
- Should therapeutic footwear be prescribed?
  - Y — —
  - N — —

### VII. Education (Fill in the blanks)
- Has the patient had prior foot care education?
  - Y — —
  - N — —
- Can the patient demonstrate appropriate self-care?
  - Y — —
  - N — —

### VIII. Management Plan
*(Check all that apply)*
- ☐ Provide patient education for preventive foot care
- ☐ Athletic shoes
- ☐ Accommodative inserts
- ☐ Custom shoes
- ☐ Depth shoes
- ☐ Comfort shoes
- ☐ Pedorthist

**Footwear recommendations:**  
- Refer to:
  - ☐ Primary Care Provider
  - ☐ Endocrinologist
  - ☐ Diabetes Educator
  - ☐ Rehab. Specialist
  - ☐ Podiatric Surgeon
  - ☐ Vascular Surgeon
  - ☐ Other — —

**Date:** ____________________________

**Provider Signature:** ____________________________

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A Clinical Practice Guideline
REFERENCES


Diabetic Foot Disorders


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