This practice guideline is based upon current clinical practice and extensive review of the clinical literature and has been developed in cooperation with the American College of Foot and Ankle Orthopedics and Medicine (ACFAOM) by the Clinical Practice Core Committee and the Diabetes Committee of the American College of Foot and Ankle Surgeons (ACFAS).

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DIABETIC FOOT DISORDERS
A Clinical Practice Guideline

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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:

040.0	Gas gangrene
357.2	Polyneuropathy in Diabetes (Report code 357.2 as secondary to 250.6x)
440.23	Atherosclerosis of the Extremities with Ulceration (Report code 440.23 as a secondary code to 250.7x)
681.11	Cellulitis/Abscess, Toe
682.6	Cellulitis/Abscess, Leg (including ankle)
682.7	Cellulitis/Abscess, Foot (except toes)
707.1	Ulcer of lower limbs, except Decubitis (Both codes 707.1 and 785.4 should be used to report an ulcer with gangrene)
713.5	Arthropathy Associated with Neurological Disorders (Charcot's) (Report code 713.5 as a secondary code to 250.6x)
730.06	Osteomyelitis/Acute/Lower Leg (Report code 730.06 as a secondary code to 250.8x)
730.07	Osteomyelitis/Acute/Ankle and Foot (Report code 730.07 as a secondary code to 250.8x)
730.16	Osteomyelitis/Chronic/Lower Leg (Report code 730.16 as a secondary code to 250.8x)
730.17	Osteomyelitis/Chronic/Ankle and Foot (Report code 730.17 as a secondary code to 250.8x)
785.4	Gangrene (any site)
V49.71	Status Post Amputation, Great Toe
V49.72	Status Post Amputation, Other Toe(s)
V49.73	Status Post Amputation, Foot
V49.74	Status Post Amputation, Ankle
V49.75	Status Post Amputation, Below Knee
V49.76	Status Post Amputation, Above Knee

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 neurologic 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 (cheiroarthropathy) 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 from 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)

of 15 days (4). 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, payor, 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.

TABLE 1 Risk factors for ulceration

- Peripheral sensory neuropathy
- Structural foot deformity
- Trauma and improperly fitted shoes
- Callus
- History prior ulcers/amputations
- Prolonged, elevated pressures
- Limited joint mobility
- Uncontrolled hyperglycemia
- Duration of diabetes
- Blindness/partial sight
- Chronic renal disease
- Older age

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 crural 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 neuropathy may commonly result in dry skin with cracking and fissuring, thus creating a portal of entry for bacteria (29,38). Autosympathectomy 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).
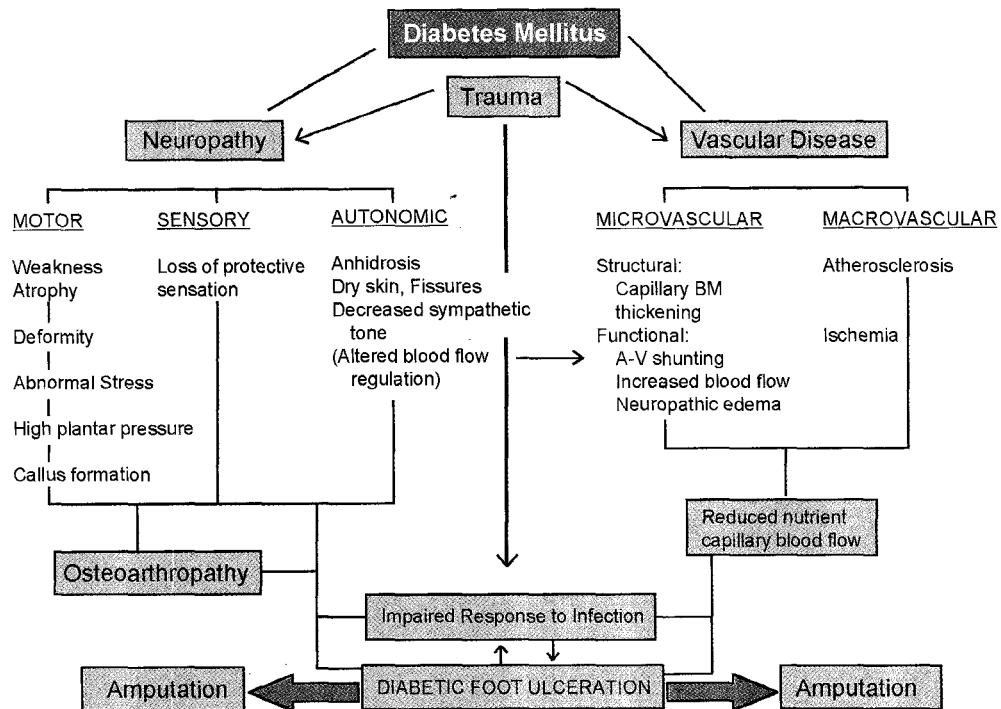
FIGURE 1 Contributing factors in the pathogenesis of ulceration.

TABLE 2 Risk factors for amputation

- Peripheral sensory neuropathy
- Vascular insufficiency
- Infection
- History foot ulcer/amputation
- Structural foot deformity
- Trauma
- Charcot deformity
- Impaired vision
- Poor glycemic control
- Poor footwear
- Older Age
- Male sex
- Ethnicity

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.

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

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
- Rhomberg's test

Musculoskeletal Examination

- Biomechanical abnormalities:
  Orthopedic deformities
  - Hammertoes
  - Bunion(s) or Tailor's bunion(s)
  - 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, nonweightbearing 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
  Bullosum 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):

Risk Categorization System

Category	Risk Profile	Evaluation Frequency
0	No neuropathy	Annual
1	Neuropathy	Semi-annual
2	Neuropathy, PVD, and/or deformity	Quarterly
3	Previous ulcer or amputation	Monthly to quarterly

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_{GG}-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 ($TcPO_2$) (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 TcPO$_2$ levels of less than 30 mm Hg, since these measures of arterial perfusion are associated with impaired wound healing (33,37,49,115,117).