Clinical Examination and Risk Classification of the Diabetic Foot

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Abstract

A consistent, thoughtful assessment of the diabetic foot is pivotal to identify patients at risk for ulceration. In this chapter, we discuss the key risk factors to screen patients for foot complications: a history of lower extremity disease, the presence of peripheral neuropathy, and foot deformities. We discuss the practical approach and background of these key risk factors and subsequently the two most commonly used classification systems for diabetic foot ulcers. Many of the risk factors for ulceration may be identified using simple, inexpensive techniques in a primary care setting. Appropriate classification of the wound becomes paramount in our efforts to document and communicate the level of risk and facilitate amputation prevention.

Foot ulceration is one of the most common precursors to lower extremity amputations among persons with diabetes [1–3]. Ulcerations are pivotal events in limb loss for two important reasons. First, they allow an avenue for infection [4], and second they can cause progressive tissue necrosis and poor wound healing in the presence of critical ischemia. Infections involving the foot rarely develop in the absence of a wound in adults with diabetes, and ulcers are the most common type of wound in this population [4]. Foot ulcers therefore play a central role in the causal pathway to lower extremity amputation [5].

The etiology of ulcerations in persons with diabetes is commonly associated with the presence of peripheral neuropathy and repetitive trauma due to normal walking activities to areas of the foot exposed to moderate or high pressure and shear forces [6]. Foot deformities, limited joint mobility, partial foot amputations and other structural deformities often predispose patients with diabetes with peripheral neuropathy to abnormal weight bearing, areas of concentrated pressure and abnormal shear forces that significantly increase their risk of ulceration [7–9]. Brand theorized that when these types of forces were applied to a discrete area over an extended period of time they would cause a local inflammatory response, focal tissue ischemia, tissue destruction, and ulceration [10]. Clearly, identification of persons at risk for ulceration is of central importance in any plan for amputation prevention and diabetes care.

Diabetic Foot Risk Classification

Preventing foot complications begins with identifying patients at risk for developing a foot ulcer. Diabetic foot screening programs are inexpensive and can be performed by technicians or nurses with very little training. In patients with signs or symptoms of loss of protective sensation caused by peripheral neuropathy, examinations should include obtaining a detailed history of ulceration and amputation of the lower extremities, and screening for the presence of peripheral artery disease and foot deformities. On top of that other patient-related factors like inadequate footwear, foot hygiene, and pre-ulcerative signs on the foot should be identified. In the updated consensus document of the International Working Group on the Diabetic Foot (IWGDF), a screening interval is added to the widely used classification system of the key risk factors [11].

Lavery et al. reported that a patient with neuropathy but no deformity or history of ulcer or amputation has a 1.7 times greater risk for ulceration compared with a patient without neuropathy [12]. Neuropathy with concomitant deformity or limited joint mobility yields a 12.1 times greater risk. Lastly, a patient with a history of previous ulceration or amputation has a 36.4 times greater risk for presenting with

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Table 2.1 The IWGDF Risk Classification System 2015 and preventative screening frequency [11]

Category	Characteristics	Frequency
0	No peripheral neuropathy	Once a year
1	Peripheral neuropathy	Once every 6 months
2	Peripheral neuropathy with peripheral artery disease and/or a foot deformity	Once every 3–6 months
3	Peripheral neuropathy and a history of foot ulcer or lower extremity amputation	Once every 3–6 months

another ulcer. These risk factors compare to the categories in the classification system promoted by the International Working Group on the Diabetic Foot [11–14] (Table 2.1) and similar classification systems described by Rith-Najarian [15] and Armstrong [16]. A comparison was made between this system and four other classification tools in a systematic review in 2011 [17]. Core values of the stratification systems were very similar, but the risk groups and number of variables that were included varied.

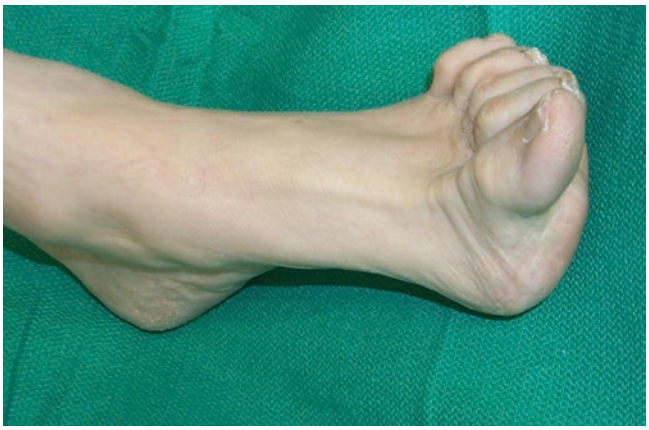

Fig. 2.1 Intrinsic muscular atrophy and foot deformity. Diabetic peripheral neuropathy also affects motor nerves, often causing atrophy of intrinsic musculature of the hand and foot. When this occurs, the extrinsic musculature work unopposed, thus causing hammering of the toes and retrograde buckling of the metatarsal heads. Thus, both the toes (dorsal) and the metatarsal heads (plantar) are more prominent and therefore more prone to neuropathic ulceration

History of Foot Pathology

History of foot disease is the strongest predictor of ulceration and amputation and the least expensive screening measure [18, 19]. It is the easiest risk group to identify, and the group most in need of frequent foot assessment, intensive education, therapeutic shoes, padded stockings, and rigorous blood glucose control. A current ulcer, past history of previous ulceration or amputation heightens the risk for further ulceration, infection, and subsequent amputation [5, 11, 17, 18]. Patients in this risk group (Risk Category 3) are about 50 times more likely to have an ulcer in the next year and 36 times more likely to have an amputation compared to patients with no neuropathy or PAD [20]. The presence of pre-ulcerative lesions such us abundant callus, hemorrhage or a blister, is a strong determinant of ulcer recurrence, especially in patients with recurrence caused by unrecognized repetitive trauma [21].

There are several potential explanations for the increased risk. Diabetic patients with a history of ulceration or amputation have all the risk factors to re-ulcerate [22, 23]. Ulceration and amputation damage the integument and the biomechanics of the foot. After healing by secondary intention, the skin and soft tissue is scarred and it may be less resilient and less pliable, so it is more prone to injury. In addition, persons with a partial foot amputation often develop local foot deformities secondary to biomechanical imbalances that may cause further foci of pressure and shear [24–26]. Structural deformities increase pressures on the sole of the foot and are associated with ulceration (Fig. 2.1). A classic example is clawing of the lesser toes and subluxation and dislocation of the metatarsophalangeal joints [26].

Peripheral Neuropathy

Neuropathy is a major component of nearly all diabetic ulcerations [27]. Loss of protective sensation is a term that is often used to describe a level of sensory loss that allows patients to jury themselves without recognizing the injury. These patients are vulnerable to physical and thermal trauma that increases the risk of foot ulceration twofold [20]. Patients with neuropathy often wear a hole in their foot much as a sensate patient might wear a hole in their stocking or shoe.

Screening for neuropathy is noninvasive, fast, and inexpensive. Several consensus documents recommend that all patients with diabetes should be screened annually for sensory neuropathy [27, 28]. There are several techniques to screen for neuropathy. The absence of protective sensation may be determined using a tuning fork, a Semmes-Weinstein 10 gram monofilament nylon wire, a calibrated vibration perception threshold (VPT) meter, or by a comprehensive physical examination.

Inspection of the feet may provide valuable clues as to the presence and severity of sensory neuropathy. Atrophy of the intrinsic muscles of the hands and feet is often a late-stage condition that is very frequently associated with polyneuropathy. When this occurs, the extrinsic muscles of the foot are unopposed, thus causing hammering of the toes and retrograde

buckling of the metatarsal heads. Thus, both the toes (dorsal) and the metatarsal heads (plantar) are more prominent and therefore more prone to neuropathic ulceration (Fig. 2.1). This condition often leads to prominent digits and metatarsal heads, and (in the face of sensory loss) has been associated with increased risk for neuropathic ulceration. Similarly, bleeding into callus is a condition which is associated with neuropathy. Patients with autonomic neuropathy may present with dry skin that is poorly hydrated.

Tuning Fork

The conventional 128 Hz tuning fork is an easy and inexpensive tool to assess vibratory sensation. The test is considered positive when the patient loses vibratory sensation while the examiner still perceives it [29]. The tuning fork is struck until it clangs, and the tip of the tuning fork is held against a bony prominence, such as the distal tip of the great toe. The patient is asked if they can feel the vibration. If they feel pressure but no vibration, they have loss of vibration sensation. In addition, the patient should be able to feel the vibration for about 20 s. If they cannot feel the vibration for 20 s, they have abnormal vibration sensation. In addition to a standard 128 Hz tuning fork, a graduated tuning (Rydel-Seiffer) fork has provided comparable results to the vibration perception testing (r, −0.90; $P < 0.001$) [30, 31]. Using the graduated tuning fork, patients indicate first loss of vibration at the plantar hallux as the intersection of 2 virtual triangles moves on a scale exponentially from 0 to 8 in a mean (AD) of 39.8 (1) seconds [32].

Semmes Weinstein Monofilament

The Semmes Weinstein monofilament is one of the most frequently utilized screening tools for identifying loss of protective sensation in the United States [28, 33]. The inability to perceive the 10 g Semmes Weinstein monofilament has been associated with large-fiber neuropathy [34, 35]. In three prospective studies, the 5.07 or 10 g Semmes Weinstein monofilament identified persons at increased risk of foot ulceration with a sensitivity of 65–91%, a specificity of 36–86%, a positive predictive value of 18–39%, and a negative predictive value of 90–95%. (Table 2.2) [18, 35, 36] The Semmes Weinstein monofilament consists of a plastic handle supporting a nylon filament. It is portable, inexpensive, easy to use, and provides excellent negative predictive ability for the risk of ulceration and amputation [37].

There are a number of important concerns regarding the Semmes Weinstein monofilament. There is wide variability in the accuracy and durability of monofilaments sold in the United States. Certain brands of monofilaments are more accurate than others [38]. Instruments made in the United Kingdom seem to have better initial accuracy and calibration [37]. Semmes Weinstein monofilaments experience material failure of the nylon monofilament and become less accurate with repeated measurements. Therefore it is important to purchase calibrated instruments and to replace them on a regular basis. In a clinical setting, it is best for the evaluator to have more than one monofilament available, as after numerous uses without a chance to "recover," the monofilament may buckle at a reduced amount of pressure, thus making it oversensitive and therefore less accurate [38]. Longevity and recovery testing results from an independent study suggest that each monofilament, regardless of the brand, will survive usage on approximately ten patients before needing a recovery time of 24 h before further use [32, 38]. Furthermore, differences in materials used in the manufacturing process and environmental factors may also change the characteristics of the monofilament [38, 39].

Testing with the Semmes Weinstein monofilament is best performed with the patient sitting supine in the examination chair with both feet level. The monofilament is applied perpendicular to the skin until it bends or buckles from the pressure. It should be left in place for approximately one second and then released [27]. The monofilament should be demonstrated on the patient's hand, so they can understand the level of pressure provided during testing. The patient should close their eyes for the foot examination. They should be instructed to say "yes" each time that they feel the monofilament and then to identify the site where they felt the monofilament. The number of sites that should be tested with monofilaments is unclear. However, because testing is noninvasive and inexpensive, the number of sites should not be a limiting factor in testing protocols. Some authorities recommend that measurements be taken at each of ten sites on the foot [40]. These include the first,

Table 2.2 10 g monofilament to diagnose sensory neuropathy

Author, year, Journal	Prevalence Ulcers %	Sensitivity / Specificity	Positive Predictive Value	Negative Predictive Value
Boyko, 1999, Diabetes Care [18]	11%	68 / 62	18	94
Rith-Najarian, 1992, Diabetes Care [35]	11%	65 / 86	39	95
Pham, 2000, Diabetes Care [36]	29%	91 / 36	34	90

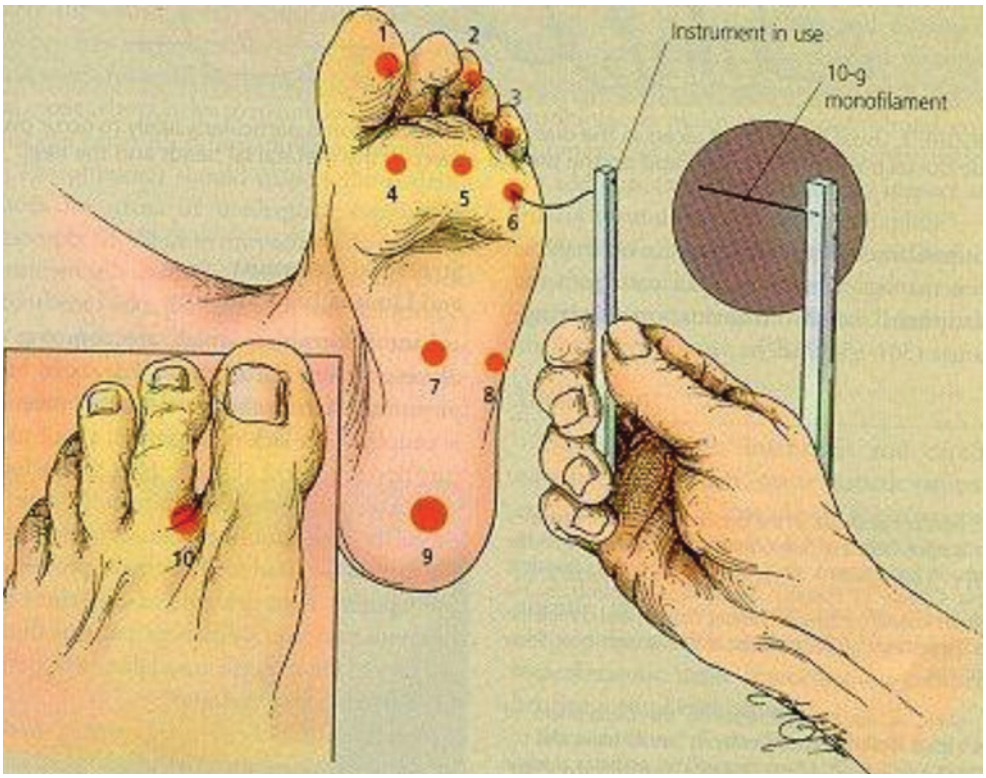

Fig. 2.2 Use of the 10-g monofilament

third, and fifth digits, plantarly, the first, third, and fifth metatarsal heads plantarly, the plantar midfoot medially and laterally, the plantar heel, and the distal first interspace, dorsally (Fig. 2.2). However, testing just four plantar sites on the forefoot (the great toe, and base of the first, third, and fifth metatarsals) identifies 90% of patients with loss of protective sensation [41].

Vibration Perception Threshold (VPT) Testing

A VPT meter is a semiquantitative tool to assess large fiber neuropathy. The VPT meter (also known as Biothesiometer or Neurothesiometer) is a handheld device with a rubber tactor that vibrates at 100 Hz. The handheld unit is connected by an electrical cord to a base unit. This unit contains a linear scale which displays the applied voltage, ranging from 0 to 100 V (converted from microns [36, 42] (Fig. 2.3). The device is held with the tactor balanced vertically on the pulp of the toe. The voltage amplitude is then increased on the base unit until the patient can perceive a vibration. A mean of three readings (measured in Volts) is generally used to determine the vibration perception threshold for each foot. "Loss of protective sensation" with VPT has commonly been considered to be about 25 V. The level of Vibration Perception Threshold testing can help to predict ulceration [43]. In a prospective cohort study Abbott and colleagues evaluated 1035 patients with diabetes, no history of a foot ulcer and a VPT greater than 25. During the follow-up period the yearly ulcer incidence was 7.2%. For every one volt increase in VPT, there was a 5.6% increase in the risk of foot ulceration [44]. VPT testing has been shown to have very good sensitivity and specificity (Table 2.3).

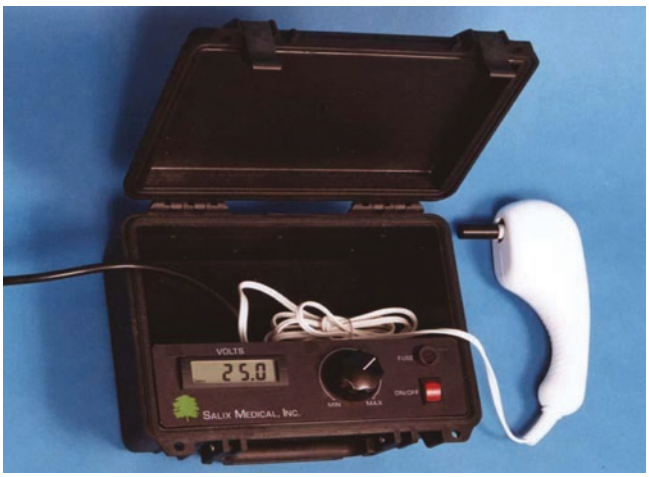

Fig. 2.3 Vibration perception threshold meter. The vibrating tactor is placed at the distal pulp of the great toe. The amplitude (measured in Volts) is increased on the base unit until the patient feels a vibration. This is termed vibration perception threshold (VPT). A VPT greater than 25 V may be an optimal combination of sensitivity and specificity for identifying clinically significant loss of protective sensation using this device

Table 2.3 Vibration Perception Threshold Testing

Author, year, Journal	Prevalence Ulcers %	Sensitivity / Specificity	Positive Predictive Value	Negative Predictive Value
Sosenko, 1990, Diabetes Care [45]	29%	83% / 87%	49%	NS
Vileikyte, 1997, Diabetes Care [46]	28%	86% / 79%	NS	NS
Armstrong, 1998, Arch Int Med [24]	33%	80% / 85%	NS	NS

NS not stated

New Neuropathy Screening Tests

Two recently described tests have been validated against the other commonly used screening test. The Ipswich Touch Test (IpTT), which assesses the ability of the patient to perceive the touch of a finger [48] and the Vibratip, a disposable vibrating stylus that can assess vibration sensation [49]. The IpTT involves lightly touching/resting the tip of the index finger for 1–2 s on the tips of the first, third, and fifth toes and the dorsum of the hallux. Direct comparison of the IpTT and monofilament testing showed almost perfect agreement, with positive predictive values indicating at-risk feet of IpTT 89%, MF 91% and negative predictive values of IpTT 77% and MF 81%. The IpTT has also been evaluated to detect reduced foot sensation in the setting of the patient's home [50]. Having a simple method to detect loss of sensation at home might improve awareness of foot disease in patients with diabetes and empower them to seek appropriate care. When activated, the VibraTip (McCallan Medical Limited, Nottinghamshire, UK) provides a stimulus of 128 Hz, mimicking the conventional tuning fork. The patient's hallux is touched twice with the rounded tip of the VibraTip, each time for approximately 1 s, while randomly activating the VibraTip on either the first or second touch. Both the Vibratip and the IpTT showed high concordance with the vibration perception threshold test ≥25 V in 83 at-risk individuals [49].

Modified Neuropathy Disability Score

Clinical assessment can be used to score the severity of peripheral neuropathy in order to identify high-risk patients. The Modified Neuropathy Disability Score is a clinical assessment scoring scheme that uses standard clinical tools. These include deep tendon reflexes of Achilles tendons, vibration sensation with 128 Hz tuning fork, pinprick, and hot and cold rods. Use of these instruments, combined into a disability score, has proven to be predictive of future diabetic foot complications [19]. In a population-based prospective study, Abbot evaluated 9710 patients with diabetes from six health districts in the United Kingdom. During the 2-year follow-up period there were 291 ulcers. Only 1.1% of patients with a Neuropathy Disability Score less than six developed a foot ulcer, and 6.3% of patients with NDS greater than six developed an ulcer [19].

Limited Joint Mobility

Neuropathy and foot deformity, when combined with repetitive or constant stress, can lead to ulceration. Characteristically, the highest plantar pressure is associated with the site of ulceration [6, 7, 51, 52]. In one study of patients with peripheral neuropathy, 28% with high plantar pressure developed a foot ulcer during a 2.5-year follow-up compared with none with normal pressure [53].

Clinicians should examine the feet for structural abnormalities including hammer or claw toes, flat feet, bunions and calluses, and reduced joint mobility to help identify pressure points that are susceptible to future ulceration. Structural deformity is frequently accompanied by limited joint mobility. Nonenzymatic glycosylation of periarticular soft tissues or tendons may contribute to limited joint motion in the person with diabetes. Neuropathy can lead to atrophy of the intrinsic muscles of the hands and feet which can cause instability at the metatarsophalangeal joint and digits [54]. Limitation of motion reduces the foot's ability to accommodate for ground reactive force and, therefore, increases plantar pressures [55–57]. Limitation of motion of the first metatarsophalangeal joint has been defined as less than 50° of passive dorsiflexion of the hallux (Fig. 2.4).

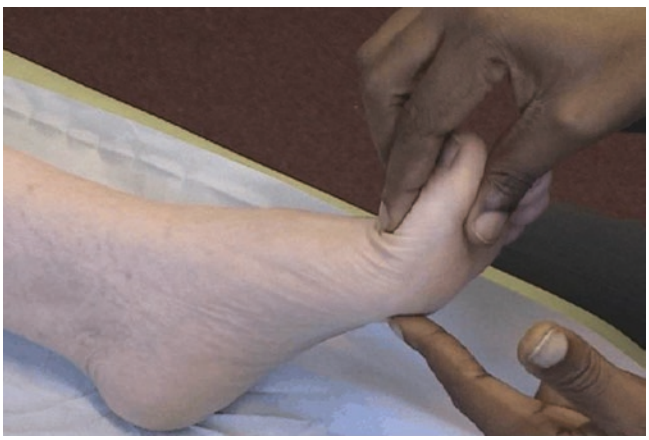

Fig. 2.4 Evaluation of first metatarsophalangeal joint dorsiflexion (limited joint mobility). Limited joint mobility is frequently encountered in patients with long-standing diabetes. This is most significant in the ankle joint (equinus) and in the forefoot. Less than 50° of dorsiflexion at the first metatarsal phalangeal joint indicates clinically significant limited joint mobility

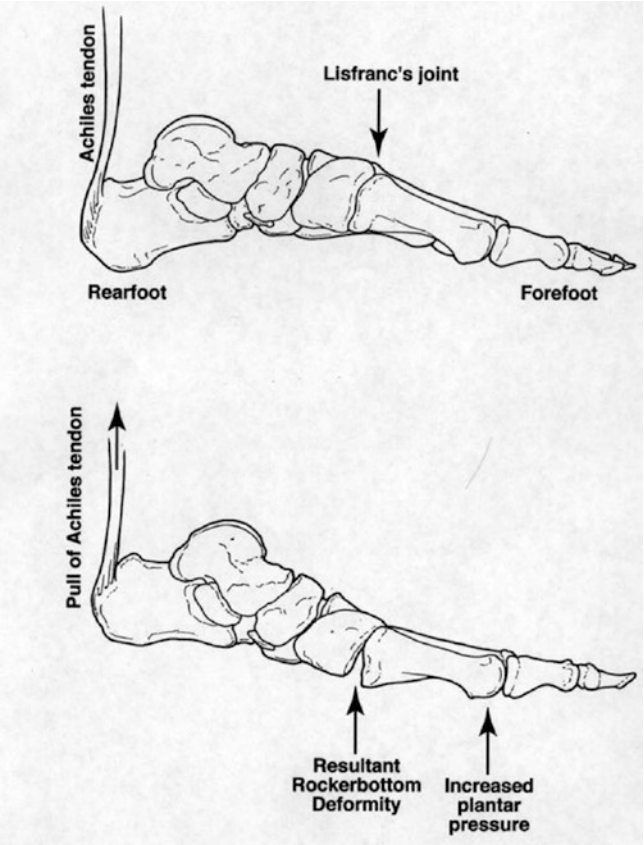

Fig. 2.5 Equinus and its relationship to elevated forefoot plantar pressure. Shortening or loss of natural extensibility of the Achilles tendon may lead to pulling of the foot into plantar flexion. This leads to increased forefoot pressure (increasing risk for plantar ulceration) and, in some patients, may be a component of midfoot collapse and Charcot arthropathy

Additionally, glycosylation may deleteriously affect the resiliency of the Achilles tendon, thereby pulling the foot into equinus and further increasing the risk for both ulceration and Charcot Arthropathy (Fig. 2.5) [58]. In a recent case control study, plantar and dorsal flexion of the feet of 87 patients with diabetes was measured and the incidence of foot ulcers was reported over a follow-up period of 8 years. Diabetes specifically reduced the plantar flexion in the feet and patients with a history of foot ulceration had significantly lower ankle joint mobility [59].

Diabetic Foot Ulcer Classification

Foot ulcers in patients with diabetes are one of the most common precursors to lower extremity amputation. Appropriate care of the diabetic foot ulceration requires a clear, descriptive classification system that can be used to direct therapy, communicate risk, and possibly predict outcome. Speaking a "common language" when communicating risk in the diabetic foot is therefore essential. A classification system, if it is to be clinically useful, should be easy to use, reproducible, and effective to accurately communicate the status of wounds in persons with diabetes mellitus. There are a variety of variables that could be included in such a system, such as faulty wound healing, compliance issues, quality of wound granulation tissue, host immunity, nutritional status, and comorbidities. However, most of these variables are difficult to measure or categorize and can complicate a system. In contrast, three relatively quantifiable factors associated with poor wound healing and amputation include depth of the wound [60, 61], presence of infection, and presence of ischemia [62].

Seven Essential Questions to Ask when Assessing a Diabetic Foot Wound

A classification system has little value if the clinician employing it does not approach each wound in a stepwise consistent, logical fashion. When employing this approach, the first four questions are useful in terms of their descriptive value. The last three questions are most useful for their predictive qualities.

1. Where Is the Ulcer Located?

Location of a wound and its etiology go hand in hand. Generally, wounds on the medial aspect of the foot are caused by constant low-pressure (e.g., tight shoes) whereas wounds on the plantar aspect of the foot are caused by repetitive moderate pressure (e.g., repetitive stress on prominent metatarsal heads during ambulation).

2. How Large Is the Ulcer?

Size of the wound plays a key role in determining duration to wound healing. To simplify wound diameter measurements, one may trace the wound on sterile acetate sheeting and tape this tracing into the chart (Fig. 2.6). The tracing can also be performed on the outer wrapping of an instrument sterilization pack (which would otherwise be discarded). Recently, many centers have begun employing digital photography and computer-driven planimetric wound area calculations. This provides for potentially more consistent, accurate measurements and, ultimately, for comparison of wound healing rates with other centers regionally and beyond. In an evaluation of the reproducibility of wound measurement techniques, Wunderlich and colleagues reported that wound tracing and digital planimetric assessment were by far more reliable than manual measurement of length and width [63].

3. What Does the Base Look Like?

When describing the base of a wound, one may use terms like granular, fibrotic, or necrotic. One may record the presence or absences of any drainage, which may be described as serous or purulent, with a further description of any odor or color.

4. What Do the Margins Look Like?

The margins tell us a lot about the wound. If adequately debrided and off-loaded, they should be well adhered to the surface of the underlying subcuticular structures with a gentle slope toward normal epithelium. However in the inadequately debrided, inadequately off-loaded wound, undermining of the leading edge normally predominates. This is due to the "edge effect" which dictates that an interruption in any matrix (in this case, skin) magnifies both vertical and shear stress on the edges of that interruption. This subsequently causes shearing from the underlying epithelium (making the wound larger by undermining) and increased vertical pressure (making the wound progressively deeper). If appropriately debrided and off-loaded, this effect will be mitigated. Nonetheless, the margins of the wound should be classified as undermining, adherent, macerated, and/or nonviable.

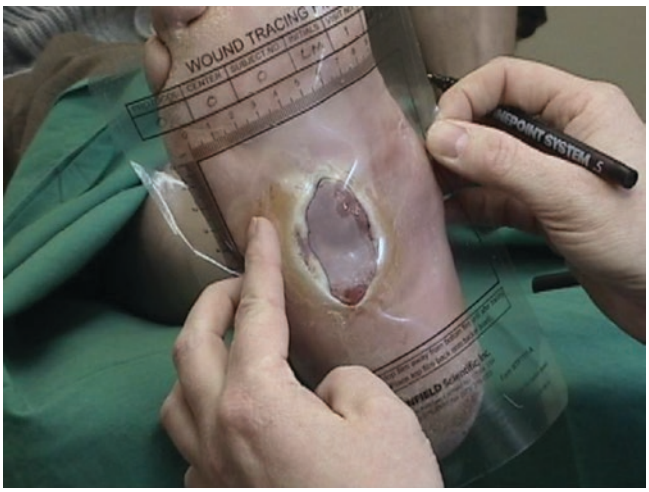

Fig. 2.6 Tracing the wound using sterile acetate sheet. Wound tracing may yield far more reproducible results in measuring wound size than simply length by width measurement

Subsequent to the first questions, which we term "descriptive," come the last three questions which we term "classifiers," These classifiers can then be used to fit a patient into the University of Texas wound classification system (Fig. 2.7). This system has evolved as a significant modification of the Wagner system (Fig. 2.8) to include concomitant depth, infection, and ischemia. While both systems have been shown to be predictive of poor outcomes, the UT system has been shown to be significantly more predictive and complete [64, 65]. Both, however, may be considered useful in a clinical scenario, depending on the preference of the clinician.

5. How Deep Is the Ulceration? Are There Underlying Structures Involved?

These two questions are so closely related that they are combined into one. There is a possible contribution of depth to ulcer healing times [65]. Depth of the wound is the most commonly utilized descriptor in wound classification. Wounds are graded by depth. Grade 0 represents a pre- or post-ulcerative site. Grade 1 ulcers are superficial wounds through the epidermis or epidermis and dermis but do not penetrate to tendon, capsule, or bone. Grade 2 wounds penetrate to tendon or capsule. Grade 3 wounds penetrate to bone or into a joint. We have known for some time that wounds that penetrate to bone are frequently osteomyelitic. Additionally, we have observed that morbid outcomes are intimately associated with progressive wound depth.

		Grade			
		0	1	2	3
Stage	A	**Pre or post-ulcerative lesion completely epithelialized**	**Superficial wound, not involving tendon, capsule, or bone**	**Wound penetrating to tendon or capsule**	**Wound penetrating to bone or joint**
	B	with infection	with infection	with infection	with infection
	C	with ischemia	with ischemia	with ischemia	with ischemia
	D	with infection and ischemia	with infection and ischemia	with infection and ischemia	with infection and ischemia

Fig. 2.7 University of Texas Wound Classification System

Meggit Wagner Grading System

Grade 1: Superficial Diabetic Ulcer
Grade 2: Ulcer Extension

1. Involves ligament, tendon, joint capsule or fascia
2. No abscess or Osteomyelitis

Grade 3: Deep ulcer with abscess or osteomyelitis
Grade 4: Gangrene to portion of forefoot
Grade 5: Extensive gangrene of foot

Fig. 2.8 Meggit Wagner grading system

Depth of the wound and involvement of underlying structures may best be appreciated through the probe-to-bone test (PBTB). The probe-to-bone test is performed by inserting a sterile blunt metallic probe into the wound. Since it was first reported in 1995, there have been varying reports about the accuracy of the PBTB [66, 67].

6. Is There Infection?

The definition of bone and soft tissue infection is not an easy one. Cultures, laboratory values, and subjective symptoms are all helpful. However, the diagnosis of an infection's genesis and resolution has been and continues to be a clinical one. While criteria for infection may be something less than clear-cut, there is little question that presence of infection is a prime cause of lower extremity morbidity and frequently eventuates into wet gangrene and subsequent amputation. Therefore, in an effort to facilitate communication and effect consistent results, the foot care team should agree on criteria for this very important risk factor.

7. Is There Ischemia?

As discussed above, identification of ischemia is of utmost importance when evaluating a wound. Ischemic wounds were found to take longer to heal compared to neuropathic wounds without deformities [68]. If pulses are not palpable, or if a wound is sluggish to heal even in the face of appropriate offloading and local wound care, noninvasive vascular studies are warranted followed by a prompt vascular surgery consultation and possible intervention to improve perfusion.

Wagner Ulcer Classifications

Several diabetic classification systems have been reported in the medical literature. This section aims to chronologically review some of the most commonly described classification systems currently used by a variety of practitioners to stage diabetic foot wounds and to discuss outcomes related to their use. One of the most frequently cited diabetic wound classification systems was first described by Meggitt [69] in 1976 and Wagner [70] in 1981. The system is based mainly on wound depth and consists of six wound grades. These include grade 0 (intact skin), grade 1 ("superficial ulcer"), grade 2 (deep ulcer to tendon, bone or joint), grade 3 (deep ulcer with abscess or osteomyelitis), grade 4 ("forefoot gangrene"), and grade 5 ("whole-foot gangrene"). This classification is outlined in Fig. 2.8.

The classification system contains three key descriptors including depth, infection, and ischemia. However, it does not consistently include these important risk factors in every ulcer grade. Infection is included in only one of the six Wagner ulcer grades, and vascular disease is only included in the last two classification grades. The first three grades are concerned only with depth. It is perhaps for this reason that they are the most commonly used, whereas the last three are largely ignored because of their limited clinical use. The descriptors Meggit and Wagner used for ischemia were forefoot and whole foot gangrene. These represent the most severe form of end-stage disease, and therefore cannot help to guide proactive interventional therapy except frank ablation of the affected site. In addition, because gangrene can be caused by infection, it may not always have a vascular origin. Since there are better diagnostic tools to assess and treat PAD, more robust criteria for ischemia will improve diagnosis, interventions, and amputation prevention.

There are several papers that have attempted to validate the Wagner classification system [71, 72]. Calhoun et al. [72] evaluated wounds that were infected and retrospectively assigned Wagner grades to them. They found that when wounds were treated according to what they considered a healthy standard of care, then success, which they defined as eradication of infection and prevention of readmission for 1 year, was frequently achieved despite wound grade. Van Acker et al. [73] found the Wagner classification to have significant association with the duration of healing of the ulcer. Armstrong et al. [65] suggested that patients with Wagner stages 4 and 5 may be grouped together as the two groups did not have separate prognostic value. In addition these patients are often referred directly to a surgeon for amputation and are rarely seen by the diabetic foot team. The system was adapted to combine medical and surgical elements of therapy to monitor the treatment of diabetic foot infection. Unfortunately, in requiring that wounds be infected as an inclusion criterion, it made assessment of this classification problematic, as Wagner wound grades 0–2 classically have no infection descriptor attached to them. In fact, the only mention of infection in this system occurs in grade 3. It is this fact that causes many to customize this system, such that it often takes on distinctly different regional characteristics. This unfortunately limits its usefulness as a standard diabetic foot classification.

Other Ulcer Classifications

In the 1980s and 1990s many authors including Forrest and Gamborg-Nelson [74], Pecoraro and Reiber [75], Arlt and Protze [76], and Knighton [77] proposed their own wound

classifications; however, these systems have not gained universal acceptance. More recent classification systems that have been proposed include the UT classification modification by Van Acker/Peter [73], the PEDIS system by IWGDF members [78], and the S(AD) SAD system proposed by Macfarlane and Jeffcoate [79, 80]. These systems will require validation to gain universal acceptance.

UT Ulcer Classification

The University of Texas Health Science Center in San Antonio (UT) proposed a classification that included depth, infection, and vascular status in 1996 [65, 81]. The classification integrates a system of wound grade and stage to categorize wounds by severity. It is based around two fundamental questions the clinician asks when assessing a wound: (1) How deep is the wound? and (2) Is the wound infected, ischemic, or both? The classification formulates into a matrix with infection and/or ischemia as the vertical axis and depth as the longitudinal axis. This system is illustrated in Fig. 2.7.

Similar to other wound classification systems, the UT system grades wounds by depth. Grade 0 represents a pre- or post-ulcerative site. Grade 1 ulcers are superficial wounds through either the epidermis or the epidermis and dermis but do not penetrate to tendon, capsule, or bone. Grade 2 wounds penetrate to tendon or capsule but the bone and joints are not involved. Grade 3 wounds penetrate to bone or into a joint. Within each wound grade there are four stages: clean wounds (A), nonischemic infected wounds (B), ischemic wounds (C), and infected ischemic wounds (D).

The Grade 0 Wound: Grade 0 wounds are pre-ulcerative areas or previous ulcer sites that are now completely epithelialized after debridement of hyperkeratosis and nonviable tissue. The diagnosis of a grade 0 wound can be made only after removal of any regional hyperkeratosis, as ulcerations may be hidden by overlying calluses. The grade 0-A wound is then a pre-ulcerative area or a completely epithelialized post-ulcerative area. The grade 0-B wound is a 0-A lesion with associated cellulitis. The grade 0-C wound is a 0-A lesion with concomitant regional signs of ischemia. The grade 0-D wound is a 0-B lesion coupled with a working diagnosis of lower extremity ischemia as defined above.

Although lesions that fall into the grade 0 category do not have a break in the epidermis and may not be classically classified as "wounds," the category is important in the identification of sites that are "at risk" for future ulceration and to monitor and prevent re-ulceration of newly healed wounds. Because there is a very high rate or re-ulceration (28–50%), the grade 0 classification allows physicians to follow the progression of wounds over time from healed to re-ulcerated.

The Grade I Wound: Grade I wounds are superficial in nature. They may be either partial or full thickness skin wounds without the involvement of tendon, capsule, or bone. The Grade I-A wound is therefore superficial partial or full thickness wound. The Grade I-B wound is an infected superficial wound. As with any neuropathic lesion, Grade I- B wounds should be examined very carefully. By definition, the Grade I-B wound implies superficial infection without involvement of underlying structures. If the wound shows signs of significant purulence or fluctuance, further exploration to expose a higher grade infection is in order. The Grade I-C wound is I-A plus vascular compromise and the Grade I-D wound is the infected I-B wound with concomitant ischemia.

The Grade II Wound: Grade II wounds probe deeper than the Grade I wounds. Grade II wounds may involve tendon or joint capsule but not bone. The reason for the distinct delineation between wounds that probe to bone and those without bone or joint involvement is because of the high correlation between probing to bone and osteomyelitis [67]. The II-A wound may therefore probe to tendon or joint capsule, but not bone. The II-B wound is II-A plus infection, again the bone and joint are not involved. The Grade II-C wound is II-A plus ischemia, and the Grade II-D wound correspond to II-B plus ischemia.

The Grade III Wound: A wound that probes to bone is categorized as a grade III wound. The modifiers are then added pending the presence of comorbid factor. The III-A wound probes to bone without local or systemic signs of acute infection. The III-B wound probes to bone with signs of acute infection. The III-C wound is identical to III-A with concomitant ischemia. The III-D wound is characterized by active infection, exposed bone, and vascular insufficiency. The criterion for each of the stages is based on clinical and laboratory data. The working diagnosis of lower extremity ischemia may be based on clinical signs and symptoms such as absence of pedal hair, absent pulses, claudication, rest-pain, atrophic integument, dependent rubor or pallor on elevation plus one or more of the noninvasive criteria (transcutaneous oxygen measurements of <40 mm Hg, ankle-brachial index of <0.80, or absolute toe systolic pressure <45 mm Hg) [83–86].

Clean ulcers may be defined as wounds without local or systemic signs of infection. The clinical diagnosis of infection in persons with diabetes is often difficult and defined by narrow, subtle parameters. Wounds with frank purulence and/or two or more of the following local signs may be classified as "infected": warmth, erythema, lymphangitis, lymphadenopathy, edema, pain, and loss of function. Systemic signs of infection may include fever, chills, nausea, vomiting, or generalized malaise [87]. This clinical diagnosis of infection is often obscured by neuropathy and possibly immunopathy [88, 89]. The diagnosis and subsequent treatment of infection may also be assisted by laboratory studies or positive deep tissue cultures or wound based curettage. When osteomyelitis is suspected, bone biopsy with appropriate pathology and culture studies is still the gold standard for diagnosis [87].

Armstrong et al. validated the predictive value of the UT classification system in 1998 [65] and noted a significant overall trend toward an increased prevalence of amputations as wounds increased in both grade (depth) and stage (comorbidity). Patients whose wounds were both infected and ischemic were noted to be almost 90 times more likely to receive a high level amputation compared with patients in a less advanced wound stage, and patients whose wound probed to the underlying bone were over 11 times as likely to receive a high level amputation. Unfortunately the study was retrospective and was not a multicenter trial. In addition, some degree of bias may have been present since the study was carried out by the center that first described the system and the clinicians using it intimately familiar with the system.

Oyibo et al. [90] compared the Wagner classification system with the UT system in a multicenter prospective longitudinal case-control study of 194 patients. The study suggested that both the UT and the Wagner classification system correlated similarly with clinical outcomes. Both systems associated higher grades with a greater likelihood of an ulcer not healing and a greater chance of limb amputation. The trend for grade of the UT classification system was slightly more robust than the trend for grade of the Wagner classification. The inclusion of comorbid factors such as infection and/or ischemia to grade (depth) when classifying an ulcer with the UT system improves description and adds to the predictive power of a wound classification system, especially for ulcers within the same grade level but at a different stage. Based on this, the UT wound classification showed promise as a more practical system.

In conclusion, it is observed that many of the risk factors for neuropathic ulceration, infection, and subsequent amputation may be identified using simple, inexpensive equipment in a primary care setting. A consistent, thoughtful assessment of the diabetic foot is pivotal to identify high-risk patients. Subsequent to the gathering of clinical data through sequential assessment, appropriate classification of the wound becomes paramount in our efforts to document and communicate the level of risk to all members of the health care team caring for the person with diabetes. These simple approaches should improve communication and facilitate amputation prevention.

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