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Diabetic Neuropathy: Pathophysiology and Prevention of Foot Ulcers

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Outline

Abstract

Diabetic neuropathy, which affects 60% to 70% of those with diabetes mellitus, is one of the most troubling complications for persons with diabetes, often leading to foot ulcers and potentially to lower limb amputations, both of which are preventable. The physiologic, structural, and functional changes associated with diabetic neuropathy and foot ulcers are discussed. Advanced practice nurses are in a unique position to implement strategies for the prevention of serious and debilitating complications from diabetic neuropathy, including foot assessment, education, and specialist referrals. Research evidence is given to support the use of the Semmes-Weinstein monofilaments to evaluate

decreased
plantar sensation, a common precursor to ulceration. Ongoing patient and family
education can emphasize the importance of preventive self-care measures.
Referrals for specialist care and therapeutic footwear can be made by advanced
practice nurses. If begun early, these interventions can prevent foot ulcers
from diabetic neuropathy, thereby improving the quality of life and reducing
healthcare costs for this chronic disease.

This article has been designated for CE credit. A closed-book, multiple-choice
examination follows this article, which tests your knowledge of the following
objectives:

1. Discuss the pathophysiology associated with diabetic neuropathy.
2. Properly evaluate patients at risk for developing foot ulcers.
3. Outline the elements of proper foot care and strategies to prevent potential
complications.

An estimated 16 million people in the United States have diabetes, and half do
not know they have the disease. Type 1 diabetes is an insulin-dependent diabetes
that occurs in about 5% to 10% of those with diabetes. Considered an autoimmune
disease, it occurs equally among males and females but is more common in whites
than in nonwhites. Type 2 diabetes is non-insulin-dependent diabetes and
accounts for 90% to 95% of persons with diabetes. Risk factors for type 2
diabetes include a family history of diabetes, age >40, overweight, and African
American, Hispanic, or Native American race. Older, overweight women are
especially at risk.1

Diabetic neuropathy (DN), nerve damage that affects the sensory,

motor, and
autonomic nervous systems, occurs in 60% to 70% of people with diabetes 1 and is
one of the most troubling complications. It often leads to foot ulcers and may
ultimately lead to lower limb amputations. The consequences of DN on the
physical, social, and emotional quality of life for patients and their families
can be enormous. Limited mobility, limited independence, inability to work, and
loss of income are only a few of the areas negatively affected by the development
of foot ulcers. Ulcers are highly preventable; therefore, prevention and early
detection of DN and its sequelae are critical to reducing such morbidity.

The economic impact of complications from DN is considerable. In 1992, about 40%
($37 billion) of the total estimated cost of diabetes was spent on hospital
care, with nearly $10 billion related to chronic complications such as DN.1 The
cost of care is increasingly important in the changing healthcare environment.
Focusing on prevention and identification of individuals at greatest risk for
foot ulcers would allow the most effective allocation of increasingly limited
healthcare resources. As insurance companies restrict the use of physician
specialists, advanced practice nurses (APNs), especially those with a generalist
practice, may care for increasing numbers of diabetic patients. The recent
enactment of direct Medicare reimbursement for APN services provided in urban
areas also may contribute to expanded practices. Thus, APNs have increased
opportunities to improve the prevention and early detection of foot ulcers.

Extensive research has been conducted to better understand the pathologic basis of DN and its impact on the development of diabetic foot ulcers and the subsequent need for lower limb amputation. Researchers have focused on sensory, motor, and autonomic changes and their interrelationships, as well as the effects of external precipitating factors such as improper footwear, all of which bring about the underlying problems of DN that contribute to subsequent development of foot ulcers: increased foot pressures and/or traumatic injury in a foot with reduced or absent sensation. In addition, recent research on the role of hyperglycemia in DN has resulted in recommendations for tighter glycemic control. Although ischemic peripheral vascular disease also contributes to the development of foot ulcers in persons with diabetes, a thorough discussion of peripheral vascular disease and its relation to peripheral DN is beyond the scope of this paper.

This article examines peripheral DN and the development of foot ulcers. A discussion of the pathophysiologic basis of the sensory, motor, and autonomic systems will be integrated with some of the research that has examined extrinsic factors that contribute to the development of diabetic foot ulcers. In addition, the role of the APN is discussed in relation to three areas critical to prevention of diabetic foot ulcers: foot assessment, patient and family education, and referrals for APN diabetes specialists, podiatrist care, and therapeutic footwear.

PATHOLOGIC BASIS

The pathologic basis of DN is a complex interrelationship of the sensory, motor, and autonomic systems. The key elements in this process are summarized in Figure

1.
Sensorimotor System

The peripheral nervous system consists of sensory and motor nerves. Sensory nerves transmit nerve impulses to the spinal cord and brain and detect changes in temperature, light touch, vibration, and pain. Motor nerves transmit impulses from the brain and spinal cord to the muscles and are responsible for muscle strength, movement, and tone. Peripheral nerves contain a series of connective sheaths that enclose nerve axon fibers. Within these sheaths are Schwann cells, which produce the myelin sheath surrounding the axons of the nerve. Myelin sheaths serve a protective function that is disrupted by hyperglycemia. This disruption leads to a segmental demyelinization process accompanied by a slowing of motor nerve conduction and an impairment of sensory perception.2-4 The effects of sensory neuropathy involve a symmetric, bilateral pattern in the lower limbs that may begin with tingling, burning, and pain that progresses to numbness and eventual loss of sensation.2 Such loss of sensation in the foot can lead to trauma from mechanical, chemical, or thermal injury; further, this trauma can go undetected because of the impaired or absent sensory perception. This painless, undetected trauma leads to inflammation and further mechanical injury and can result in foot ulceration and infection that could lead to amputation.5

In addition to the sensory loss associated with DN, several changes in motor function can also occur; they are referred to as intrinsic motor factors. Because motor nerves control muscle strength, decreased motor nerve function can result in atrophy of the small muscles in the foot. This in turn leads to an imbalance of flexor and extensor muscles in the toes, resulting in clawing or curling under of the toes. Clawing, a typical foot posture in peripheral neuropathy, shifts the foot position so that the toes bear less weight and the metatarsal heads become more prominent. This shift in foot position increases weight bearing on the metatarsal heads. Subsequent pressure on these more prominent metatarsal heads often is manifested by an altered gait. As this pathologic process continues, the soft tissue that covers these bones is exposed to repeated shearing stress (ie, horizontal movement) as the tissue rubs against footwear, especially during walking. Continuous exposure to shearing stress causes the skin to react by increasing keratin production, thus forming a thicker epidermis, or callus, that can become very thick if not removed. At the site of callus formation, foot pressures become elevated.

Although callus formation is initially a protective response, as time progresses a thin layer of tissue develops between the callus and the affected metatarsal head(s). This tissue, caught between these two forces, is subjected to additional shearing stress that may lead to inflammation, bleeding, tissue destruction, and eventual ulcer formation.5-9

All these changes can contribute to limited joint mobility, and can also be worsened by such joint limitations. Some suggest that preserving ankle range of motion, because of its vital role in walking, is particularly important. Ankle joint restrictions lead to poor foot adaptation, poor shock absorption, increased weight-bearing load, and ultimately abnormally high foot pressures that can progress to ulcer formation.[8,10] Thus, loss of sensation combined with musculoskeletal changes in the foot can bring about abnormalities in a patient's gait and elevated foot pressures that can result in ulceration.

These intrinsic factors, occurring in the insensate foot, are aggravated by extrinsic motor factors that further increase the risk of foot ulceration. Extrinsic factors include improper footwear, mechanical trauma such as stubbing the foot or careless cutting of nails, thermal injury from burns, and decubitus heel ulcers from immobilization.[6,8,10-13] In the normal foot, discomfort from these extrinsic factors would cause a change in gait and use of a different part of the foot to bear the stress. This allows the foot to remain intact, as compensatory limping attempts to shift weight off the area of discomfort. However, in the foot with impaired sensation, the patient's inability to feel trauma contributes to increased susceptibility to further injury. Even minor foot trauma can progress to ulceration and infection.[6]

Autonomic System

The autonomic nervous system directly influences peripheral circulation in the extremities by supplying sympathetic adrenergic fibers that regulate arteriole vasomotor tone and blood flow through arteriovenous shunts. Arteriovenous shunts directly connect an arteriole and a venule, bypassing the capillary bed where the exchange of gases, nutrients, metabolites, and heat occurs. Failure of sympathetic control results in arteriolar vasodilatation, causing a sequence of events that includes reduction in peripheral resistance, an increase in arteriovenous shunt flow, an increase in cutaneous blood flow, and ultimately increased foot temperatures.[14-17] Thus, the diabetic foot is warm to the touch secondary to the increased arteriovenous blood flow. This can create a false sense of security. If a patient has a warm foot, he or she may assume that circulation is intact and that the risk of injury is minimal, but actually the contrary is true: the warm insensitive foot can easily result in a truly painless foot ulceration.

An additional consequence of increased arteriovenous shunting is the development of distended dorsal veins in the foot. The increased blood that is bypassing the capillary bed via the increased arteriovenous shunting may result in increased capillary pressure, which can produce a fluid shift from the vascular to the interstitial spaces, thus causing neuropathic edema in the foot.[15,16] This edema can further increase foot pressures and can contribute to the formation of foot ulcers.

One final mechanism that complicates this process is diabetic anhidrosis, a sudomotor dysfunction that leads to distal, decreased sweating that results in dry, scaly, cracked skin in the feet.[18] Almost all persons with peripheral neuropathy experience diabetic anhidrosis,[16,19,20] which predisposes them to a foot infection and/or ulceration.[16]

Thus, the sensory, motor, and autonomic systems contribute to the development of neuropathy and the potential development of foot ulcers.

Glycemic Control

In addition to the effects of the sensorimotor and autonomic systems, current research has focused on the role of hyperglycemia in the pathogenesis of DN and foot ulcers. The Diabetes Control and Complications Trial was a large, longitudinal, multisite, randomized clinical trial that compared intensive diabetes control to conventional treatment in persons with type 1 diabetes. The intensive group received three or more daily insulin injections or used an insulin pump to maintain blood glucose levels as close to normal as possible. Those in the conventional group received one or two daily insulin injections. In those who entered the study with no evidence of neuropathy, tight glycemic control reduced the risk of developing neuropathy by 69%. In those who had evidence of neuropathy when they entered the study, there was a 57% reduction in the rate of progression of chronic neuropathic complications with tight control.[21,22] These dramatic improvements in the prevention and progression of DN with tighter glycemic control have led to the recommendation for more aggressive management of hyperglycemia in those with type 1 diabetes. In addition, the researchers suggest that these findings can be applied to those with type 2 diabetes, although they caution that diet should remain the primary treatment modality in this group.

Although research has identified many of the mechanisms that contribute to DN and foot ulcers, the pathogenesis is complex and a great deal remains to be learned. Meanwhile, APNs are in a unique position to implement strategies in the prevention of serious and debilitating complications from DN. There are three critical areas on which APNs can focus when caring for patients with diabetes: thorough and frequent assessment of the feet, patient and family education, and referrals for specialist care and therapeutic footwear.

FOOT ULCER PREVENTION

Assessment of the Feet

All patients with diabetes should have a thorough annual foot assessment that includes the condition of the skin and nails and the presence of any foot deformities. Table 1 presents an overview of foot assessment for diabetics. It is particularly important to identify those who have a high risk of developing foot ulcers. High-risk individuals include older persons with diabetes, those with a longer duration of diabetes, and those with foot deformities such as Charcot's joint or hallux (great toe) deformity. Charcot's joint is a chronic, progressive, degenerative disease of one or more joints identified by swelling, bleeding, heat, bone changes, and joint instability that can result from DN. High-risk individuals also include those with a previous history of lower extremity ulcer or amputation and those with decreased sensory perception. Two types of sensory perception have been examined as predictors of risk for foot ulcers: vibratory and pressure sensations of the feet. Although helpful in evaluating high-risk individuals, decreases in vibratory sensation occur later in the course of DN than changes in pressure sensation. Therefore, standardized evaluation of cutaneous pressure sensation of the feet is of primary importance in the identification of individuals at risk for foot ulcers.

As discussed earlier, patients with diabetes who have sensory neuropathy typically show a gradual loss of sensation on the plantar surface of the foot. Eventually they reach a threshold of insensitivity that, when combined with elevated foot pressures and/or trauma, puts them at risk for the development of plantar ulcers.

Methods for evaluating pressure sensation measure either the actual pressure or the cutaneous sensation perception at specific points on the foot. Measures of actual foot pressures are used primarily in research studies.23 Clinical use is limited because of their expense, time required, and the technical and physiologic sophistication necessary to use and maintain some of the equipment and to interpret the results. In contrast, measures of pressure sensation are easy and quick to perform, reliable, extremely sensitive, and inexpensive.12,24-27

The Semmes-Weinstein (SW) monofilament test is an effective method that can be used to evaluate patients at risk for foot ulceration due to a sensory deficit. The SW monofilaments-calibrated nylon monofilaments attached to a handle-are used to measure a patient's ability to feel a point of pressure at specific sites on the plantar surface of the foot (Fig. 2). These monofilaments generate a reproducible buckling pressure and are identified by manufacturer-assigned sizes from 1.65 to 6.65. The higher the number of the monofilament, the stiffer and more difficult it is to bend, and therefore the more force one must apply to bend the monofilament for the patient to feel the pressure. The monofilaments most frequently used to screen for sensory neuropathy are 4.17, 5.07, and 6.10. Forces required to bend these monofilaments are 1, 10, and 75 g of force, respectively. The lighter the force the patient can identify (ie, the smaller the monofilament number), the more intact his or her sensation and the less risk for foot ulceration.

Sites normally checked are over the metatarsal heads and toes, the medial and lateral midfoot, and the heel (Fig. 3). The monofilament is applied perpendicular to the skin with enough force to cause the monofilament to buckle for approximately 1 second. The monofilament must be lifted away from the site rather than dragged across the skin. It may be helpful to let the patient experience what the monofilament will feel like by applying it to the palm first. The patient then is asked to close the eyes to eliminate visual cues and to respond "yes" each time he or she feels a monofilament. The APN presses the monofilament to the skin so that it buckles one of two times and asks the patient to identify which time he or she was touched, the first time or the second. It may be helpful to check each site more than once, especially to establish a baseline for a new patient or to verify changes from previous evaluations. If repeated testing is done, sensation is considered intact if the patient correctly identifies the touch two of the three times. The sites should be tested in random order so that patients do not know which site is being assessed at any given time.1

Research has determined the monofilament size that is most predictive of protective sensation. Individuals with normal feet are able to detect the 4.17 monofilament. The 5.07 monofilament is used to identify those who are more likely to develop foot injuries. Persons who can feel the 5.07 monofilament have protective sensation. However, failure to detect sensation in any tested area with the 5.07 monofilament indicates that the patient lacks protective sensation and thus is more likely to develop neuropathic foot injuries.12,26

Risk categories that are helpful in predicting the likelihood of neuropathic ulceration are linked to recommendations for frequency of follow-up

foot care
and type of footwear adaptations 27-30 (Table 2).

Risk category 0 identifies those with intact sensation of the feet. No other
risk factors, such as severe foot deformities, absent pedal pulses, or a history
of foot ulcer or amputation, are present. Education regarding proper foot care
and well-fitting shoes, as well as the importance of maintaining consistent
control of their diabetes, should be reinforced with each visit. Patients should
be made aware that their risk level may change and should be informed about the
problems they should share with their healthcare providers. Annual 1 or biannual
30 assessments should be made.

Those in risk category 1 have lost protective sensation. They are considered to
be at high risk, and "high-risk" chart stickers should be used.1 White 30
advocates the addition of soft insoles that minimize shear and vertical stress.
Biannual foot examinations by a healthcare professional are recommended.

Risk category 2 identifies those who, in addition to loss of protective
sensation, have a foot deformity, but do not have a history of a plantar ulcer.
They should be fitted for custom-molded insoles, multilayered insoles made from
a mold of the plantar surface of the feet. Professional foot assessments should
be made every 3 to 4 months.

The highest-risk category, category 3, adds a history of ulcer or amputation to
the criteria in categories 0 through 2. Custom-molded shoes are necessary to
prevent further problems in these individuals. In addition, foot and footwear
assessments are recommended every 3 months 1 or every 1 to 2

months.[29,30]

Ongoing evaluation of patients with diabetes at risk for ulcerations from DN is an integral part of working with diabetic patients. Use of the SW monofilaments to assess sensory perception must be a routine and consistent part of foot assessment when caring for individuals with diabetes. Office, clinic, and hospital personnel, as well as patients themselves, should be instructed to remove the patient's shoes and socks; failure to remove footwear has been identified as a major barrier to foot assessment.[31] Adding the evaluation of sensory perception with SW monofilaments to a thorough foot assessment at each patient visit can more accurately identify patients at risk and lead to improved preventive intervention, thereby reducing the need for lower limb amputation.

Education

Education is a crucial component in the prevention of foot problems in patients with diabetes, and APNs are in an ideal position to assist these patients in learning important self-care practices. Research studies evaluating patients' knowledge regarding diabetes have demonstrated extensive deficits. Masson et al.[32] assessed the extent to which patients with diabetes who had new foot ulcers were aware of risk factors and preventive care for foot ulceration. Only 29% of patients with an ulcer and 59% of patients without an ulcer considered themselves at risk for foot ulceration. Farrant et al.[33] found that >25% of patients with type 1 diabetes and >50% of those with type 2 diabetes did not realize that loss of sensation in the feet was a complication of diabetes.

Other research, however, has demonstrated the benefits of intensive patient education in reducing the incidence of ulceration.[34,35] Valente and Nelson [36] suggest combining patient education with individualized foot screenings. Talking about foot care can be more personalized if each principle is addressed during an examination of the patient's feet. If patients can see how each point is related to their own feet specifically, they become more aware of their personal risk factors and may more easily incorporate preventive behaviors and foot assessments into their daily routine. Interventions to protect and cushion the feet are important for all persons with diabetes, not just those with DN. Table 3 presents basic guidelines for foot care and recommendations to emphasize when purchasing new shoes.

Research also supports the benefits of diabetes education for healthcare providers. Aguila et al.[37] demonstrated that physicians who were aware of risk factors such as a prior history of foot ulcers were more likely to prescribe preventive foot care behaviors. In their evaluation of an intervention designed to improve patients' foot care practices, Litzelman et al.[35] identified an unexpected benefit: the thoroughness of foot care given by healthcare providers was improved as well. They also discussed the benefits of systems interventions such as flagging charts to remind providers to assess the feet and mailing postcards to remind patients of appointments. In addition, their research supported the effectiveness of developing contracts with patients and suggested that the more detailed and specific the foot care behaviors, the more improvement there was in patient self-care. General principles such as "avoid injury" were not as helpful as specific instructions such as, "do not go barefoot inside or outside."

Healthcare providers and patients and their families should receive ongoing education in the management of diabetes risk factors and complications. Education for APNs may be particularly important for those whose practice includes patients with a variety of medical diagnoses, not just diabetic patients. Care by a diabetes APN or physician specialist may not be as widely covered by managed care insurance plans, and APNs may see increasing numbers of patients with diabetes in their practices. Harley 38 described components of a comprehensive physical examination for evaluating patients with diabetes that may be helpful to APNs who are adding patients with diabetes to their caseloads. Healthcare providers must work with patients to ensure they are receiving the information and learning the skills and proper treatment techniques that are critical to the successful prevention and management of long-term complications from diabetes.37

Referral

In addition to rigorous foot assessment, evaluation of sensory perception, and education, the generalist APN must be able to identify when appropriate referrals are warranted. Referrals to an APN diabetes specialist, a podiatrist, and a pedorthist are an integral part of preventing foot ulcers in high-risk patients. In their nursemanaged foot care clinic, Kelechi and Lukacs 39 have developed a comprehensive lower extremity assessment form that encompasses screening and clinical measures useful in identifying high-risk

individuals.
Evaluation and treatment by the diabetes APN or podiatrist are especially important when patients require care of their nails, calluses, fissures, infections, or ulcers.

If it is determined that the patient would benefit from therapeutic footwear, referral to a certified pedorthist is warranted. A pedorthist fills a written prescription to provide specialized insoles, external shoe modifications, or customized shoes. Properly fitted therapeutic footwear can relieve high foot pressures, reduce shock, reduce horizontal shear from foot shifting within the shoe, and accommodate foot deformities. Typically, a cushioned insole is the initial adjustment and can be recommended before DN occurs. Commercial insoles purchased over the counter are appropriate at this time. Additional changes would require a prescription and include four basic types of alterations:

* In-depth shoes can add up to 3/8" of depth throughout the entire shoe to accommodate foot deformities.
* This added depth also could accommodate a total contact insert, a custom-made insole developed from a model of the patient's feet.
* External shoe modifications are a third type of alteration. These modifications are numerous and include variations in heel height and heel support. Configuration of the shoe soles with some type of rocker potential to minimize the movement of inflamed and painful joints, and extensions to the sides or back of the shoes for stabilization are additional external shoe modifications.
* The fourth type of alteration is less common and is indicated in cases of extensive deformities such as partial foot amputations. These patients require custom-made shoes developed from a cast of their feet.

Any combination of these alterations is possible, depending on the characteristics of each foot and the therapeutic goals to be achieved. This most often is a collaborative process involving the diabetes APN, the podiatrist, the pedorthist, and the patient.40

Since 1993, Medicare Part B has included shoe benefits for special footwear when persons with diabetes are certified by their healthcare provider. Patients are permitted one of two groups of therapeutic footwear adaptations each calendar year: one pair of depth-inlay shoes and three pairs of inserts, or one pair of custommolded shoes with inserts and two additional pairs of inserts. Financial coverage is 80% of the Medicare-allowed payment.

The benefits of properly fitted therapeutic footwear in preventing foot ulcers are well supported by clinical research. Uccioli et al.41 found that the patients wearing protective footwear had 50% fewer ulcer relapses compared to the control group. Chantelau and Haage 42 examined duration of wearing time as a factor in recurrence of neuropathic foot ulcers. After 3 years of follow-up, those who wore protective footwear >60% of the day had 50% less ulcer relapses than those who wore protective footwear

Studies of protective footwear have identified lack of intervention by healthcare providers and lack of adherence by patients as two major barriers to adequate prevention of foot problems. Masson et al.32 found that 78% of patients with foot ulcers and 83% of patients with no history of ulcers had never had their feet measured for regular or therapeutic footwear. Of the 22% in the ulcer group who had been measured for footwear, the most recent measurement was 5 years earlier. One reason noted for limited adherence is that the shoes are not fashionable and are not accepted, particularly by women.41,43 However, the recent fashion shift in women's shoes away from pointed-toe, high-heeled footwear to shoes with wider toe boxes and thicker, more stable heels may help increase use of more appropriate footwear.

CONCLUSION

Peripheral DN affects more than half of patients with diabetes, and foot ulceration is a preventable complication in those with peripheral neuropathy as well as those without neuropathy. Better understanding of the pathophysiologic basis of foot ulcers can assist APNs in providing thorough, regular foot assessments with sensory evaluations, patient and family education, and appropriate referrals to podiatrists and pedorthists. If begun early, these interventions for health promotion and disease prevention can help practitioners and their patients identify early signs and symptoms of diabetic foot neuropathy before extensive complications occur, thereby improving the quality of life for those with diabetes and reducing healthcare costs for this chronic disease.

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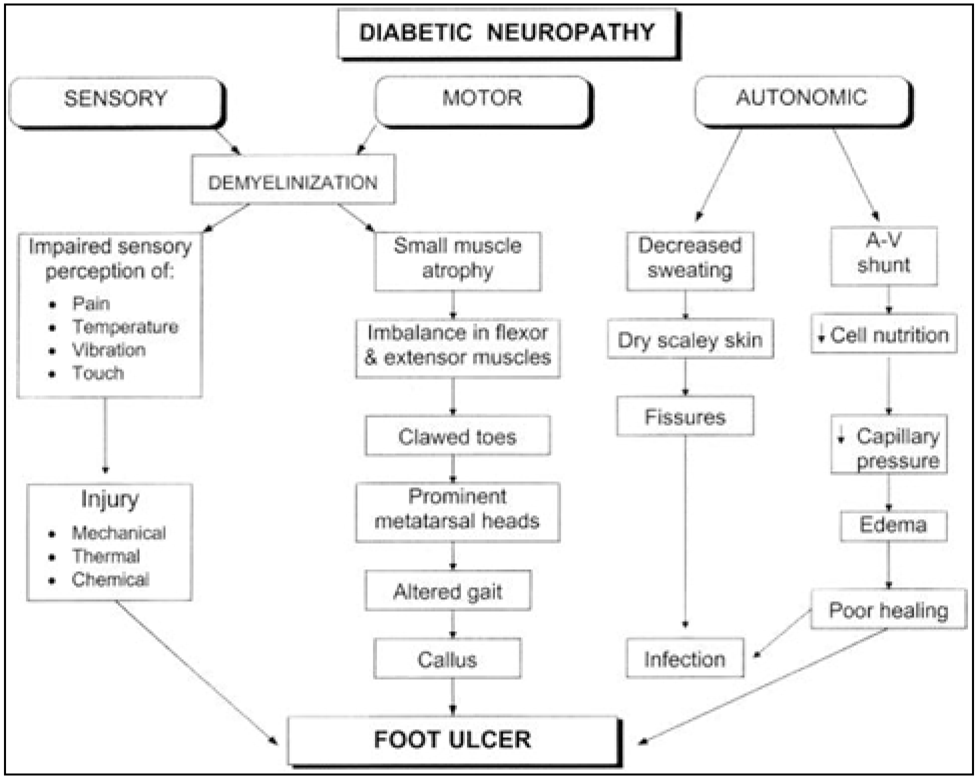
Key Words: diabetic neuropathy; foot care; prevention
Section Description

Editor's Note: In 1998, we will publish six articles for which 1-3 credit hours may be earned as part of a CNS's learning activities. Exam questions are provided at the end of this article for your consideration. See the answer/enrollment form following the article for additional information regarding the program.

Figure 1



Diabetic Neuropathy: Pathophysiology and Prevention of Foot Ulcers.
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Figure 1 . Pathophysiology of foot ulcers from diabetic neuropathy (adapted from references 5 and 23).

TABLE 1

Skin	Nails	Deformities
Color	Thickening	Clawed toes
Temperature	Discoloration	Muscle wasting
Absence of sweating	Ingrown	Charcot's
Dry	Split, broken	Gait disturbances
Scaly	Mycotic	Absence of fat pad over metatarsals
Fissures	Build up of debris under nail	Hallux deformity
Macerations	Deformed	
Calluses		
Bunions		
Corns		
Lesions		

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TABLE 1 FOOT ASSESSMENT

Figure 2

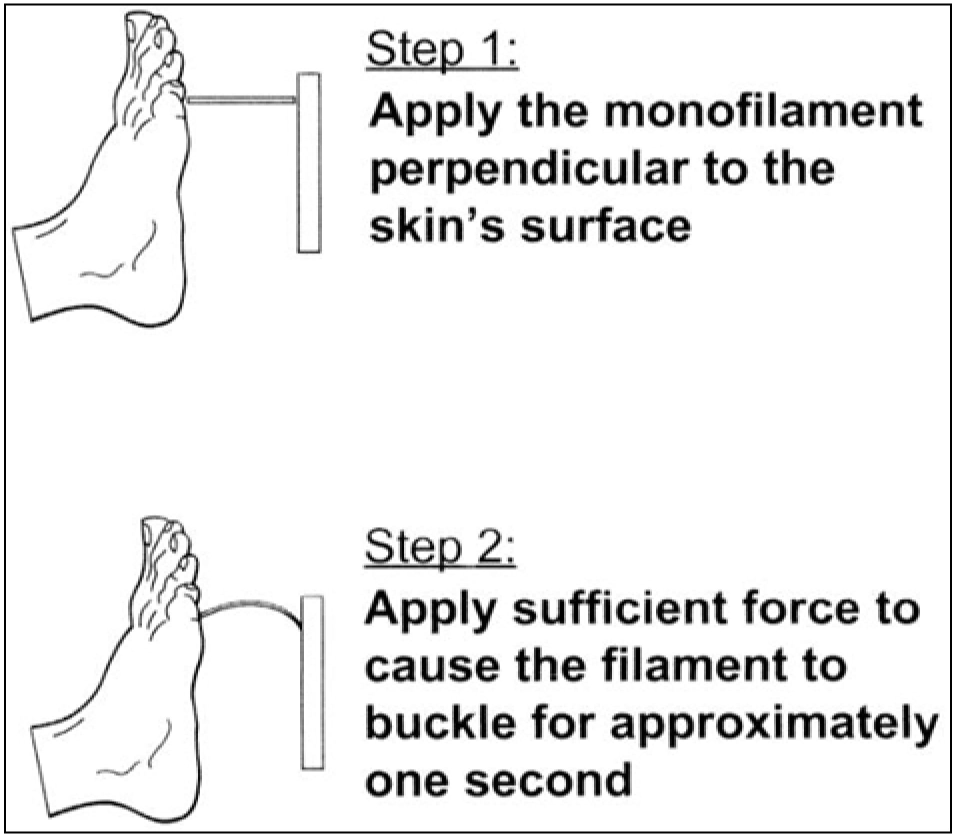

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Figure 2 . Proper use of the Semmes-Weinstein monofilament.

Figure 3

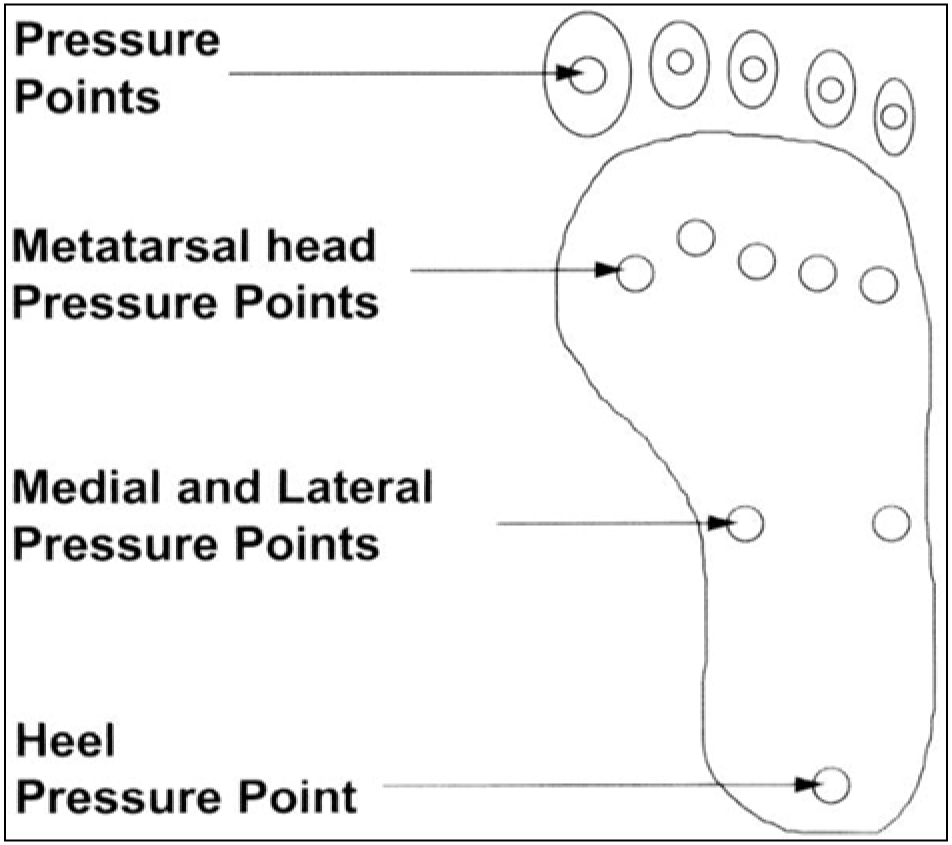

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Figure 3 . Pressure points on the plantar surface of the foot.

TABLE 2

Foot risk	SW 5.07 sensory perception	Foot deformity	Previous ulcer or amputation	Foot and shoe assessments	Insole adaptations	Shoe adaptations
0	Present	No	No	Every 6–12 months	Optional	Over the counter
1	Absent	No	No	Every 6 months	Shear reducing	Over the counter
2	Absent	Yes	No	Every 3–4 months	Custom molded, multiple density	Depth or custom molded inlay
3	Absent	Yes	Yes	Every 1–2 months	Custom molded, multiple density	Custom molded

Adapted from: White, 1994[30] (primary), and Rith-Najarian, Stolusky, Gohdes, 1992;[27] Collier, Kinion, Broadbeck, 1996;[28] Gillis W. Long Hansen's Disease Center, 1993.[29]

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TABLE 2 FOOT RISK CATEGORIES, ASSESSMENT FREQUENCY, AND FOOTWEAR ADAPTATIONS

TABLE 3

General care and hygiene	Shoes	Hosiery
Never walk barefoot on hot sand, stones, asphalt pavements, or in the house. Avoid extremes in temperature. Check temperature of bath water before entering. Protect feet against sun burn. Never use hot water bottles or heating pads on feet. Never soak feet in hot or cold water. Never warm bare feet in front of a fireplace. Wear socks in bed if feet feel cold. Wash your feet daily. Inspect your feet daily and use a mirror to see the soles of your feet. Spread toes. Examine each interspace for evidence of corns, blisters, or sores. If toes are curled under, try to straighten and look at the underside with a mirror. Keep web spaces of feet dry at all times. Have a professional foot exam each year. Do not treat corns, calluses, or ingrown toenails with over-the-counter remedies. Use lanolin (not alcohol)-based lotions on feet daily. Do not apply lotion in-between the toes. Cut toenails straight across with clippers, not scissors or razor blades; not too short. Use a beveled orange wood stick to remove debris from around and under the nail beds. Smooth corns and calluses gently with a pumice stone.	Buy comfortable well-fitted shoes. Buy shoes made of calfskin or leather. Have both feet measured each time you buy shoes. When buying shoes, wear socks and stand up when feet are being measured. Buy shoes with laces. These cover more of the foot, distribute pressure around the sides and top, and allow adjustment for swelling. Avoid slip-on shoes, shoes with pointed toes, and sandals, especially sandals with thongs between the toes. Wear shoes with a wide toe box, such as sneakers or running shoes with laces. Do not wear shoes with heels higher than 1 inch as they increase pressure on the metatarsal heads. Buy new shoes late in the day. Feet enlarge during the day, and shoes that fit in the morning may be too tight in the afternoon. Remove new shoes 1 hour after wearing. Break in new shoes gradually, adding 1 hour wearing time each day. Do not wear any shoes longer than 6 hours without removing. Each pair of shoes fits differently so pressure will be distributed differently. Alternate shoes daily to allow perspiration in shoes to evaporate. Check shoes before wearing for small stones or puckered, bunched up soles.	Wear socks that are 100% cotton, or blends. If wool socks are worn, check for irritation or bunching of sock in shoe. Do not wear mended socks or stockings. Do not wear socks or stockings with seams. Be sure socks are not wrinkled in shoe. Do not wear socks with holes in them. Put on a pair of clean socks daily. Do not wear hosiery that constricts circulation. Wear knee-high hosiery that has a wide band.

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TABLE 3 GUIDELINES FOR FOOT CARE, SHOE SELECTION, AND HOSIERY