

Diabetes & Your Practice

The Clinical Challenge of Diabetes Related Neuropathy

It's important to understand the complex inter-relationships between the body's different systems.

Objectives

After reading this continuing education article, the podiatric physician should be able to do the following:

- 1) Identify the various clinical presentations of the diabetic neuropathic patient.
- 2) Be able to describe the various skin problems that occur in the neuropathic patient.
- 3) Be able to describe the various types of pain that the neuropathic patient presents with.
- 4) Describe the various types of neuropathy and how they relate to the patient clinically.
- 5) Be familiar with the types of muscle weaknesses and gait imbalances that are caused by diabetic neuropathy.
- 6) Identify the etiology of the various bone and joint abnormalities that are part of diabetic neuropathy.
- 7) Identify the causal relationship between callus buildup and ulcer formation.
- 8) Be familiar with psychological neuropathy in the diabetic patient.

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An answer sheet and full set of instructions are provided on pages 176-178.—**Editor**

By **Kenneth B. Rehm, DPM, and Lenny Horwitz, DPM**

Foot problems remain among the most common presenting complaints to physicians' offices and

the most common cause of hospital admissions among diabetic patients in this country. Neuropathy is a major contributing factor, especially when accompanied by peripheral vascular disease and abnormal stresses. It

is estimated that about 25% of diabetic patients have some form of neuropathy on clinical assessment. The clinical features were first described about 200 years ago; and most comar

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monly diabetic neuropathic patients present with a mosaic of complaints that include the following:

Skin problems and ulcerations

Pain and/or numbness

Muscle weakness and gait problems

Bone and joint abnormalities

Psychological problems dealing with their neuropathic situation

The following discussion will focus on these most common problems.(Figure 11)

It is likely that one or more of



Figure 1 Notice decrease in circulation to the toe from low pressure influences



Figure 2 severe dry skin typical of autonomic neuropathy

the above will be the overriding problem for the patient. They all may co-exist, inter-relate and cause other associated problems. Not uncommonly, a patient will present with severe pain, burning and/or a feeling of numbness and stiffness, "like a tight stocking" or "like a brick" in the feet, while at the same time, may or may not exhibit a true loss of sensation. This loss of sensitivity can contribute to excess callus formation under pressure points and skin breakdown that results from abnormal pressures on the feet, (Figure 1) along with other contributing factors of which the patient may be unaware, and that may potentially lead to infection, gangrene and amputation. These complaints may be accompanied by muscle weakness, bone and joint problems, disfigurement and indeed, the psychological effects that all these have on the individual.

The etiologies of these complaints are not black and white; they are multi-faceted and they interplay with the other elements of diabetic pathology, but each will be discussed individually as it relates to the different types of neuropathy.

I. Problems with Skin

In the majority of cases, the cause of skin problems is multi-factorial.

A diabetic patient may present with dryness of the skin, cracks, fissures, increase in callus buildup and ulcerations. (Figure 2) A substantial portion of these skin problems re-

lates to autonomic neuropathy. Two consequences of autonomic neuropathy in the diabetic foot are sudomotor dysfunction and changes in vascular flow within the skin of the sole of the foot. Dry, cracked or fissured skin is a common problem in people with diabetes because of two reasons: lack of skin lubrication and alteration of the blood flow in the microscopic vessels of the skin.

Decreased subtalar joint mobility has been associated with elevated plantar pressures that lead to callus and ulcerations.

Skin lubrication is a function of healthy oil and sweat secretion by the sebaceous glands. In the presence of autonomic neuropathy, these sweat glands atrophy.

The other contributing factor to dry skin on the feet is that the AV shunts, located on the soles of the feet, but not the dorsum, are inappropriately dilated. Normally the stimulation of sympathetic nerves to the AV shunts keep them tightly shut and the blood flows to the skin surface through nutrient capillaries. Normally used by the body to aid in resisting the effects of cold weather, these channels can open up and redirect the blood flow away from the surface of the skin back toward the central core of the body. These AV shunts are located in the ear lobes, the fingers, the tip of the nose and the soles of the feet; therefore, these are the areas where frostbite occurs first. When the nervous input to these channels is damaged from autonomic neuropathy, the shunts are not kept consistently shut because they dilate. This allows the blood to bypass the surface of the skin, creating a possible overload on the venous system, causing pooling of blood in the feet. This shows up as pedal edema, further reducing arterial flow to the feet.(Figure 5) This

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reduction in blood flow causes the feet to be cold and damages the integrity of the skin. This, in combination with poor lubrication, causes the skin of the feet to become dry, cracked, fissured and hard. The skin cannot function to protect the feet from injury or invasion of bacteria, putting them at risk for damaging cuts, sores, lesions, ulcers, infections and even amputation.

Callus Formation

One of the most common presenting complaints in patients with diabetic neuropathy is excess callus formation. In autonomic neuropathy, the amount and rate of callus buildup is increased, creating more pressure on the plantar surface of the foot. This increase in the formation of callus is proportional to the amount of external pressures put on the skin. These pressures are of three

types: Direct pressure, friction stress and shearing stress, which result in two basic formations of callus tissue: Nucleated keratomas and shearing callus. Nucleated keratomas are usually associated with direct pressure caused by prominent metatarsal heads or other bony prominences in weight or pressure bearing areas. Shearing callus is usually associated either with friction or shearing stress, which is created secondary to biomechanical imbalance.

Plantar callus formation is a marker for high plantar pressures. (See Figure 11) Large plantar callus buildup itself causes an estimated 30% increase in plantar pressure, affecting the deeper tissues and forming even more callus. (Figure 14) Increased load or body weight on certain foot deformities causes more direct pressure and therefore more peak plantar pressures, and even more callus formation than the neuropathic patient would have if

The overall risk for amputation in diabetic patients is increased 15 fold over non-diabetics.

he/she did not have a pressure bearing structural foot deformity.

Significant foot deformity, abnormal soft tissue thickness, altered biomechanics, posture and gait parameters, such as speed and instability, have all been associated not only with direct pressure-related callus formation but also with friction and shearing stress-related callus situations.

Callus buildup is also a function of motor neuropathy, where muscle atrophy occurs, thereby creating imbalance of the muscles. An example is the development of more prominent metatarsal heads due to the extensor tendons contracting unopposed as a result of the weakening of the foot flexors and intrinsics. Even at rest, the toes are pulled into a contracted, claw-like position. As this occurs, the fat pad is displaced distally, thereby removing the plantar fat pad's protective shock absorbing bed for the already prominent condyles of the metatarsal heads. In addition, the existent fat pad becomes atrophied due to metabolic challenges.

Non-enzymatic Glycosylation

Non-enzymatic glycosylation of many proteins in the body play a role in changing the mechanical properties of tissue. In the foot, the development of calluses is fostered by non-enzymatic glycosylation by increasing the stiffness of the skin, decreasing elasticity and its ability to withstand friction and shear. Keratin in the stratum corneum of the diabetic foot has been shown to be glycosylated compared with nondiabetic skin. This results in a reduced capacity of the skin to properly distribute plantar pressures. Nonenzymatic glycosylation has been shown to play a significant role in the observed limitation of motion in many

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Figure 3 Charcot Foot
Neuropathic Osteoarthropathy

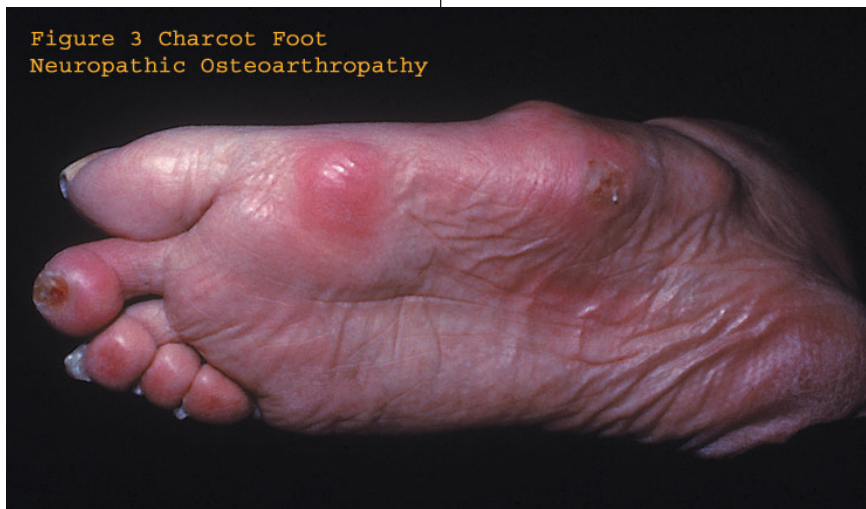


Figure 4 Neuropathic ulcer with faulty healing leading to gangrene of the digits



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joints of the body in persons with diabetes, including the joints of the foot and ankle.

It has been found that limitation of motion of the 1st metatarsal-phalangeal joint is associated with increased pressure under the 1st metatarsal head, leading to increased callus and ulceration. (Figure 10) Conversely, it has been found that persons with calluses and ulcerations due to increased pressure under the 1st metatarsal head had significantly diminished dorsiflexion at the 1st metatarsal phalangeal joint and limited 1st ray mobility. (Figure 13) Decreased subtalar joint mobility has been associated with elevated plantar pressures that lead to callus and ulcerations. Higher interdigital pressures between the 4th and 5th toes commonly leading to heloma molle and eventual ulceration have been associated with limited joint mobility due to nonenzymatic glycosylation in these joints.

Although calluses form in order to better protect the foot, callus is unyielding, fixed tissue that is extremely prone to injury from shearing and friction. Calluses may actually cause increased pressure in the areas beneath them; thus it is not uncommon to find an ulcer under a callus.

Any time there is a buildup of callus tissue in a neuropathic patient by whatever mechanism, there is a risk for ulceration. Complicating matters, neuropathic patients cannot be relied upon to report problems because foot lesions may be painless; without supervision and education, such a patient may allow a minor lesion to progress to a serious state.

Diabetic Ulcerations

The relationship between diabetic neuropathy and foot ulceration was recognized by a British surgeon over a century ago: Pryce remarked that "it is abundantly evident that the actual cause of the perforating ulcer was peripheral nerve degeneration", and that "diabetes itself may play an active part in the causation of the perforating ulcers." Over 50 years ago, Joslin realized that the de-

velopment of foot lesions was not an inevitable consequence of diabetes, and that most problems were potentially preventable. Even still, the overall risk for amputation in diabetic patients is increased 15 fold over non-diabetics.

Diffuse autonomic neuropathy, especially involving peripheral sympathetic fibers, is frequently present in foot ulcer patients and is even considered a major contributing cause of foot and lower extremity ulcerations. This is due to increased blood flow and arteriovenous shunting in the absence of large vessel disease, leading to cracking and fissuring of the skin as well as increased lower extremity edema.

Foot ulcerations are usually associated with diffuse distal-symmetric polyneuropathy and may involve both motor and sensory nerves.

Neuropathy of the motor nerves in the foot may result in muscle wasting, foot deformities and abnormal pressure points.

Sensory Neuropathy

Sensory neuropathy may result in pain, decreased position sense, numbness or loss of protective sensation, resulting in gait changes and abnormal pressure points. Abnormal pressure points may lead to morbid callus formation and potential ulcerations. These conditions allow the patient to tolerate poorly fitting shoes. (Figure 7) Loss of joint position sense interferes with foot function and loss of pain and/or protective sensation allows the patient to tolerate noxious stimuli. (Figure 8)

Our understanding of the etiopathogenesis of foot ulcerations

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Figure 5
Severe edema in the
foot of a patient
with autonomic
neuropathy



Figure 6 Digital Contracture
secondary to motor neuropathy



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have progressed in recent years. Earlier, vascular disease and infection were considered to be the two major causes. Research has shown that loss of protective sensation and insensitivity, reduced vibration perception (a measure of large fiber dysfunction), high foot pressures and abnormalities of autonomic function (such as reduced sweating and dry skin), increased peripheral blood flow and associated arteriovenous shunting are all strongly associated with ulceration. In isolation, each of these components is not sufficient to cause ulceration but in combination they form a causative pathway.

In a report from the Manchester Diabetic Foot Clinic, neuropathy can be implicated as an etiologic factor in 90% of over 600 episodes of ulceration. By extrapolation, the common pathway to amputation includes the component causes of neuropathy (diabetes-related pathophysiology), minor foot trauma (an environmental event), a consequent ulcer, faulty healing, and then gangrene. (Figure 4) The same studies showed that 61% of diabetic amputations were related to neuropathy. These studies confirm the role of neuropathy in the pathogenesis of foot ulcers and amputation.

Therefore, a foot that is insensate as a consequence of neuropathy does not spontaneously ulcerate; the neuropathy serves only as a permissive factor. It is a combination of neuropathy and pressure that leads to ulceration, and the pressure may come from a number of different sources.

Studies of pressures and loads under normal and neuropathic feet have confirmed that high foot pressures are commonly found under neuropathic feet, especially those with a history of ulceration. Recent studies have shown that high foot pressures are a major cause of ulceration in susceptible feet. It is report-

Studies have shown that trimming of corns and calluses decreases plantar pressure by an average of 29%.

ed that 28% of neuropathic feet with high foot pressures ulcerated within a 2 1/2-year period. Callus tissue that forms as a response to dry skin (from autonomic neuropathy) and high pressures may in itself further exacerbate the problem. Other studies have shown that trimming of corns and calluses decreases plantar pressure by an average of 29%.

Pressure

Pressures that contribute to foot ulcerations may be from external factors (extrinsic pressure or stresses), such as foreign bodies and ill-fitting footwear (Figure 7); or intrinsic factors, such as limited joint mobility, abnormal foot shape (such as a Charcot-type of foot), and high

foot pressure. Further, intrinsic stresses may develop as a result of the characteristic deformity of the diabetic neuropathic foot, where unopposed action of the long extensor tendons leads to clawing of the toes and resultant prominence of the metatarsal heads. Extrinsic pressures produce an ulceration in which the skin is clearly broken. Intrinsic pressures produce ulcerations that are initially not open, but occur under hard callused skin.

Damage to a foot by direct vertical external forces can occur in one of three ways:

1. Continuous low pressure from two-five pounds per square inch for a period of 10-12 hours can create damage to the skin and subcutaneous tissues, as with tight shoes. (Figure 7) These are very low pressures, because if a shoe applies greater than five pounds per square inch to the foot, the person cannot put the shoe on the foot. Nearly all ischemic ulcers created by continuous pressure occur on the sides of the foot. This is due to pressures being concentrated over bony prominences and in areas of smallest radius of curvature. A person

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Figure 7 ulcer due to skin-on-shoe friction



Figure 8 ulcer caused by stepping on a sewing needle in an insensate foot

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with an insensate foot may allow the pressure to be sustained for many hours. The problem is created when arterial inflow pressure is less than the external pressure applied. Damage is created by ischemic necrosis that develops because the blood flow from the capillaries which supply the soft tissues is compromised. (Figure 1)

2. *A direct injury* can create sudden high pressure for a short period of time, eliciting as much as 600-1000 pounds per square inch. This is the type of extrinsic pressure that stepping on a foreign object may deliver to the foot, creating an acute wound. (Figure 8) Common foreign objects are sharp pins, glass, toothpicks, nails and small pebbles that get caught in the shoe. An opening in the skin is created by the very high sudden pressure over a small area and damage is created by the mechanical disruption of tissue. (Figure 9)

3. *Repetitive stress* can create moderate pressure from 25-75 pounds per square inch, creating breakdown of the skin. This type of pressure is usually associated with thousands of repetitions, such as in walking. Damage to the skin is created through enzymatic autolysis of tissue. (Figure 15)

Damage by intrinsic forces can occur in the following ways:

1. *Spreading infection* can cause moderate pressure on the internal tissues of 25-75 pounds per square inch. Pressure of the purulence on the infected tissue causes the infection to spread through the soft tissues.

2. *Shear stresses* from either anteroposterior shear, which tears tissues in a linear fashion in the foot, or medialateral shear, which tears tissues from side to side. These problems happen because of biomechanical problems with gait.

3. *Friction pressures* which create a rubbing on the skin. This problem usually occurs with too-tight shoes or in bed-bound patients who have friction created between their skin and the sheets.

The equation $\text{Pressure} = \text{Force} / \text{Area}$ is important when considering the mechanism of diabetic foot ulceration due to neuropathic changes.

These forces can be thought of

as a mechanical input to the foot. However, the magnitude of the forces does not necessarily reflect the magnitude of damage to the tissue. To quote Dr. Paul Brand: "Pressure is the critical quantity that determines the harm done by the force." To explain, it can be said that more damage can be done by a force transmitted through a few plantar prominences than by the

same force distributed over a larger area of the plantar surface of the foot. Therefore, distributing the weight of the person over a larger area through shoes and orthoses is a valuable therapeutic measure.

II. Pain and/or Numbness

Painful diabetic neuropathy is perhaps the most challenging ele-

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ment of caring for the diabetic patient because the cause of the pain is not well understood, and therefore it is not uncommon for the patient to be treated in a semi-empirical manner.

We do understand, though, that pain involves C fibers, which are small-diameter, unmyelinated slow conduction velocity fibers, and A and D fibers, which are small and myelinated and have faster conduction than large myelinated fibers. These nerves fire in a hectic pattern and cause pain.

The first consideration when a person with diabetes presents with severe pain is to determine that the painful symptoms are caused by diabetic neuropathy. The most likely neuropathic etiology is distal symmetric polyneuropathy (DSP). Pain from other causes may respond well to appropriate therapy once the diagnosis is made. Non-DSP-related pain in a diabetic patient includes peripheral vascular disease, femoral neuropathy, tarsal tunnel syndrome, spinal stenosis, reflex sympathetic



Figure 9 Malperforans ulcer caused by repetitive moderate pressure



Figure 10 ulcer associated with increased pressure under the 1st metatarsal head

dystrophy. These must be ruled out first before consideration is given to treating the diabetic neuropathy.

In one study, it was found that only 65% of the diabetic patients who were referred to a pain center for treatment of diabetic peripheral neuropathy actually had distal symmetric polyneuropathy. Non-DSP patients present with a different clinical picture than those with DSP. For instance, tarsal tunnel syndrome generally has a unilateral presentation. Electrophysiological tests are often needed to make a definitive diagnosis of tarsal tunnel syndrome. The hands and feet are equally involved in neuropathy stemming from pernicious anemia. Lead poisoning has more proximal than distal nerve involvement.

Clinical Diagnosis

Quantitative tests are not routinely necessary to establish DSP. The clinical diagnosis can often be

made by a detailed neurological exam. DSP involves both small and large nerve fibers and involves more than one nerve. Small nerve fiber assessment is not easily done in the neurological exam. Therefore, it may be occasionally necessary to examine the thermal perception threshold or pseudomotor function, if the diagnosis is in question after the neurological exam is performed, to determine the presence of small fiber involvement. The typical pattern is that the toes are worse than the ankles and the lower extremity is worse than the upper extremity. It is bilaterally symmetrical and there is distal and dying-back (glove and stocking) nerve damage. Pain due to DSP is often brought to the attention of the physician because the patient is seeking relief from pain. The pain is usually worse at night (as opposed to the end of the working day), is exacerbated by rest and is relieved rather than made worse by movement.

Once it has been determined that the pain is secondary to DSP, further categorization should then take place. Acute painful neuropathy lasts less than 12 months and is related to metabolic abnormalities rather than structural abnormalities. The typical patient is the newly di-

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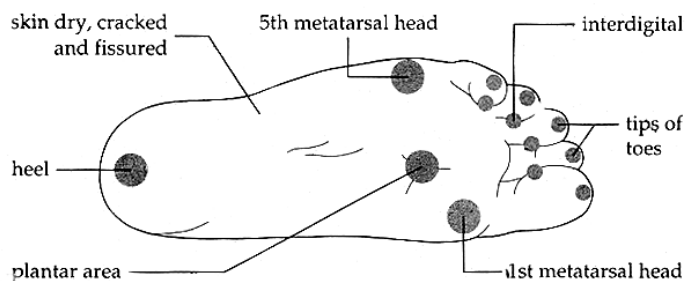
agnosed diabetic patient that just started on insulin. The course is self-limited and responds well to analgesic therapy with nonsteroidal anti-inflammatory medication.

Chronic painful diabetic neuropathy typically occurs after 8-12 years duration of diabetes. Its onset is gradual and subtle and typically lasts more than 12 months. This is usually associated with structural abnormalities such as an abnormal

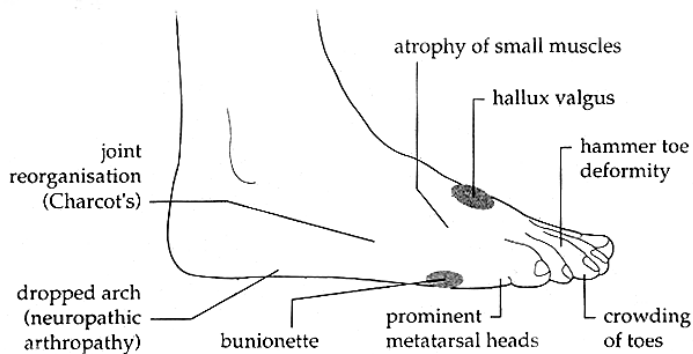
vasa nervorum causing local nerve ischemia, nerve fiber loss, axonal atrophy and degeneration, nodal and endoneurial swelling, myelin wrinkling and wallerian degeneration. The pain persists for years and relapses are common. Treatment is more complex and is complicated by psychological factors that are present in a patient with chronic pain.

Neural function is generally improved with intensive glucose control, but pain may actually worsen. As nerve structure and function worsen, the pain threshold may be exceeded and may proceed to the point where sensation decreases and the foot becomes numb. Depending on where the patient is on this continuum, an improvement in nerve function could either cause the pain to drop below the pain threshold or increase the pain. Regardless of the change in pain symptoms, glucose control is still desirable and should be initiated in all patients.

Figure 11 Plantar callous is a marker for high pressure and ulcer formation

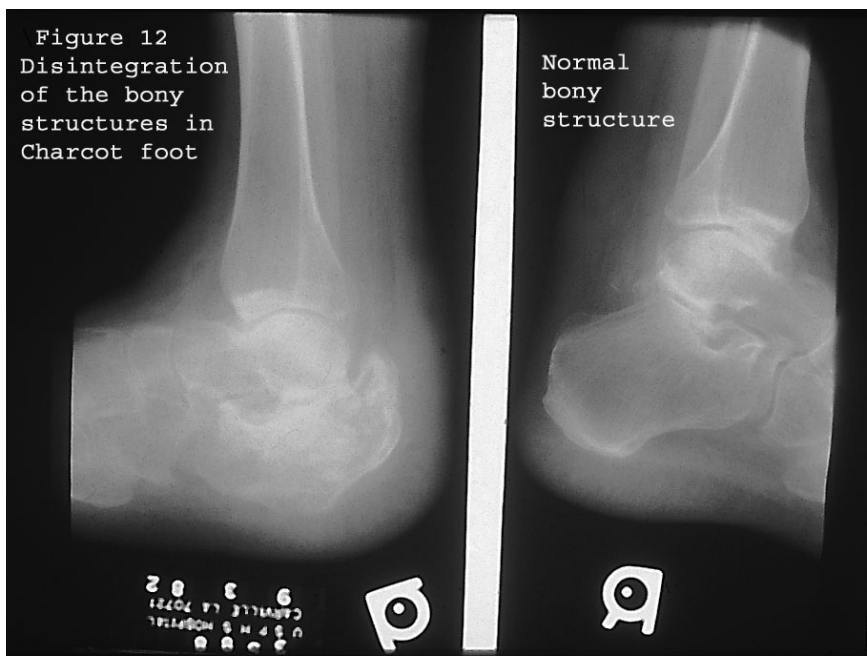


Areas of callous formation and neuropathic ulceration



The Neuropathic Foot

Figure 12 Disintegration of the bony structures in Charcot foot



Normal bony structure

Distal Symmetric Polyneuropathy

The pain of distal symmetric polyneuropathy is of three types.

Dysesthesia pain is usually described as a "burning sensation", "sunburn-like pain", "tingling skin" or having a painful sensation caused by something that would not ordinarily hurt, such as bed sheets or stockings. This type of pain is caused by an increased firing of damaged or abnormally excitable nociceptive fibers, especially sprouting regenerating fibers in the cutaneous or subcutaneous levels. Standard treatments for this type of pain are capsaicin or gabapentin. Capsaicin is considered ideal for this type of pain because it only penetrates to the sub dermal layer. It is a natural extract from hot peppers and comes in a cream base that is either .25% or .075% concentrations. Initially, the application of capsaicin may produce a burning sensation by stimulating substance P into the dermal area, but continued use of topical capsaicin typically produces desensitization by blocking the nociceptive afferent nerve fibers. It can decrease the pain 40-70% but may take 2 to 3 weeks before maximum pain relief is realized.

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Gabapentin (Neurontin) is being used successfully for the treatment of painful diabetic neuropathy, but was originally developed for the treatment of complex partial seizures. A common mistake is to prescribe too low of a dose. Larger doses (2400 to 3600 mg in divided doses per day) may be necessary to achieve a level of therapeutic pain relief.

Paresthesia pain is commonly described as "pins and needles", "electric-like", "numb", "aching feet", "knife-like", "shooting pains", "lancinating pains" or "I feel like my feet are in ice water." These types of pain are thought to come from both physiologic and structural changes in affected nerves.

These changes produce paresthesia pain by the following mechanisms:

1) spontaneous activity and increased sensitivity of damaged afferent axons in the dorsal root ganglion

2) loss of segmental inhibition of large myelinated fibers on small unmyelinated fibers

3) ectopic impulses generated from demyelinated patches of myelinated axons, or

4) increased firing caused by physiologic stimulation of endings of nociceptive afferents (nervi nervorum) that innervate the nerve sheaths themselves.

5) The above abnormalities causing structural changes such as nerve fiber loss, axonal atrophy and degeneration, nodal and endoneurial swelling, myelin wrinkling and wallerian degeneration.

Systemic as opposed to topical therapy is often required. Medications that have shown to be moderately successful include the following: tricyclic antidepressants, mexiletine, carbamazepine, phenytoin, alpha-lipoic acid, and tramadol.

Muscular Pain

Muscular Pain is usually described as "a dull ache", "night cramps", "band-like sensation", "charlie-horses", "drawing sensations", "toothache-like pains", "knotting up", and "muscle spasms." Muscular pain is associated with motor neuropathy. The condition is believed to be caused by injury to the motor neurons, in that demyelinated patches develop. Ectopic impulses to the muscle can be

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APWCA Ends Grandfathering, Plans Annual Conference

The one-year-old **American Professional Wound Care Association (APWCA)** is a multidisciplinary organization whose membership represents every health care specialty involved in wound management. Having demonstrated impressive growth in its initial year, the organization has announced that it will close grandfathering for DPM's to join as of Dec. 31, 2002.

Founding fellows include the following podiatrists, some of which serve on the Medical Advisory Board: doctors David Armstrong, Barry Block, Alan Catanzariti, Jerry Ferritto, Robert Frykberg, John Giurini, Arthur Helfand, Guido LaPorta, John Mattiacci, Robert Mendicino, David Novicki, Jeffrey Robbins, Michael Trepal and others.

The APWCA serves to represent your interests, and they've clarified this by challenging a number of current less-than-desirable regulations, among them: global fees that are inappropriate for wound care, length-of-stay guidelines, the inefficiency of required over-documentation, disallowances for needed services, and other mandates. APWCA uniquely offers innovative, interactive, multidisciplinary education, with cutting edge presentations unavailable from other organizations (seminars, e-mail newsletters, indexed journal, etc.).

APWCA's annual conference will be held March 27-29, 2003. The Symposium will have a variety of multidisciplinary speakers, the interactive general lectures will feature the latest research techniques (including morphing images of wound healing), and there will be an optional multi-track non-surgical and surgical precourse. A partial list of these break-out sessions is expected to include tracks in plastic surgery with lab, internal fixation with lab, vacuum assisted wound closure, and antibiotic beads. The conference will be held at the Hilton Philadelphia Airport, which will accommodate over 400 DPMs, MDs, DOs, RNs and other healthcare practitioners. Last year's annual seminar had 200 attendees with standing room only.

Many other seminars throughout the country are available to APWCA members at reduced rates, and APWCA's dues structure is very affordable.

For more information circle #162 on the reader service card. ■

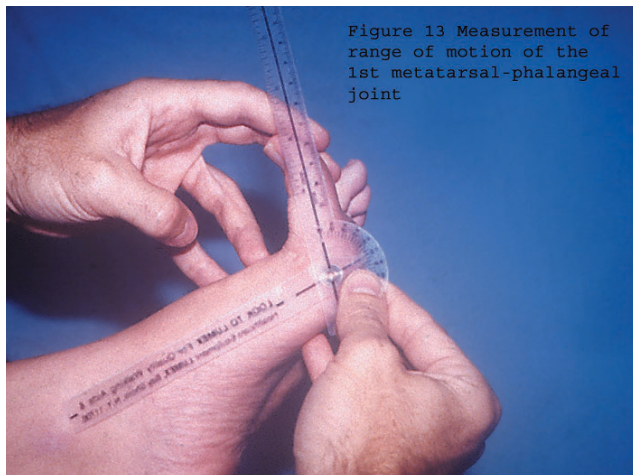


Figure 13 Measurement of range of motion of the 1st metatarsal-phalangeal joint

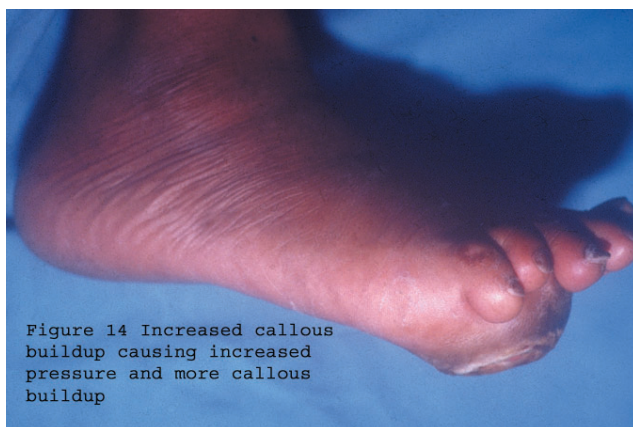


Figure 14 Increased callous buildup causing increased pressure and more callous buildup

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generated from these demyelinated patches and result in muscle spasms and pain. This creates a vicious cycle that was first described by Livingston in 1925. This cycle is created by a reflex loop that involves a nociceptive input activating motor neurons within the spinal cord that cause muscle spasms that in turn stimulate the muscle nociceptors and feed back to the spinal cord to sustain the cycle of spasms and pain.

Treatment for muscle pain is geared at breaking "Livingston's vicious cycle" by using NSAID's, pain medications, and muscle relaxants such as Metaxalone.

Focal Neuropathy

Focal neuropathies are believed to be due to an acute ischemic event to a nerve or when a nerve is compressed in a specific body compartment. These can be a significant contributor to painful diabetic neuropathy. Focal ischemic neuropathy tends to have a sudden onset, to be asymmetric in distribution, and to be self-limiting, as its pathogenesis is different from the more common DSP. Mononeuropathy is an example of this type and can be very painful. This "nerve-trunk" pain is usually described as aching in character and is experienced in the cutaneous area supplied by the specific nerve involved. It is presumed to be caused by increased firing following physiological stimulation of the endings of nocicep-

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tive afferent neurons innervating the nerve sheaths.

Focal compression or entrapment neuropathies tend to have a gradual onset, tend to occur in an asymmetric distribution, (although bilateral distribution is possible), and often have a progressive course. A common example would be tarsal tunnel syndrome, where the posterior tibial nerve becomes entrapped in the tarsal tunnel. Symptoms may include paresthesias or numbness in the distal foot with the heel spared, or the heel may be involved. Pain usually worsens as the day progresses and with activity. Therefore, the pain is generally worse at the end of the day and improves with resting the foot, a different scenario from the pain associated with DSP. Also, unlike DSP, treatment may require steroid injections or surgical intervention.

Numeric Treatment Algorithm

A numeric scoring system and treatment algorithm is now being used to initiate treatment for painful diabetic neuropathy. When 0 is no pain at all and 20 is the worst pain imaginable, a score of 5 or above mandates treatment; and the treatment is geared to the specific type of pain the person has. Re-assessments are done every 6 weeks and the pain score is re-evaluated. If the pain score drops to less than 5, then medications are decreased or discontinued. All medications are usually discontinued within 12 months of therapy; however, reoccurrences are not uncommon. If this is the case, then the medication for the specific type of pain the patient is experiencing should be re-started.

This treatment algorithm is highly successful and should be used as a treatment model for painful diabetic

neuropathy. In addition, control of pedal edema has been shown to be a useful adjunct in the overall treatment of peripheral neuropathy. Future treatments that show promise include topical clonidine, gabapentin cream, pre-gabalin therapy, intravenous alpha-lipoic acid and pulsed electromagnetic wave (Diapulse®) therapy.

III. Muscle Weakness and Gait Problems

Muscular pain is usually accompanied by other signs of motor neuropathy which affect muscle tone and strength, as well as changes in foot structure and gait patterns. Initially, the small intrinsic muscles begin to atrophy due to a

loss of appropriate motor nerve innervation. This leads to a loss of the stabilizing effect they provide, and the foot is overpowered by the long flexors and extensors. This unbalanced contraction leads to digital contracture and prominent metatarsal heads. (Figure 6) Neuropathy of the motor nerves supplying the lumbrical muscles and the interossei results in claw toes, hammer toes and mallet toes. Less weight is borne



Figure 15 Heel ulcer due to friction pressure between the skin and sheets

on the toes and more weight is distributed to the metatarsal heads because of the retrograde forces placed on the metatarsal heads, creating higher pressures, calluses and ulcerations.

The prominent joints of the toes are susceptible to skin-on-shoe friction (Figure 7); and in the insensate foot this can lead to heloma durum, blisters, ulcers, bone infection and gangrene. Not uncommonly, the anterior tibial muscle becomes weakened and the opposing gastrocnemius will contract, creating a functional equinus deformity. The ability to dorsiflex the foot during swing phase of gait is compromised as well as the ability to dorsiflex the foot during stance phase of gait. These problems result in excess pronation, more pressure on the medial side of the foot and an propulsive steppage gait pattern.

Physical Therapy

Physical therapy and addressing the biomechanical abnormalities caused by the motor neuropathy should be an important part of the therapeutic program for motor neuropathy. These therapies should include stretching exercises and appropriate shoes and orthoses, and even tendo-Achilles lengthening should be considered in some cases.

Besides DSP, there is another type of diffuse neuropathy that commonly produces pain in diabetic patients. This is termed proximal motor neuropathy, also known as amyotrophy or femoral neuropathy. This tends to affect older Type 2 diabetic patients, particularly males,

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and is distinguished by a relatively acute onset of pain and weakness in the proximal muscles of one or both lower extremities. Evidence derived from nerve biopsies suggests a vascular etiology, perhaps associated with micro-infarction in the nerve trunk and occlusion of the epineural arteries. In this type of neuropathy, there is no evidence that recovery is in any way linked to improvement of blood glucose control.

IV. Bone and Joint Abnormalities

Anyone who treats the diabetic foot is familiar with the most extreme example of diabetic foot deformity, the Charcot foot neuropathic osteoarthropathy, which is a common presentation in any podiatric physician's office and its etiology is strongly associated with neuropathy. (Figure 3) The Charcot foot is hot, erythematous and edematous, with bounding pulses and prominent veins. Patients may have moderate pain and discomfort even in the presence of loss of sensation. There is usually a recent history of minor trauma. Frank fracture, osteomyelitis and cellulites must be ruled out. The patient with Charcot foot is afebrile and the white blood cell count is normal. There are two neuropathic components to the development of neuropathic osteoarthropathy.

The first is the involvement of autonomic neuropathy, essentially resulting in an equivalent of a sympathectomy to the arteries of the foot. The arteries dilate and AV shunts develop in these feet. The increased blood flow allows for the increased absorption of calcium from the bones in the feet and precipitates their disintegration, the collapse of the joints and therefore the deformed Charcot foot. (Figure 12). In addition, the gross neuropathic changes in the ligaments contribute to the spontaneous dislocation of the foot. Ligaments and joint capsules are thought to be weakened at their insertions into bone by hyperemic resorption, allowing complete dislocation to take place.

The second neuropathic component is loss of protective sensation, a complication of DSP. Because there is relative insensitivity, the pa-

tient continues to walk, developing stress fractures and further bone destruction. In addition, the ligaments and joint capsule are thought to be stretched by the abnormal stresses applied to the joints, allowing them to go beyond their normal range of motion, and contributing to the collapse of the foot. Treatment is to off-weight the foot either with a total contact cast or other non-

weight-bearing devices. Non-weight bearing should continue until temperatures normalize and there is x-ray evidence of consolidation of fractures and fragmented bone. Surgery during the acute destructive phase should be avoided because it may accelerate the destructive process. Surgery should be attempted in the later phase only

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when attempts at conservative care have failed to establish a stable, plantigrade foot or prevent recalcitrant plantar ulceration.

V. Psychological Problems Dealing with the Neuropathy

Neuropathy itself can trigger severe psychological effects. Paul Brand, MD, in a lecture given at the Gillis W. Long Hansen's Disease Center in Carville, LA explains this. According to Dr. Brand, the sense of touch is considered our validating sense. Upon seeing something unfamiliar, a person's first desire is to touch the item, not to smell, hear or taste it. This sense of touch makes the object real. Without it, the reality of an object is not confirmed and therefore not validated.

When a patient loses foot sensation, the feet are regarded as "unreal", as are any associated problems (e.g., ulcerations). The patient then becomes detached from his or her foot problems—even disgusted to the point where a serious complication often is neglected.

For this reason, Dr. Brand points out, the podiatric physician must encourage patients to "like" their feet and hence identify with them. Offering positive observations (e.g., compliments for nicely kept toes, a well-shaped foot, healthy-feeling skin or patent circulation) can help the podiatric physician gain the patient's trust. Adding a reassuring touch during this exchange also can be empowering.

Such small talk might sound like a trivial waste of time, but it encourages the patient to take responsibility for his or her own health care and thereby override these negative effects.

In addition, any time a person is in chronic pain, there is a psychological profile associated with that, including depression. This patient should be treated as any chronic pain patient would be, including being prescribed medication for depression. The astute practitioner will do his patients a great service by having a high index of suspicion regarding these psychological issues.

Prevention

Prevention is said to be the best treatment. At this point, there is no definite prevention pathway for dia-

betic neuropathy. It is clear, however, that several factors play an important role in altering its course. Good glucose control will delay, prevent or slow the progression of DSP. There are some promising drugs on the horizon, but adequate clinical trials are currently not available. Treatment of the risk factors for the development of neuropathy and offering treatment for the specific type of pain will help alter the development of further complications of the neuropathy.

When considering the risk factors, it is important to recognize that there are modifiable and non-modifiable risk factors. The DCCT and UKPDS studies have offered conclusive evidence that better glucose control prevents or slows the progression of diabetic neuropathy. Hypertension, elevated cholesterol, smoking and alcohol abuse have yet to be proven conclusively to be modifiable risk factors, but they have been shown to prevent or slow the progression of retinopathy and/or nephropathy. There is no reason to believe that the progression of neuropathy would not be similarly affected. At any rate, a prudent lifestyle that employs the control of all these factors should be encouraged by everyone.

Non-modifiable risk factors include older age, longer duration of diabetes, HLA DR 3/4 genotype, and height. Many studies show that men are at greater risk for neuropathic complications than women, but recent analysis of this data resulted in the hypothesis that the risk factor is actually the height of a person because longer nerves are more prone to nerve damage.

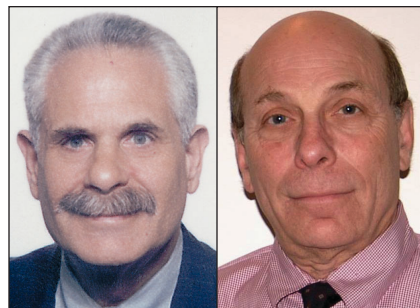
In summary, it can be said that diabetic neuropathy wears many faces but none of these faces wear a smile. It can be a devastating complication of diabetes. An astute practitioner and a motivated patient can certainly be an effective team in controlling the progression of diabetic neuropathy. This requires aggressiveness, a high index of suspicion, early intervention, taking on the role of teacher and motivator and being a true patient advocate.

Podiatric physicians have an excellent opportunity to provide this kind of care to the neuropathic patient. Patients suffering from neuropathy must be aggressive in choos-

ing a healthcare practitioner that addresses their neuropathic complaints completely, and assertive in acquiring the kind of care they need. They also must take responsibility for their own healthcare and aggressively seek a healthy lifestyle. With proper control and attitude on the part of the patient and practitioner, diabetic neuropathy can certainly wear a face with a more pleasant countenance. ■

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EXAMINATION

See instructions and answer sheet on pages 176-178.

- 1) What percentage of diabetic patients is estimated to have some form of neuropathy?
- A) 95%
 - B) 75%
 - C) 10%
 - D) 25%
- 2) Which of the following is not associated with diabetic neuropathy?
- A) Vasospastic disease
 - B) Dry skin
 - C) Hammertoe deformity
 - D) Plantar ulceration
- 3) Which of the following is associated with autonomic neuropathy?
- A) Oily skin
 - B) Tinea Pedis
 - C) Onychomycosis
 - D) Dry, cracked and fissured skin
- 4) Which of the following factors is not associated with dry feet in the diabetic patient?
- A) Inappropriate shoes
 - B) Atrophy of the sweat glands
 - C) Arteriovenous shunts
 - D) Damaged nervous inputs
- 5) One of the most common presenting complaints in diabetic neuropathy is:
- A) Morton's neuroma
 - B) Tarsal Tunnel syndrome
 - C) Sciatica
 - D) Excess callus formation
- 6) All of the following represent different types of pressure that contribute to callus formation except:
- A) Direct
 - B) Pooling of the blood
 - C) Friction stress
 - D) Shearing stress
- 7) Common types of callus formation seen in the diabetic patient include the following:
- A) Nucleated keratoma
 - B) Shearing callus
 - C) Heloma
 - D) All of the above
- 8) Motor neuropathy is related to:
- A) Disuse atrophy
 - B) Ligamentous laxity
 - C) Muscle atrophy creating imbalance of the muscles
 - D) Overuse syndrome
- 9) The following muscles commonly weaken as a result of motor neuropathy in the diabetic patient except:
- A) Lumbrical muscles
 - B) Interossei
 - C) Anterior tibial
 - D) Extensor hallucis brevis
- 10) Non-enzymatic glycosylation in the foot does all the following except:
- A) Changes mechanical properties of tissues
 - B) Increases the stiffness of the skin
 - C) Decreases elasticity of the skin
 - D) Adds moisture to the skin
- 11) Plantar callus formation is a marker for
- A) Tinea pedis
 - B) Onychomycosis
 - C) Subungual exostosis
 - D) High plantar pressures
- 12) Contracted digits can cause the following:
- A) Heel spur syndrome
 - B) Plantar fasciitis
 - C) Displacement of the plantar fat pad distally
 - D) Onychocryptosis
- 13) Nonenzymatic glycosylation has been shown to play a significant role in all of the following except:
- A) Limitation of motion of the 1st metatarsal phalangeal joint
 - B) Change in the keratin of the stratum corneum
 - C) Hypoglycemic episodes
 - D) Decreased Subtalar joint mobility
- 14) Higher interdigital pressures can lead to the following:
- A) Morton's neuroma
 - B) Heloma molle
 - C) Metatarsus adductus
 - D) Onycholysis
- 15) The relationship between diabetic neuropathy and foot ulceration was first recognized:
- A) Over 10 years ago at the Hansen's Disease Center in Carville
 - B) Over 20 years ago at

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EXAMINATION

(cont'd)

Joslin Diabetes Center

- C) Over 40 years ago at Case Western Reserve University
- D) Over 100 years ago by a British surgeon

16) Foot ulcerations in the diabetic patient are usually associated with all of the following except:

- A) Loss of protective sensation
- B) Distal symmetric polyneuropathy
- C) Increased plantar skin creases
- D) Reduced vibration sensation

17) Trimming of calluses decreases plantar pressure by:

- A) 9%
- B) 19%
- C) 29%
- D) 39%

18) Damage to a foot by external forces can occur in all of the following ways except:

- A) Burns
- B) Continuous low pressure
- C) A sudden high pressure injury
- D) Repetitive stress

19) Damage to the foot by intrinsic forces can occur in the following way:

- A) Scratching the foot to excess
- B) Crush injury to the toe
- C) Spreading infection
- D) Blister formation

20) The following is an important formula when considering the mechanism of diabetic foot ulcerations:

- A) $\text{Area} = \text{Force}/\text{Pressure}$
- B) $\text{Force} = \text{Area}/\text{Pressure}$
- C) $\text{Pressure} = \text{Force}/\text{Area}$
- D) $\text{Pressure} = \text{Area}/\text{Force}$

SEE INSTRUCTIONS AND ANSWER SHEET ON PAGES 176-178.