# An Overview of the Diabetic Foot: Pathogenesis, Management and Prevention of Lesions

**Marvin E. Levin***

## ABSTRACT

Diabetic foot problems are not very glamorous. Nevertheless, the diabetic foot is the most common complication of diabetes, greater than retinopathy, nephropathy, heart attack and stroke combined. Throughout the world, foot lesions and foot infections are the leading causes of hospitalization and prolonged hospital stays for diabetics. Diabetic foot ulceration is the result of trauma to an insensate foot. Infection frequently follows and unless there is aggressive intervention, amputation becomes the end point. The management of diabetic foot problems is complicated and frequently requires the attention of many specialists. The single most important factor in preventing amputation is detailed and repeated education of the patient in foot care.

Physicians, clinics and hospitals who educate patients provide aggressive ulcer and infection treatment and who prescribe therapeutic shoes have reduced their amputation rate by 50%. This should be the goal of everyone involved in the management of the diabetic patient.

## INTRODUCTION

Diabetes is one of the oldest diseases known to mankind. The Ebers Papyrus of 1500 B.C. mentions its symptoms and suggests treatment. However, the history of gangrene of the foot goes back to Biblical time, when, in Chronicles II, the first case of gangrene of the feet, perhaps due to diabetes, is described.

The relationship between diabetic neuropathy, the insensitive foot, and foot ulceration was recognized by Pryce, a British surgeon, over a century ago. He stated that, "It was abundantly evident that the actual cause of the perforating ulcer was peripheral nerve degeneration and that diabetes itself played an active part in the causation of the perforating ulcer" (1).

The diabetic foot is especially vulnerable to amputation because of the frequent complications of peripheral neuropathy (PN), infection and peripheral arterial disease (PAD). A combination of this triad leads to the final cataclysmic events, gangrene and amputation. More than fifty-six thousand plus major amputations due to diabetes were reported in 1987in the United States (2). Fifty percent of all non-traumatic amputations are performed on the diabetic. Fifteen percent of all diabetics will develop a foot ulcer during their lifetime (3). Most of these are the result of PN and the insensate foot which leads to painless trauma and ulceration.

Diabetic foot lesions are not very glamorous and do not generate as many publications as do other complications of diabetes. However, in the United States the annual incidence of lower extremity leg and foot ulcers is 2,00,000. Add to these 56,000 major amputations and we have over a quarter of a million lower extremity diabetic complications. This is greater than all the other complications of diabetes combined, coronary artery disease 1,01,000, stroke 27,000 blindness 6,900, kidney failure 5,900(4).

Diabetic foot problems are a major cause of hospitalization and prolonged hospital stays. Twenty percent of all diabetic persons who enter the hospital do so because of foot problems (5). In the series of Smith et al, foot problems were responsible for 23% of the hospital days over a two-year period (6). At the Indian Institute of Diabetes in Bombay, India, more than 10% of all admissions for diabetes are primarily for foot management. More than 70% required surgical intervention and in more 40% of those interventions there was a toe or limb amputation (7). In the U. K. more than 50% of bad occupancy of diabetics are due to foot problems (8). It is obvious from these figures that throughout the world diabetic foot problems are a major cause of hospitalization, morbidity and mortality.

### Pathogenesis of Diabetic Foot Lesions

The signs and symptoms of either ischaemia or neuropathy may predominate. However, neither is present to the total exclusion of the other. The clinical picture is therefore the result of complications stemming from a combination of both. Peripheral neuropathy is the leading cause of most diabetic foot lesions. A majority of patients who enter the hospital because of diabetic foot lesions do so because of ulceration secondary to painless trauma. The various pathways leading to ulceration, infection, gangrene, and amputations are shown in Figure 1.

The exact incidence of PN is difficult to assess. Most studies estimate the incidence of clinical neuropathy to be 10 to 20%. However, this percentage may increase to as much as 50% after twenty-five years of diabetes. While most diabetic foot ulcers are due to

* *Clinical Professor of Medicine, Washington University School of Medicine, St. Louis, Missouri, U.S.A.*

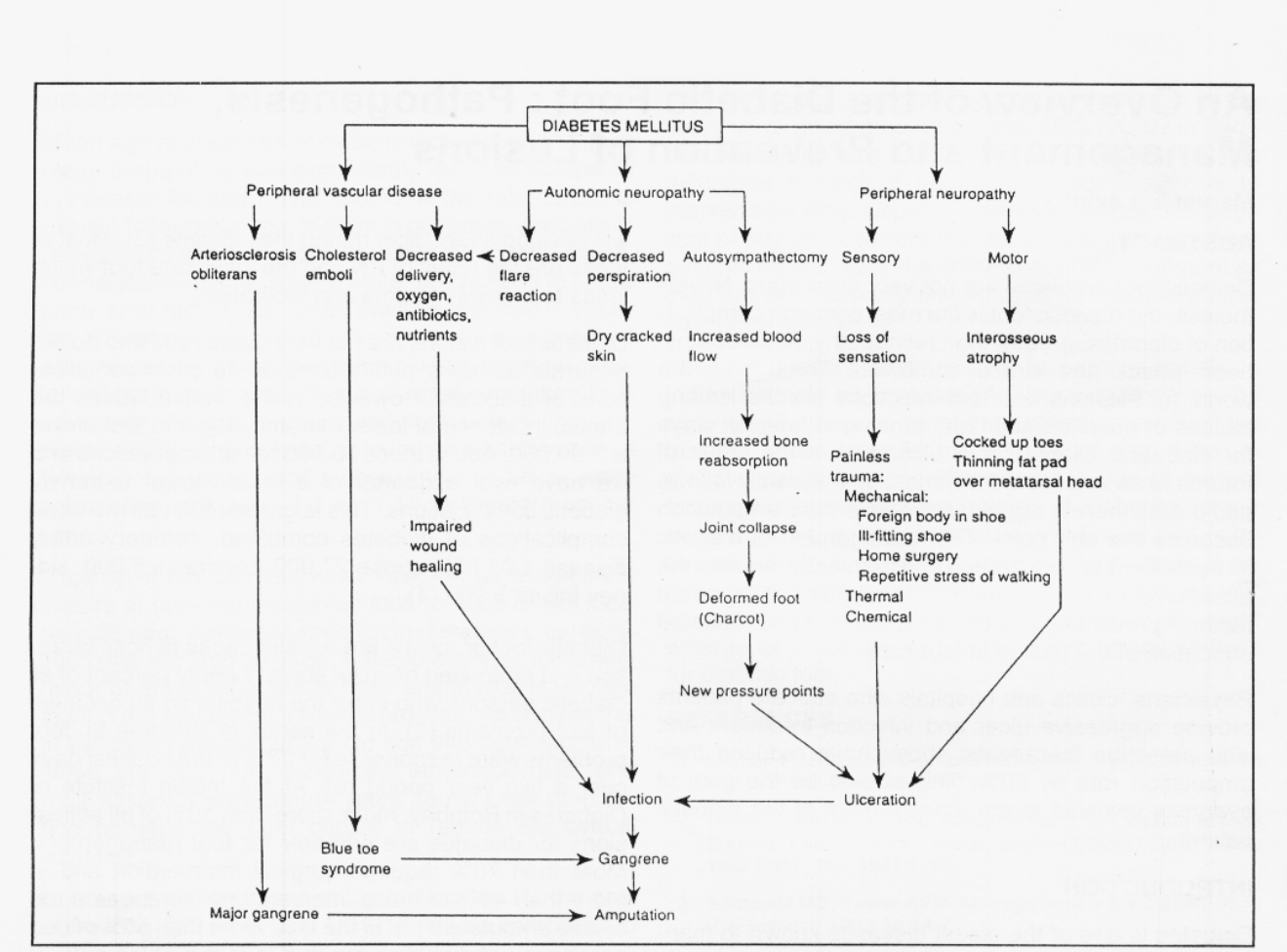


Fig. 1 Pathogenesis of diabetic foot lesions. Adapted from Levin ME. "Pathogenesis and management of diabetic foot lesions" In Levin ME, O'Neal LW, and Bowker JH, eds. The Diabetic Foot, 5th ed. St. Louis, Mosby Year-Book, 1993.

peripheral neuropathy and the insensate foot, it must be remembered that painful symptoms are common in-patients with PN. The series of Veves et al has shown that painful symptoms are frequent in-patients with PN irrespective of the presence or absence of foot ulceration (9). Diabetic patients may have an insensitive foot and still experience painful symptoms. Painful symptoms were present in 33% of the foot ulcer group (9). Therefore painful and painless PN are not necessarily two separate clinical conditions and may co-exist. It should also be kept in mind that when a patient with a diabetic foot ulcer which has been painless suddenly develops pain in the ulcer it may be indicative of worsening infection.

The most important effect of peripheral neuropathy on the diabetic foot is the loss of sensation, making the foot vulnerable even to trivial trauma. A break in the skin, even though it is inconspicuous and minuscule can become a portal of entry for bacteria. Unsuccessfully treated infection leads to gangrene and amputation.

## Special Diabetic Foot Problems

### The Heel

The heel of the diabetic patient is particularly vulnerable to trauma. When the diabetic patient is required to have bed rest for any length of time, such as when hospitalized, particular attention must be paid to the heel. Because of loss of sensation, the patient tends to keep the heels in the same position. This results in pressure necrosis causing the skin to break down. Infection and gangrene can follow. These patients should have their heels inspected at least once and preferably twice a day. If erythema is present, aggressive protective intervention must be instituted.

Prevention is critical. This is best achieved by turning the patient, using heel protectors, and an air suspension mattress. The available heel protectors may not stay in place; therefore frequent checks are required.

### Foot Deformities

Foot deformities frequently lead to ulceration. Diabetics are particularly prone to develop cocked up toes which can result in pressure at the top of the tip of the toes. I refer to this as the "tip-top-toe

syndrome". This deformity is frequently associated with a thinning or shifting of the fat pad under the first metatarsal head. These areas, the tops and tips of the toes, and the area under the first metatarsal head are therefore very vulnerable to ulceration and infection. The ideal treatment is prophylactic surgery to straighten these toes while the circulation is still good. When prophylactic surgery cannot be carried out, these patients should wear a shoe with a larger toe box or an in-depth shoe with a cushioned insole to protect the toes and metatarsal head area. Bunions are common and frequently lead to ulceration and infection. Prophylactic surgery is the ideal treatment.

The Charcot foot is the classic diabetic foot deformity. The Charcot foot develops in four stages. In the first or acute stage the patient usually presents with a history of mild trauma and a hot, red, swollen foot with bounding pulses. This must be differentiated from cellulitis. Once infection has been ruled out and a diagnosis of Charcot foot has been established, the treatment is non-weight bearing. This is best accomplished with a contact cast. If the patient is allowed to ambulate, the second stage of the Charcot foot develops, with the breakdown of the bones of the foot, resulting in fractures. The x-ray at the time of the patient' s initial visit may be perfectly normal. Calcification of the interosseous arteries is rarely found. The second stage develops in a two to three week interval. Repeat x-ray may show fractures, usually at the tarsometatarsal joint but not infrequently at the distal ends of the metatarsals. The third stage of the Charcot foot is characterized by foot deformity. The foot takes on a club foot appearance and a rocker bottom configuration. Treatment at this stage requires the use of special molded shoes. If the patient continues to walk on unprotected feet, the fourth stage ensues with development of a plantar ulceration in the area of the arch. The ulceration can become infected, leading to gangrene and amputation (10).

## Exercise for Diabetics with Foot problems

Exercise is an important modality in the management of diabetes. However, in-patients with PAD and PN, weight bearing exercises such as jogging, prolonged walking, treadmill, and step exercises may need to be curtailed or avoided. The presence of an active foot ulcer is an absolute contraindication for weight bearing exercise. Patients who have a healed ulcer must take special precautions when exercising. Scar tissue is not good tissue and is vulnerable to the sheer forces of walking. Patients with PAD, PN and an insensate foot can do a variety of non-weight bearing exercises such as swimming, bicycling, rowing, chair and upper body exercises.

Diabetic persons, particularly those with PAD, PN and previously healed ulcerations, should have specific and detailed instructions in foot care and techniques for decreasing foot pressure before undertaking an exercise program.

Physical therapists and personnel in exercise centers should discuss with the referring physicians the type of exercise program suitable for the diabetic.

## The Immunosuppressed Diabetic

The healing of foot ulcers in immunosuppressed renal transplant diabetics is markedly impaired. These patients also have a higher amputation rate (11). In most kidney transplant series, limb amputation is required for at least 15% of kidney recipients short-term and one third of the ten year survivors (12).

## Diabetic Foot Ulcers

Ulcers occur in the diabetic foot because of repetitive stress on insensitive feet. When repetitive stress continues, the foot develops hot spots, callus build-up, pressure necrosis and ultimately ulceration. Ulcerations occur most often at the site of the maximum pressure and excessive callus build-up, usually over the metatarsal heads, especially the first, and on the plantar surface of the hallux. Patients who develop ulcers have increased foot pressures (13). Increased pressure can be the result of foot deformities. Decreased flexibility of the foot, probably due to glycosylation of tendons and ligaments can also cause increased foot pressures. There are several techniques for decreasing plantar foot pressures in the diabetic. Removing a callus can reduce pressure at that site by at least 30% Wearing cushioned shoes (14) and pressure reducing hosiery (15) can also be beneficial. Patients should not go barefoot, not only because of possible trauma, but because pressures are significantly higher when walking barefoot than when wearing cushioned shoes. On a rare occasion it may be necessary to alleviate internal pressure, e.g., by removing the sesmoid bones or metatarsal heads. When a patient presents with ulceration on the dorsum of the foot, it is due to trauma. When the presentations is on the side of the foot, it is most likely due to an ill-fitting shoe.

Persistent and untreated ulceration in the diabetic foot leads to lower limb amputation in 84% of all the cases (16).

## Management of Diabetic Foot Ulcers

Table 1 lists the steps in the management of diabetic foot ulcers (17). X-rays are necessary to rule out

osteomyelitis, gas formation, the presence of foreign objects and asymptomatic fractures. It is my feeling that any foot with ulceration or infection should be x-rayed.

**Table 1**
**Management of diabetic foot ulcers**

I. A. Evaluation
   B. Depth of penetration
   C. X-ray
      1. Foreign body
      2. Osteomyelitis
      3. Subcutaneous gas
   D. Location
   E. Biopsy
   F. Blood supply (non-invasive vascular studies)
II. Debridement, radical
III. Bacterial cultures (aerobic and anaerobic)
IV. Metabolic control
V. Antibiotics
   A. Oral
   B. Parenteral
VI. Do not soak feet
VII. Decrease oedema
VIII. No weight bearing
   A. Bed rest
   B. Crutches
   C. Wheelchair
   D. Contact casting
IX. Improve circulation (vascular surgery)

*Adapted from Levin ME. "Pathogenesis and management of diabetic foot lesions" In Levin ME, O'Neal LW, and Bowker JH, eds. The Diabetic Foot, 5th ed., St. Louis, Mosby Year-Book, 1993.*

Treatment of a foot ulcer requires establishment of depth and degree of ulceration. What appears to be a superficial ulceration may be only the tip of the iceberg. There may be penetration deep into tissues. Vigorous debridement of the ulcer must be done to establish the degree of penetration and to remove all necrotic tissue. Debridement should be carried out to healthy tissue. The ulcer following debridement will, in all probability, be larger than it was at presentation. Eschars should be completely removed. Whirlpool is not the method of choice for debridement. When the foot is insensitive, minor debridement can be carried out at the bedside. However, in many cases the patient must be taken to the operating room for the adequate debridement under anesthesia. Taylor and porter have demonstrated that aggressive foot debridement, and, when indicated, revascularization resulted in long term salvage of 73% of threatened limbs even in high-risk patients (18).

Biopsy should be considered when the ulcer appears at an atypical location, e.g., not over the metatarsal heads or the plantar surface of the hallux, when it can not be explained by trauma, and when it is unresponsive to aggressive therapy. On numerous occasions biopsies of atypical ulcers have revealed malignancies, both primary and metastatic.

Infection is a common and major complication of diabetic foot wounds. Infection leads to microthrombi formation, causing further ischaemia, necrosis, and progressive gangrene. Massive infection is the most common factor leading to amputation. Lichter et al reviewed the laboratory results of a large series of patients with serious pedal infections (19). In this series the sedimentation rates were significantly elevated, mean 58.6 mm/h. Surprisingly, the mean white count was only 9,700. Therefore, one should not depend on white counts alone as a measure of the seriousness of the foot infection. As with other series, they found the lesions to be polymicrobial, 72% having gram positive cocci and 49%, gram negative. Nine percent had gram negative anaerobes (19). There is a high correlation between foot ulcers, infection and other diabetic complications. Lichter et al found that 67% of these patients had retinopathy, 70% nephropathy, 80% peripheral neuropathy, 91% decreased pulses, 69% hypertension and 40% atherosclerotic heart disease (19).

The selection of an oral antibiotic or parenteral antibiotic for the treatment of a diabetic foot infection is based on medical judgement. It should be kept in mind that many diabetic foot infections contain gram negative organisms. Therefore the oral antibiotic chosen should be effective for gram positive and gram negative organisms. The criterion for hospitalization and treatment with parenteral antiobiotics includes patients who are septic, febrile, and have leukocytosis and deep infection. The patient with what appears to be a minor infection on the plantar surface of the foot and evidence of infection on the dorsum of the foot, suggested by erythema and frequently oedema, should be hospitalized. Even though the patient is not septic, there is high probability that severe infection exists deep in the foot. Patients with infection and severe PAD should be hospitalized and evaluated for arterial by-pass surgery. The worst scenario leading to amputation is ischaemia and infection. Patients with PAD should be given parenteral antibiotics to achieve higher concentration of antibiotics in the peripheral tissues than can be achieved by oral therapy alone. Furthermore, the antibiotic of choice frequently can only be given parenterally.

If an oral antibiotic is selected, it is my opinion that the diabetic patient should not be told to take the medication and return in one week. Infection in the diabetic can deteriorate rapidly within twenty-four hours. It is therefore my recommendation that diabetics on oral therapy should be seen within a few

days following institution of therapy. They must be carefully instructed to notify the physician at once should there be an increase in redness, drainage, pain, odor, or evidence of lymphangitis. While many of these patients have insensate feet, the development of pain is indicative of deep infection and requires immediate attention. The development of a bad odor also indicates worsening infection and frequently the presence of anaerobes.

It is very important that patients with infection monitor their blood sugar level closely. A rising blood sugar level strongly suggests worsening infection, even though other signs and symptoms of a worsening infection are absent.

When infection is not responding to aggressive debridement and antibiotic therapy, the wound should be debrided again and recultured, as the flora may have changed. Chronic recurrent or resistant infection suggests the presence of osteomyelitis. Impending or developing gangrene also suggests possible progression of infection. Indications of worsening infection are noted in Table 2.

**Table 2**
**Worsening infection: Indications**

*Signs and Symptoms*
- Increased:
  - Drainage
  - Erythema
  - Pain
  - Temperature
  - Malodorous
  - Lymphangitis
  - Lymphadenopathy
  - Gangrene

*Laboratory*
- Increased:
  - Blood Sugar
  - WBC
  - Sedimentation Rate

Osteomyelitis is a frequent complication of diabetic foot ulcers and infection. Osteomyelitis may be difficult to detect on a clinical basis. Newman and coworkers showed that in biopsy-proven osteomyelitis only one third of the patients had clinically suspected osteomyelitis (20). If bone is visible or the ulcer can be probed to bone, the probability of the presence of osteomyelitis is extremely strong. Scanning techniques for osteomyelitis are not always successful. The triple-phase scan with technetium lacks specificity (21). Magnetic-resonance imaging (MRI) is proving to be a helpful technique. However, Newman and coworkers (22) found the use of labeled-leukocyte Indium$^{111}$ scanning techniques to be superior to magnetic resonance imaging (22). Bamberger, Daus, and Gerding established prognostic factors for preventing amputation in the face of osteomyelitis (23). They found that in patients without necrosis, gangrene, or the presence of swelling, the use of antimicrobial therapy active against the isolated pathogens given intravenously for at least four weeks or combined intravenously and orally for 10 weeks predicted a good outcome without the need for ablative surgical procedures (23).

Lipsky et al have recently reviewed soft tissue and bone infection in the diabetic foot (24).

Metabolic control is essential. It has been well demonstrated in a number of studies that leukocyte function is impaired in the presence of uncontrolled diabetes. Blood sugar levels should be kept below 200 mg/dl and as close to euglycaemia as is reasonable.

Soaking the feet has no benefit, although it has been traditional approach. Soaking can lead to maceration and further infection. Because of the insensitive foot, soaking may take place in water that is too hot, resulting in severe burns. Chemical soaks can result in chemical burns (25).

Oedema is frequently present. Elevation of the feet, no more than the thickness of one pillow, can be beneficial. Higher elevation may impede circulation.

Avoidance of weight bearing is essential. These patients have insensitive feet and because the ulcer is not painful, they continue to walk. The result is an increase in pressure necrosis, forcing bacteria deeper into the tissues, and causing failure to heal. The use of crutches and wheelchairs is seldom successful in achieving total and consistent avoidance of weight bearing. Many patients with PN have ataxia, making the use of crutches potentially dangerous. The best method for avoidance of weight bearing in appropriately selected patients is the use of the contact cast. The contact cast allows the patient to be ambulatory but essentially avoids weight bearing by redistributing the weight and decreasing the pressure on the ulcerated area (26, 27).

When an ulcer does not heal despite good metabolic control, adequate debridement, parenteral antibiotic therapy and avoidance of weight bearing, the impaired healing may be caused by vascular insufficiency. Mills, et al, found that all appropriately treated neuropathic ulcers and forefoot injections healed in-patients with palpable pedal pulses. If foot pulses were absent and arteriography confirmed large-vessel occlusive disease, foot lesions and

infections healed when concomitant revascularization was done (28).

The worst scenario for impaired would healing or the clearing of infection may be vascular insufficiency. Ankle or brachial indexes of less than 0.45 or transcutaneous oxygen pressure < 30 mm Hg and certainly those under 20 mm Hg are highly predictive that the infection will not resolve and that the ulcer will not heal. For example, Pecoraro and colleagues found a 39-fold increased risk of early wound failure if the average peri-wound transcutaneous oxygen pressure was under 22 mm Hg (26). Vascular surgery should always be considered in these cases. The importance of peripheral arterial reconstruction was demonstrated by LoGerfo and associates. In 2883 extreme distal arterial reconstructions, they found a statistically significant decrease in every category of amputation, a decrease that correlated precisely with increasing the rate of dorsal pedis artery by-pass (29).

Hyperbaric oxygen delivered by the hyperbaric chamber has been reported to be helpful in healing diabetic foot ulcers (30). Hyperbaric oxygen delivered by hyperbaric boot is totally ineffective. It must be kept in mind that the hyperbaric oxygen is used in conjunction with all of the aggressive treatments outlined in Table 1.

Experimental studies have suggested that a combination of topical growth factors and hyperbaric oxygen may be beneficial in improving wound healing (31).

## Topical Treatment of Foot Ulcers

The use of topical therapy goes back to ancient times, when an unbelievable number of substances were used to treat wounds, ranging from wine to human excreta (Table-Talk, XCII "Of God' s Works," Martin Luther 1483-1546). Today the list of topical agents remains long and continues to grow. Currently resins and enzyme therapy to aid in debridement are advocated by some. Although these are of some benefit, they represent adjunct therapy and should not be substituted for aggressive surgical debridement. It has been traditional to use povidone-iodine (Betadine), acetic acid, hydrogen peroxide, and sodium hypochlorite (Dakin' s solution).

Although these substances destroy surface bacteria, they are cytotoxic to granulation tissue and may delay wound healing (32). It is therefore my belief that these substances should be either avoided or used for the briefest periods.

The benefits of topically applied antibacterial agents, silver sulfadiazine 1%, polymixin B with bacitracin and neomycin, and gentamicin sulfate may be helpful (32). Topical antibiotics along may not be satisfactory, and the use of oral or parenteral antibiotics in conjunction with topical therapy is frequently necessary. Cleansing agents and types of dressings can make a difference in the rate of wound healing, Moist dressings seem to aid wound healing (32).

Despite aggressive therapy and adequate circulation, some diabetic foot ulcers heal slowly or not at all. Current investigative studies with topically applied platelet-derived growth factors to these foot ulcers have shown these factors to be important adjunct therapy in would healing (33, 34). Bentkover and Champion have shown the cost effectiveness of wound care centers and the use of platelet releasate (35). Platelet-derived wound-healing formula is an autologous solution extracted from the alpha granules of the patient' s platelets. This extract is applied to the ulcer daily by the patient in an outpatient setting. The platelet-derived wound healing formula contains several growth factors. They are platelet-derived growth factor, angiogenesis factor, epidermal factor, transforming growth factor ß, and platelet factor 4. Recent work has shown that interactions between factors and the extracellular matrix are of central importance in the process that causes wounds to close. Transforming growth factor ß appears to be a central player in many of the steps of wound healing, inducing angiogenesis, acting as a chemoattractant for macrophages and fibroblasts, regulating self-proliferation, and stimulating extracellular matrix (36).

The impediments to wound healing are listed in Table3.

**Table 3**

**Impediments to wound healing**

1. Vascular
   a. Atherosclerosis
   b. Increased viscosity
2. Neurologic
   a. Insensate foot
   b. Decreased flare reaction
3. Infection
   a. Inadequate debridement
   b. Poor blood supply
   c. Microthrombi
   d. Hyperglycaemia
   e. Decreased polymorphonuclear neutrophil function
   f. Polymicrobial infection
   g. Changing bacteria
   h. Osteomyelitis
4. Immunosuppression
5. Mechanical
   a. Oedema
   b. Weight bearing
6. Poor nutrition
   a. Low serum albumin level
7. Decreased growth factors
8. Poor patient compliance

9. Delayed treatment and referral

*Adapted from Levin ME: "Pathogenesis and management of diabetic foot lesions" in Levin ME, O' Neal LW, and Bowker JH, eds. The Diabetic Foot, 5th ed., St. Louis, Mosby Year Book,1993.*

## Post-treatment Management of Healed Diabetic Foot Ulcers

Even though the diabetic foot ulcer has healed, the job is not complete. The underlying aetiologies responsible for the ulcer, such as foot deformity, calluses, and increased pressure are still present. In addition, scartissue from previously healed ulcers is not strong tissue and is thus vulnerable to the shearing forces of walking. Special measures are therefore necessary to protect the vulnerable sites of previous ulceration. These include education of the patient in walking, for example, taking shorter steps and decreasing overall walking. Patients whose jobs require standing or walking, such as waiters or waitresses, may need to change jobs. Therapeutic shoes play a very important role in preventing recurrence of these ulcers.

## Special Shoes

The use of special therapeutic shoes is critical in preventing ulceration or recurrence. Patients who have cocked-up toes require a shoe with a bigger toe box. The patient with a markedly deformed foot, such as the Charcot foot, need a specially molded shoe. An in-depth shoe with a plastic-like material, such as plastazote insole, is frequently required to redistribute the weight away from the previously ulcerated site and thus prevent recurrence of ulcer. The importance of special shoes was clearly demonstrated at a study at King' s College in London, which showed an 83% recurrence of ulcers when patients returned to wearing regular shoes; with the use of special shoes, there was only a 17% recurrence of ulceration (37).

## Teamwork

The management of the diabetic foot requires the interaction of many medical disciplines (The table 4). A team approach is needed that will save the foot, not amputate it.

**Table 4**
**Team members involved in the care of the diabetic foot**

1. Primary physician
2. Endocrinologist
3. Diabetologist
4. Podiatrist
5. Nurse educator
6. Physician' s assistant
7. Enterostomal nurse
8. Infectious disease sepcialist
9. Neurologist
10. Vascular surgeon
11. Orthopedist
12. Physiatrist
13. Pedortrist
14. Orthotist
15. Physical therapist
16. Prosthetist
17. Occupational therapist
18. Social worker
19. Home care nurse

## Patient Education

Of all the approaches to prevent ulceration and to save the diabetic foot, the most important is patient education. Despite our current knowledge, physicians cannot totally prevent PAD and PN. However, the patients can be educated in proper foot care, and can learn how to prevent injury and detect lesions as early as possible.

At the time of the office visit and while the shoes and socks are off, the nurse or physician should review the do' s and don' ts of foot care with the patient. This objective cannot be adequately accomplished simply by handing the patients a list of instructions. The instructions should be explained and questions should be encouraged and answered, so that the patients can attain a better understanding of the importance of foot care (Table 5). These instructions should be carried out at least once a year or even more often. The effectiveness of educational program in reducing amputation has been noted by Malone and coworkers (38) and more recently by Litzelman (39).

*Adapted from Levin ME: "Pathogenesis and management of diabetic foot lesions" In Levin ME, O' Neal LW, and Bowker JH, eds. The Diabetic Foot, 5th ed. St. Louis, Mosby Year Book, 1993.*

**Table 5**
**Patient-instructions for the care of the diabetic foot**

1. Do not smoke.
2. Inspect the feet daily for blisters, cuts and scratches. The use of a mirror can aid in seeing the bottom of the feet. Always check between the toes.
3. Wash feet daily, dry carefully, especially between the toes.
4. Avoid extremes of temperatures. Test water with hand, elbow or thermometer before bathing.
5. If feet feel cold at night, wear socks. Do not apply hot water bottles or heating pads. Do not use an electric blanket. Do not soak feet in hot water.
6. Do not walk on hot surfaces such as sandy beaches, or on cement around swimming pools.
7. Do not walk barefooted.
8. Do not use chemical agents for removal of corns and calluses, corn plasters or strong antiseptic solutions.

9. Do not use adhesive tape on the feet.
10. Inspect the insides of shoes daily for foreign objects, nail points, torn linings and rough areas.
11. If your vision is impaired, have a family member inspect feet daily, trim nails and buff calluses.
12. Do not soak feet.
13. For dry feet, use a very thin coat of lubricating oil or cream. Apply after bathing and drying the feet. Do not put oil or cream between the toes. Consult your physician for detailed instructions.
14. Wear properly fitting stockings. Do not wear mended stocking with seams. Change stockings daily.
15. Do not wear garters.
16. Shoes should be comfortable at time of purchase.Do not depend on them to stretch out. Shoes should made of leather. Purchase shoes late in the afternoon when feet are the largest. Running or special walking shoes may be worn after checking with your physician. Purchase shoes from a shoe salesperson who understands diabetic foot problems.
17. Do not wear shoes without stockings.
18. Do not wear sandals with thongs between the toes.
19. In the winter, take special precautions. Wear wool socks and protective foot gear such as fleece-lined boots.
20. Cut nails straight across.
21. Do not cut corns and calluses: follow instructions from your physician or podiatrist.
22. See your physician regularly and be sure that your feet are examined at each visit.
23. Notify your physician or podiatrist at once should you develop a blister or sore on your foot.
24. Be sure to inform your podiatrist that you are diabetic.

*Adapted from Levin ME: "Pathogenesis and management of diabetic foot lesions" In Levin ME, O' Neal LW, and Bowker JH, eds. The Diabetic Foot, 5th ed., St. Louis, Mosby year Book,1993.*

## CONCLUSIONS

Diabetic foot ulcers are common. If treatment is delayed or improper treatment is given, these lesions can lead to infection, gangrene and amputation. Physicians and clinics that avoid delay in the treatment of the diabetic foot ulcer, exercise aggressive therapy for these ulcers, provide revascularization when indicated, use therapeutic shoes, practice the team approach, and repeatedly educate patients in foot care have reduced their amputation rates by 50% (37,40). This should be the goal of everyone who cares for patients with diabetes.

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