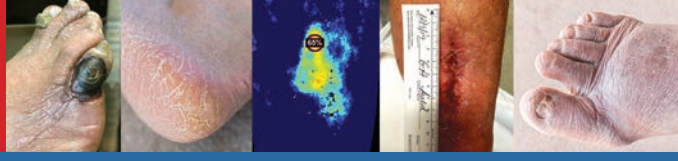

The Wound Is Closed… Now What? So What?

The closure of a wound does not define the endpoint of treatment.

BY KENNETH REHM, DPM

Note: *Part 1 of this article deals with why the closure of a wound does not define the endpoint of treatment but rather heralds the next phase in the management of a more complex condition.*

When a person has their wound treated at a credible institutional wound care center or other well-regarded center of excellence for wound healing, the care they receive is expected to be the epitome of best practice, defined by up-to-date evidenced-based standards of care in this discipline. That person, in fact, would not be disappointed in this regard because treatments of the diabetic wound are usually state-of-the art. Yes, the most sophisticated topical medications, dressings and grafts probably would have been used; the well-regarded algorithms of care, however, seem to stop dead-end after the wound is closed. The patient, then, is usually discharged into the frays of the muddled disorganized abyss posing as our health care system.

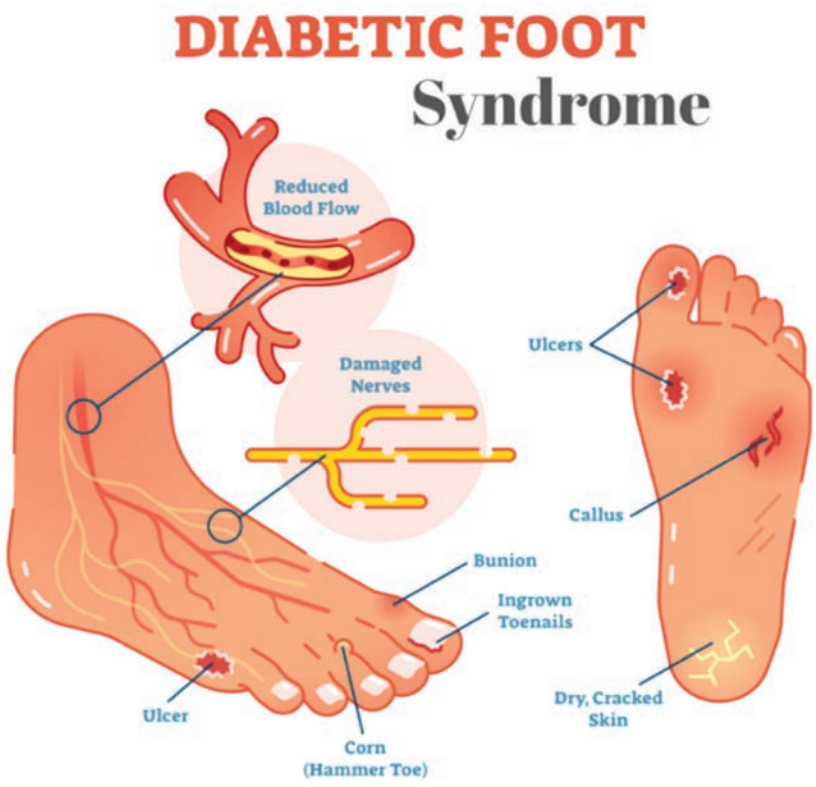

Figure 1: Diabetic Foot Syndrome: neuropathy-vasculopathy-dermatopathology

This is the experience of many and is discussed by those who advocate for a more holistic approach toward wound treatment.[1] Roland Renyi, researcher and past chair of the Lindsey Leg Club Foundation, emphasizes that chronic wounds are usually a sign of a wider systemic disease; and lack of communication between the inter-disciplinary providers is putting these very patients at risk for treatment failure, reoccurrence of the wound, and a worsening scenario that may very well result. Consequently, perhaps related to a limited allotment of time and resources, patients often experience a lack of consistency, planning and information regarding the management of their conditions, along with referral mechanisms that are not responsive to their immediate needs.

In this author's experience, here is an ongoing problem and a burgeoning obstacle to care, especially in the patient with diabetes who suffers from a foot or lower extremity wound. Wound care clinicians are most often aware of these matters, but the system is not set up for automatic inclusion of these supra-wound issues. In addition, it should be pointed out that most wounds are not treated by wound specialists; and by extension of logic, then, most foot wounds are not treated by podiatric physicians and surgeons who would be trained in surgery, biomechanics, wound care

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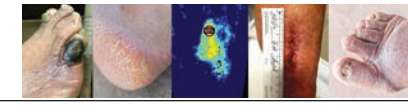

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and the complexities surrounding diabetic foot disease. These areas of expertise need to be an obligatory part of our treatment systems if we are to do justice to the proper treatment and prevention of diabetic foot wounds.[1]

Does closure of a wound define the endpoint of treatment or should it herald the next phase in management of a more complex condition? Because it is generally agreed that diabetic foot ulcers are the most common and problematic chronic skin wounds that wound care physicians treat, let's answer this question in terms of the closed diabetic foot wound (DFW).[2]

Consistent with the above statement that wounds are an extension of a wider systemic disease, ulcerations of the foot in someone who has diabetes are no exception. This paper will address the circumstances that mandate a more encompassing view of diabetic foot wounds and therefore more inclusive care. If the diabetic foot health professionals continue the limited scope of their treatment algorithms, even though the field has made significant gains in recent years, morbid outcomes will still prevail.

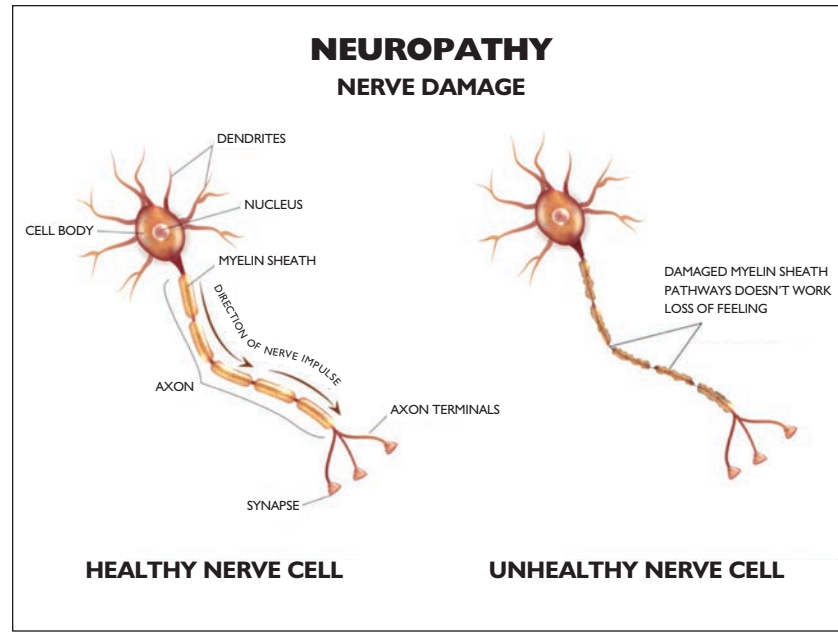

Figure 2: Nerve damage causing neuropathy in diabetes

DSPN is a critical factor in the development of over 82 percent of DFW's.

The Neuropathies

Let's analyze the development of a DFW, which is an end result of Diabetic Foot Syndrome (Figure 1). The patient with a diabetic foot wound would likely have diabetes-related comorbidities. Distal symmetric polyneuropathy (DSPN), for instance, is arguably the most common complication that affects the foot in diabetes; and is the most common form of diabetic neuropathy, representing approximately 75 percent of cases. Furthermore, DSPN is a critical factor in the development of over 82 percent of DFW's.[3,4] The small nerve fibers are typically affected earlier in the disease process and this results in pain and paresthesias (Figure 2). As the disease progresses, damage to large nerve fibers can result in numbness and loss of protective sensation, which in turn is a contributory factor in the etiology of Charcot neuroarthropathy, gait instability and resultant falls.[4] Having loss of protective sensation, especially when combined with persistent structural foot deformities, limited range of motion of weight bearing joints and bony prominences associated with high pressure areas forecasts unaccommodated focal areas of repetitive friction, shearing and direct pressure (Figure 3). These all serve as permissive factors for foot ulcerations. This does not stop when diabetic foot wounds are closed.

Figure 3: examples of 3 types of pressure that pose risk to the foot in diabetes

There are other types of neuropathy that contribute to the development of diabetic foot wounds. Peripheral Autonomic neuropathy causes

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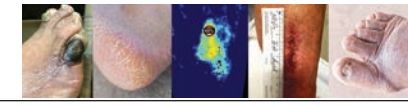

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a loss of autonomic sympathetic tone in peripheral vessels and vasomotor paresis. This results in arteriovenous shunting of the subcutaneous vascular network, which raises the venous blood pressure and causes neuropathic edema. This edema impedes fresh oxygenated blood flow into the lower extremity and could facilitate the formation of ulcerations, especially in those that demonstrate some degree of peripheral arterial disease (Figure 4).

Figure 4: Edema related to venous shunting and peripheral arterial disease in diabetes

Sudomotor paresis due to autonomic neuropathy leads to a dysfunctional thermoregulatory mechanism with compromised sweat glands and impaired ability to sweat. Blood perfusion to the deeper layers of the skin is increased and can lead to overheating of the skin, lack of humidification of the skin and cooling by evaporation. Arguably, this could result in dryness of the skin and loss of its protective function with increased risk of injury. Pedal anhidrosis shows up as dry shiny skin, which then becomes atrophic and cracks. These cracks on the feet become hard and callused. which then serves as a nidus for fungal and bacterial infection. Autonomic neuropathy is a major contributing factor in the development of Charcot foot (neuropathic osteoarthropathy or neuroarthropathy), alterations in skin thickness, and medial arterial sclerosis, a condition defined by deposits of calcium in the medium and small muscular arteries that normally do not cause luminal narrowing. These effects of autonomic neuropathy are associated with a two-fold higher risk for ulceration and a three-fold higher risk for all types of amputations of the feet.[5]

Oxidative stress may lead to increased buildup of advanced glycation end-products (AGE's) and to cross-link formation of the extracellular matrix in all tissues.[5,7]

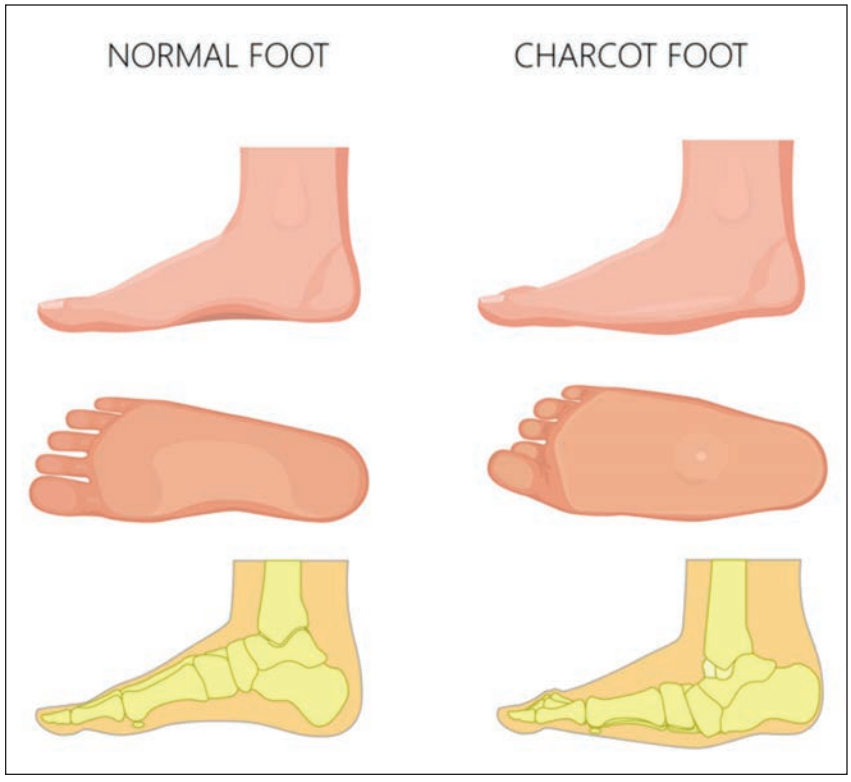

Figure 5: Charcot Foot causing midfoot collapse

Motor neuropathy associated with diabetes was brought to the surface and elucidated by neurologists Hugh Garland and Derek Taverner in their 1953 paper on myelopathy, after twenty years of the subject being non-existent in the literature.[6] They considered this condition as a proximal motor pathology accompanied by pain in the hip and thigh with wasting of the quadriceps and loss of reflexes in the legs. There is now a broader understanding of diabetic motor neuropathy; and we appreciate that along with autonomic neuropathy and sensory neuropathy, these conditions can act individually or together as a precedent event to such conditions as Charcot neuroarthropathy, (Figure 5). which often leads to midfoot collapse and increased pressure over bony prominences, which result in morbidities associated with DFW's. Additionally, we see weakness and atrophy in the intrinsic muscles of the foot, the lumbricales and interosseous muscles, which stabilize the digits. There is an imbalance created between the flexors and extensors; and with this instability present, an 'intrinsic minus' foot deformity may occur. Digital contractures form and hammertoe, mallet toe as well as claw toe deformities develop (Figure 6), predisposing the

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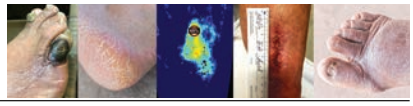

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patient to digital ulcerations over prominent bone and joint areas. In addition, direct pressure, shearing and friction is increased to the forefoot because of the progressive declination of the metatarsal heads. The result is the formation of nucleated keratosis as well as dispersive callus formation (Figure 7) heralding potential ulcerations and infection. In addition, the pathologic functioning of the 1st metatarsal phalangeal joint and a high arch formation predisposes the neuropathic patient to significant biomechanical and gait abnormalities.

Most neuropathies affect all three types of nerve fibers to varying degrees; and damage to sensory, motor and autonomic nerves can occur at the same time. A mixed clinical picture is often the result, where the effects on a target organ is a consequence of the damaging effects from the multi-neuropathic influences.

Oxidative Stress and Non-Enzymatic Glycosylation

These neuropathic conditions are strongly associated with oxidative stress leading to increased buildup of advanced glycation end-products (AGE's) and to cross-link formation of the extracellular matrix in all tissues.[5,7] This damaging process inflicts structural connective tissue changes that result in limitation of motion of both the joints of the hand and foot in about 40% of patients. In particular, stiffening and loss of flexibility occurs in the gastrocsoleus complex and a tight Achilles tendon is a result, thereby creating an equinus deformity limiting a person's ability to dorsiflex during gait. This aspect of the neuropathic limb is another contributing factor to the damaging aspects of Charcot neuroarthropathy and the subsequent midfoot collapse previously discussed. The biomechanical instability created allows additional force to be generated on areas of skin that is pathologically thick and non-pliable. The shearing, friction and direct pressure imposed on both the skin and the underlying defective scaffold serve as permissive factors in the development of callus. Due to the influence of the mixed neuropathic picture, this callus formation is likely to be thick and xerotic, and left without intervention, ulcerations would ensue—adding to the register of risk factors for amputation in persons with diabetes.

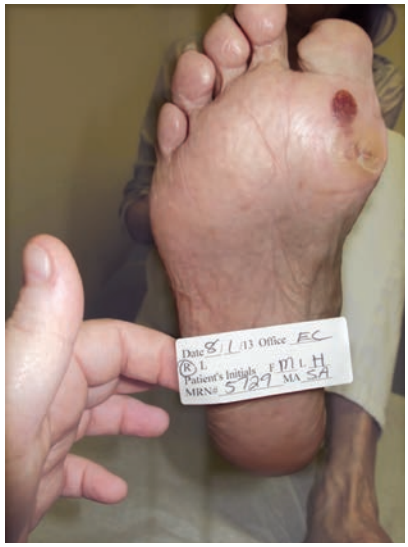

Figure 7: High arch in a person who has diabetes with pathologic functioning of 1st metatarsal phalangeal joint with nucleated keratoma and ulcerative callus

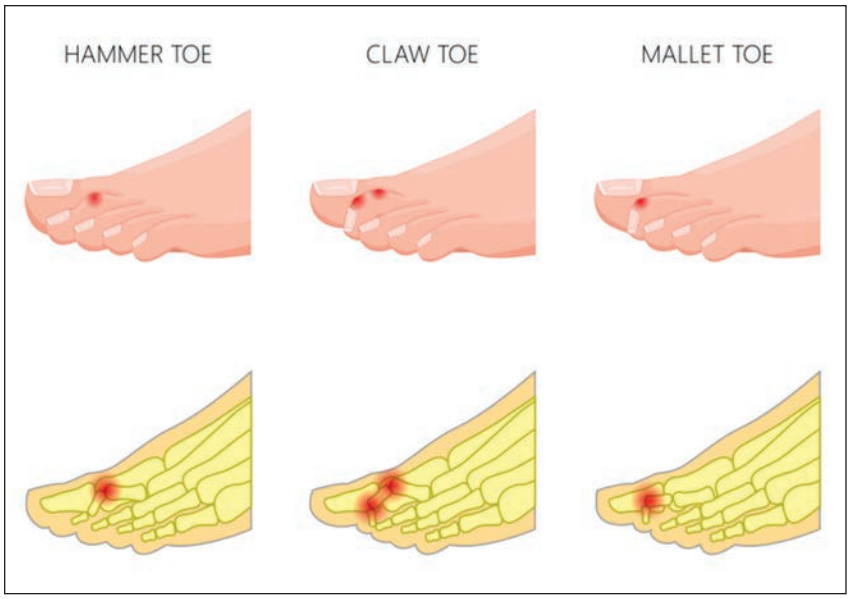

Figure 6: Digital contractures caused by an imbalance created between the flexor and extensor tendons

These damaging progressive changes can be mitigated, even after the wound is closed, by embracing a lifestyle that limits this process.

What is important to know about oxidative stress and non-enzymatic glycosylation is that these damaging progressive changes can be mitigated, and certainly should be addressed, even after the wound is closed, by embracing a lifestyle that limits this process.

The Effects of PVD

When peripheral vascular disease, a ubiquitous occurrence in the progression of diabetes, is present, foot ulcerations then are more likely to be accompanied by a high risk of slow healing, infection, gangrenous changes and eventual amputation. Keeping in mind that a closed ulceration poses a risk for future ulcerations, then caring for a wound after it closes appears to rival the importance of all the sophisticated and expensive treatment rendered before closure.[8] Is this concept incorporated into standard wound care algorithms? Are the opportunities to augment circulation exercised?

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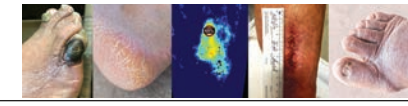

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Skin Involvement

The skin is perhaps the most visible of the organs affected by this devastating discordance of pathology. The characteristics of skin in persons with diabetes are clearly different than in those without, because in diabetes, the skin is not spared the damaging effects that this metabolic disease has on other organ systems. Skin involvement associated with both DM1 and DM2 is almost universal and is a predominant source of common complications which often lead to ulcerations, infections, gangrene and amputation.[9] These complications are present in 79.2% of people with diabetes. A study of 750 patients with diabetes found that the most common skin manifestations were cutaneous infections (47.5%), xerosis (26.4%), and inflammatory skin diseases (20.7%).[10] Incidentally, Individuals with type 2 diabetes are more likely than those with type 1 diabetes to develop these cutaneous manifestations.

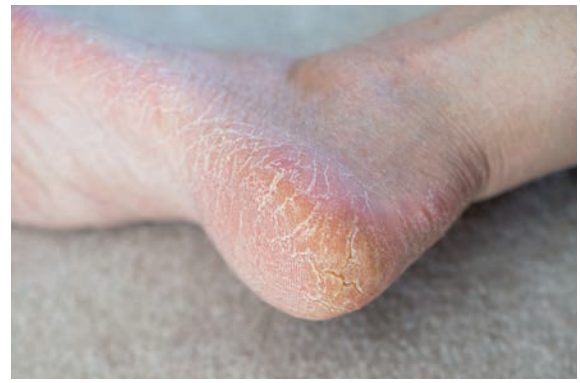

Figure 8: Skin fissures and xerosis due to autonomic neuropathy and angiopathy in a diabetic pose a high risk for ulceration

With diabetes, the progressive changes in the skin reflects the advancing nature of the individual's disease, mirroring the variability in the status of their glycemic control. Functional properties of the epidermis and dermis are not only affected by hyperglycemia, but relative ischemia of the skin is postulated to be contributory. This ischemia can be a result of previously discussed arterio-venous shunting due to autonomic neuropathy, microvascular or macrovascular disease.[9] It is important to point out that ischemia stemming from the same vascular shortcomings have widespread injurious impact, not only on the skin, but on all end organs and tissues perfused by these compromised vascular channels.

There are conflicting reports about whether patients with diabetes demonstrate a normal hydration state of the stratum corneum; however, diminished activity of the sebaceous glands together with impaired skin elasticity, without any impairment in the stratum corneum barrier function, seems to be a consistent finding, provided there is no physical insult to this barrier, such as superficial or deep fissures. These functional changes to the stratum corneum are similar to the changes that take place in the normal aging process, but in diabetes, these changes are expedited and are very similar to senile xerosis.[11,12]

The ominous scenario of ulceration, leading to infection, gangrene and amputation can originate from skin fissures developing in diabetics.

The ominous scenario of ulceration, leading to infection, gangrene and amputation can originate from skin fissures developing in patients with diabetes (Figure 8). It is generally considered that these most often develop from problems associated with autonomic neuropathy, and newer studies indicate a correlation of the deep fissures with diabetic angiopathy. This suggests that improving blood supply might be effective in preventing these. Deep fissures that extend into the dermis are especially concerning as they pose a higher risk of ulcerations than the superficial ones in that they are likely to compromise the protective skin barrier mechanisms.

Other frequently reported skin-related expressions of diabetes in the skin include abnormalities of sweating. Decreased sweating, often accompanied by compensatory hyperhidrosis elsewhere, is a result of autonomic neuropathy. Skin thickening, simulating scleroderma, is reported to be present in as many as 30% of persons with diabetes.[9] This pathogeny is a function of untoward collagen formation and collagen cross-linking as well as abnormal fibrosis taking place mostly in the dermis. Immunologic abnormalities, especially involving the function of the polymorphonuclear leucocytes, are present, placing the person at risk for fungal, yeast and bacterial infection.[13]

Preserving an acidic pH in the stratum corneum is of vital importance in maintaining the health of the acid mantle layer and epidermal barrier homeostasis.

Addressing the topic of pH of the skin in diabetes, it must be pointed out that preserving an acidic pH in the stratum corneum is of vital importance in maintaining the health of the acid mantle layer and epidermal barrier homeostasis. High pH can deplete the skin of essential oils and lipids, a key component of the acid mantle layer, and compromise

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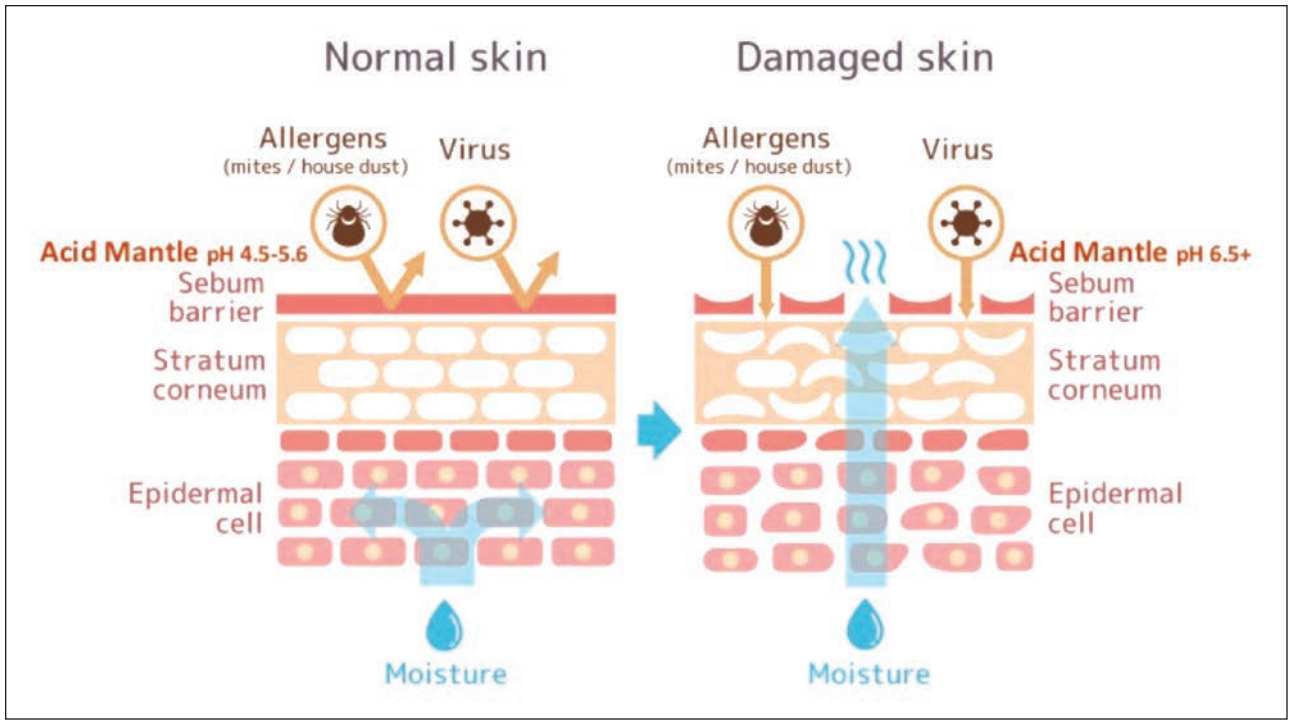

Figure 9: Diagram of healthy and damaged skin denoting the pH of the acid mantle

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the protective functions of the skin (Figure 9). This can lead to irritation, inflammation, colonization of unwanted bacteria as well as dehydration that causes dryness of the skin, creating its own problematic cycle of adverse events. The pH of healthy skin is acidic, which in turn promotes the natural healing processes of the skin in creating a healthy microenvironment, thus inhibiting the growth of pathogenic bacteria and reducing damaging proteolytic activity.[14] **PM**

Part 2 of this article will discuss what should be done, after the wound is closed until complete healing takes place, to make the system work to address the critical supra-wound issues.

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