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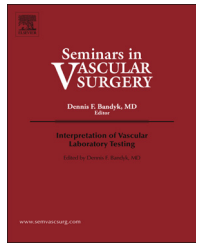

Offloading the diabetic and ischemic foot: solutions for the vascular specialist

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ABSTRACT

For generations, the use of techniques to defer skin pressure and protect the lower-extremity wound has been a cardinal goal to achieve therapeutic success and healing. Choosing the appropriate postoperative offloading device or shoe is often difficult, as it is challenging to merge optimal mechanical protection with clinical realities and patient needs. The gold standard for offloading remains the total contact cast, yet it receives minimal utilization in the clinical setting. Other devices have shown benefit, including the removable cast walker, instant total contact cast, and depth inlay shoes, for preventative measures. Ultimately, any plantar, lower-extremity wound must receive some form of external pressure reduction to reach acceptable rates of healing. Future technologies will aid these measures by providing body-worn constant monitoring systems and more effective offloading via patient-specific exoskeletons. This review is a supplemental update on the available wound offloading modalities based on logic-driven research regarding pressure relief across the diabetic neuropathic or impaired perfusion foot.

1. Introduction

The importance of offloading therapy and devices cannot be overstated in the care of the patient with diabetic peripheral neuropathy and critical limb ischemia (CLI), as the global incidence of foot ulcerations leading to amputation and increased mortality is staggering. Diabetes mellitus is central to this epidemic, with diabetes rates expected increase by >50% in the next 20 years [1]. This epidemic will have drastic consequences for universal human health, as this rapid disease dissemination is linked to distal extremity neuropathy and development of atherosclerotic occlusive disease; symptomatic gateways leading to ulceration and amputation, with long-term morbidity and mortality [2–4].

Peripheral arterial disease (PAD) affects 8 to 10 million Americans, roughly 1% will develop CLI [5]. Although the progression to CLI in the PAD patient may be multivariate and unpredictable, it is apparent that patients with CLI and tissue loss (Fig. 1) experience significant morbidity and require attempts at endovascular or surgical revascularization. Key to the prevention and resolution of an active ulcerative wound is the abolition of direct pressure and repetitive trauma to the wound site. Therefore, healing of any wound, whether primary or surgically induced, can be obtained in the perioperative time frame when appropriate offloading is established in parity with strong patient compliance.

There are four dominant pathways for the development of foot ulcers [6]. These include:

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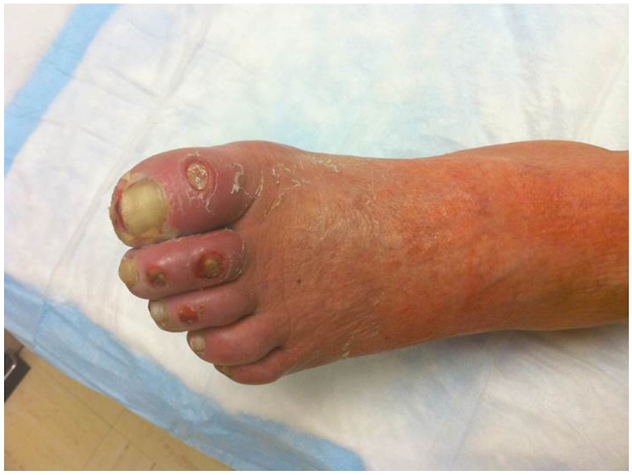

Fig. 1 – Photo of foot with multiple ischemic wounds caused by excessive skin shear stresses due to inappropriately sized shoes.

1. a combination of diabetic neuropathy, deformity, callus, and elevated peak skin pressure;
2. CLI caused by PAD;
3. penetrating trauma; and
4. ill-fitting shoes, or friction-induced skin trauma.

In the search for the cost-effective resolution to resolve these wounds, logic-driven research has resolved itself to answering three principle inquisitions regarding proper management:

1. What are we going to remove from the wound in order to facilitate healing?
2. What are we going to apply to the wound to expedite healing?
3. What steps can we take to prevent recurrence?

A primary evaluation of the patient with a foot ulcer is adequacy of arterial perfusion by arterial physiologic testing. Once adequacy of perfusion for healing has been confirmed, "what can be done to facilitate wound healing?" should be addressed. In limbs with a neuropathic ulcer, the initial management should include regular debridement by sharp surgical device. Proper debridement involves the removal of the necrotic tissues present in the wound bed and the surrounding hypertrophic dermal callus [7]. Wound callus debridement was found to reduce peak plantar pressures by >25% [8]. Debridement of the wound bed is thought to correct several abnormalities, including local excision of necrotic tissue, control of infection, and redirecting the nonhealing ulcer toward a more normal wound-healing sequence. Newer biologic means, such as enzymatic collagenases or maggot therapy, offer their own advantages in terms of cost, time, and targeted specificity of tissue debridement.

Answering the second question of what to apply to the wound is more complex. There are a multitude of wound care options available, each with advantages and limitations specific to the etiology of each wound. Currently, dressings that allow for moist wound healing and control excess wound exudate are the preferred choice. Adjunctive therapies may show some adjunctive value in the wound healing regimen, therefore, clinicians may consider using a combination of skin equivalents, growth factors, granulocyte colony-stimulating factors, and hyperbaric oxygen therapies as needed [9]. Negative pressure wound therapy is very successful when implemented after surgical wound debridement by facilitating exudative wound clearing and a creating a healthy wound bed suitable for surgical closure by skin graft, suture, or secondary intention [10,11].

Elevated plantar foot pressures, particularly in the presence of neuropathy or PAD, are among the strongest risk factors for ulcer development. The lack of sensation and decreased metabolic response renders patients unable to organically detect repetitive stress and trauma to their plantar tissues. When the "gift of pain" is absent, the patient is not aware of the potential damaging skin trauma in performing daily activities, and is unable to alter forces beneath their feet. This ungoverned propagation continues silently, developing into an open wound and a nidus for infection. Currently, the total contact cast (TCC) remains as the gold standard for offloading the active diabetic wound, except in the case of severe ischemia [12,13]. Therefore, it is important to posit the multitude of options available to the physician with respect to best patient care, including TCC, removable cast walkers, and orthotic footwear.

2. TCCs

The use of the TCC was first popularized by Dr. Paul Brand through his work with neuropathic Hansen's Disease patients in Carville, Louisiana in the 1960s [14]. This type of cast obtained its name because of the way it intimately contacts the exact contours of the plantar foot. By extending from the distal toes, across the bony prominences of the metatarsal heads and bony midfoot, and beyond the back of the heel, a TCC takes the direct forces that would be applied to any singular site and grossly distributes them across the cast length. By continuing to transfer direct force pressures up the posterior cast wall, wounds are preserved from direct, forceful trauma, preventing the mechanism that initiated ulcerative development [15–17].

Laboratory research has shown that TCCs can reduce the pressure at the site of a neuropathic ulcer by 84%–92%, and additional studies have shown that TCCs can heal a majority of diabetic neuropathic ulcers in 6–8 weeks [18–20]. Not only is the TCC effective in improving healing, but it can also be used to help control edema and deformation in the initial phases of Charcot neuroarthropathy [21]. Because of its definitive ability to reduce the concentrated plantar pressures that exist on bony prominences, TCC is the best technique to offload plantar pressure of the diabetic foot [7].

Although TCCs possess the advantages listed here, there are drawbacks as well. A TCC is a modification of a traditional fracture cast that requires some skill to apply, because, if applied incorrectly, the skin lesion can worsen, produce additional tissue injury, or allow for soft-tissue infection to progress unchecked (Fig. 2). Typically, the TCC is changed every 1 to 2 weeks, which can be associated with patient