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Physiology and Prevention of Heel Ulcers: The State of Science

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Abstract

The prevalence of heel ulcers across settings is high and is increasing. Prevention of ulcers requires knowledge of their etiology and the scientific basis for preventive care. The interaction between external pressure and the heel vasculature is central to the prevention of heel ulcers. This paper focuses on the prevention of heel pressure ulcers. The physiology of heel tissue perfusion, the effect of external pressure on heel perfusion as well as what is known about strategies to reduce external pressure and approaches to improve heel skin blood flow will be discussed. It is only through understanding of the physiology of heel tissue perfusion and its relation to external pressure that effective preventive measures to reduce heel skin breakdown can be adapted in clinical practice.

The incidence of heel pressure ulcers in the United States is increasing¹. The heel or calcaneus is subjected to repetitive trauma during normal walking and high pressures when patients lie supine or use the heel for a pivot point when in bed. Pressure ulcers in general affect physical and psychosocial function² and impose financial burden on the family and society.³ The annual cost of pressure ulcer treatment in the United States is estimated to range between \$5 billion and \$8.5 billion.⁴ Hospital-acquired pressure ulcers lengthen hospital stay⁵ and nosocomial pressure ulcers represent an incremental cost of \$12,186 per patient.⁴

The prevalence of heel pressure ulcers is high across settings. In hospitalized patients, it ranges between 10% and 18%.¹ Heel ulcers also occur in rehabilitation units and at home. Adult men (n=170) hospitalized on a rehabilitation unit were followed for 12 months to examine pressure ulcer prevalence and incidence.⁶ Forty-six pressure ulcers occurred (27%), with the second most common occurrence in the heel-ankle area (44%). Heel ulcers continue to be prevalent after patients are discharged home. A report on the prevalence of pressure ulcers on 177 home health agencies over 19 states found that the prevalence rate for each agency ranged between 0.5% to 35.7% with 14% of ulcers present on the heel.⁷

Heel ulcers occur in the elderly and the very young. Gunningberg, Lindholm, Carlsson, & Sjoden (1999) studied patients (n=124) over the age of 65 and found that the third most common location of pressure ulcers was the heel (19%).⁸ Meanwhile, heel pressure ulcers have been reported in children. A national survey of pressure ulcer prevalence showed that in 1998, heels were the second most frequent site of pressure ulcers (18.1%) in pediatric patients under the age of 21 (n=138).⁹

Healing of heel ulcers is slow and may result in disruption of mobility with iatrogenic consequences. Heel ulcers often are associated with arterial disease where perfusion pressure is limited due to arteriosclerotic disease or with diabetes where neuropathy interferes with the protective sensation, resulting in repetitive trauma.¹⁰ Regardless of etiology, heel ulcers are associated with impaired perfusion to the heel. Thus, this paper will address prevention of heel ulcers. Specifically, this paper will describe the anatomy of the heel, heel tissue perfusion, the effect of external pressure on blood flow, as well as what is known about strategies to reduce external pressure to the heel and approaches to improve heel skin blood flow.

The Heel

The heel or calcaneus is the most posterior portion of the foot (Figure 1). It is the largest bone in the foot and often bears the brunt of body weight. The dorsalis pedis and lateral plantar arteries supply the heel area. The venous flow in the foot is bi-directional, from the depth of the plantar foot to the superficial dorsal system. The heel pad has an average thickness of 18mm with a mean skin thickness of 0.64mm. Bundles of collagen fibers form the cup ligament, which is attached to the periosteal membrane of the calcaneus by the septa. The fibrous septa form large sealed compartments retaining the fat. The low viscosity of the retained specialized adipose tissue helps dissipate the energy due to walking or external pressure, thus reinforcing shock absorption.¹¹

Heel skin is affected as people age. The capacity for shock absorbency of the heel pad declines with age.¹² The mean blood flow response is inconsistent in elderly at-risk individuals.¹³ Ryan, Thoolen, & Yang (2001) applied external pressure to the skin of the

heel for 10 minutes in 14 volunteers. Elderly subjects showed less resilience of the skin than that of younger subjects.¹⁴

Heel Skin Perfusion/Oxygenation

Tissue Perfusion

Tissue perfusion determines tissue viability and is a function of oxygen delivery, tissue oxygen transport, and oxygen consumption of the cells. Oxygen delivery to the skin depends on adequate peripheral blood flow. Tissue oxygen transport involves the delivery of oxygen and nutrients to the skin and tissue. As oxygen leaves the red blood cells in the capillaries, its diffusion into the surrounding tissues is determined by its partial pressure.¹⁵ High arterial oxygen partial pressure in the vasculature accelerates the rate and depth of oxygen penetration into tissues. When perfusion is adequate, tissue oxygen tension increases. When perfusion diminishes, hemoglobin releases more oxygen but consumption may outstrip availability, resulting in low tissue oxygen tension. Since tissue oxygen utilization is constant and the volume of consumption is quite small, measurement of tissue oxygen partial pressure is a sensitive index of small changes in local blood flow.¹⁶

The regulation of blood flow through the arterioles and arteriovenous anastomotic channels is governed by the nervous system in response to reflex activation by temperature receptors and by higher centers of the central nervous system. External pressure and modulators of the sympathetic nervous system, including pain and blood volume, affect perfusion. Pain results in reduced blood flow.¹⁷ Hypovolemia and

hypothermia lead to skin vasoconstriction. Dehydration and blood loss result in decreased circulatory volume.¹⁸

The subcutaneous bed is one of the first tissues to vasoconstrict to compensate for an acute reduction in circulating volume. It also is the last to regain normal perfusion after restoration of the circulation.¹⁹ When there is enough blood supply to meet the tissue oxygen demand, tissue perfusion is adequate.²⁰

Heel ulcers and external pressure

Heel ulcers result from damages to heel tissue. A heel ulcer may present as a persistent erythematous area, a bluish skin discoloration, a fluid-filled blister, an open wound, or an eschar. Heel pressure ulcers occur on persons of all ages and in all settings including the hospital, nursing home, and patient's residence.¹

Heel ulcers, like pressure ulcers on any site in the body, are caused by pressure that exceeds capillary closing pressure and results in ischemia.²¹ Both high external pressures applied for short durations or low external pressures applied for long durations over bony prominences cause localized areas of cellular necrosis.²¹ Superficial tissue ulceration also is due to mechanical forces acting on localized areas of skin and subcutaneous tissue.²² When patients are in bed and immobile, the source of external pressure can be a bed surface, tight bed-covers, or pressure and friction generated when the leg becomes restless. Heel skin blood flow is reduced with high external pressure.

Patients who have many risk factors such as friction, shear, and moisture are more likely to develop heel ulcers in the hospital when compared to those that have few risk factors.²³ The heel may be more prone to tissue breakdown than some other parts of the

body because it has a small subcutaneous tissue volume with pressure exerted directly on bone.

Reactive hyperemia

An important determinant of heel skin perfusion is external heel pressure. When external pressure is applied to the heel with walking, lying supine in bed with the heel on the bed, or using the foot as a pivot to turn while in bed, flow is decreased or ceases. The cutaneous arterioles below the occlusion dilate.

When pressure is released, dilation of vessels results in increased flow beyond baseline. The increase in flow is visible as erythema or reactive hyperemia. The cutaneous resistance vessels function in autoregulation of blood flow and reactive hyperemia.²⁴ Xakellis et al. (1993) hypothesized that the normal hyperemic response to external pressure is the result of a compensatory vasodilatation of dermal circulation that serves to minimize the damage from the compressive pressure.²⁵

Reduced hyperemic response may result from capillary damage, endothelial dysfunction, capillaries being plugged by leukocytes, or failure of the capillaries to dilate.²⁶ Reduced hyperemic response means reduced perfusion. Lack of the hyperemic response or nonblanchable erythema is a sign of impaired blood supply and tissue destruction and is classified as a stage I pressure ulcer.²⁷

Changes in the hyperemic response

The hyperemic response of the heel skin to external pressure has been examined in several studies. Meinders and colleagues (1996) measured skin microvascular

responses of the footsole to changes in externally applied pressure.²⁸ Skin microvascular blood flow was measured in healthy volunteers (n=11), during and after external mechanical pressure of different magnitudes. Pressures above 40 kPa stop skin microvascular blood flow. Releasing the applied pressure results in a hyperemic response, which increases when the applied pressure increases from 40 to 80 kPa. Higher pressures do not influence the amplitude in skin microvascular response, but result in a longer delay to maximal hyperemia.²⁸

Mayrovitz and his colleagues²⁹ explored the hyperemic response of heel skin. Higher post-pressure heel skin perfusion was recorded in 11 healthy women using laser-Doppler Imaging scan. Heel perfusion was significantly reduced upon pressure loading and during loading while a significant hyperemic response was seen for up to 10 minutes after pressure release.

Mayrovitz and Smith³⁰ (1998) went on to examine the effect of loading and unloading on heel perfusion using laser Doppler Imaging to examine heel blood perfusion in 11 vascularly normal persons before pressure loading (10 minutes), during pressure loading (40 minutes) and after off-loading (20 minutes). Loading was done with subjects supine and one heel on a transparent plate through which heel blood perfusion data were obtained during loading.³⁰ Heel perfusion was rapidly and significantly reduced on loading ($P < 0.01$) with the greatest reduction within the central heel area; perfusion remained uniformly depressed throughout the loading interval while off-loading was associated with a rapid onset hyperemia which exceeded baseline ($P < 0.01$) for 10 minutes.

Mayrovitz et al. then studied the effect of load magnitude and duration on hyperemia response of the heel in 14 vascularly normal women.³¹ A sequential local heel loading with graded magnitudes (30-140 mmHg) and durations (2.5-20 min) were applied. All heel loads and durations resulted in hyperemic responses, with the largest increase in peak response occurring between heel loads of 60 and 120 mmHg. Recovery times also increased with both load duration and magnitude.³¹

The above studies showed that hyperemic response occurs after a period of external pressure with vascular occlusion and relief of external pressure. Time taken for the returning of previous skin blood flow depends on duration and magnitude of external pressure. The relation of hyperemic response of the heel and external pressure has only been studied in healthy subjects and showed that external pressure reduced heel perfusion. Hyperemic responses reaction occurred with relief of pressure. Higher external pressure produces a longer hyperemic response time. Some researchers further suggest that pressure ulcer development may be related to a lack of physiologic increase in blood flow after pressure is relieved.^{25,26} Others propose that repeated cycles of ischemic-reperfusion increase tissue injury.³²

Reducing Heel Pressure

The two approaches to reduce heel pressure are the use of support surfaces and heel specific devices. Little literature is available about either, and the combination of approaches has not yet been systematically examined in those at-risk for heel pressure ulcers.

Support Surfaces

A Medline search was conducted examining support surfaces and heel pressure. Studies in the support surface section are included here if they addressed heel interface pressure, heel microcirculation, or incidence of pressure ulcer development (Table 1). The effectiveness of various support surfaces for reduction of pressure ulcer and or wound healing has been examined in many studies. The physiological mechanism of their benefit has not been widely evaluated.

Most of the studies use interface pressure between heel and bed surface as the criterion for measuring the effectiveness of support surfaces. Studies comparing different types of pressure reduction mattresses showed that the interface pressure of the heel remained greater than the buttock and the trochanter.^{33,34} Most of the support surfaces had exceeded the acceptable capillary closing pressure,³⁵ or had no significant reduction on heel pressure.³⁶⁻³⁸ Heel interface pressure remains lower in pressure reduction/relieving surfaces than in conventional hospital mattresses.^{36,39,40}

Heel specific devices

Less work has been done on heel specific devices than on support surfaces as prevention for pressure ulcers. A Medline search was conducted and literature is included here if it addresses heel devices and pressure.

There are a variety of commercial heel pressure relief/reduction devices and they can be categorized in the following groups.

- * Heel protectors/heel pads: these heel protectors are usually made of polyester fibers, soft fabric, foam pad, or sheepskin. Some products have built-in, heat-

activated skin protectant. They are good for protection of the heel against friction.

- * Foot product with plastazote that molds to the shape of the foot to reduce shear.
- * Foot boots: protectors that contain gelastic material or gel pad to contour around the heel.
- * Heel elevation devices: are made of foam or inflatable air chambers. Relief of heel pressure is achieved by having an opening at the heel location of the device.

Some data exist that compared heel devices (Table 2). Studies that compared various heel devices found that heel protectors that are made up of foam were able to maintain heel skin integrity.⁴¹ When comparing hospital pillows with inflatable heel elevation devices, pillows were able to keep heels off the bed more often than heel elevation devices.⁴² In another comparison study of 13 heel-protecting devices, an ordinary head pillow was shown to be the most effective pressure-reducing device.⁴³ The best heel pressure relief/reduction product has not been identified. In fact, proper fitting of the device, patient activity level, and leg movements all affect the effectiveness of heel specific devices. Many devices are used in clinical practice to relieve/reduce heel pressure yet there remains little scientific evidence as to which is best. More studies on the efficacy of heel pressure relief/reduction products used on at-risk patients are needed.

Strategies to improve heel skin blood flow

Few studies focus solely on improving heel skin perfusion. Studies on wounds showed that relieving wound pressure markedly improve tissue oxygen tension.⁴⁴ Local wound heating was also found to improve tissue oxygen tension in post-operative

patients.⁴⁴ Pressure ulcer healing rate increased with warming of ulcerated area.⁴⁵ Electrostimulation has been shown to improve oxygen delivery and perfusion pressure to patients' legs with impaired arterial circulation.⁴⁶ The effect of intermittent pneumatic compression of the legs was studied in persons who had impaired distal perfusion. Data showed that this intervention resulted in a significant increase in foot skin perfusion.⁴⁷ None of these therapies i.e., electrical stimulation, external heat, and or intermittent pneumatic compression, has been examined to determine their effect on heel pressure ulcer prevention with the dependent variable being pressure ulcer development.

Heel skin blood flow has been studied during intermittent cycles of pressure-relief.⁴⁸ Mayrovitz and Sims (2002) measure heel skin blood flow in 20 healthy subjects using laser-Doppler. Mattresses that provided cycles of pressure-relief increased heel skin blood flow. Hyperemia during pressure relief compensated for flow deficits during pressure. However, this method has only been applied to healthy individuals.⁴⁸

Relieving heel pressure remains to be an important strategy to improve heel skin blood flow. Strategies to improve heel skin flow to prevent pressure ulcers have not been tested in patients at risk for pressure ulcers.

Conclusion

Tissue ischemia predisposes to heel ulcer formation. The integrity of heel tissue is maintained by adequate heel skin perfusion. Heel perfusion is directly affected by external pressure or internally by arteriole disease, diabetes, circulatory problems, changes in the hyperemic response, and age. Preventing heel tissue breakdown involves reducing external pressure, and improving heel perfusion and oxygenation.

Preventive measures should aim at reduction of the magnitude and duration of external pressure. This includes daily assessment of heel skin for color and changes in integrity, removing supportive hose daily for skin assessment, and subjecting the heels to minimal external pressure. Keeping heels off the bed with pillows is the best documented approach in relieving heel interface pressure. Special pressure-reduction beds/mattresses reduce heel pressure better than conventional hospital beds.

The most effective heel pressure relief device is yet to be identified. Strategies used to increase blood flow in other populations included electrical stimulation, external warmth, and intermittent compression. These have not been explored in patients at high risk for heel pressure ulcers. To date, little is known about effective strategies to improve heel blood flow in at risk patients.

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Figure 1. Medial aspect of foot

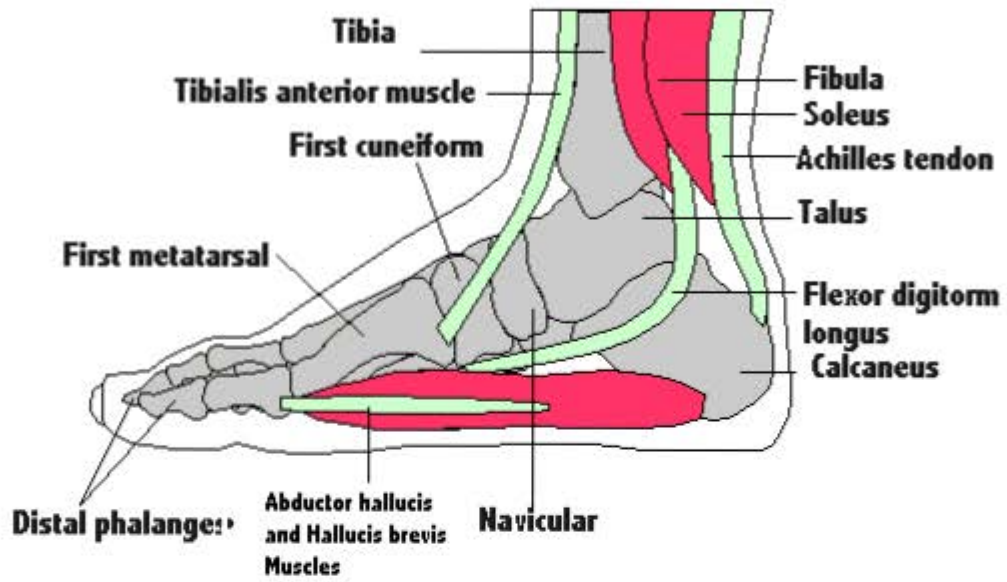


Table 1 Comparison studies on pressure-reduction products and the effect on heel pressure.

Author & year	Purpose of study	Independent variables	Measuring construct	Sample	Outcomes in relation to heel pressure
Thompson-Bishop & Mottola ³⁶ (1992)	To compare the pressure-reducing properties.	11 different pressure-reducing devices & standard hospital mattress	Mean heel pressure readings using an electropneumatic pressure transducer	13 healthy adult volunteers	Mean heel pressure readings on the pressure-reducing support surface were lower than on the standard hospital mattress, none of them is capable of preventing tissue ischemia if the subcutaneous pressure is three to five times higher than the interface pressure.
Allen et al. ³³ (1993)	Pressures were measured under five anatomical sites with patients in supine and sitting positions	2 different air-fluidized beds	Interface pressure	10 subjects	Though pressures did not rise above the accepted capillary closing pressure, on either bed, heel pressures were 2.67 times greater than buttock pressure.
Hedrick et al. ³⁴ (1993)	Examine effects of products at interface sites of sacral, trochanter, and heel	7 pressure-reducing products	Mean pressure readings at the interface sites	15 healthy volunteers	No product had acceptable readings at the heel site*.

Whittemore et al. ⁴⁰ (1993)	Determine tissue interface measurements obtained under the sacrum and heel bony prominences in the supine and 45-degree Fowler's positions.	Static air overlays, replacement mattresses, and a standard hospital mattress	Tissue interface pressures	25 healthy subjects	Heel interface pressure measurements were significantly higher than sacral interface pressure measurements. There was no significant difference between supine and 45-degree Fowler's positions with respect to tissue interface pressures
Abu-Own et al. ³⁹ (1995)	To assess the effects of compression on the skin	Conventional hospital bed and low air-loss bed system	Microcirculation of the heel using laser Doppler fluxmetry and interface pressure	Ten patients at risk of developing pressure ulceration and ten healthy young volunteers	Heel microcirculation is vulnerable to compression. The low air-loss system maintained sufficiently low interface pressure to prevent cessation of heel microcirculation.
Fontaine et al. ³⁷ (1998)	Compare pressure and shear in a controlled laboratory setting	3 support surfaces	Interface pressure and shear	11 healthy subjects	Pressure measurements taken at the heels were not significantly different for any of the surfaces
Hardin et al. ³⁸ (2000)	Compare the effectiveness of 2 pressure-relieving surfaces	Low-air-loss mattress & static fluid mattress	Tissue interface pressures	73 post-op transplant patients	Pressure did not differ significantly at the heel site

* An acceptable reading is below the capillary closing pressure of 32mmHg.

Table 2 Comparison studies on heel specific devices and the effect on heel pressure.

Author & year	Purpose of study	Independent variables	Measuring construct	Sample	Outcome in relation to heel pressure
De Keyser et al. ⁴³ (1994)	Examine the pressure-reducing effects of heel-protecting devices	13 different heel protecting devices	Vertical measurement of heel pressure	40 elderly patients	An ordinary head pillow was shown to be the most effective pressure-reducing device.
Zernike ⁴⁹ (1994)	Compare heel pressure relieving devices	4 pressure-reducing devices	Skin integrity over a period of 12 days	41 patients with fracture femurs	Meticulous nursing care is the critical element, use of eggshell foam and form splints helps
Tymec et al. ⁴² (1997)	Examine the effectiveness of preventing heel ulcers	Hospital pillows versus a commercial heel elevation device	Heel interface pressures were taken with patients in supine and right lateral tilt positions.	52 patients	There was no significant difference between groups in incidence of pressure ulcers. Patients using the heel elevation device developed pressure ulcers significantly sooner
Randolph et al. ⁵⁰ (1999)	Evaluation of orthosis purported to decrease pressure on heel while walking	Multipodus System orthotic device	Pressures exerted on the plantar surface of the hindfoot, midfoot, & forefoot	10 subjects	Heel pressure was reduced significantly compared to the ordinary shoes using both the flat bottom and the rocker bottom boot.

