

EFFECTS OF SUPPORT SURFACE RELIEF PRESSURES ON HEEL SKIN BLOOD PERFUSION

HN Mayrovitz, N Sims, MC Taylor, L Dribin, College of Medical Sciences, Nova Southeastern University, Fort Lauderdale, FL

INTRODUCTION

Pressure ulcers due to sustained unrelieved or inadequately relieved pressure, are an important clinical, humanitarian and economic problem.¹⁻³ Pressure dependent blood flow changes play a major role in the skin breakdown process with the greatest breakdown frequency at sites of bony prominences. The heel is particularly prone to such effects⁴, in part because of its relatively lower resting blood perfusion level⁵, and higher amounts of experienced surface pressure when under load⁶⁻⁹. Local blood flow decreases during heel loading⁵ and flow recovery after unloading are involved in the breakdown process¹⁰⁻¹². Previous work has shown that when the pressure supporting the heel was cycled at different rates, the average blood flow over complete cycles was significantly greater when the level of pressure to which the heel was released was zero (full release) as compared to a nonzero pressure value (partial release).¹³ However, because only two levels of pressure relief were investigated, complete and partial, the blood flow effects of intermediary levels of pressure relief are unknown. Thus the present study sought to characterize the flow responses of the heel under conditions in which the heel was supported with a uniform pressure magnitude and duration but with three separate relief pressure levels.

METHODS

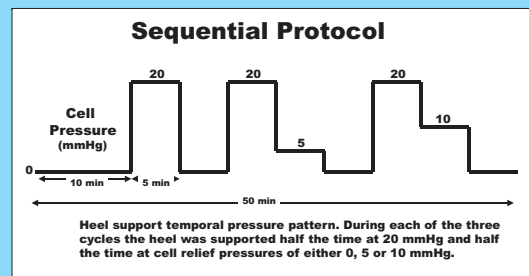
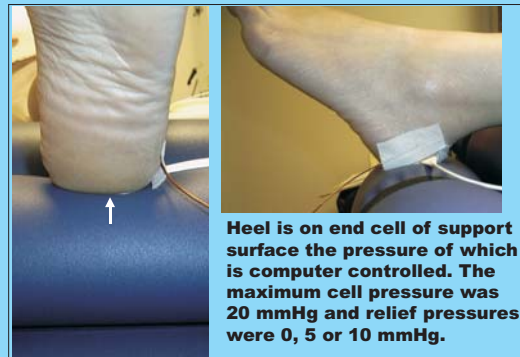
Subjects: Twelve volunteers (5 male) randomly drawn from medical school students were tested. Subjects were free of lower extremity vascular disease with pre-test ankle-brachial pressure indices of 1.13 ± 0.02 . None were taking medications that would impact on vascular reactivity. Group features (mean \pm sem) were for age 29.8 ± 3.1 years, height 66.4 ± 1.2 inches and for weight 148 ± 7 pounds. Systolic, diastolic and mean blood pressures were normal at 107 ± 7 , 67 ± 2 and 80.3 ± 2.6 mmHg. No subject had diabetes or any notable medical history.

Protocol and Support Patterns: Subjects lay on a support surface with their heel positioned on the end cell of a support surface. Pressure in this supporting cell was under computer control, and could be made to vary between a constant upper limit of 20 mmHg and a variable lower limit of 10, 5 or 0 mmHg on a cyclic basis. The overall test sequence was 50 minutes. The first cyclic pattern was initiated after a baseline recording interval of 10 minutes in which the heel was not loaded (0 mmHg). Tests were conducted in a room with a well-controlled ambient temperature.

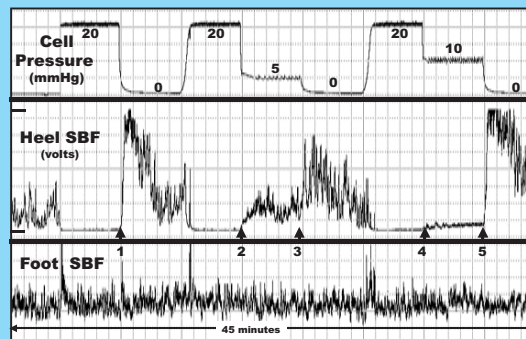
Blood Perfusion: Heel skin blood perfusion (SBF) was monitored continuously with a thin, flat laser-Doppler probe affixed to the heel at the site of surface contact with tape. A second probe was placed on the foot dorsum just proximal to the union of the great and second toe. Foot SBF was used to judge if systemic changes in SBF occurred during the procedure. At the end, the biological zero of both laser-Doppler probes were determined using a thigh cuff that was inflated to 40 mmHg above systolic blood pressure for two minutes. The biological zero value was subtracted from all laser-Doppler raw values.

Interface Pressure: At the end of the sequence, heel interface pressures (IP) were measured by a sensor placed between the heel and the supporting cell. Six measurements of IP at each cell pressure were averaged.

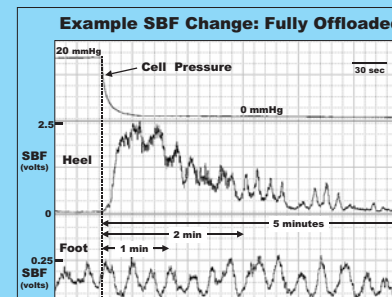
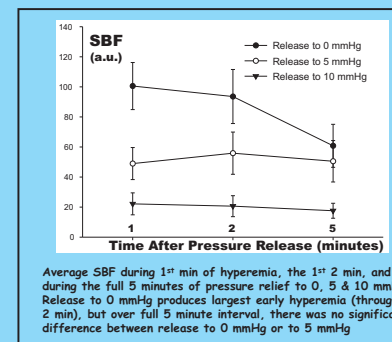
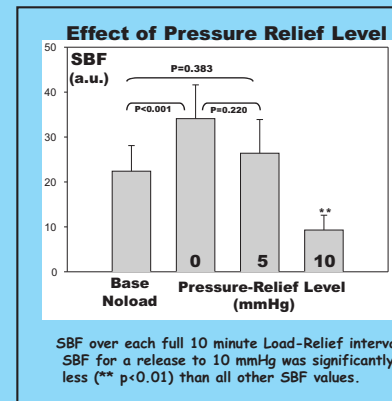
METHODS



Typical Experimental Responses



RESULTS



Interface Pressures: With end cell internal pressures set at 20, 10 and 5 mmHg, interface pressures (mmHg) were 140.9 ± 8.5 (range 109-176), 78.6 ± 1.3 (73-83) and 44.2 ± 3.1 (39-60). This wide variation among subjects within cell pressures reflects the dependence of IP on multiple factors such as foot position, body habitus and heel shape. These IP levels indicate that the maximum cell pressure (20 mmHg) was greater than the average systolic pressure (107 mmHg), the cell pressure of 10 mmHg was slightly less than the group average diastolic pressure (80.3 mmHg), and the support cell pressure of 5 mmHg was less than the group average diastolic pressure.

Features of SBF Responses: Cell pressurization to 20 mmHg caused a decrease in SBF to a level that was at or close to the biological zero. This indicates that the maximum cell pressure caused a heel ischemia for all or most of its application. Foot dorsum SBF was not affected by either cell pressurization or pressure relief, indicating that heel SBF change was a localized phenomena. The SBF change accompanying pressure relief depended on the relief pressure level. Thus, release to zero mmHg was always associated with significant hyperemia, release to 5 mmHg normally had some hyperemia, and release to 10 mmHg cell pressure had a marginal or absent hyperemic response. When the hyperemia was low or absent during the relief pressure, subsequent release to 0 mmHg was always associated with a significant further flow increase.

CONCLUSIONS

Results emphasize the important role of pressure relief level in dynamic surfaces for pressure ulcer prevention. Full alternation appears superior to partial or no alternation in achieving good levels of perfusion in heels. Since no standard exists as to what 'alternating' means, it is prudent to know full details of the exact nature of any product's alternating cycle before purchasing.

These findings, and other data¹⁵⁻¹⁷, indicate that a suitable non-zero relief pressure depends on the relation between a patient's diastolic blood pressure and tissue forces on heel blood vessels. Thus, lower blood pressures likely need lower pressure-relief levels, a concept well worth keeping in mind if dealing with patients who are hypotensive. The present results apply strictly if normal hyperemia potential is present. Impacts of depressed vascular responsiveness and/or diminished hyperemic reserve on qualitative and quantitative aspects are unknown. However, it is suspected that for such conditions (diabetes or peripheral vascular disease), relief-pressure would need to be reduced. Characterizing these patient groups represents an important major investigative challenge.

References

1. Allman R. Clinics in Geriatric Medicine, 1997;13:421-436. 2. Barczak CA et al. Advances in Wound Care 1997;10:18-26. 3. Scheer RM, Longene DC. J Wound Ostomy Continence Nurs 1998;25:36-43. 4. Gruff AM, Bryant J, Benlich N. Orthop Nurs 2000;19:63-69. 5. Abu-Owen A et al. Eur J Vasc Endovasc Surg 1995;9:327-334. 6. Ek A, Gustavsson G, Lewis D, Scand J Rehabil Med 1987;19:121-126. 7. Counsell C et al. J Enterostomal Ther 1990;17: 150-153. 8. Allen V, Ryan DW, Murray A. Br J Clin Pract 1993;47:195-197. 9. Allen V, Ryan DW, Murray A. Br J Clin Pract 1994;48:125-129. 10. Mayrovitz HN et al. Ostomy/Wound Management 1997;43:16-26. 11. Mayrovitz HN, Smith J. Microcirculation 1998;2:227-233. 12. Mayrovitz HN, Smith J. Clinical Physiology 1999;19:351-359. 13. Mayrovitz HN. Advances in Skin and Wound Care (2002, in press). 14. Mayrovitz HN, Leatham J. Microvasc Res 2001;62:74-78. 15. Nielsen HV. Clinical Physiology 1982;2:447-457. 16. Nielsen HV. Danish Medical Bulletin 1984;31:425-438. 17. Nielsen HV. Acta Physiol Scand 1991;143(Suppl 603): 85-92.