Blood perfusion hyperaemia in response to graded loading of human heels assessed by laser-Doppler imaging

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Received 6 October 1998; accepted 30 December 1998

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Summary

Heel pressure ulcers are important clinical, humanitarian and economic problems arising in part from localized blood flow deficits during loading and inadequate flow recovery. Because there are few data available with regard to the intrinsic physiological responses of heel skin to pressure-induced ischaemia, the present study was undertaken to characterize the main features of the post-loading hyperaemic response. Laser-Doppler perfusion imaging was used to measure hyperaemia in 14 vascularly normal women who were subjected to sequential local heel loading with graded magnitudes (30–140 mmHg) and durations (2·5–20 min). Peak heel perfusion produced by local heating to 44°C for 5 min was used as a comparison standard. All heel loads and durations resulted in hyperaemic responses, with the largest increase in peak response occurring between heel loads of 60 and 120 mmHg. During this transition, peak hyperaemia increased from about 32% to 79% of the local maximal microvascular vasodilatory capacity. Recovery times also increased with both load duration and magnitude, with the longest recovery time being about 7·5 min. Hyperaemic responses and recovery times were analytically dependent on the heel load pressure duration product, with evidence of suppression of the peak response at 1500 mmHg min and a levelling off of recovery time at higher pressure durations. These findings serve to characterize normal physiological perfusion responses to pressure-induced ischaemia at an anatomical site prone to pressure ulceration. The results suggest the possibility of a 'critical' heel loading, above which a near-maximum response is elicited and beyond which vasodilatory recovery potential is blunted.

Keywords: blood flow, ischaemia, microcirculation, pressure ulcers, vasodilation.

Introduction

Pressure ulcer development, resulting from sustained and unrelieved pressure during extended cardiovascular, orthopaedic and other surgical procedures, as well as in other acute and long-term care settings, is an important clinical, humanitarian and economic problem (Papantonio *et al*., 1994; Regan *et al*., 1995; Allman, 1997). Many factors contribute to this (Kemp *et al*., 1990; Jesurum *et al*., 1996; Tourtual *et al*., 1997) but, in most instances, the final common pathway is associated with blood flow changes within pressure-loaded tissue. These detrimental blood flow changes affect the skin breakdown process in multiple ways (Kosiak, 1959; Sanada *et al*., 1997; Mayrovitz, 1998a), usually at sites of bony prominences including sacral, greater trochanter and heel regions (Meehan, 1990; Cheneworth *et al*., 1994). The heel is particularly prone to such effects, in part because of its relatively lower resting blood perfusion level, higher amounts of experienced surface pressure when under

load and the possibility of further compromise of local blood flow when lower extremity arterial disease is present. Although localized blood flow changes during heel loading and flow recovery after unloading are factors that affect skin breakdown processes, there are few data available with regard to heel skin responses (Mayrovitz & Smith, 1998). Because of the importance of this issue (Barczak *et al*., 1997) and the need for basic information, the present report is concerned with the investigation and characterization of skin blood perfusion hyperaemia after the application of standardized local heel loads for variable durations. Such information is targeted at providing further insights into the way in which this physiological response to pressure-induced ischaemia manifests itself at this clinically important anatomical site. To obtain this information, a fixed weight load was applied sequentially to the heel of 14 women for durations of 2·5–20 min followed 1 week later by the application of graded load magnitudes for a fixed duration of 10 min. In all cases, the blood perfusion hyperaemic response after load removal was measured by laser-Doppler imaging and characterized in terms of its magnitude and recovery time.

Methods

Subjects

Fourteen female volunteer subjects (four African Americans, five Hispanics and five Caucasians) ranging in age from 24 to 61 years (43·7 ± 2·8 years) participated in this study. Absence of significant lower extremity arterial disease was confirmed in each participant based on screening with bilateral nuclear magnetic resonance (NMR) flowmetry (Mayrovitz & Larsen, 1996; Mayrovitz, 1998b) and ankle-brachial systolic pressure indices (ABI) obtained using standard Doppler ultrasound at the posterior tibia and dorsal pedis arteries. Average flow (Q) measured at mid-calf determined by NMR flowmetry was 27·4 ± 2·3 and 31·1 ± 3·0 ml min^{-1} for right and left legs respectively. Corresponding ABIs were 1·1 ± 0·02 and 1·0 ± 0·02. Blood pressures measured with standard pressure cuffs also verified that average systemic blood pressure was within normal ranges (124·3 ± 6·0 to 78·3 ± 3·1 mmHg.). No subject had a history of vascular disease, diabetes or was taking vasoactive medication.

Initial procedures

Subjects entered a testing laboratory that was temperature controlled (22–23°C). They were asked to remove shoes and socks and were placed in a prone position on an examination table with their feet hanging over the edge of the table. A light blanket was placed over the subject. The location of the centre of the calcaneal prominence on the posterior surface of the left heel was marked for later use as a reference site for tissue loading.

Blood perfusion assessments

A laser-Doppler imaging (LDI) system (Moor Instruments, Devon, UK) was used to measure skin blood perfusion via a laser beam (780 nm) aimed at the heel skin with subjects lying prone. Although similar in principle and some data-processing respects to standard laser-Doppler, a major advantage of LDI is its ability to acquire perfusion via non-contact measurements over a larger area (Seifalian *et al*., 1994; Wardell *et al*., 1994; Abbot *et al*., 1996). Low-power laser light is directed across the skin by a moving mirror that executes a raster pattern. This permits data acquisition over a defined tissue area that, on analysis, allows the inclusion of spatial heterogeneity effects, thereby facilitating a more accurate view of tissue perfusion changes in response to vasoactive stimuli. As is standard for laser-Doppler measurements, results are reported in arbitrary units (a.u.). All measurements were obtained at a distance of 30 cm from the skin. Each scan encompassed an area of ≈2·0 cm × 2·0 cm (90 × 90 pixels) and took ≈50 s for each scan. System calibration was done before each experimental day using a standard phantom target consisting of a 0·5% solution of 10-μm-diameter polystyrene spheres in sterile water.

Fixed load magnitude protocol

With subjects in a prone relaxed position, the laser beam was centred initially on the previously made reference mark on the heel. After 10 min of subject rest and acclimatization, a single LDI baseline scan was obtained of the left heel before any loading. A stainless steel rod measuring 20·8 cm in length, 1·27 cm in diameter (D), with mass (m) of 206 g was

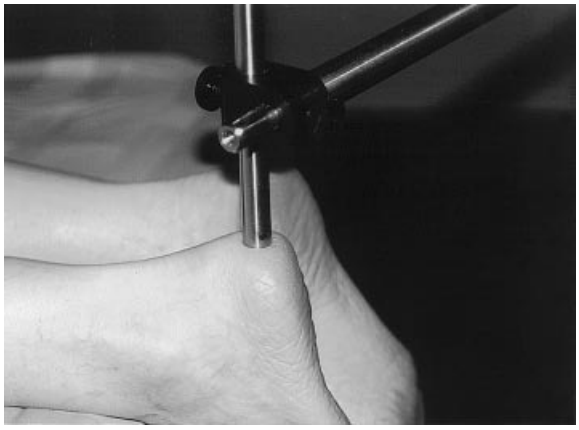

Figure 1 Load applied to the posterior surface of the calcaneal tuberosity.

then placed vertically on the loading site with the support of a specially constructed stand (see Fig. 1). The pressure, P, produced by the rod was estimated by calculating $P = ma/A$, where $m =$ mass of rod (g), $a = 980$ cm s^{-2}, $A =$ rod cross-sectional area $= \pi D^2$. Using the numerical values in the expression for P and multiplying by $7{\cdot}5 \times 10^{-4}$ to convert from cgs units to mmHg, yields a pressure of 119·52 mmHg, which was then rounded to 120 mmHg. Similar calculations were used for all rods. The rod remained in place for 2·5 min and was then removed and an LDI scan started immediately. LDI scans were started every minute after off-loading until the heel blood perfusion had returned to baseline values. A new baseline scan was then acquired, and the rod was placed on the same site for 5 min. LDI scans were obtained every minute after off-loading, and the procedure was repeated for additional loading durations of 10 and 20 min.

Maximal vasodilatory response to local heating

After completion of the loading sequence, and following an additional rest interval of 5 min, a new baseline LDI scan was obtained. A small circular heater probe (≈2·1 cm in diameter) was then centred on the mark on the same site that had been loaded previously. A small piece of silk tape was used to affix the preheated (to 35°C) probe to the skin lightly. The heater was then activated to reach a skin interface temperature of 44°C, which was maintained for 4 min. Thereafter, the heater was removed, and an LDI scan was obtained immediately.

Fixed load duration protocol

One week later, subjects returned to the laboratory and were again placed in a prone position. The left heel was remarked if necessary and, after a 10-min rest period, a single baseline LDI scan was obtained. The site was then loaded serially with stainless steel rods calculated to induce skin pressures of 30, 60, 120 and 140 mmHg. The rods, all 1·27 cm in diameter, measured 5·2, 10·4, 20·8 and 24·1 cm in length, with corresponding masses of 52, 104, 206 and 241 g. Each loading period lasted 10 min. A new baseline LDI scan was acquired before each loading period. Immediately after load removal, LDI scans were obtained as described previously, and vasodilatory response to heat was measured.

Data reduction and analyses

LDI data were analysed using processing software (Moor Instruments, version 3·0). Uniform areas, equal to the area of skin in contact with the loading rod and surrounding the centre point, were used to assess tissue perfusion on each heel region. The spatial mean of all pixels within this region was used to determine perfusion. Baseline and hyperaemic perfusions were determined using the same areas. Parameters used to characterize the magnitude of the post-loading hyperaemia were the perfusion ratio (post-load perfusion/baseline perfusion) and the post-load perfusion expressed as a percentage of the peak heat response. Hyperaemia duration was assessed by its recovery time, defined as the time for the post-load perfusion to return to baseline. Data in text and tables are reported as means ± SEM.

Results

Sequential scan images

Figure 2 shows typical LDI perfusion images obtained before, and for 7 consecutive minutes after, loading a heel with 120 mmHg for 10 min. The image is rendered in pseudocolour with deep blue representing the contours of the lowest perfusion and

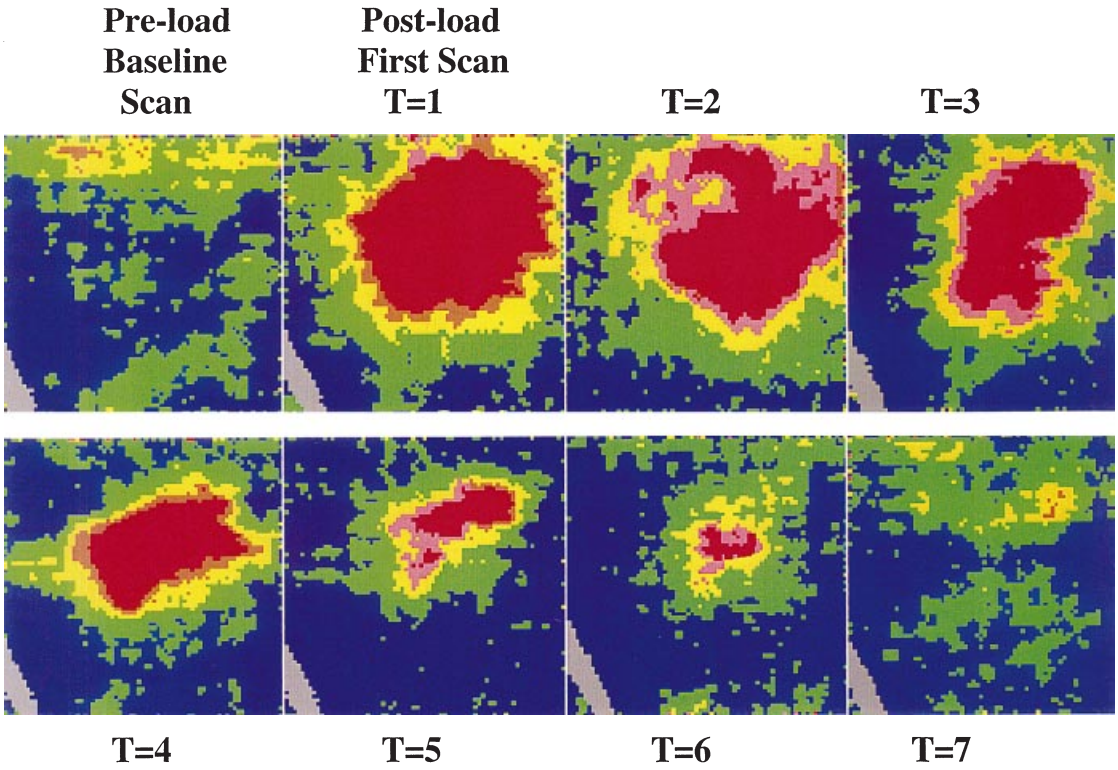

Figure 2 Perfusion images before and after loading a heel with 120 mmHg for 10 min. Top left: the perfusion image before loading (labelled baseline), and the adjacent image (T1) was taken immediately after removal of the 120 mmHg load. Each of the subsequent images are 1 min apart and are labelled consecutively as T2 to T7. The scan area for each image corresponds to a heel area of 2·0 cm × 2·0 cm. For the coloured renditions, deep blue represents the lowest perfusion and red the highest. Intervening perfusion levels are rendered in light blue to green to yellow in ascending order. The centre of each of the images is approximately the centre point of where the heel load was placed. After the initial hyperaemic scan, there is a contracting zone of perfusion elevation approximately circumscribing the previously loaded centre with an eventual return to near baseline.

deep red the highest. Intervening perfusion levels are rendered in light blue to green to yellow in ascending order. These colour renderings are controllable by the equipment according to selectable perfusion ranges. All images are at the same settings. The centre of each of the images is approximately the centre point of where the heel load was placed. After the initial hyperaemic scan, the images show a contracting zone of perfusion elevation approximately circumscribing the previously loaded centre. It is also noted that, by 7 min, the hyperaemia appears visually to have returned to baseline.

Maximal heat response

The maximum blood perfusion increase subsequent to local skin heating provides an index of the maximum vasodilatory capacity of each subject. Although baseline perfusion values may vary in individuals when assessed at different times, if responses are normalized to the local maximal response measured during the same session, the variability may be decreased. In Table 1, the group mean perfusion values obtained before heating, after heating and their ratio are shown for the fixed load magnitude experimental session and for the fixed load duration session. Comparing the fixed load vs. fixed duration sessions (1 week apart), during the fixed load sessions, subjects tended to have higher preheat and post-heat perfusions, although neither was significantly different (P>0·10). However, the peak/baseline ratios were very close (8·30 ± 1·40 vs. 8·03 ± 1·70), suggesting that the overall group vasodilatory capacity at each session was similar.

Table 1 Heel skin response to 44°C heating.

n = 14	Preheat baseline	Post-heat peak	Peak/baseline
Fixed load	102·0 ± 28·0	614·5 ± 93·6	8·30 ± 1·40
Fixed duration	67·3 ± 7·0	522·0 ± 111·9	8·03 ± 1·70

Data are means ± SEM.

Post-loading recovery perfusions

Overall peak hyperaemic responses after heel load removal are shown in Fig. 3 for the fixed load (A) and fixed duration (B) protocols. During the off-loaded recovery interval, hyperaemic perfusions are expressed as a percentage of the maximum perfusion produced by the standardized heat provocation. As seen in Fig. 3A, the 120 mmHg load was associated with a substantial peak hyperaemic response, even for the shortest load duration of 2·5 min (49·4 ± 6·4%). Maximum responses occurred after load durations of 5 min (74·1 ± 9·9%) and 10 min (71·3 ± 6·5%). After 20 min of loading, the peak hyperaemic response, after load removal, was less than that produced by either the 5- or 10-min loading durations, being 57·9 ± 6·4% of the heat response. As shown in Fig. 3B, the 10-min loading protocol was associated with a progressive increase in peak hyperaemic perfusion up to a load magnitude of 120 mmHg. The greatest change occurred between 60 and 120 mmHg, with corresponding perfusions of 31·9 ± 4·8% and 79·0 ± 11·7%. After loading with 140 mmHg, perfusion (73·5 ± 10·8%) did not differ significantly from that at 120 mmHg. Similar response patterns were obtained if post-load perfusion was expressed relative to preload baseline, as summarized in Table 2.

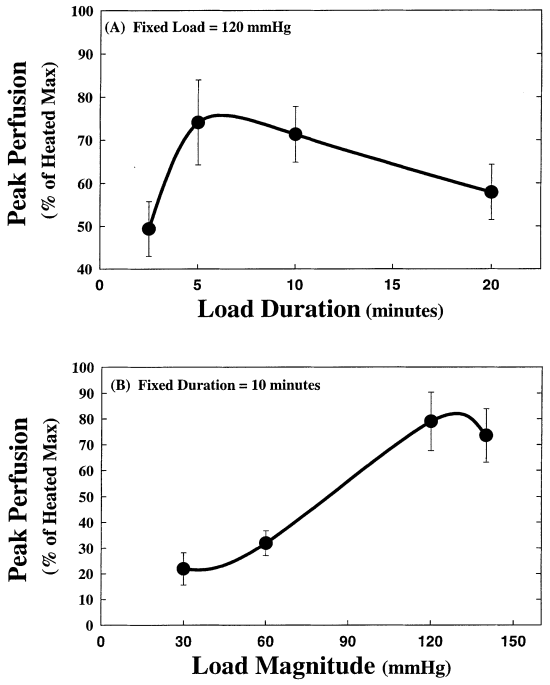

Figure 3 Peak post-loading perfusion hyperaemia. Peak hyperaemic responses after heel load removal expressed as a percentage of the maximum heat response for a fixed magnitude of 120 mmHg (A) and for a fixed duration of 10 min (B). Data points are means ± SEM connected via a best-fit spline curve. Fixed load and fixed duration data were obtained 1 week apart but from the same subjects.

Hyperaemia recovery time

Recovery times (T_r) increased with load duration (D) and load pressure (P), as shown graphically in Fig. 4 (means ± SEM data in Table 2 for reference). Nonlinear regressions were used to fit all recovery time data ($n = 56$) for each protocol (14 subjects × four durations or four load magnitudes). These yielded the following best-fit relations: $T_r = 2{\cdot}58D^{0{\cdot}36}$ ($r^2 = 0{\cdot}419$, $P<0{\cdot}001$) and $T_r = 0{\cdot}12P^{0{\cdot}79}$ ($r^2 = 0{\cdot}600$, $P<0{\cdot}001$) for fixed load and fixed duration protocols respectively.

Hyperaemic response dependence on pressure–time product

Combining the data for fixed magnitude and fixed duration protocols permits an assessment of the relationship between the post-load hyperaemic response and the load pressure–duration product ($P*D$). Figure 5A shows the resultant peak/baseline perfusion ratio as a function of this pressure–time product. The perfusion ratios (Q) obtained for each of the $P*D$ load products ($n = 8$) are fitted by a quadratic function yielding the equation $Q = 0{\cdot}338 + 0{\cdot}006\,(PD) - 0{\cdot}000002(PD)^2$ with an r^2 of 0·802.

Recovery time dependence on pressure–time product

Figure 5B shows the post-loading recovery time (T_r) as a function of the pressure–time product for each of the pressure–time combinations ($n = 8$). The best-fit quadratic regression yields the relationship between

Table 2 Perfusion ratios and recovery times.

Load magnitude = 120 mmHg			Load duration = 10 min		
Duration (min)	Perfusion ratio (peak/baseline)	Recovery (min)	Pressure (mmHg)	Perfusion ratio (peak/baseline)	Recovery (min)
2·5	2·73 ± 0·38	3·57 ± 0·25	30	1·17 ± 0·13	1·93 ± 0·27
5	4·10 ± 0·49	4·79 ± 0·33	60	2·46 ± 0·78	3·42 ± 0·36
10	4·72 ± 0·82	7·07 ± 0·68	120	4·79 ± 0·65	5·79 ± 0·45
20	3·76 ± 0·62	7·57 ± 0·84	140	5·04 ± 0·90	5·71 ± 0·40

Data are means ± SEM.

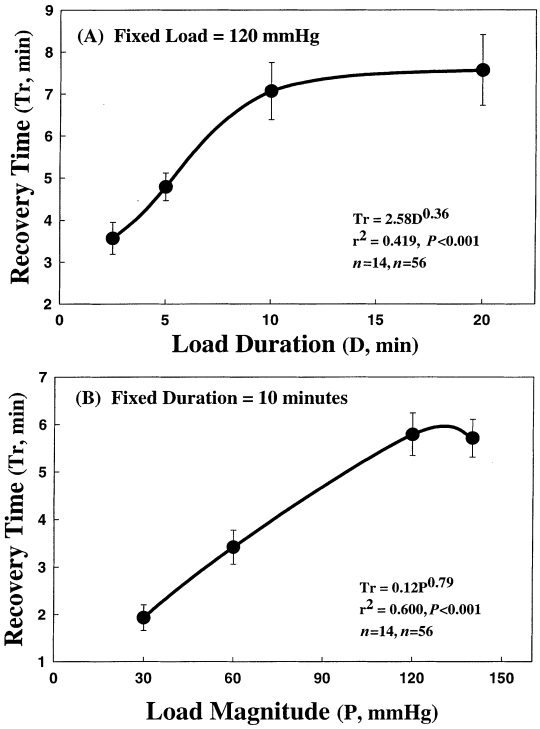

Figure 4 Hyperaemia recovery times. Time for post-loading perfusion to return to preload baseline for fixed magnitude loading (A) and for fixed duration loading (B). Fixed load and fixed duration data were obtained 1 week apart but from the same subjects.

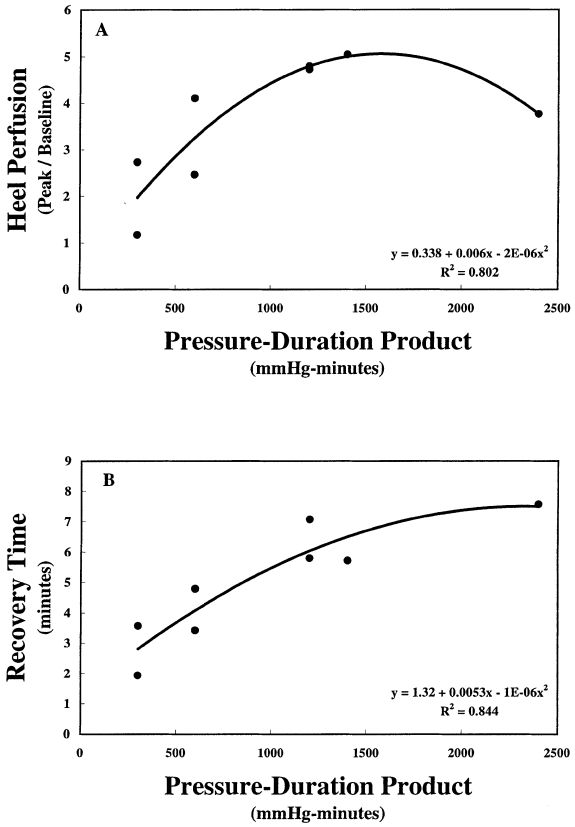

Figure 5 Dependence of hyperaemia peak and recovery on load pressure–duration. The peak/baseline perfusion ratio (A) and recovery time (B) as functions of pressure–duration product are fitted by non-linear quadratic regressions with parameters as shown. The data set is of the combined fixed load and fixed duration protocols. Data points are mean values.

T_r and the P^*D product given by $T_r = 1{\cdot}32 + 0{\cdot}0053(PD) - 0{\cdot}000001(PD)^2$ with an r^2 of 0·844.

Discussion

Hyperaemic responses and recovery times

The main new experimental findings relate to the peak hyperaemic response and associated recovery times subsequent to localized heel loading. Peak post-loading responses are shown to depend on applied load magnitude and duration of application. Heels loaded for 10-min intervals show peak hyperaemic responses that increase with increasing load magnitudes up to 120 mmHg, with a threshold-like large

increase between 60 and 120 mmHg. Comparisons with peak perfusion levels elicited by localized skin heating show that post-loading responses are associated with a near-maximal vasodilatory state, with largest post-pressure responses about 79% of the heat response. When heels are loaded with a fixed magnitude of 120 mmHg for 5–10 min, similar peak hyperaemic perfusion levels (74% of heat response) are found. Accounting for the combined effects of load magnitude and duration via the pressure–duration product shows that heel post-load hyperaemia increases up to a level of about 1500 mmHg min, but beyond this there is a tendency for the hyperaemic response to turn downwards. However, as the largest pressure–duration data result from a load duration of 20 min, it is not yet known whether a similar downward trend would be observed with an equal pressure–duration product derived from a shorter duration and larger pressure. But the present data set provides initial evidence that 20 min of continuous loading may have compromised the physiological post-loading vasodilatory response. A previous study using 40 min of heel loading during continuous supine lying reported a peak hyperaemic response that was about 3·4 times the resting unloaded baseline (Mayrovitz & Smith, 1998). This is near that found for 20 min of loading used in the present study (3·76). This may indicate that the compromising effect occurs within a 10–20 min time frame.

Hyperaemic recovery times increased with both load duration and magnitude and showed a progressive increase and dependence on loading pressure–duration product. A maximum recovery time of about 7·5 min was associated with the largest pressure–duration during 20 min of loading. Previous work has shown a recovery time of about 15 min after 40 min of continuous heel loading (Mayrovitz & Smith, 1998). Thus, the maximum recovery time found using a localized heel load in the present study is consistent with the doubled recovery duration associated with twice the maximum loading duration obtained under those bed-lying conditions.

Potential clinical implications

The most common site for pressure ulcer development is the sacral region, with the heel–ankle site being the second most common (Schue & Langemo, 1998). The development of such ulcers is associated with significant patient morbidity and costs, thereby prompting considerable research focused on prevention strategies. Key basic aspects in this regard are related to the nature of physiological responses to tissue loading among normal and compromised individuals.

Comparisons of post-loading hyperaemic responses at the sacrum and gluteus maximus muscle of geriatric patients, healthy young and elderly subjects revealed a suppressed response in geriatric patients (Schubert & Fagrell, 1991a). This finding suggested that early assessments of such deficits may be useful in predetermining patients at high risk of pressure ulcer development. Deficits in peak hyperaemic response have also been detected over sacrum and gluteus maximus muscle areas in patients with spinal cord injuries (Schubert & Fagrell, 1991b). Post-loading recovery times assessed by skin temperature changes were reported to correlate with pressure ulcer risk in elderly nursing home patients (Meijer *et al.*, 1994), and peak hyperaemic responses measured thermographically on skin overlying ischial tuberosities were reported to increase with duration of seated loading from 5 to 15 min (Barnett & Ablarde, 1995).

Heel pressures measured in bed-lying subjects are in the range of 90–140 mmHg (Ek *et al.*, 1987; Thompson-Bishop & Mottola, 1992). Such values not only exceed capillary pressure but are also greater than the mean arterial pressure of most patients, the level at which skin blood perfusion is reduced to zero in limbs (Holstein *et al.*, 1979) and back (Larsen *et al.*, 1979), although even lower external pressures produce these effects in some patients. Forearm local compression data suggest that, at pressures of about 50 mmHg and greater, the potential for autoregulation may be lost (Holloway *et al.*, 1976).

Many pressure ulcer prevention strategies recognize the need for providing patients with suitable pressure relief at appropriate intervals. It has long been recognized that the use of alternating pressures, whereby tissues are periodically rendered completely free of load, are effective in reducing or eliminating ulcer occurrence (Kosiac, 1961). If such pressure relief was provided for intervals of 5 min, pathological

tissue changes in experimental models were minimized or eliminated even at pressures up to 240 mmHg for 3 h. The present finding that the time required for hyperaemia recovery after load removal ranged from about 5 to 7·5 min may therefore explain in part the physiological basis for this observation. Thus, the implication is that, if a sufficient duration of recovery flow is provided at appropriate intervals, irreversible tissue injury may be avoided. However, it is also clear from the present findings that recovery responses are linked to both the magnitude and the duration of tissue loading. When the pressure–duration product exceeded about 1500 mmHg min, the peak increase in post-loading hyperaemic flow did not increase further or actually decreased. Although the importance of the combined pressure–duration loading aspect on pressure ulcer development is well recognized, the present findings suggest that a possible pathophysiological suppression of vasodilatory potential may be involved mechanistically. Based on the present combined results, it would appear that, to protect against the pathological sequelae to heel loading, an appropriate course of action is to attempt to limit the total loading pressure–duration to less than 1500 mmHg min and to provide periodic complete or near complete off-loading for durations of 7–8 min. For lesser pressure–duration loading, a decreased pressure relief interval would appear to be permissible based on the present data. However, it is important to note that these findings are for individuals who do not have abnormal lower extremity vasculatures or compromised microvascular vasodilatory reserve. The previously cited work clearly shows that there are subsets of patients and conditions in whom this normality is not present, either by virtue of co-existing disease or by transient suppression, as may occur during surgery and recovery. These factors need to be taken into account with regard to permissible loading and duration of pressure relief combinations. Specification of these parameters must await systematic assessment in individuals with known deficits in vascular function.

Acknowledgement

The research support of Smith & Nephew Wound Management Division, Largo Florida, is gratefully acknowledged.

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