Vulnerability of the heel to ulceration in bed-bound persons is related to pressure-induced blood flow decreases. Periodic pressure reduction is a clinical strategy to help prevent ulcers by allowing blood flow reperfusion during intervals of off-loading. The magnitude and duration of the resulting hyperemia is related to the duration and magnitude of the prior interval of ischemia. Previous work has shown that if healthy individuals lie supine with their heels in contact with a controllable support surface that allows the heel to be either partially or completely off-loaded, hyperemia features depend on the pressure-relief magnitude during offloading.1-4. Similar effects can be shown with graded local pressure ischemia.5 In the case of supine lying, if off-loading is characterized by the magnitude of interface pressure between heel and support surface during pressure-relief, an inverse relationship between hyperemia and relief pressure is demonstrated, with the greatest hyperemia occurring with complete off-loading (zero interface pressure). If relief pressure is greater than zero, some blunting of the hyperemic response is observed. But, in healthy persons, whether off-loading is partial or complete, average heel blood flow (over a complete load-off cycle) results in a net heel blood flow that exceeds the apparent flow deficit during the loading interval.6 This finding is consistent with the concept that in healthy persons, hyperemia, pressure, pressure-relief, more so than compensates for pressure deficits during pressurization. But, as these previous results strictly apply to normal physiological hyperemic response capacity, effects that a reduced hyperemic reserve may have are unclear. Herein we report on preliminary observations regarding the possible impact of the diabetic condition on the general features of heel loading and partial and complete pressure-relief hyperemia.

Subjects: Persons with diabetes mellitus (DM, n=13), and without DM (NO-DM, n=15) participated. For (DM vs. NO-DM) data (mean ± s.e) were as follows: Abb. 1: 114±0.04 vs. 113±0.02; Height: 67.3±0.9 vs. 66.9±1.1 inches; Weight: 205.2±17.4 vs. 156±9.1 lbs, p < 0.05. Age: 66.3±6 vs. 70±2 years, p < 0.05. Blood Pressure: 121.8±7.2 vs 127.7±4.8; diastolic 75.8±2.6 vs. 72±4.2. mean, 95.3±0.6 vs. 92±1.25 mmHg. Duration of DM was 7.5±1 years. Five subjects were on insulin; remainder on oral medication for type II DM. HbA1c for the group was 8.5±2.2 and their morning blood sugar level averaged 144±33 mg/dl.

Protocol: Subjects lay on a support surface with their left heel positioned on the end cell of a support surface (Figure 1). Pressure in this cell was under computer control, and could be made to vary between 20 mmHg and a variable lower limit of either 5 or 0 mmHg. The test sequence was initiated after supine rest of 15 minutes during which the heel was not loaded (0 mmHg, Figure 2). Tests were conducted in a room with a well-controlled ambient temperature. Room temperature was 24.0±0.4°C at the start and 24±3°C at the end.

Heel Blood Perfusion: Heel skin blood perfusion (SBF) was monitored with a Laser-Doppler probe on the heel (Figure 3). The probe was at the site of contact of the heel with the support surface. A second probe, inserted in a heater, was on the foot dorsum. Heater temperature could be rapidly raised to 45°C while monitoring local SBF responses. This heat response was used to provide an index of the relative hyperemic potential for each subject. Skin temperature at non-heated sites on the foot dorsum and heel were measured with an infrared thermometer prior to the experiment start and at the end of the experiment. Skin temperatures did not differ between groups nor were there significant changes at the skin sites from start to finish. For dorsum and heel skin, temperatures were 33.1±0.3°C and 32.3±0.4°C respectively. Example data are shown in Figure 4.

Interface Pressure: At the end of each experimental sequence, heel interface pressures were measured in a pressurized heater with a pressure that was placed between the heel and the supporting cell (Figure 5). The cell was pressurized to the levels corresponding to those used during the test-sequence and six measurements of IP were made at each cell pressure. Averages of the six measurements were used to report interface pressures.

In both DM and NO-DM persons, partial heel off-loading results in a reduced hyperemic response as compared to complete off-loading. But, in persons with DM there is a significantly reduced hyperemia for complete off-loading. One explanation of these results is that the diabetes-related reduced microvascular vasodilatory capacity is not exceeded during the partial relief, but is exceeded during complete pressure relief. The presence of a lesser magnitude of hyperemic response is suggested by the reduced heel response findings herein, by specific assessments of foot skin responses7 and by numerous other studies8,9. Accordingly, differences in hyperemic response become unmasked only when maximum hyperemia can be established, which is only during complete off-loading.

For both groups, hyperemia, even during partial off-loading, appears to be adequate to compensate for the prior interval of ischemia. This follows since a flow area ratio (AR) of 2.0 would just be sufficient, theoretically, to compensate for the flow ischemic interval. What then accounts for the “overcompensation” seen during complete off-loading? It has been suggested that hyperemic responses to heel loading and off-loading do not just depend on the ischemia associated with the pressure-induced flow reduction10,11. It may be that the “excess” flow serves additional physiological needs; if true, this implies that the larger hyperemic response present with full pressure-relief, is in fact a needed flow response to compensate for sustained intervals of loading and off-loading. By extension, this suggests that a reduced hyperemia during complete off-loading as found in the DM group, may be problematic if widely present in the diabetic population. Further work is needed to investigate and clarify this concept.

## References

12. Sharp TJ, Unsworth LC, Rucker RS. Characterizing hyperemia in diabetic tissues: influence of interface pressure and pressure-relief magnitude for AR and Q0 (p<0.05). As shown in the table above, 0 mmHg, compared to relief of 5 mmHg, showed a greater AR and Q0 only in the NO-DM group. Further, for the DM group, full pressure relief was associated with significantly reduced AR and Q0 compared to NO-DM for HbA1c. For HbA1c, which characterizes the SBF response on foot dorsum, a significantly reduced value was observed in DMS (p<0.05).