Effects of Rhythmically Alternating and Static Pressure Support Surfaces on Skin Microvascular Perfusion

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ABSTRACT: The use of periodic pressure relief mattresses and overlays as a means to minimize the risk of pressure ulcer development is an attractive concept. The dynamics of the inflation–deflation cycle of this modality gives rise to rhythmical variations in both tissue interface pressure and microvascular blood perfusion to the skin at risk. A theoretical basis underlying the potential benefit of using dynamic vs. static support surfaces is presented and evaluated by exposing the trochanter of 10 subjects to 60 minutes of continuous loading by a dynamic mattress overly (Alamo™) and a static pressure low air–loss mattress (Mediscus™). Blood perfusion was determined at the trochanter in 10 subjects using Laser Doppler flowmetry. Following a 30 minute no–load baseline interval each subject was exposed to the dynamic pressure for a period of 60 minutes. On a subsequent visit each subject followed a similar protocol on the constant pressure bed at an interface pressure equal to the subject's previously measured average value. Results showed increases in skin temperature and flowmotion frequency with both surfaces, but no detectible difference in mean skin blood perfusion in subjects whether exposed to static or dynamic external pressure load for 60 minutes.

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Prevention and treatment of pressure ulcers are major concerns for health care providers in acute care, rehabilitation, nursing homes and home care facilities. The epidemiology of pressure ulcers in the United States is currently poorly defined, but the data available suggest that pressure ulcers are a serious health problem. It has been suggested that from 3 to 10 percent of all hospitalized patients develop pressure ulcers during their hospital stay.[1] The incidence rises steadily with increased age, and in the hospital-

ized elderly, the prevalence may increase to 20 to 32 percent.[2] The cost of the problem is enormous, both in terms of individual human suffering and in terms of the financial expense to society. It is estimated that the cost to heal one pressure ulcer may range from $5000 to $40,000.[3] Nursing care costs may increase as much as 50 percent for patients with pressure ulcers. An approximate fourfold increase in risk of death has been reported with the development of a pressure ulcer among geriatric patients and those in nursing homes.[4] Complications of pressure ulcers include osteomyelitis and sepsis.

Many methods are available to relieve pressure for the prevention of skin breakdown. These methods include air–mattresses, foam, water mattresses, low–air loss specialty and air–fluidized therapy beds. Each modality has its devotees and, of course, an associated cost. The use of alternating pressure has also been advocated as a method for preventing skin breakdown. With this method the patient can be supported on a mattress overlay. The overlay's pressure varies with time in an alternating manner. Such support surfaces may have additional benefits for reasons which will be discussed in the next section. This report is concerned with the comparison of such an alternating pressure surface (termed dynamic surface) with a high quality non–pressure varying mattress (termed static surface).

Theoretical Differences Between Dynamic and Static Modalities

One rationale for the use of dynamic (alternating pressure) vs. static support surfaces is based in part on the differences with which these two modalities theoretically effect the blood flow in the underlying blood vessels. Compressive forces acting on the skin surface are transmitted to the underlying blood vessels and have the effect of increasing the net inwardly directed pressure acting on blood vessel walls. As illustrated in Figure 1, this "external" pressure (Pe), which is the force per unit area acting on the body surface in contact with the supporting surface, has the tendency to compress the blood vessels in the underlying tissue. This excess pressure thereby tends to reduce the diameter (D) of the blood vessels which supply nourishment to the tissues. As a consequence of this reduction in vessel diameter there is a tendency for the blood flow (F) to the tissue to be reduced assuming that the perfusion pressure (Pa–Pv) remains the same or is decreased.[5] Since according to hemodynamic theory, the blood flow is dependent on the inverse fourth power of the vessel diameter, blood flow reductions accompanying even small diameter reductions are in a sense amplified in a nonlinear fashion.[6] If the flow reduction is sufficiently large or its duration sufficiently long, the dependent tissue is at risk of ischemia and of developing related pathology.[7,8] Depending on the vascular status of the patient, skin breakdown may or may not develop. When the external pressure causing this flow–reducing process is at a constant (static) level, the amount of flow reduction is in part determined by the net reduction in hemodynamic conductance of the microvasculature in the region exposed to the external pressure.

Because of the nonlinear relationship between blood flow and vessel diameter, decreases and increases in vessel diameter from any given level do not have a symmetrical effect on blood flow changes.[9] This concept is illustrated in Figure 2. In this figure, the relative blood flow changes associated with a dynamically time varying blood vessel diameter are compared with a non–varying vessel diameter. In both cases, the average diameter of the vessel is the same; for the static case, the vessel diameter remains at some fixed value whereas for the dynamic case, the vessel diameter varies rhythmically. Based on well established hemodynamic analyses, the resultant blood flow pattern will be of a form as shown in the figure.[5,10] This result assumes that the diameter change is concentric in that the vessel maintains a cylindrical shape. If the variation is asymmetrical yielding an elliptical cross section, then the results will be qualitatively similar but with some modification in the absolute values. For the specific illustrative example shown in the figure, it may be seen that average blood flow in the vessel undergoing periodic diameter change (dynamic average) is actually 1.75 greater than that in the vessel with an unchanging diameter (static average). This flow difference occurs even though the unchanging vessel diameter is precisely equal to the average diameter of the time varying vessel.

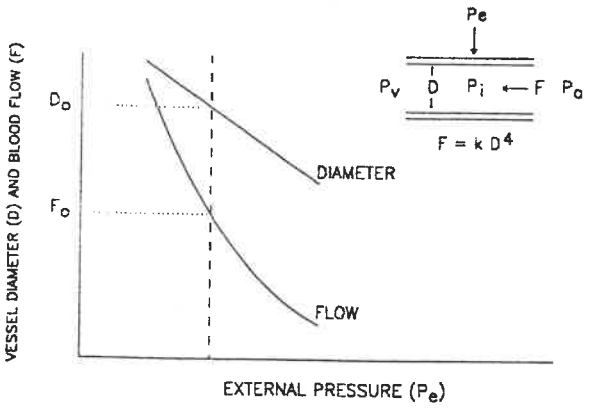

Figure 1: Effects of static external pressure on diameter (D) and blood flow (F) of underlying blood vessels. Insert shows a vessel segment with an intravascular pressure Pi being acted on by an external compressive pressure Pe. Pa and Pv designate the upstream and downstream pressures respectively. The graph illustrates the effect of Pe on both D and F. Fo represents a conceptualized flow level below which the tissue becomes compromised and Do as the corresponding vessel diameter.

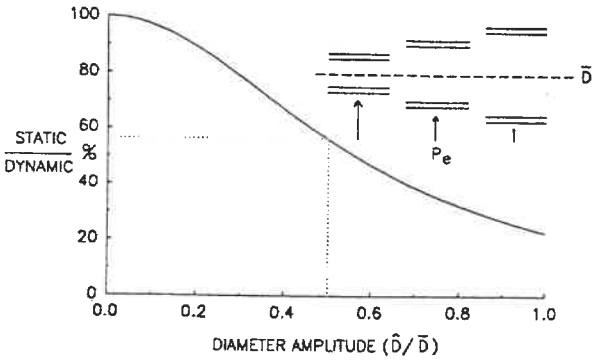

Figure 3: Quantitative prediction of the average blood flow deficit with static vs. dynamic loading. Inset shows a vessel segment exposed to different levels of average external pressure (Pe) with the length of the arrows being proportional to the magnitude of the pressure. The graphic shows that the flow in a static vessel (expressed as a percentage of the flow that would occur in a corresponding dynamically varying vessel), decreases as the vessel diameter change increases. The amplitude of the diameter change is expressed as the ratio of peak–to–peak diameter change (D) to the vessel average diameter (D).

If two blood vessels have the same average diameter and are exposed to identical perfusion pressures, and one vessel has a fixed diameter and the other one has a rhythmically varying diameter, then the amount by which the flow in

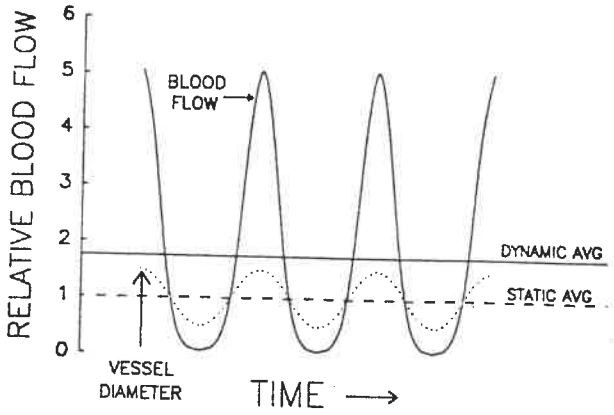

Figure 2: Temporal changes in blood flow theoretically predicted for dynamic vs. static pressure loading. The time averaged relative blood flow for dynamic loading (dynamic avg) is larger than for static loading (static avg) because of the nonlinear relationship between vessel diameter and blood flow. The dotted curve shows the assumed blood vessel diameter change.

the latter exceeds that in the former depends on the amplitude of the diameter change. This feature is illustrated in Figure 3, which shows the flow in a static vessel expressed as a percentage of the flow that would occur in a corresponding dynamically varying vessel. This ratio is seen to decrease as the amplitude of the diameter change increases. For convenience, this amplitude is expressed as the ratio of the peak–to–peak diameter change (D) to the vessel's average diameter (D). Note that when the diameter amplitude is zero there is no difference in flow between the static and dynamic vessel. As the diameter amplitude increases the flow in the static vessel as compared with that in the dynamic vessel becomes progressively less. Thus on these theoretical grounds it appears that the larger the diameter amplitude the larger is the net blood perfusion, all else remaining constant. Phrased somewhat differently, a blood vessel that is caused to rhythmically change diameter from full closure to twice its mean value will in fact have a fourfold greater blood flow than if the blood vessel were static at the same average diameter. The physical mechanism causing such dynamic diameter changes during exposure to an alternating pressure surface is schematically illustrated in the inset of Figure 3. There, a longitudinal section of a blood vessel at three different levels of external pressure (Pe) is illustrated with the length of the arrow proportional to the pressure level. As the

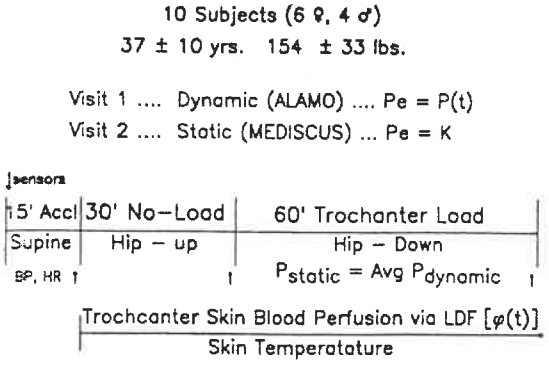

Figure 4: Protocol Summary

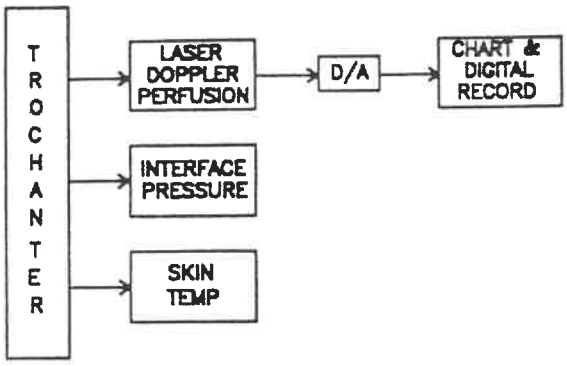

Figure 5: Data acquisition schema and parameters.

alternating pressure surface goes through its cycle, the external pressure varies with time and causes the diameter to change in a manner similar to that schematically illustrated. With a static support surface no such rhythmical changes in blood vessel diameter would occur and thus no blood flow augmentation would be anticipated.

With these theoretical underpinnings in mind, the goal of the present investigational study was to determine if such blood flow augmentation would be detectible in normal subjects when exposed to external pressures produced by a dynamic surface as compared with a static surface.

Methods

Protocol. Figure 4 summarizes the main points of the protocol used. Ten non-selected and apparently healthy volunteer subjects (six female and four male) were used. All subjects received verbal explanation of the study and signed an informed consent in accordance with the ethical standards of the institutional IRB. Subjects were instructed to change into cotton surgical scrub pants. Age and weight (mean and sd) were 37 ± 10 years and 154 ± 33 lbs respectively. One subject was obese. Subjects were free of any clinical signs and symptoms of disease and denied any history of health problems. The laboratory environment was controlled to exclude external stimuli and temperature fluctuations, only soft background music was played. Blood pressure measurement prior to and during data procurement established that no subject was hypertensive. The protocol sequence began with the subject supine. During this time a laser Doppler flow (Laserflo, model BPM 403, Vasamedics, Inc., St. Paul, MN) probe (which was previously affixed to an interface pressure sensor (Gaymar Industries, Inc., Orchard Park, New York) was placed on the left trochanter. The theory and applications of laser Doppler flowmetry has been well reviewed elsewhere.[11] With this method coherent laser light (780 nm, 2 mw) produced by an infrared diode laser is coupled to the skin via a flexible fiberoptic cable. The light penetrates the skin surface to a depth of 1 to 1.5 mm. When moving red blood cells (rbc) are intercepted by the incident photons there results a scattering of the light which is subsequently detected by a photodector in the same cable bundle as the incident light. The shift in the frequency of the detected signal contains information which is proportional to the mean velocity (U) associated with all moving cells contributing the detected scattered light (a tissue volume of about 1 to 1.5 mm^3). Doppler shifts imparted to the scattered photons are of the order of 0.3kHz /mm/sec. The amplitude of the frequency spectrum of the Doppler shifted photons is proportional to the number of scattering events which have occurred. This in turn depends on the number of moving scatterers (rbc's) and is thus proportional to the number of moving rbc's in the tissue (V). The product U x V is the tissue blood perfusion (Q) and is proportional to blood flow differing from it only by a length constant. There are three separate analog signal outputs from the instrument we use; U, V and Q. In use, each signal is routed to a dedicated microcomputer where the signals are converted to digital format (a/d conversion) and stored in computer memory for post-processing. Simultaneously, the signals are displayed in real time on a chart recorder. A

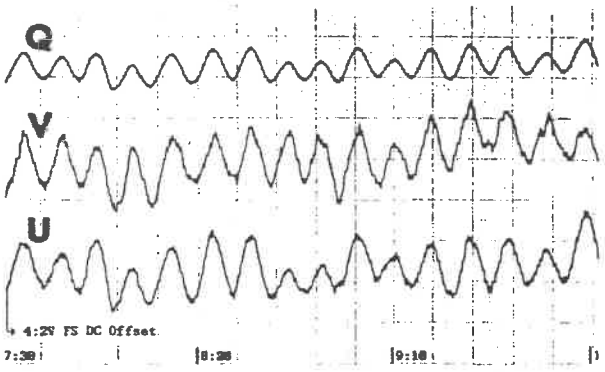

Figure 8: Demonstration of rhythmic variations in perfusion (flowmotion) recorded in a subject. Q=total perfusion as recorded by laser Doppler, V=associated blood volume changes, U=mean red cell velocity changes.

lying on. Higher skin temperatures are generally associated with greater microvascular blood perfusion.

Flowmotion frequency. Normal skin (and other tissues) demonstrate rhythmical variations in blood perfusion unrelated to respiratory or heart rhythms. An example may be seen in Figure 8 in which the rhythmic changes in total perfusion (Q), blood volume (V) and mean red cell velocity (U) for one subject are illustrated.

Flowmotion was observed in all subjects studied. Figure 9 compares the flowmotion frequency as measured at baseline and during a post–load recovery interval after the 60 minutes of loading had been completed. The results show a significant increase in flowmotion frequency which was present independent of the type of surface used.

Comparison of dynamic vs. static perfusion. The composite data for the trochanter skin perfusion during no–load and loaded conditions for all subjects is shown in Figure 10 with perfusion expressed as a percentage of the no–load perfusion. Comparisons of the pressure loaded perfusion values between dynamic and static surfaces were made using analysis of variance (anova) with time as the repeated measure. With this statistical procedure the individual perfusion values at each of the time points during pressure loading are taken into account and one seeks to test for the presence of an overall difference between the perfusion values on the two surfaces. The main

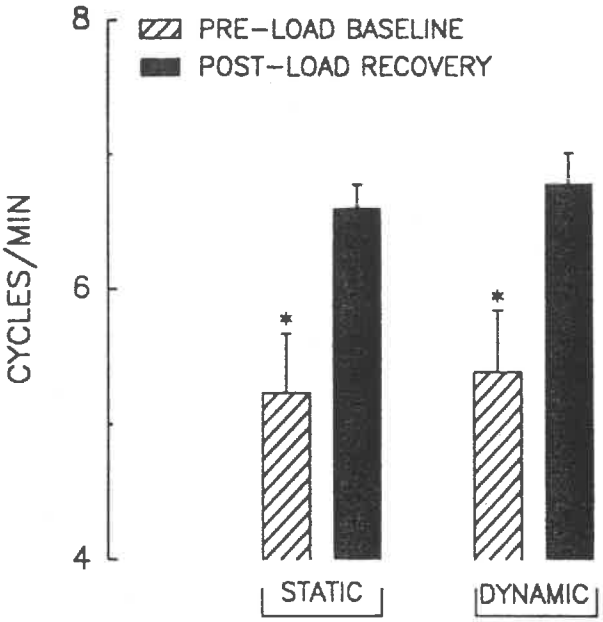

*Figure 9: Comparison of flowmotion frequency (cycles/minute) before and after 60 minutes of pressure loading. Both surfaces resulted in an increase in frequency. *=p<0.05.*

finding is that there was no significant difference detected between surfaces (dynamic vs. static) with regard to the blood perfusion status of the skin microcirculation. This is true whether the comparison is made on the basis of absolute perfusion units (mv) or when analyzed in terms of relative changes from baseline levels as in Figure 10.

Skin perfusion vs. interface pressure as a figure–of–merit. As a final, albeit ancillary point, the question of the relationship between tissue interface pressure and skin blood flow was investigated in two of the subjects. To accomplish this, the trochanter perfusion was monitored while the interface pressure was varied with the subject lying on the Mediscus mattress. The results are shown in Figure 11 where the trochanter skin flow is represented by the Greek symbol (psi) and the measured interface pressure by the symbol p. It can be seen that the flow–pressure relationship is linear for both subjects with the flow decreasing with increasing pressure. However, the slopes and intercepts of these lines are quite different, as indicated visually and by the linear regression equations for each subject shown in the figure. The vertical dotted line corresponds to an interface pressure of 32 mmHg. It is important to note that blood flow continues to increase

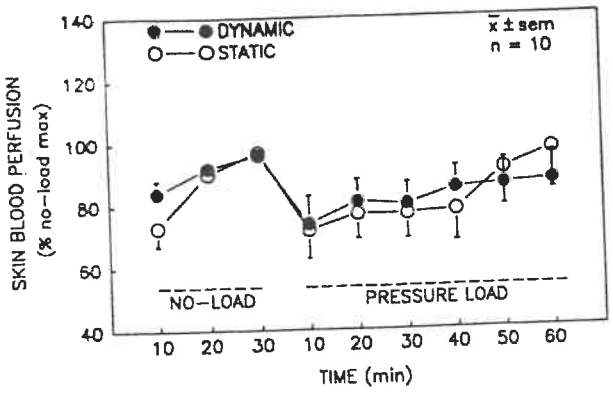

Figure 10: Comparison of relative skin perfusion for dynamic and static loading for all subjects.

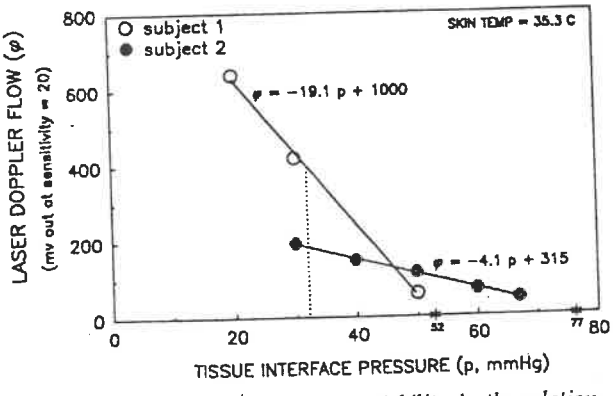

Figure 11: Demonstration of the variability in the relationship between skin blood perfusion and tissue interface pressure in two subjects.

even for values below this level despite the classical notion that at this pressure level the flow is not compromised. Secondly it is important to note that there is significantly different blood perfusion levels between these two subjects at precisely the same interface pressure. We would thus conclude that the actual skin perfusion is a significantly better figure–of–merit for the assessment of the effects of bed surfaces than is interface pressure by itself. This is true in the measurements associated with the healthy normal subjects herein studied and would likely be more important in the assessment of patients who have compromising vascular or other debilitating states present. Similar concerns regarding the use of interface pressures has been put forward by others.[12]

Discussion

One of the main findings of the present study was that there was no detectible difference in mean skin blood perfusion in normal subjects whether exposed to static or dynamic external pressure load for 60 minutes. From the point of view of skin perfusion enhancement, this finding would seem to suggest that over the pressure range herein studied, the dominant factor influencing perfusion was the mean level of interface pressure. In the present design this level was maintained constant on a subject–by–subject basis under both static and dynamic loading conditions. At first glance these results would appear to be inconsistent with the theoretical expectation of a greater perfusion with dynamic loading conditions. Reasons for the apparent discrepancy from the results as predicted by the hemodynamic considerations outlined previously are somewhat speculative but there are at least two factors worthy of further consideration.

In order for flow augmentation to be realized (dynamic vs. static) part of the pressure induced blood flow reduction would have to have been attributable to compression and resultant diameter reduction of underlying supplying and draining blood vessels. It is only under these conditions that the hemodynamic benefit of the phasic changes in vessel diameter can be realized. In the normal healthy subjects used in the present study it may be that no such significant diameter reductions occurred. This would be explainable on the basis of the low mean interface pressure (32 mmHg over all subjects) and the normal structure and integrity of the extravascular matrix between skin and underlying vessels. Indeed, the maximum overall mean perfusion was reduced only to about 80% of the no–load values after one hour of loading. Such changes could easily be attributable to pressure related changes only in a subset of surface capillaries. Thus the finding of no detectible differences between the static and dynamic modalities may simply be attributable to the combination of the small flow reduction produced in each case and the absence of significant vessel diameter reduction.

An alternate and possibly coexisting possibility to account for the absence of a flow difference in this healthy subject population is the integrity

of the subject's vascular reserve. When local compression occurs, which causes the tissue region to be at risk of ischemic damage, autoregulatory processes result in healthy individuals. Among these processes is vasodilation of arteriolar vessels in the peripheral border region of the zone of compression. The skin and underlying blood vessels normally have a high degree of intercommunication with each other which allows blood to enter the affected zone from border vessels below and adjacent of the risk zone.[13] The subjects used in the present study, being free of vascular abnormalities, would likely have such an intact anastomosing network and adequate vascular reserve to at least partially compensate for the external pressure induced changes. Under these conditions, an augmentation in dynamic flow would only be evident if such compensating mechanisms were defective.

The two mechanisms on which we have speculated may have masked the predicted flow benefit of the dynamic modality are based on the presence of; 1) a small pressure induced flow reduction 2) functionally normal tissue, 3) normal vascular structure, and 4) adequate blood flow reserve. As is well known, most patients in whom the use of pressure sore prevention methods are required will unlikely satisfy these conditions. Even in the absence of accompanying disease processes, aging itself can influence the microvascular status.[14–16] One may speculate then that it is in these patients that the theoretically predicted flow augmentation may have its greatest potential. However, this hypothesis remains to be tested.

An additional new result from the present study was the finding of a significant increase in the flowmotion frequency following 60 minutes of compression. This result occurred with both dynamic and static loading conditions. Flowmotion is the term used to describe the rhythmical variations in blood velocity and flow which are often observed within the microvasculature of skin and other tissues.[17–19] The flowmotion waves are not directly related to cardiac or respiratory rhythms and are thought to be due to spontaneous variations in arteriolar diameter (rhythmical vasomotion).[18,20] The functional role of this type of flow behavior is speculative but is thought to be related to flow regulation within the microvasculature. Alterations in amplitude and frequency have been detected in certain pathological conditions.[21,22] The significance of the increase in frequency following the 60 minutes of compression is not clear, although there are several possibilities.

It has been shown that both spontaneous arteriolar vasomotion and flowmotion frequencies tend to increase with increasing temperature and intravascular pressure.[19,23,24] The present results have shown that during the hip–down loading sequence there is a rise in skin temperature which may in part account for the increase in flowmotion frequency. Additionally during the compression phase it is likely that there is a rise in the intravascular pressure in arterioles proximal to the compression site due to the distal compression. As noted, such a pressure elevation could also account for the present findings. A final possibility is that the increase in flowmotion frequency represents an autoregulatory response to aid in the perfusion of the compromised compressed tissue. Such an increase in blood flow associated with an increased vasomotion frequency has been previously postulated.[5,9,10,18]

In summary, a theoretical basis underlying the potential benefit of using dynamic vs. static support surfaces was presented and was evaluated by exposing the trochanter of 10 subjects to 60 minutes of continuous loading by a dynamic mattress overlay (Alamo) and a static pressure mattress (Mediscus). The results revealed no significant difference in trochanteric skin blood perfusion between the two loading conditions presented by the two different supporting surfaces. Explanations for the absence of the predicted flow augmenting effects of dynamic vs. static loading in the normal subjects herein studied were offered. It was suggested that the predicted perfusion augmentation of dynamic loading would more likely be manifest in patients with compromised vasodilatory reserve and other forms of circulatory impairment. Testing of this hypothesis in an at–risk clinical population is urgently needed.

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