The Importance of the Microenvironment of Support Surfaces in the Prevalence of Pressure Ulcers

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Abstract. Soft tissue breakdown is a major cause of disablement in the United States. External pressure has been the most frequently discussed stress factor in the formation of ulcers. Analysis of published data on the prevalence of pressure ulcers and interface pressures at various anatomic sites indicate a nearly non-existent or slightly negative correlation between prevalence and interface pressure for the general and the spinal cord injured populations respectively. This lack of direct relation suggests the major influence of environmental factors in addition to mechanical factors (pressure, shear strain and friction, etc.,) on ulcer formation and indicates the need for control of the support surface microenvironment. However, most of the reported results from studies evaluating support surfaces focus mainly on pressure relief and neglect to address adequately the environmental contributing factors of ulcer formation. Studies directly relating primary stress factors and tissue viability with prevalence and incidence of pressure ulcers are needed to better understand the benefits of pressure relieving support surfaces and to improve the effectiveness of prevention and treatment interventions. The effects of microenvironment on support surface function are reviewed here to aid the healthcare providers in the choosing of the most appropriate support surface to meet the patient's needs.

1 Introduction

1.1 Pressure Ulcer Prevalence and Incidence, Economic Cost

Soft tissue breakdown is a major cause of disablement in the United States affecting an estimated 1.3 to 3 million patients [1,2] and the total cost of associated health care is $8 billion/yr [3]. Miller and Delozier [4] estimated that treating patients with pressure ulcer cost $1.335 billion (average charge was $21,675) in 1992. The average charge for treating pressure ulcers was nearly $37,800 or approximately $17.2 billion in total cost for the year 2003. The prevalence (the number of patients with ulcers divided by the number of patients at risk for ulcers) and incidence (the number of patients who develop a pressure ulcer after admission to a hospital) of pressure ulcers in the acute care settings has remained steady around 15.3% and 7.6% respectively for the period 1999-2004 [5] It is universally accepted that a reduction of pressure between the body and the support surface

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interface will reduce or prevent the occurrence of pressure ulcer. Landis' [6] observation (using a microinjection method) of 32mmHg capillary pressure has been interpreted as a threshold above which pressure ulcer occurs and is often used by the healthcare industry as a guideline for testing the effectiveness of a support surface. The healthcare industry and healthcare providers have been very active to develop new products and treatment and prevention guidelines to reduce the complexity and occurrence of pressure ulcers. The objective of this chapter is to present the relationship between interface pressure and the occurrence of pressure ulcers at various anatomic sites from published literature and briefly review the role played by factors other than pressure in the development of pressure ulcers.

1.2 Current Definition of PU

A pressure ulcer is localized injury to the skin and/or underlying tissue usually over a bony prominence, as a result of pressure, or pressure in combination with shear and/or friction [7].

1.3 Major Factors

External pressure has been the most frequently discussed stress factor in the formation of ulcers. Other primary stress factors associated with ulcer formation are shear, friction and the resulting deformation of the soft tissues.

1.4 Current Methods/Procedures to Prevent PUs

Currently, there are more than 200 support surfaces [8] available that aim to either redistribute or reduce interface pressures below the 32mmHg threshold. These pressure relieving and pressure reducing support surfaces have been widely applied with the hope of reducing the frequency of ulcers. The effectiveness of these myriad support surfaces is usually tested by their ability to reduce interface pressures, usually in a healthy population. Many studies [9-19] have been published by researchers, clinicians and the support surface industry to aid the healthcare staff in choosing the right support surface for their patients. These studies mostly evaluate or compare the different support products using interface pressure measurements alone, mainly because they are readily available, easy to measure, non-invasive and reasonably repeatable. Randomized control trials are costly, limited in number of similar subjects participating and require long duration to complete.

1.4.1 Status of These Methods

Despite all these efforts by the healthcare providers and the healthcare industry, the rate of occurrence of hospital stays with pressure sores has increased by 63% from 280,000 cases in 1993 to 455,000 cases in 2003 [20]. 72% of patients with pressure ulcer were 65 years of age and older, and 19% of patients were between 45 and 64 years of age [20]. The five most common conditions for pressure sore related hospital stays were septicemia, pneumonia, urinary tract infection, aspiration pneumonitis and congestive heart failure. The common concomitant conditions for those admitted primarily for pressure sores were paralysis, spinal cord

injury, substance abuse, malnutrition, multiple sclerosis, stroke and senility. This has led many researchers to revisit the issue of the role played by pressure in the development of pressure ulcers and the effectiveness of the pressure relieving support surfaces.

1.4.2 Reasons for Limited Success

However, due to paucity of other relevant data, healthcare providers continue to rely on research studies reporting mainly interface pressure to select pressure relief support devices and fail to adequately address all the other factors contributing to the formation of ischemic necrosis [21-23]. Berlowitz and Brienza reviewed the literature relevant to the pathophysiology and pathogenesis of pressure ulcer and noted that pressure ulcers are the result of deep tissue damage and that eliminating of pressure is not necessarily the highest priority for prevention and treatment of deep tissue damage [24]. As a result, these expensive support surfaces provide mixed, or in some cases, no benefit at all to the patients. Correlating the interface pressure measured for the various pressure relieving support surfaces with the relevant prevalence or incidence information will be very useful in understanding the role of pressure in reducing pressure ulcer and the effectiveness of the pressure relieving systems. Bader and White also pointed out that tissue viability could be compromised in elderly patients making them more susceptible to pressure sores during surgical procedures [25].

2 Relationship between the Prevalence of Pressure Ulcers and Interface Pressures

At different anatomic regions the occurrence of the ulcers and the corresponding body interface pressures are highly variable depending on the tissue health, thickness, support characteristics, and the method of measurement. Figure 1 shows the distribution of pressure ulcers at various anatomic locations for the general population and the SCI patients. The overall prevalence of pressure ulcer in the acute care setting was reported to be stable around 15% for the years 1999-2004 [5]. Except for occiput, elbow and heel locations, the distribution of pressure ulcers was similar between the two populations.

The weighted average interface pressure by the number of subjects at various anatomic locations as reported by the surveyed publications is shown in Figure 2. The interface pressure for the healthy volunteers and the general population is lower compared to the SCI population except for the occiput [27]. The higher interface pressure in SCI patients is due to a lack of innervation, resulting in disuse atrophy, rapid bottoming out, more tissue deformation, total compression of the paraplegic tissues and obstruction of blood flow. During weight bearing, the rapid reduction of paraplegic soft tissue thickness (bottoming out) creates higher pressure gradients and larger shear effect in the thinner soft tissues between the support surface and the bony prominence. [26]

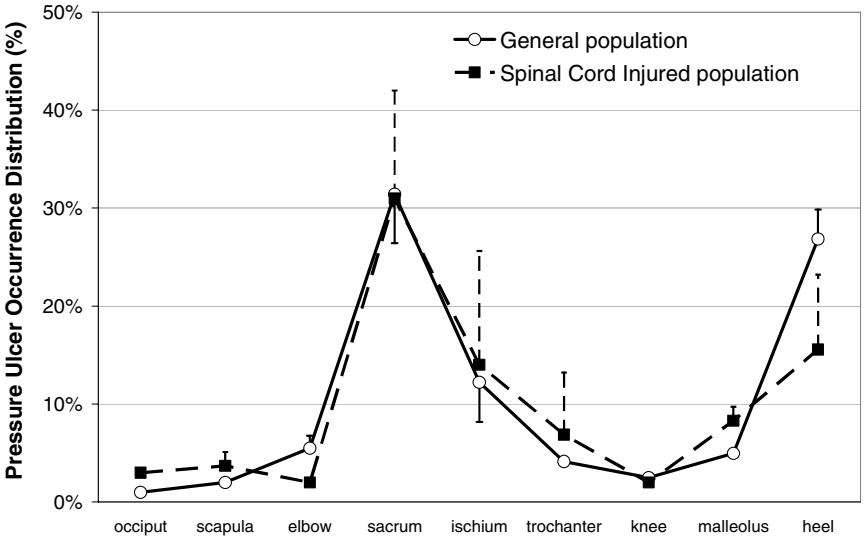

Fig. 1. The graph shows pressure sore occurrence distribution (weighted average) at various anatomic locations for the general and SCI population. Standard deviations are not shown if the data was from a single study [27]. (Used with permission).

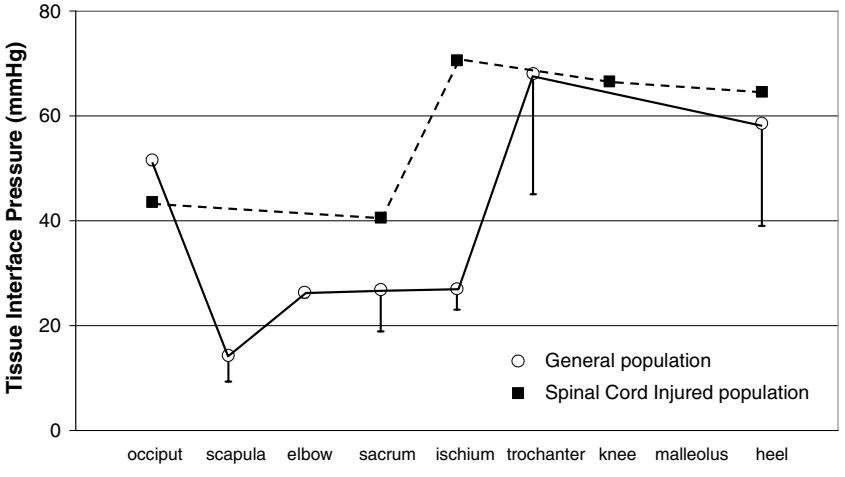

Fig. 2. The graph shows tissue interface pressure (weighted average) at various anatomic locations for the general and SCI population. Standard deviations are not shown if the data was from a single study [27]. (Used with permission).

Table 1. Correlation analysis between reported interface pressure and pressure ulcer occurrence distribution (weighted averages) for the general population and SCI patients at selected anatomic locations [27]. (Used with permission).

	General Population		**Spinal Cord Injury Patients**	
Location	Occurence in % (range)	Interface Pressure in mmHg (range)	Occurrence in % (range)	Interface Pressure in mm Hg
Occiput	1	49 (23-51)	3	44
Sacrum	31 (26-37)	24 (12-43)	31 (14-37)	41
Ischium	12 (8-17)	26 (22-29)	14 (9-35)	71
Heel	27 (23-30)	61 (14-102)	16 (11-26)	65
Correlation Coeff	**-0.118**		**-0.191**	

A correlation analysis was done between the interface pressures and the prevalence for 4 locations where the data were available for both the general population and the SCI patients (Table 1) [27]. The authors found a slightly negative correlation for both the general population of ulcer patients and the SCI patients. Both groups had the same distribution for pressure ulcer at sacrum (31%) but the SCI patients had a 70% higher interface pressure compared to general population of ulcer patients. In the case of heel ulcers, the general population had a much higher occurrence (27% vs. 16%) compared to SCI patients while the interface pressure was actually smaller (61mmHg vs. 65mmHg) [27].The differences between the two populations indicate the change in weight bearing ability of soft tissues in the absence of innervations.

The review article acknowledged the various limitations and shortcomings of using data from multiple sources, years, surfaces, patient groups, investigators, measurement technologies, missing data, small sample size and lack of standardization regarding support surfaces, methodology and reported data [27]. A good analysis would require a long duration randomized controlled study with continuous or at least frequent measurement of interface pressure at various anatomic sites along with other confounding factors (temperature, moisture, friction, immersion etc.) for each of the support device to be tested and relate it to the rate of ulcer formation.

3 Microenvironment

3.1 What Constitutes Microenvironment?

There are more than one hundred biomechanical and pathophysiological factors identified to contribute to the formation of ischemic necrosis of the skin and soft tissues [1]. External pressure has been the most frequently discussed but other primary stress factors associated with ulcer formation are shear [28], friction and the resulting deformation of the soft tissues. The secondary or environmental factors important in bed immobility are temperature, moisture, duration of the applied

load, tissue atrophy, and posture. Microclimate is the effect of temperature, humidity and airflow at the patient support contact. These factors influence tissue quality by reducing the strength and the stiffness of soft tissues and increase the coefficient of the friction of the skin.

The data used for analysis (shown in Table 1) has many significant limitations and indicates the need for better controlled studies investigating the relationship between stress, environmental factors and pressure ulcer prevalence or incidence for the support surfaces currently used. While the published data has many limitations, the analysis presented here indicates no direct or positive relationship between interface pressure and the distribution of pressure ulcers at the anatomic locations. This lack of direct relation between interface pressure and the frequency of ulcers at anatomic sites suggests the major influence of secondary factors at these anatomic sites on ulcer formation and mandates the control and reporting of these factors at the microenvironment of the support surface.

3.2 Shear Stress

3.2.1 Effect of Shear

Shear stress is generated in the soft tissues by the tangential force component of the body weight gravity force on the contact area externally and by the parallel and opposite tangential force on the bony prominence internally (Figure 3). Tangential forces acting on the skin develop shear stress in the tissues through friction and cause the tissue layers to slide with respect to each other. The amount of sliding depends on the looseness of the connective fibers between the tissue layers and create tissue deformation and stretching. If the fibers are tight, the skin and

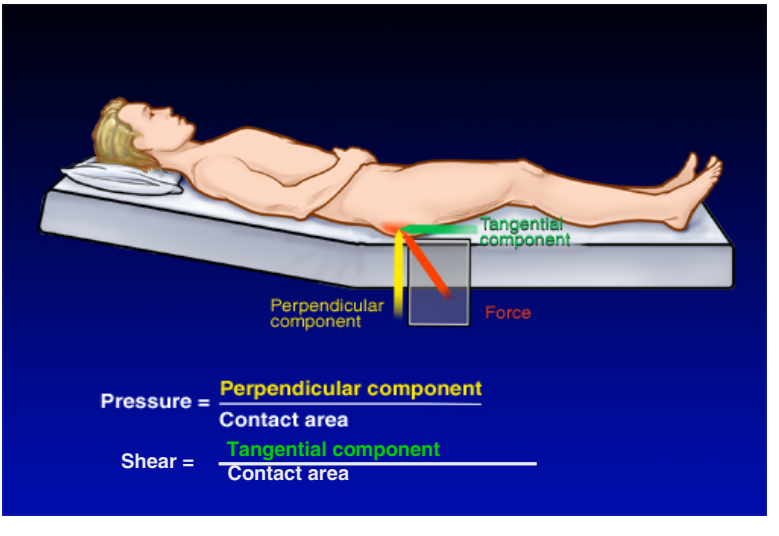

Fig. 3. Illustration of force components of pressure and shear stress on the body. (Courtesy of Cleveland Clinic).

subcutaneous tissue will be subjected to higher shear stress and more stretching; if the fibers are loose, there will be more sliding and lower shear stress and less stretching.

3.2.2 How Is It Minimized?

Shear stress is reduced by a decrease in the tangential force and an increase in the contact area. Loose covers and increased immersion in the support medium also increase contact area and further reduce shear stresses. When shear induced tissue sliding is present, vessels approaching the skin surface perpendicularly will bend and occlude at the connective layers between the tissue planes. Thus, shear will increase the effect of pressure in reducing flow through the blood vessels [29]. Conversely, if shear stress is reduced, tissues can tolerate higher pressures without blood flow occlusion.

3.2.3 Limitations / Difficulties

The shear occlusion of perforating deeper perpendicular blood vessels will create larger ischemic volume of soft tissues than pressure occlusion of deep surface vessels. There are no good proven and validated methods for measuring shear stress in a patient care environment.

3.3 Pressure

3.3.1 Effect of Pressure

The pressure stress in the soft tissues arise from the gravity force component perpendicular to the external contact area and from the body weight acting through the nearest bony prominence (Figure 3). Sustained pressure can stretch soft tissues and blood vessels, causing multiple microthrombi around the point of maximum compression [30]. In the design of support surfaces the objective is to increase contact area by greater “immersion” allowing the body to sink deeper into the support, distributing the force and reducing the pressure. The cyclic transfer of weight from high-pressure areas is the guiding principle underlying alternating pressure support systems. The alternating pressure creates pressure gradients, which are related to shear stress stretching and may damage adipose tissues and capillaries which are lacking tensile strength [28,31]. Interestingly, Knox reported in his article that a only a weak negative relationship existed between skin surface temperature and interface pressure [32].

3.3.2 How Is It Minimized?

The effect of pressure and shear stress are similar on the blood flow to the tissues. Both stresses reduce blood flow and occlude tissue perfusion [33]. In the absence of shear, tissues can tolerate nearly twice as much surface pressure before the loss of tissue perfusion [31]. Thus, a simple reduction of tissue stretching can nearly double the tissues’ ability to withstand pressure without the development of ischemia. Many support surfaces take advantage of this fact and reduce shear, thus improving the weight-bearing tolerances of the soft tissues [34]. Lim et al [35] have introduced a promising concept of “Off-Loading” sitting by partially removing the ischial support to reduce excessive pressure under buttocks.

3.3.3 Limitations / Difficulties

Two groups of patients, those with neurological disorders (spinal cord injury, stroke, head trauma) and the elderly, account for most of the reported prevalence of pressure ulcers. The similarity is not surprising since the loss of muscle strength, skin and muscle proteins, and muscle mass with age are similar to the losses observed with neurological disorders. The changes in the mechanical strength and susceptibility to external loads of the skin and subcutaneous tissues in neurological disorders indicate a significant reduction of tissue viability from normally innervated tissues. With the ageing of the population, these soft tissue changes are increasing in importance and may override the benefits of pressure reduction alone.

3.3.4 Tissue Deformation Due to Shear Stress and Pressure

Compression of soft tissues and their deformation under externally applied forces are the limiting factors in the fitting of body support systems in bed supports and wheelchair seating. Tissue pressure and strain are also important determinants of comfort on beds and mattresses. Studies have reported information about the significant effect of externally applied pressure on the dimension changes of muscle, fat and skin and the compatibility of tissues and cushion for load transfer [26]. Figure 4A shows the unloaded ("free hanging") muscular and subcutaneous tissues in a normal subject and Figure 5A the unloaded atrophied tissues of a paraplegic subject due to disuse. The extent of tissue atrophy is more clearly indicated by the increased reduction of tissue thickness upon loading of the paraplegic shown in figure (5B) from that of the normal loaded tissue, illustrated in figure (4B). Reduction of tissue tone in paraplegia is also indicated by the extensive lateral tissue bulge near the trochanter in Figure (5B), suggest increased coupling of vertical load to transverse elongation in the loaded tissues indicating tension and shear loading in the lateral direction. This lateral "bulging" and transverse distortion of the paraplegic tissues under vertical compressive loading indicates the need for precise matching of shape and material of the support surface to the unloaded tissue contours to prevent the lateral "bulge" [26].

Rapid reduction in tissue thickness was evident with increasing surface pressures for both normal and the denervated tissues. The paraplegic tissues were maximally compressed or indented and bottomed out under their own body weight at the bony prominences without additional weight. The additional external weight caused no further reduction of tissue thickness; it increased surface and deep pressures only. On the normal tissues, however, the additional weight could cause further reduction of thickness at all locations except at the male trochanter. The transverse images of the soft tissues and bony prominences showed thicker tissues in normal subjects at every pressure, leading to the realization that higher pressure gradients from skin to bone must exist in the paraplegic than in the normal tissues [26]. Recent studies by Linder-Ganz et al [36]. using open-MRI scans also reported that internal tissue loads are significantly higher in paraplegics. Bottoming out of the paraplegic tissues showed the increased risk of tissue trauma caused by total compression and the resulting obstruction of blood flow.

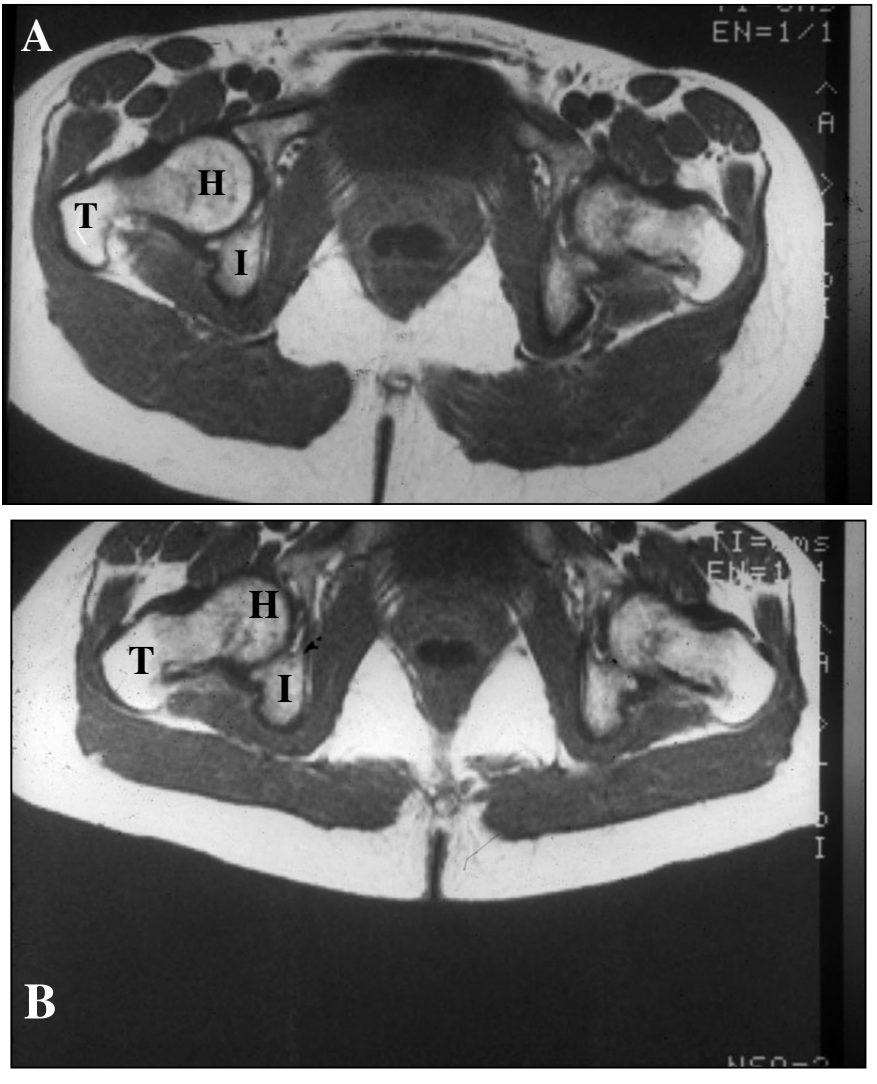

Fig. 4. Transverse magnetic resonance image of normal female near ischial tuberosities [26]. (A) Tissues without external support, “free hanging”. (B) Loaded tissues on the right side with flat cushion support below. T: Trochanter of femur; H: head of femur; I: Ischium.

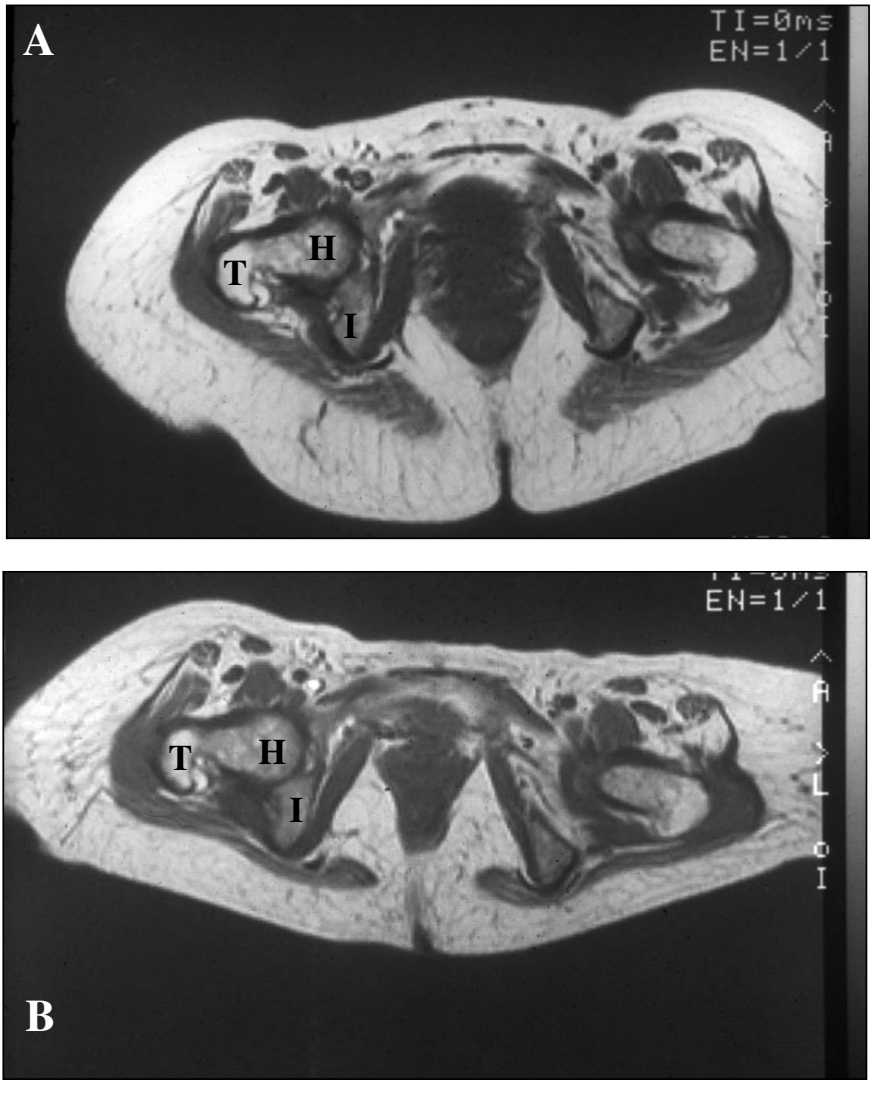

Fig. 5. Transverse magnetic resonance image of paraplegic female near ischial tuberosities [26]. (A) Tissues without external support, "free hanging". (B) Tissues on the right side with flat cushion support below. Note blood vessel distortion by the mechanical environment. T: Trochanter of femur; H: head of femur; I: Ischium.

Sae-Sia et al studied the effect of pressure loading over a 2hr period on the sacral skin blood flow (SBF) and skin temperature in acute spinal cord injury patients [37]. They observed a negative change in SBF during pressure loading along with a higher initial positive slope of the SBF reactive hyperemia response compared to patients with orthopedic trauma and healthy subjects. This suggests microvascular dysfunction in acute SCI subjects and indicates that turning interval guidelines need to be revisited for acute SCI patients.

3.4 Friction

3.4.1 Effect of Friction

Friction is a phenomenon, which describes the surface's ability to prevent motion due to forces tangential to the contact area (Figure 6). The tangential or friction force depends on the perpendicular force and the coefficient of friction at the contact and it is independent of the contact area. When the cover of the support surface is designed to allow movement over its foundation, due to lower coefficient of friction, the slippage occurs between the cover and the bed and not within the tissue layers and the tension in the skin is decreased without stretch and occlusion of blood vessels (Figure 6) [27].

3.4.2 How Is It Minimized?

The tangential or shear force is reduced most effectively by decreasing the coefficient of friction on the support surface (Figure 6A). The effect of high coefficient of friction is shown in Figure 6B [27].The effectiveness of properly inflated air, water, and viscous fluid or gel supports rests on these principles. Combinations of these biomechanical principles are commonly used in modern support surfaces to create a better physical environment for tissue survival.

3.4.3 Limitations / Difficulties

Measuring friction in a patient care environment is not easy. If the interface surface is frictionless, patient stability will be affected as patients may slide or be unable to maintain posture.

3.5 Temperature

3.5.1 Effect of Temperature

Elevating body temperature increases the metabolic activity of the tissues by 10% per degree Celsius of temperature rise, thus increasing the need for oxygen and energy source at the cellular level. If the patient has impaired circulation from local pressure and shear then the tissues will starve and release contents of lysozymes inducing autodigestion of cytoplasma. The metabolic activity may also cease from lack of energy and the accumulation of waste products. It has been shown [29] that pressure induced tissue injury accelerates with increasing body temperature (see Table 2). An equally significant effect of increases in skin temperature is the induction of the sweat response and the potential accumulation of moisture in the skin at the skin-support interface. This local perspiration increases

friction, shear, maceration and may weaken skin in the area [38]. Also, the adverse effects of fever should not be overlooked [30]. Interestingly, Knox observed that a negative correlation existed between core body temperature and skin surface temperature [32].

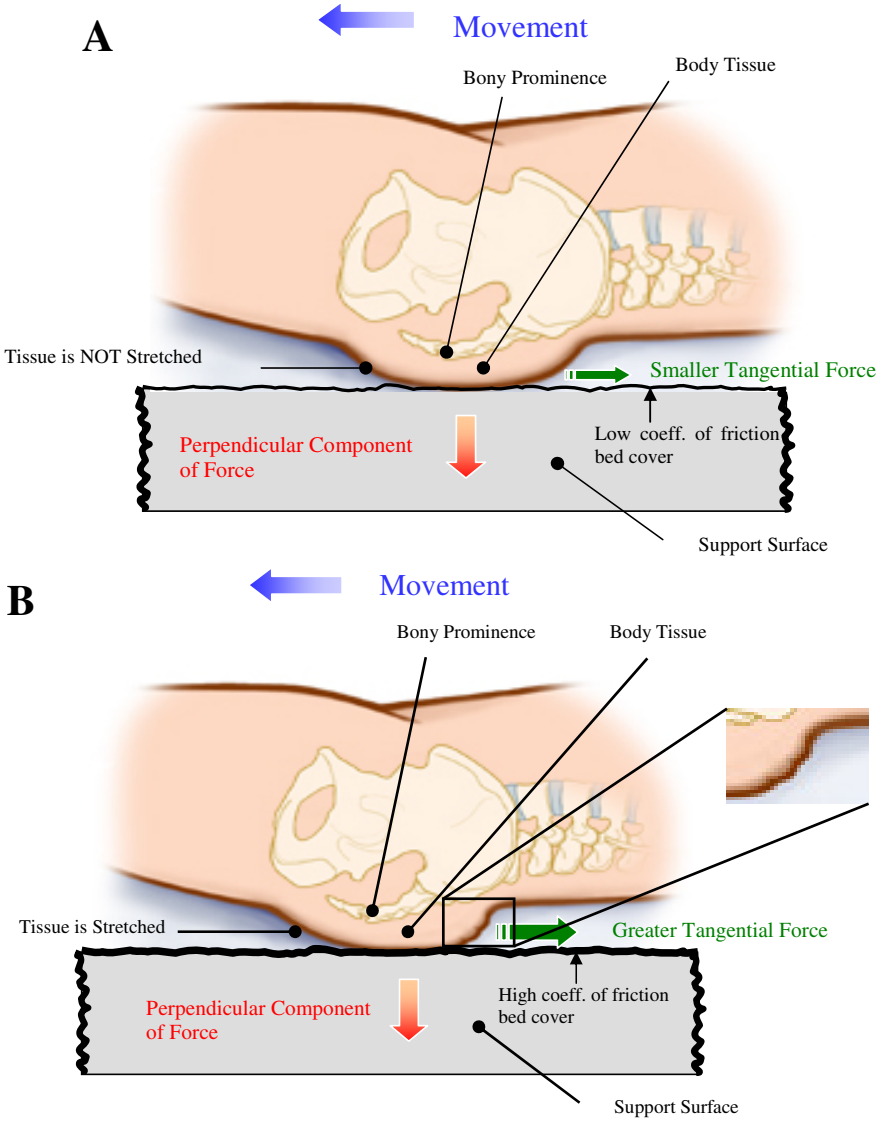

Fig. 6. (A) The illustration shows the lack of skin stretch in the case of low coefficient of friction between the cover and the bed. (B) The illustration shows the skin stretched on one side and folding of tissues on the side opposite to the direction of movement (see inset) in the case of high coefficient of friction between the cover and the bed. Force components on the support [27]. (Used with permission).

Table 2. Pressure Induced Tissue Injury Accelerates With Increasing Body Temperature [29] (Used with permission)

Experimental pig model	@ 100 mm Hg 5 hrs
25°C	no break down
35°C	partial thickness injury
45°C	full thickness breakdown

3.5.2 How Is It Minimized?

Temperature is usually maintained by varying the airflow at the patient/support surface interface area. Low and High-air loss mattresses are able to maintain optimal body temperature by cooling the skin. Reducing body temperature aids in preventing or minimizing infection and fever. Studies have suggested that a 5 degrees C reduction in skin temperature would have an effect similar to that of the most expensive support surface available [38].

3.5.3 Limitations / Difficulties

Excess cooling at the interface may cause some discomfort to the patient and may also enhance muscle tension.

3.6 Moisture

3.6.1 Effect of Moisture

Hydration of the weight-bearing skin opens a new set of destructive influences on skin integrity. Moisture from sweating or from urinary or fecal incontinence will hydrate the skin, dissolve the molecular collagen crosslinks of the dermis and soften the stratum corneum (maceration). Skin maceration results in the reduction of the stiffness, the near complete loss of the connective tissue strength and in the erosion of the dermis under the action of shear forces. Another result of skin hydration is the rapid increase of the coefficient of friction of the epidermis, which promotes adhesion of the skin to the support surface and produce elevated shear, easy sloughing and ulceration. Compounding the destructive effect of stress is hydration diluting the natural skin acidity, reducing antibacterial properties of the epidermis leading to easier sepsis. A recent study [39] attempted to delineate the differences in pathophysiology and histopathology in patients with both incontinence and pressure ulcer lesions. They observed that there is no justification for singling out moisture lesions from pressure ulcer lesions.

3.6.2 How Is It Minimized?

The clinician has two excellent technologies for controlling the microclimate at the skin-support surface interface. The dynamic low- and high-air-loss [40] and the air-fluidized support systems are designed to reduce stress and temperature, evaporate moisture and prevent heat accumulation and cool the interface with the support surface. The evaporation of one kilogram of water from the skin at the support surface will remove 580 Kcal of heat from the body through the “latent heat of vaporization” [41]. Thus the cooling power at the rate of the total water

loss through the skin for an average person with 1.8m^2 skin surface and at the normal sweat rate of 26.7g/m^2/hr, the heat loss is 27.9Kcal/hr. With proper design and nursing care aimed to maintain physiologic water balance, dynamic air loss supports are able to control interface pressure, shear, friction, and the temperature and moisture of the support environment.

3.6.3 Limitations / Difficulties

The operating cost of these air-loss systems can be significant and are not usually reimbursable in the home-care environment.

4 Summary and Conclusion

It is not the intention of the authors to discount the usefulness of clinical support surfaces. On the contrary, the authors wish to remind the healthcare providers that the usefulness of any pressure relieving support surface depends on the control of the microenvironment and not just pressure alone. It is therefore important for healthcare providers to understand the influence of the microenvironment in the development of pressure ulcers when selecting and applying a support surface for their patients. More research is still needed to understand the interactions between the microenvironment and pressure sore occurrence.

4.1 Areas for Future Research

A recent study has proposed endothelial dysfunction as an additional risk factor for pressure ulcer [42]. Endothelial dysfunction is defined as impaired vasodilation to specific stimuli which results in reduced vasodilation, proinflamatory state and prothrombotic state. Statins and angiotensin receptor blockers improve endothelial function indicating good potential for future research.

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