Introduction

Unrelieved pressure upon weight-bearing tissues can produce lesions, identified by their etiology as pressure ulcers (AHCPR, 1992). The prevalence of pressure ulcers for elderly nursing home residents has been estimated between 2.3% and 28%. (AHCPR, 1992; Brandeis, Ooi, Hossain, Morris, & Lipsitz, 1994; Smith, 1995; Young & Burns, 1981). The prevalence rate among other populations with mobility impairments is even higher; it has been estimated that between 50% and 80% of persons with spinal cord injury will develop a pressure ulcer (Gosnell, 1973; Richardson & Meyer, 1981; Rodriguez & Garber, 1994; Salzberg et al., 1996). Even the lowest of these estimates aptly demonstrates that pressure ulcers are a significant health care problem.

According to 1999 Health Care Financing Administration (HCFA) data, inadequate attention to prevent pressure ulcers was the most frequently cited quality of care deficiency in the long-term care setting (Lyder 2000). The costs associated with the management of U.S. pressure ulcers exceed $6.4 billion annually (Marwick, 1992). This burden is reflected in health policy as Healthy People 2010 includes the measurable prevention objective of decreasing pressure ulcer prevalence 50% by the year 2010 (NPUAP, 2000).

Sitting-acquired pressure ulcers are a particularly significant problem for wheelchair users. The percentage of pressure ulcers caused by sitting in wheelchairs is difficult to ascertain, but estimates in the literature suggest that it is between 36% and 50% for the at-risk elderly population (Zacharkow, 1984). The inadequacy of wheelchair fit contributes to the development of pressure ulcers (Zacharkow, 1984; Lim, 1988). In assessing risk, individuals with mobility impairments that prevent independent repositioning, as well as those that are bed and chair-bound, tend to be at the greatest risk for pressure ulcer development (Allman, 1997). Several studies have indicated that the use of pressure-reducing wheelchair seat cushions designed to maintain tissue integrity will reduce the incidence of sitting-induced pressure ulcers (Bardsley, 1984; Shaw, 1993; Lim, 1988; Shaw, 1996; Conine, 1994; Geyer, 2001). Tissue integrity is maintained by reducing pressures near bony prominences, accommodating orthopedic deformities through immersion, enveloping irregularities at the seating interface to reduce high pressure gradients, and controlling heat and moisture.

Review of the Science

While most agree that the primary cause of pressure-induced tissue necrosis is occlusion of capillary blood flow resulting in an ischemic injury (Kosiak, 1961), the impairment of lymphatic drainage and/or interstitial fluid flow have also been proposed as primary causes of pressure ulcers (Krouskop, 1983; Krouskop, Reddy, Spencer, & Secor, 1978; Reddy & Cochran, 1981). Nonetheless, external pressure has long been the focus of etiological investigations on the mechanics of pressure ulcer formation (Crenshaw & Vistnes, 1989; Kosiak, 1961). Pressure measurements are also common in clinical settings where support surfaces are evaluated relative to their potential for risk of pressure ulcers. Other mechanical factors studied include pressure gradient, shear force, and tissue deformation, although investigations studying these factors are far less common than those focusing on pressure alone.
While considering the pressure ulcer problem it is important to note that factors other than mechanics influence pressure ulcer formation. Many interrelated factors, intrinsic as well as extrinsic, have been shown to predispose load-bearing tissue to mechanical damage (Crenshaw & Vistnes, 1989). An extended accounting of pressure ulcer formation must include not only mechanical factors as a primary causative agent, but also other contributing agents such as friction, heat, moisture, incontinence, malnutrition, and an altered level of consciousness (AHCPR, 1992; Evans, Andrews, Chutka, Fleming, & Garness, 1995; Mawson, Siddiqui, & Biundo, 1993). The importance of these various factors also depends upon the particular patient population considered. For example, persons with spinal cord injury are at greatest risk subsequent to loss of sensory and motor functions, loss of vasomotor control and tone, changes and abnormalities in morphology of bone and soft tissue, and altered neuromuscular activity (spastic or flaccid) (Ferguson-Pell, 1990; Mawson et al., 1993; Rodriguez & Garber, 1994). Salzberg followed 219 individuals with SCI for six years and found that 176 (80.4%) had a history of at least one pressure ulcer (Salzberg et al., 1996).

The mechanics of pressure ulcer formation are characterized by several key elements including the magnitude, direction and the distribution of forces over the body surface and the tissue deformations associated with those forces. Extrinsic pressure acting upon weight-bearing tissue is defined by the distribution of forces over an area of tissue. The direction of the forces range from perpendicular to the tissue surface to tangential to the tissue surface. The typical loading conditions will include a combination of normal and shear forces. The forces on the surface of the tissue are transmitted into the tissue and cause deformation, resulting in internal stress and strain of the tissue that can lead to necrosis. As such, tissue deformation is seen as a measure of the net effect of these external reaction forces (Brienza, Iñigo, Chung, & Brubaker, 1993; Levine, Kett, & Ferguson-Pell, 1990; Reddy, Palmieri, & Cochran, 1981).

Coincidentally with tissue deformation is an increase in internal tissue pressure levels. The levels of internal pressure are dependent upon the heterogeneous mechanical properties throughout the soft tissues and the directions in which these tissues are distended by the applied loading. If the tissues are confined so that no redistribution of tissue mass can occur or if loading is applied hydrostatically, the soft tissues can withstand relatively high pressures without significant risk of tissue damage (Levine et al., 1990). Only when pressure is applied non-uniformly are tissues strained and consequently put at risk of tissue damage. While sitting, the soft tissue of the buttocks is not contained; therefore, support surface reaction forces can result in internal strain and ischemia. If excessive forces are not relieved, the result is often morbidity and ensuing tissue necrosis. The variability in normal forces is sometimes described as vertical shear (Bennett, Kavner, Lee, Trainor, & Lewis, 1984) or is quantified in terms of "gradients" of force or pressure (Garber & Krouskop, 1982). As vertical shear increases, the probable effect is an increase in the deformation of the tissue and, therefore, in the risk of tissue damage.

Much previous research has been directed at attempts to establish an interface pressure threshold beyond which pressure ulcers will form. Interface pressure has been used extensively as a tool for predicting the clinical effectiveness of various support surfaces and for comparing products. The validity of this approach, however; has come into question as a wide range of interface pressures have been found to occlude capillary flow. Values ranging from 11 to 120 mmHg have been shown to have this effect (Clark, 1988). More recent research has gone beyond assuming that tissue necrosis is a result of ischemia due to external pressure alone.

A number of techniques are available to assess the effects of pressure on tissue responses including measurements of transcutaneous oxygen tension (TcPO₂), Laser Doppler flowmetry
(LDF), thermography, transcutaneous and in vivo biochemical analyses, ultrasound propagation properties and various imaging techniques. Current investigations are focusing on the physiological, biochemical and biomechanical characteristics of tissue and their interactions.

TcPO$_2$ quantifies the tissue oxygen tension as oxygen diffuses from the dermal capillaries through the epidermis to the skin surface. This technique requires the application of external heat (approximately 45° C in adults) which dilates the dermal capillaries and reduces the resistance of the stratum corneum to oxygen diffusion (Tremper, 1984). TcPO$_2$ has been used as a measure of the perfusion of the skin in response to external loading (Bader, 1986; Bader & Gant, 1988; Liu et al., 1999; Sangeorzan et al., 1989; Xakellis, Frantz, Arteaga, & Meletiou, 1991).

LDF measures the capillary blood flow of the skin 1-6 mm below the surface. It utilizes the Doppler shift of laser light backscattered from moving red blood cells to provide a continuous and non-invasive measure of blood flow in the tissues (Sacks et al., 1988). LDF has also been used as a measure of skin perfusion (Bennett, 1961; Bennett et al., 1984; Ek, Lewis, Zetterqvist, & Svensson, 1984; Johnson & Park, 1979; Mayrovitz, 1993; Sacks et al., 1985; Sanada et al., 1997; Xakellis & Frantz, 1990). Some recent studies have measured perfusion with both TcPO$_2$ and LDF techniques. Of note is the finding by Xakellis that LDF continued to decline beyond the point where TcPO$_2$ reached zero. This may indicate that interface pressures lower than those causing capillary closure may also lead to tissue hypoxia (Xakellis et al., 1991).

In 1990, Bader used TcPO$_2$ to monitor changes produced by the application of various loading regimens to the sacrum and ischial tuberosities of normal and spinal cord subjects (Bader, 1990). In 1997, Sanada used LDF to monitor pressure-induced changes in blood flow over the bony prominences of surgical patients (Sanada et al., 1997). Both studies revealed a normal response to pressure as an increase in perfusion that has been recognized as active hyperemia (Bader, 1990; Mayrovitz, 1993; Sanada et al., 1997). Abnormal responses (a failure to increase perfusion in response to pressure) have been explained in terms of impaired vasomotor response. The results of these investigations reflect the limitation of using interface pressure as a sole indicator of threshold for pressure ulcer formation.

Reddy et al. investigated the effects of external pressure on interstitial fluid dynamics by using a mathematical model. According to their theoretical model, for a given pressure, the time in which the interstitial fluid volume decreases by half represents the tolerance time/threshold for pressure ulcer formation (Reddy, Cochran, & Krouskop, 1981a; Reddy, Palmieri, & GV, 1981b). The similarity between the pressure-time relationships of Reswick and Rogers and the interstitial fluid volume-pressure-time relationships observed by Reddy support the theory that slow viscous flow of interstitial fluid and ground substance play a role in tissue necrosis (Reddy et al., 1981a; Reswick & Rogers, 1976). Utilizing the same mathematical model, Reddy et al. proposed that it was the pressure gradient that induced the flow of interstitial fluid and thus, they proposed that pressure gradients may be more significant in pressure ulcer etiology than interface pressure (Reddy et al., 1981a). The work of Swain et al. demonstrated that pressure gradients were indeed proportionally larger in subjects with the highest interface pressure readings (Swain, 1997).

Attempts to monitor the interstitial fluid pressure in the soft tissues using the wick-catheter technique followed. Reddy et al. believed that the collagen network supports a substantial portion of the externally applied load with only a fraction of the load being
transmitted to the interstitial fluid (Reddy et al., 1981a). In general this study indicated that the relationship between the external load and the interstitial fluid pressure at 2-5 mm below the skin was a non-linear one, i.e., the interstitial fluid pressure tended to be lower than the external pressure. Reddy et al. propose that when interstitial fluid is squeezed out of a tissue region, direct contact of the cells induces stresses that may cause rupture and interrupt vital cell functions including collagen synthesis. This breakdown would continue even after the load was removed (Reddy et al., 1981a).

Reddy also believes that lymphatic flow could be impeded by mechanical stresses and/or the lymphatic vessels themselves might be damaged in response to excessive fluid flow. Subsequent accumulation of metabolic waste products may lead to tissue necrosis. The research of Miller and Seale support this theory (Miller & Seale, 1981). Isotopic (technetium) techniques enabled them to trace the radioactivity in dog hind limbs during external compression with a dead weight cylindrical device. The lymphatics cleared the tracer from the interstitial fluid until the external pressure reached 60-70 mmHg, then lymph flow decreased to zero with further increases in pressure.

Recent investigators also note the importance of biochemical responses. Transcutaneous biochemical analysis such as sweat analysis has been investigated by Polliack et al. (Polliack, Taylor, & Bader, 1993). Based on the hypothesis that specific metabolites may be used as an indicator of soft tissue damage, they collected thermally induced sweat following the application of different loading regimens on the forearms of normal subjects. Results indicated that tissues subjected to pressure ischemia produced a general increase in concentrations of lactate, chloride, urea and urate as well as a decreased sweat rate. Further work is now in progress using this methodology to analyze sweat samples collected from tissues subjected to varying amounts of pressure ischemia.

Measurements of circulating plasma levels of mediators of immunoactivation (ICAM-1 and IL-2R ) were measured in able-bodied, spinal cord injured and spinal cord injured subjects with pressure ulcers by Segal et al. (Segal, Gonzales, Yousefi, Jamshidipour, & Brunnemann, 1997). Results indicated that the spinal cord subjects with pressure ulcers had the highest levels of such markers of inflammation. The authors contend that these easily quantifiable mediators may have diagnostic, prognostic and therapeutic value in predicting or differentiating subgroups of patients who will vary in the severity or the healing of their wounds (Segal et al., 1997).

The mechanisms underlying the etiology of pressure ulcers are apparently not well-understood. And the elucidation of the relative significance of the physiological, biochemical and biomechanical mechanisms will require multidisciplinary research. Therefore, while interface pressure may aid in selecting the best support surface for a specific individual based on that individual’s relative responses, interface pressure alone is not sufficient to evaluate the efficacy of a particular device or class of devices. Many factors make the results of support surface studies difficult to compare. For example, the testing protocols, test postures, techniques used to measure interface pressure and the sampling groups vary considerably. Lack of knowledge regarding additional tissue responses makes it difficult to determine which parameters should be assessed to determine the efficacy of specific interventions. While efforts to standardize the performance characteristics of support surfaces are in progress, for now, the best evidence regarding the effectiveness of support surfaces appears to be the outcome of a decrease in the incidence of pressure ulcers associated with specific interventions, coupled with multiple measures of tissue response.
Clinical Issues

Contradictions exist in the literature regarding the clinical benefits of commercial cushions designed to reduce the risk of sitting-acquired pressure ulcers. Previous research has not revealed any one cushion that consistently provides the lowest pressure measurements for all subjects (Shaw, 1996; Sprigle, 1989; Garber, 1984). Studies comparing polyurethane foam slabs to custom contoured foam cushions failed to demonstrate significant differences in the incidence of pressure ulcers (Lim, 1988; Conine, 1993). Conversely, a subsequent study by Conine comparing a polyurethane foam slab to the Jay cushion showed a significantly lower incidence of pressure ulcers in the Jay group (25%) compared to the foam group (41%)(Conine, 1994). It is of note that Conine's study included the provision of a seating evaluation and appropriate modifications to the wheelchair. Thus, a higher level of internal validity was attained than in previous studies where the effects of the cushion were confounded with those of the chair.

Another recent investigation revealed that higher interface pressure measurements were associated with a higher incidence of sitting-acquired pressure ulcers (Brienza, 2001). Brienza et al. found interface pressure measured on wheelchair seat cushions was higher for subjects who developed sitting-acquired pressure ulcers compared to those who did not develop pressure ulcers.

In general, a lack of standard methodology has hampered the interpretation of previous study’s results. Additional and more comprehensive clinical trials are necessary to provide the strong evidence required by third party funding agencies and to provide clinicians the information necessary to make good clinical decisions.

Policy and regulatory issues

As medical devices, wheelchair seat cushions are subject to the general controls of the Federal Food Drug & Cosmetic Act (Title 21 Code of Federal Regulations Part 800-1200). Cushions are classified as either Class I or Class II medical devices and are exempt from pre-market notification [510 (k)] requirements if they meet two criteria. First, the cushion may not be significantly changed or modified from its original form as marketed in the U.S. prior to May 28, 1976. Second, no Food and Drug Administration regulation requiring a pre-market approval application may have been published in regard to the cushion. The majority of new cushions meet these criteria, are referred to as being "grandfathered", and are exempt from pre-market approval. When cushions are exempt from the extensive evaluation required for pre-market approval, there is little incentive for manufacturers to determine the functional characteristics of available cushions or to develop objective evaluative criteria.

Under the current Medicare system, new cushions are submitted to the HCFA alphanumeric group for assignment of a billing code. Existing codes differentiate among cushion types by either cushion thickness or component materials. This coding system is limited and does not represent the wide range of products and/or the differentials in manufacturing costs of the various cushions. Although HCFA pricing groups created a new category with a higher reimbursement rate to help ease payment inequities (E0192), few objective criteria and no functional performance standards exist to define this or any of the other categories. Despite the fact that most manufacturers seek E0192 categorization for their cushion products, a recent study revealed that only 20 of the 216 cushions available on the market were coded as E0192 (Sprigle, 2000).
Both the suppliers of the coding policy (HCFA) and the demanders (manufacturers, clinicians and consumers) recognize the negative consequences of the existing system. As a result of its limitations, Medicare beneficiaries are being denied access to medically necessary and clinically appropriate seating interventions, manufacturers are having codes rather arbitrarily assigned to their products with no guarantee of consistency, and clinicians have limited objective criteria with which to discriminate among cushions. The need to modify the existing coding system is apparent. However, both problem definition and the presence of viable solutions are hampered by a lack of research regarding objective cushion criteria and functional performance standards. Although efforts are proceeding in an attempt to solve the problem, the lack of research outcomes in the form of objective criteria and performance standards is proving to be the primary barrier to policy modification.

Decision-making on the part of policymakers, in this case, the SADMERC/DMERC medical directors and the alpha-numeric work group, requires a review of the appropriate medical literature to find support for a particular course of action. In the case of the problems associated with seat cushions and pressure ulcers, the evidence in the literature is insufficient. In a 1998 letter to a seat cushion industry group the SADMERC Medical Advisor stated, "To our knowledge there is no proof from adequate clinical trials that any cushion or any design is superior to any other in producing better health outcomes or that any cushion or any specific feature is associated with better outcomes" (Nelson, 1998). This lack of evidence is due, in part, to the nature of assistive technology research that does not lend itself easily to the traditional medical model of double-blind, cross-over, randomized control trial methodology. The wheelchair user population is diverse and subgroups tend to be small and geographically scattered. This makes conducting clinical trials logistically difficult. In addition, assistive technology research is under-funded in contrast to other areas of medical technology research. Research in this area that is industry-sponsored lacks credibility/objectivity and the FDA pre-market exemption for most new cushions provides little incentive for even this limited industry-sponsored research.

Summary of Findings

- Pressure ulcers are a significant healthcare problem for wheelchair users.
- The mechanisms underlying the etiology of pressure ulcers are not well understood.
- Various measurement techniques have been used to investigate the many factors and markers thought to be related to pressure ulcers.
- The interpretation of clinical research is complicated by a lack of standardized methodologies and thus limits the strength of evidence provided by past research.
- The lack of evidence in the literature also is reflected in the barriers to appropriate coding policy and, therefore, clinical application of appropriate seating interventions.
References


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