

Wheelchair Seating: Preventing and Treating Decubitus Ulcers with Friction, Shear, and Pressure Management

This article does the following:

1. Describes the scope of the problem of decubitus ulcers for people in wheelchairs and for our healthcare system
2. Discusses the four local factors causing decubitus ulcers that seating devices could most influence: pressure, shear, temperature and moisture
3. Explores how seating interface devices can potentially prevent or help heal decubitus ulcers with new methods of friction and shear reduction
4. Introduces a new device, GlideWear Seating Interface Technology™

Introduction

Prolonged sitting by a person in a wheelchair exposes that person to a high risk of decubitus ulcers. Decubitus ulcers are lesions ranging from areas with “intact skin with non-blanchable redness” (stage I) to full thickness tissue loss with exposed bone, tendon or muscle” (stage IV) (National Pressure Ulcer Advisory Panel 2007). We use the term "decubitus ulcer" rather than "pressure sore" or "pressure ulcer" because, as explained in this article, pressure is only one of many factors leading to formation of a lesion.

Human Cost

It is difficult for most people to understand the devastating consequences that can result from decubitus ulcers. For a person who normally uses a wheelchair, it can mean months of bed rest and hospitalization.

In addition, after a decubitus ulcer has healed, the skin never fully recovers. Scarring, adhesions and tissue loss in the wake of a decubitus ulcer heighten future risk. Finally, as a person ages, tissue and circulation gradually become less resilient and viable.

Because of the effects of aging, the margin of safety for people using wheelchairs narrows year by year, and the likelihood of something triggering a skin breakdown increases. Decubitus ulcers can destroy careers, upend lifestyles, reduce independence and lead to depression. They can ultimately lead to repeated amputations reaching the trans-pelvic level. Septic conditions can be very difficult to control and lead to death. Fifty percent of all admissions and 8% of all deaths at specialized spinal cord-injury hospitals are due to decubitus ulcers (Thomas 2005).

Numbers of People Affected

It is difficult to estimate precisely the number of wheelchair users who have had or currently have a decubitus ulcer. The following statistics help.

Decubitus Ulcer Generation Factors

Virtually all decubitus ulcers form under or very near weight bearing bony prominences.

More than 2.5 million decubitus ulcers are treated each year in the United States (Reddy et al. 2006). The two major groups of people who have decubitus ulcers are the disabled elderly and people with spinal cord disabilities (Thomas 2005).

Trying to break out the number of wheelchair users who have decubitus ulcers is difficult. One estimate from 1996 was that 25% of people with spinal cord injury develop pressure ulcers each year (Salzberg 1996). The most recent data on the number of people with traumatic SCI is approximately 259,000 according to the National Spinal Cord Injury Statistical Center (NSCISC 2009). Using those statistics, the number of people with SCI who develop a ducubitus ulcer each year could be estimated at approximately 65,000.

People with SCI are not the only users of wheelchairs who battle decubitus ulcers. Other diseases such as multiple sclerosis and stroke can also result in compromised protective sensation. Therefore, the number of users of wheelchairs with decubitus ulcers is certainly much greater than 65,000. The number at risk is greater yet.

Direct Costs of Treatment

According to data from the Center for Medicare Services (CMS), the average cost per ulcer for Medicare patients with a decubitus ulcer was \$43,180 in fiscal year 2007 (Armstrong et al. 2008). This means a conservative estimate of the annual direct healthcare costs related to treatment of decubitus ulcers for people using wheelchairs is about \$2.8 billion (65,000 x \$43,000). Estimates by Reddy et al. 2006 put total United States expenditures on the treatment of decubitus ulcers as high as \$11 billion. Therefore the costs are in the multiple billions.

In seating, the most frequently involved areas are tissue in the area(s) of the sacrum, coccyx, ischial tuberosities and greater trochanters. Four local factors contribute to the generation of decubitus ulcers: pressure, shear, temperature and moisture. Systemic factors (e.g., vascular health, muscle tone, nutrition, age, etc.) and global factors (client education, motivation, lifestyle, program follow-up, etc.) also contribute to ulcer generation. (Koziak 1961; Roaf 1976).

In this article, we focus on the local factors contributing to decubitus ulcer formation: pressure, shear, temperature and moisture. It is these factors that wheelchair seating devices can most affect.

Pressure

Anytime a person is supported by a surface – while lying down, sitting, or standing upright – weight-bearing pressure is applied to body tissue.

Pressure in the scientific sense acts perpendicular to the skin surface. Pressure is calculated in units of force per unit of area. The greater the area over which a given force is applied, the lower the average pressure. For a seated person, peak pressure loads will tend to be concentrated near bony prominences; the ischial tuberosities, greater trochanters, coccyx, and sacrum. That tendency may be mitigated by cushioning materials and by strategic contouring of the support surface.

The most widely accepted interpretation of decubitus ulcer formation relies on pressure producing ischemia which deprives cells of oxygen and nutrients. Over time, this causes tissue cells to die (Daniel et al. 1981; Dinsdale 1974). Related theories implicate ischemia and reperfusion (Houwing et al. 2000), impaired interstitial fluid flow (Reddy et al. 1981), and lymphatic drainage (Miller et al. 1987).

These interpretations have come under increasing scrutiny. Ischemia and ischemia-related events are certainly important but may not be the sole or even primary cause of tissue death. Skeletal muscle tissue cells of rats can survive two hours of complete ischemia but have been shown to die within 15 minutes when a load causes tissue shear deformation (Linder-Ganz et al. 2006).

Other clinical observations and research are also beginning to show the limitations of the ischemia model. That model would predict that by locating and sufficiently off-loading areas of peak pressure on the sitting surface, decubitus ulcer generation can be avoided. Practitioners fitting wheelchair seat cushions have traditionally been guided by external pressure limits between 30 and 40 mmHg which correspond to accepted values for blood capillary pressure (Bennett et al. 1984).

Use of interface pressures between the seat of the wheelchair and the sitter's body, however, has not proven to be a good predictor of decubitus ulcer formation. A systematic review by Reenalda and colleagues discovered "a weak qualitative relation" between interface pressure and the development of decubitus ulcers. In fact, the study concluded that "no quantification of the predictive or prognostic value of interface pressure can be given" (Reenalda et al. 2009).

If pressure induced ischemia cannot fully explain cell death, something else must be contributing to ulcer formation besides peak pressure. More and more attention and research is focusing on shear and friction.

There are several new international collaborations between clinicians and industry focusing on friction/shear and decubitus ulcer formation. They include:

- The Shear Force Initiative of the National Pressure Ulcer Advisory Panel (NPUAP)
- European Pressure Ulcer Advisory Panel
- Japanese Society of Pressure Ulcers

(Call et al. 2007)

Shear and Friction

Pressure is important in relation to shear because it is one of the factors which **ENABLE** friction forces to occur.

In fact, a frequent comment at meetings of the Shear Force Initiative was that the term “pressure ulcer” should perhaps be changed to “shear ulcer” (NPUAP Newsletter Spring, 2008). No doubt this attention means there will be substantial research efforts on studying shear. While such research is needed desperately, it does not offer immediate new direction to wheelchair users or clinicians.

Friction forces act parallel (or tangential) to the skin surface and produce shear stresses and strains within the skin and underlying tissue. Friction causes one surface to resist sliding across another surface that it is in contact with. Shear loads are caused by two phenomena working in combination: pressure and friction (Carlson et al. 1995). Friction resists both sliding and the tendency to slide. Both sliding and the tendency to slide are important in the context of wheelchair seating because damaging friction/shear loads can, and almost always, exist without ongoing sliding motion. Pressure is important in relation to shear because it is one of the factors which **ENABLE** friction forces to occur.

The equation governing the magnitude of a friction/shear load is that it can be no greater than the pressure load multiplied by the Coefficient of Friction (COF). COFs range from near zero to greater than one.

If we define:

- **L_f** as the **friction load** on the skin
- **L_p** as the **pressure load** on the skin
- **COF** as the **coefficient of friction** of the “slipperiest” interface between skin and support surface

Then:

- **L_f** cannot be greater than **L_p x COF**
i.e., **L_f ≤ L_p x COF**

The interesting thing is that the COF value varies widely for various material combinations. That variation is great enough to reduce friction/shear load peaks from 25% to 75%. That reduction is sufficient to significantly increase the margin of safe function for the soft tissue in at-risk locations.

There are two general categories of loading which can damage tissue:

- **Dynamic Loading**
- **Static Loading**

Pressure and friction are **both present** in dynamic and static loading contexts.

In **dynamic loading**, many cycles of pressure coupled with friction/shear occur. Running or walking dynamically loads the feet – this example is also considered *repetitive loading*. **Static loading** is present for a person in a stationary position, lying, sitting, or standing. The skin and underlying soft tissue distorts as body weight is borne by the cushion. Sitting in a chair having a downward inclined seat surface to accommodate a hip extension contracture or with a reclined back rest are examples of scenarios where the *tendency to slide* is particularly high. Seat design and postural support help prevent some sliding, but otherwise, friction is the force preventing sliding.

Research about the damaging effects of friction/shear on tissue is not new.

In the context of dynamic loading, especially of the feet and the hands, there is extensive research on the negative effects of friction and shear. In the 1950s P.F.D. Naylor (Naylor 1955) and later Sulzberger and colleagues (Sulzberger et al. 1966) conducted skin rub tests. Naylor's words best sum up the results: "there is an inverse relationship between the frictional force and the number of rubs required to produce a blister." By reducing the "frictional force," more rubs could be tolerated. This research by Naylor and others was primarily intended to answer questions about skin damage due to extended periods of ambulation or manual labor, but it also tells us something about how dynamic friction/shear loading during transfers, lifts, and shifts on a surface harms skin.

In the context of static loading, such as when positioned in a chair, there is also clinical experience and research. Carlson and Payette in 1995 described techniques to minimize friction/shear in wheelchair seating through orientation of sitting support surfaces, positioning of footrests, and the use of low-friction materials for seat covers. Guttman in the 1970s attributed a larger role to shear than pressure in reducing vascular supply (Guttman 1973).

Bennett and associates in the 1980s conducted tests on 39 subjects seated on a hard seat with subjects divided into three groups: persons with paraplegia, elderly, and controls. The test measured skin pulsatile blood flow and pressure and shear at the seat/skin interface at locations 2 to 3 cm lateral to the ischial tuberosities. The tests revealed the following:

- All groups developed about the same median pressure values, 52 to 60 mmHg
- Median shear values for the groups with paraplegia and the elderly were roughly three times those of the controls
- Median rates of pulsatile skin blood flow volume among the groups with paraplegia and the elderly were one-third those of the controls (Bennett et al. 1984).

Seat Interface Materials and Their Effect on Decubitus Ulcer Formation

Stekelenburg and associates recently conducted rat studies that isolated the effects of ischemia and shear loading. The investigators found that two hours of ischemic conditions caused by a tourniquet resulted in reversible tissue changes whereas two hours of static loading by an indenter induced irreversible damage. The areas damaged corresponded to a region of high shear strain values as determined in separate experiments (Stekelenburg et al. 2007).

Other studies of static loading using animal and finite element modeling suggest that shear deformation of tissue initiates short-term tissue damage. After damage initiation, ischemia may accelerate injury due to hypoxia, glucose depletion, and acidification (Ceelen et al. 2008; Linder-Ganz et al. 2007).

Isolating cause-and-effect in decubitus ulcer formation generated by static loading is beyond current medical knowledge. However, research and clinical evidence indicates that friction/shear play a potentially significant role in ulcer development. We should continue to seek more precise knowledge, but the evidence we have right now is clear. We have no real excuse for not applying available friction management techniques and technologies. A critical purpose of a seating system, in addition to strategically redistributing weight bearing pressures, should be to reduce friction/ shear at key areas of the seating interface such as those underneath bony prominences.

This section focuses on the layer or layers of materials between the seated persons' clothing and the "body" of the seat cushion. We refer to these materials as "seat interface materials". Articles providing clinical guidance rarely address seat covers. This is unfortunate because those materials can have a profound effect on ulcer prevention and treatment.

The goals of the seat interface materials should be:

- **First** – The seat interface materials should be flexible and stretchable in all directions. This allows areas of bony prominences to sink into the recesses of the sitting support surface without developing tension loads within the covering fabric. Those tension loads in the cover fabric (sometimes called "tenting" or "hammocking"), to the extent that they develop, can apply their own load to bony prominences, and for that matter, to any convex surface feature of the contacting anatomy (Carlson et al. 1995).
- **Second** – The seat interface materials should not form ridges, such as those formed by thick creases in material. Thick ridges, especially under a bony prominence, can exacerbate peak pressure.
- **Third** – The seat interface materials should be moisture and air permeable. This helps to transport moisture and heat from the contact areas.

These requirements are well-known and most seat covers incorporate such materials.

What is apparently not well understood is that the seat cover offers an opportunity to address friction, the fourth local ulcer generation factor. Distinct low friction interface regions can be designed and built into seat covers. Low friction areas should be targeted to the most vulnerable areas – the bony prominences. Safe, stabilizing friction levels should be maintained elsewhere.

Clothing is at least one layer of material interposed between the skin and the sitting support surface. One layer of clothing is generally better than two or more. The more clothing, the greater the potential for thick creases to form in the at-risk areas. All rear pant pockets, rivets, and belt loops should be removed. Ideally, the clothing should be breathable, thin, flexible and stretchable. Fashion and cosmetic concerns, however, may affect this decision.

Another set of materials typically found between the skin and the sitting support surface is the seat cover. Materials in seat covers vary tremendously.

For seating professionals, it is relatively easy to determine whether seat interface materials meet the first three criteria listed above related to stretchability, absence of ridges, and moisture and air permeability. It is much more difficult to determine the ability of the various interfaces to manage friction and their effect on the positioning of the seated person. It is with regard to these two goals – friction management and proper positioning – that a new technology from Tamarack Habilitation Technologies is directed.

In 2009, Tamarack Habilitation Technologies completed development of its GlideWear™ Seating Interface Technology. GlideWear™, as shown in Fig. 1 and Fig. 2, is designed to work in concert with currently available pressure management cushions. An international patent application is pending on the design.

GlideWear™ Seating Interface Technology

Fig. 1



Fig. 2



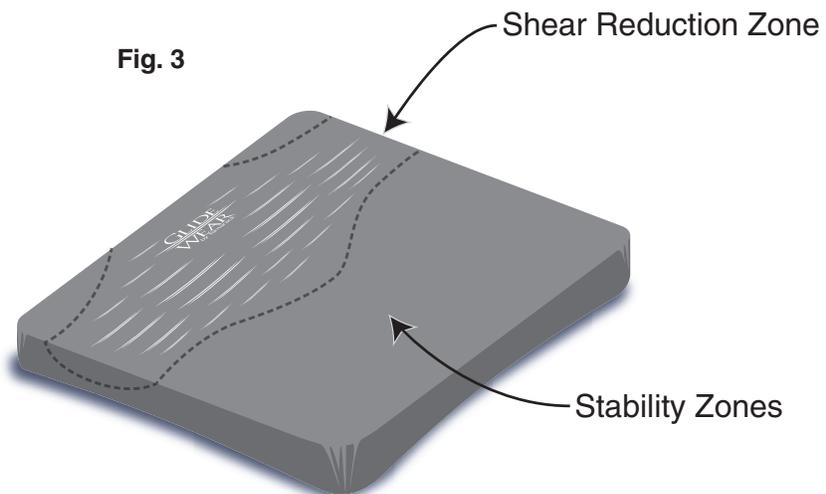
GlideWear™
has several
features and
benefits.

Shear Reduction Zone

GlideWear creates a seating interface with specialized zones. As shown in Fig. 3, the “Shear Reduction Zone” is an ultra-low COF interface for positioning under the sacrum, coccyx, ischial tuberosities and greater trochanters. These are the areas of the sitter’s body most in danger of decubitus ulcer formation due to shear and pressure loading.

Stabilization Zones

GlideWear also creates three “Stabilization Zones” as shown in Fig.3. The purpose of the Stabilization Zones are to help the person using a wheelchair to maintain a stable, functional posture within the chair. Unlike the Shear Reduction Zone, therefore, the Stabilization Zones allow the higher friction in the areas of the body that can readily tolerate higher load forces.



The Stabilization Zones are constructed to be located under the thighs and the postero-lateral portions of the gluteus maximi. These are areas of the body least vulnerable to decubitus ulcer generation.

High Body and Cushion Conformability

The GlideWear device ensures maximum conformability with the user’s body and with the cushion. The GlideWear device is extremely stretchable. The interface stretches at least 90% in each direction. This prevents “tenting” or “hammocking” over bony prominences.

Moisture and Air Permeability

The GlideWear device is highly permeable to air and moisture. This allows moisture and heat to dissipate from the contact areas, especially those areas of greatest concern under the person’s coccyx, ischial tuberosities and greater trochanters. Breathability and insulation of the cushion itself will ultimately determine the degree of moisture and air permeability and heat dissipation.

Conclusion

Decubitus ulcers present a health threat to regular users of wheelchairs.

Ulcers are a serious problem for over 65,000 people with wheelchairs and cost our healthcare system more than \$2.8 billion. Pressure cannot be ignored as a contributing cause of decubitus ulcers, but research strongly suggests it is only part of the picture. One other part of that picture is friction and shear loading. Practitioners should actively consider methods to unload friction and shear from the body/seat interface.

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