A Review of Deep Tissue Injury Development, Detection, and Prevention: Shear Savvy

Amit Gefen, PhD; Karen J. Farid, DNP, MA, CWON; and Ira Shaywitz, MD

Abstract
Pressure ulcer prevention strategies include the prevention, and early recognition, of deep tissue injury (DTI), which can evolve into a Stage III or Stage IV pressure ulcer. In addition to their role in pressure-induced ischemia, shearing forces are believed to contribute substantially to the risk of DTI. Because the visual manifestation of a DTI may not occur until many hours after tissues were damaged, research to explore methods for early detection is on-going. For example, rhabdomyolysis is a common complication of deep tissue damage; its detection via blood chemistry and urinalysis is explored as a possible diagnostic tool of early DTI in anatomical areas where muscle is present. Substances released from injured muscle cells have a predictable time frame for detection in blood and urine, possibly enabling the clinician to estimate the time of the tissue death. Several small case studies suggest the potential validity and reliability of ultrasound for visualizing soft tissue damage also deserve further research. While recommendations to reduce mechanical pressure and shearing damage in high-risk patients remain unchanged, their implementation is not always practical, feasible, or congruent with the overall plan of patient care. Early detection of existing tissue damage will help clinicians implement appropriate care plans that also may prevent further damage. Research to evaluate the validity, reliability, sensitivity, and specificity of diagnostic studies to detect pressure-related tissue death is warranted.

Keywords: pressure ulcer, shear, deep tissue injury, pressure redistribution, rhabdomyolysis, ultrasound


Potential Conflicts of Interest: none disclosed

The most recent estimates published by the Agency for Healthcare Research and Quality (AHRQ) reveal the incidence of pressure ulcers in United States hospitals increased 80% from 1993 to 2006 (from 301,944 to 503,300); during the same time frame, admissions to hospitals for a primary diagnosis of a pressure ulcer increased from 35,800 to 45,500. Pressure ulcers are associated with increased morbidity and mortality. An experimental, retrospective analysis of pressure ulcer quality assurance data conducted from October 1997 to October 2002 to ascertain the relationship among the occurrence of nosocomial full-thickness pressure ulcers, healing, and mortality revealed 68.9% of people who developed full-thickness pressure ulcers died within 180 days. Although these deaths were not all related to pressure ulcer pathology, they suggest severe pressure ulcers and end of life are common co-occurrences, an observation underscored by a consensus statement issued by the 2008 Skin Changes at Life’s End (SCALE) Expert Panel.

Litigation and reimbursement issues add to the burden of pressure ulcers in healthcare. Since October 2008, the Centers for Medicare and Medicaid Services (CMS) no longer reimburse healthcare expenditures related to nosocomial pressure ulcers. Identifying and documenting pressure ulcers “present on admission” has become a focus in hospitals and nursing homes in order to avert reimbursement and litigious repercussions.

The National Pressure Ulcer Advisory Panel (NPUAP) classifies pressure ulcer severity in one of six categories, ranging from Stage I pressure ulcers (the least severe, although Farid et al demonstrated substantial disruption of the underlying vasculature is believed to occur in nonblanchable erythema) to Stage IV pressure ulcers, with visible soft tissue loss extending...
into deep muscle and possibly to the bone. Deep tissue injury (DTI) and unstageable are two recently added classifications. The NPUAP defines a Stage I pressure ulcer as “a localized area of nonblanchable erythema with intact skin.” However, a pressure-related intact discolored area of skin (PRIDAS), a classification that includes Stage I nonblanchable erythema, can exist over or be a symptom of a more serious DTI. An observational, retrospective, correlational study by Farid et al. showed if the damage is great enough or the patient’s condition is such that the injured tissues cannot recover, the PRIDAS will evolve into a serious wound classified as a DTI, the precursor of a Stage III and Stage IV pressure ulcer. Because pressure ulcer staging is dependent on visible skin characteristics, great potential for misclassifying pressure-related injury exists until necrosis is evident. DTIs can remain undetected for days before or after a PRIDAS appears because the skin often does not immediately change color, and it then may take several more days before skin necrosis is manifested. As a DTI evolves, it changes appearance with continued decomposition of the underlying dead tissue. Often, not until clinicians observe a purple, demarcated lesion, classified by the NPUAP as a DTI, or a necrotic eschar (“unstageable”), is the wound clearly documented in the medical record. In patients with very dark skin, a DTI (purple lesion) may not be visible at all, especially in the area of the gluteal fold where skin color is commonly darker. According to a review of forensic literature, by the time dead skin becomes a more detectable black eschar, the original DTI at the level of the bone (the death of the underlying tissues) is approximately 2 weeks old. Regardless of whether the patient has been in the facility 2 weeks or less, the pressure ulcer then is classified facility-acquired “unstageable,” even though most likely was present underneath on admission.

Wound specialists are drawing new insights into the importance of certain blood and urine laboratory results, which may help pinpoint the time since death of the tissues involved in a DTI. In addition, a prospective, observational in vivo study by Aoi et al. on patients is indicative of the increased interest in the use of highly sensitive ultrasound technology in the understanding of DTI development, from isolated friction and shearing injuries to early muscle changes to advanced tissue decomposition.

Although some DTIs may be unavoidable due to skin failure or uncontrollable events such as accidents that occur before admission or a cardiac/respiratory arrest during the admission, many DTIs are preventable. Increasing knowledge of positioning techniques that reduce internal soft tissue loading, including compression, tension, and shear, as well as understanding how positioning interventions work, can very likely reduce pressure ulcer incidence. Such knowledge is also valuable for the education of patients and their caregivers. However, an inherent problem encountered in a comprehensive pressure ulcer prevention program is the seemingly unpredictable nature of pressure ulcers. A prospective study by Curry et al. on the hospital incidence of pressure ulcers suggests DTIs can occur as a result of a febrile episode, a period of cardiovascular instability, or possibly a transient respiratory acidosis several days before. These events can occur regardless of turning patients every 2 hours, applying heel protectors, and getting patients out of bed, sometimes under the most difficult circumstances (unpreventable pressure ulcers).

The purpose of this review is to explore the role of time in the appearance of a DTI and, in addition to pressure ulcer risk scores, how shearing and changes in the patient’s overall condition or position, however transient, may lead to rapid destruction of the deep tissues.

The Role of Shear in the Development of DTIs

According to a mathematical study of shearing forces by Gefen et al., shear can result when external frictional forces stretch the superficial top layers of the skin as it slides against asupportingsurfaceand/or internal tissues shearslides deeper tissue layers over their contiguous surfaces — eg, the sliding of subcutaneous soft tissues over muscle layers and the muscle layers over bony prominences. Frictional forces on the skin and internal tissues shear both contribute to pressure ulcers in general and DTI in particular. These two types of forces are known to interact; the magnitude of internal shearing load is influenced by external frictional forces — eg, internal shear will increase around the ischial tuberosities and especially the sacrum when the backrest of the patient’s bed is elevated. Additional factors involving individual internal anatomy and tissue mechanical properties strongly affect internal shear. The AHRQ Quality Indicators Tool Kit resource on pressure ulcer prevention and management recommends maintaining the head of the bed at the lowest possible angle compatible with patient’s treatment plan to manage shear. However, even if the head of the bed is low, because it is higher than the foot, shearing can still occur due to the effects of gravity. Unfortunately, many patients need the head of the bed higher than
30° because of respiratory indications. Thus, more pressure ulcers occur on the sacrum and heels than any other part of the body. Although sacral shearing is the most common example, shearing can occur on any part of the body, even inside a cast. In a case study, Farid et al stress the neutral positioning of the foot (right angle to the leg) while the total contact cast (a walking cast) is applied because the slightest downward angle of the foot would cause “pistoning” with each step and result in shearing of the soft tissues on the foot.

Carlson quotes Dr. Paul W. Brand, an orthopedic surgeon and the originator of total contact casting, from the National Hansen’s Disease (Leprosy) Program, Carville, LA: “There are two types of force that occur on the sole of the foot: total contact casting; one is vertical force at right angles to the foot, which causes direct stress on the tissues. The other is horizontal force, or shear stress, which is parallel to the surface of the foot and occurs in association with acceleration and deceleration. Of the two forces, shear stress is more damaging than pressure. It should be noted that in any real-life situation, pressure and shear act together; both are necessary for an increase in the intercellular friction between two contacting surfaces (e.g., skin and support). For shear to occur, pressure must push the two against each other.”

Still, Brand’s work emphasizes the importance of studying, quantifying, and reducing shear in soft tissues. Degloving injuries such as those that occur when limbs are caught in machinery accidents are a very visible result of shearing. Although degloving injuries are caught in machinery accidents are a very visible result of shearing, the longitudinal layering arrangement of the cells and bundles of muscle fibers are packed together by connective tissue (perimysium) with different stiffness properties than those of the fibers. Hence, packed bundles of muscle fibers tend to slide upon each other when the tissue is weight-bearing. Even inside the muscle fibers (cells), the molecules are arranged in longitudinal layers that enable them to ride over each other as they contract, ultimately affecting a contraction of the entire muscle. When muscle tissue experiences shearing, the deformity of the cell membranes is disrupted, spilling the contents into the extracellular fluid.

Hence, a DTR involving muscle destruction actually creates rhabdomyolysis (breaking apart of muscle) can cause a false elevation in the myoglobins. Histologically, these patients died (3.4%) of the group contracted rhabdomyolysis as the result of drug ingestion (prescription and illicit) and alcohol. Acute renal failure (ARF) was present in 218 patients (46%), and 16 of these patients died (3.4%). Urine myoglobin was detected in only 19% of the cohort.

Abdominal pain, nausea, vomiting, and weakness are the initial symptoms in the majority of cases. The urine may turn pink as a result of myoglobinuria (myoglobinuria is the presence of myoglobin in the urine). The urine may appear dark brown (“chocolate urine”) as the myoglobin is oxidized in the bladder. Myoglobin is a protein that is released from damaged skeletal muscle. Myoglobin is a water-soluble protein that is filtered through the glomeruli of the kidneys. Myoglobin can cause a false elevation in the myoglobin. However, a review of the literature by Bagley determined the half-life of myoglobin is approximately 4 to 6 hours, and the discoloration of the urine may be missed. Another sequela of rhabdomyolysis is an elevation of CK in the bloodstream. CK is an enzyme specific to muscle tissue. When CK is elevated to more than 1,000 IU/L (normal CK is 60 – 400), some muscle destruction can be assumed. A retrospective review of 1,362 patients at an acute care center by Melli et al showed 475 patients (>30%) with an acute neurovascular illness with serum CK had more than five times the upper limit of normal (>975 IU/L), indicating skeletal muscle damage (rhabdomyolysis). Of the 475 patients, 46% of the group contracted rhabdomyolysis as the result of drug ingestion (prescription and illicit) and alcohol. Acute renal failure (ARF) was present in 218 patients (46%), and 16 of these patients died (3.4%). Urine myoglobins were detected in only 19% of the cohort.

An elevation of the cardiac enzyme creatinine kinase mitochondrial-B (CKMB) can cause a false elevation in the CK (and vice versa), but an elevation of the CK with a
negative test for troponins can be interpreted as a sign of skeletal muscle injury.\(^{40,44}\)

Blood levels of CK usually start to rise during the first 12 hours of muscle destruction, peak during the second 12 hours to 3 days, then start to decline after the third day post-muscle death. If a patient is brought into the emergency room (ER) with a history of an unwitnessed fall, impaired consciousness, and/or high fever, sitting long hours in a chair and unable to stand, or with existing pressure ulcers, among other similar immobility histories, an order for CK levels to be done in the ER and again in 24 hours, and a urine myoglobin would be warranted.\(^{39}\) Elevated blood urea nitrogen (BUN) and creatinine in a patient with no history of renal failure warrants a test for CK, because elevated CK and myoglobins in the blood can lead to renal failure.\(^{46}\) CK and presence of myoglobin in the urine are possible indications that a DTI has occurred, even though it may not yet be visible on the skin surface. Although elevated CK is not specific to DTI, according to literature reviews,\(^{45,48}\) the subsequent appearance of a DTI within 1 week after the admission with an elevated CK in the first 24 hours of admission may lend credibility to an argument of “present on admission,” providing there is no other muscle injury, such as trauma. Current research is looking at additional biomolecules as potential biomarkers that might provide an earlier alert to the occurrence of skeletal muscle death compared to CK. These additional molecules are heart-type fatty acid binding protein (H-FABP), myosin, and troponin-I (TnI).\(^{49}\) Testing these molecules has so far been done in animal (rodent) models only.\(^{50,51}\) Although the initial results are promising, further work is needed to test their applicability to human patients in general and DTI specifically.

If the DTI is extensive and myoglobinuric levels become highly elevated, the patient may go into acute renal failure, the tubules of the kidneys becoming clogged with precipitating proteins. In this case, the BUN levels in the blood will rise along with creatinine and potassium (K+) levels. This is more likely to happen if the patient’s intravascular volume becomes contracted — i.e., if increased fluid shifts from intravascular fluids into the extracellular compartment or inadequate intake of fluids is noted. Usually, as soon as the clinician notes increases in the BUN and creatinine, IV fluids will be administered to perfuse the kidneys. If the serum K+ levels increase excessively, the patient will probably need hemodialysis.\(^{40,46}\)

Another initial sign of rhabdomyolysis is a drop to below normal in serum calcium (Ca++). This can occur even before the serum CK level begins to rise.\(^{44}\) This may be related to the fact that the dead muscle immediately shortens and exhibits a sustained contraction (rigor mortis), which requires concentrated levels of Ca++ within the muscle cells to drive the contraction. Rigor mortis of the muscle continues, causing a palpable thickening (a lump), for about 36 to 72 hours until the muscle cells actually decompose and break apart.\(^{37}\) This may be an explanation for an early drop in the serum Ca++ not associated with drops in serum albumin.\(^{52}\) Rebound hypercalcemia may occur in about 43% of those patients suffering acute renal failure as a result of rhabdomyolysis if diuresis recovers (see Table 1).\(^{52}\)

### Table 1. Chemistry of rhabdomyolysis\(^*\)

<table>
<thead>
<tr>
<th>Byproduct of muscle breakdown</th>
<th>Test (normal)</th>
<th>Onset (hours)</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Creatinine phosphokinase (CK)</td>
<td>Serum CK (60–400 IU/L)</td>
<td>Rises over first hour to 12 hours from the time of muscle death</td>
<td>Half-life of 36 hours</td>
</tr>
<tr>
<td>Myoglobin (UMGL)</td>
<td>Urine (0)</td>
<td>Rapidly excreted in urine (dark amber “tea-colored” urine) in 1–6 hours after muscle death</td>
<td>Depends on amount of muscle damage: clears hours to days after muscle death</td>
</tr>
<tr>
<td>Calcium (Ca++)</td>
<td>Serum calcium (8–10 mg/dL)</td>
<td>Rapid drop after muscle death (without drop in albumin)</td>
<td>7 days or more</td>
</tr>
</tbody>
</table>

\(*\) These chemistry changes are markers of rhabdomyolysis (muscle death) that may occur with a DTI in certain anatomical locations (eg, trunk).

Ultrasound and Detection of Friction, Shear, and Early Pressure Ulcers (Rhabdomyolysis and DTI)

Although understanding the chemistry of muscle destruction is helpful to appreciate the effects of shear and possibly indicative of the presence of DTI before the appearance of visual changes, there is no proof yet of the predictive value of chemistry for DTI. Nothing surpasses the ability to actually visualize the presence of shear injury and/or DTI even before it becomes visible to the naked eye. Ultrasound technology has been advancing exponentially over the past 2 years, and it seems quite likely that in the future ultrasound can be used for skin imaging on admission and/or to determine the extent and severity of pressure-related intact skin disfigurements and disruptions, possibly eliminating the need for staging completely.\(^{11}\) Wendelken et al\(^{13}\) describe several chronic wounds of varying etiologies using digital photography and ultrasound images, demonstrating the advantages of each evaluation tool in assessing these wounds and the high
A laboratory study by Rippon et al. compared histological samples of pig skin and human cadaver skin to ultrasonographic imaging of the same samples and found a very high correlation (P < 0.0001) between the two measurement modalities.

Utilizing ultrasound to evaluate shear and resulting DTI has several advantages. Ultrasound is a hands-on, real-time examination that, because it does not utilize radiation, allows anatomic structures to be viewed while interacting with the patient during the imaging. Another advantage is the speed at which it can be performed. The equipment consists of a ultrasound machine with its probe and, depending on the structures being studied, can be very portable.

Understanding ultrasound terminology with regard to image description and evaluation is important for interpreting the resulting report (see Table 2). In an ultrasound scan, tissues reflect the sonic impulses emitted by the probe and the reflections are transmitted back the to transducer. The transducer converts these reflected impulses into images in shades over a range from white (strong reflections) to black (no reflections). Normal fascia appears as a bright hyperechoic structure. Subcutaneous tissue is isoechoic (the same brightness) of skeletal muscle. The difference between normal subcutaneous tissue and skeletal muscle is that the septae of the subcutaneous tissues do not lay in lines or layers. A thick, continuous, hyperechoic band (fascia) usually separates subcutaneous fat from muscle and tendon (structures such as the heel do not have muscle tissue). Normal thick tendon and cortical bone appears as well defined, linear, smooth, continuous bright with posterior shadowing (homogeneous hypoechoic area) (see Figures 1 and 2). Thus, detailed images of the fascia, superficial and deep soft tissues, and bones are produced. Subtle abnormal changes in these tissues also can be detected. Ultrasound holds promise in the detection of DTI because it is an excellent imaging modality to evaluate the musculoskeletal system. A prospective study by Deprez et al. using numerical simulations, physical acquisitions on pressure ulcer mimicking phantoms, and in vivo experiments on rats “demonstrated that ultrasound elastography is a promising technique for pressure ulcer detection, especially at an early stage of the pathology, when the disease is still visually undetectable.” One of the earliest signs of muscle death in the first hours after injury, identified in experimental animal studies by Gefen et al., is “stiffening” compared to the surrounding normal muscle. This very early muscle response, because of the sensitivity of the ultrasound to differences in tissue consistencies, was detectable in the Deprez study.

Table 2. Ultrasound terminology and definitions relevant to pressure ulcers and deep tissue injuries

<table>
<thead>
<tr>
<th>Echotexture</th>
<th>The homogeneity (all bright or all darkness) versus non-homogeneity or coarseness (mixed areas of brightness and darkness) of an object</th>
</tr>
</thead>
<tbody>
<tr>
<td>Echogenicity</td>
<td></td>
</tr>
<tr>
<td>Hyperechoic</td>
<td>The ability of tissue to reflect ultrasound waves — the higher the echogenicity of the tissues, the brighter they appear on ultrasound imaging</td>
</tr>
<tr>
<td>Hypoechoic</td>
<td>Structures seen as brighter or darker on conventional ultrasound imaging relative to surrounding structures due to reduced reflectivity</td>
</tr>
<tr>
<td>Isoechoic</td>
<td>Seen on imaging as equally as bright as the surrounding tissues due to similar reflectivity</td>
</tr>
<tr>
<td>Anechoic</td>
<td>Structures lack reflectors and therefore appear black on ultrasound</td>
</tr>
</tbody>
</table>

Figure 1. Example of high-frequency ultrasound of planter left heel surface showing soft tissue disruption. Blue arrow: Achilles tendon; orange arrow: calcaneous; green arrow: red and blue dots representing increased blood flow associated with hyperemia and inflammation as a result of friction.

Figure 2. Example of high-frequency ultrasound posterior left heel scan showing soft tissue disruption. Blue arrow: Achilles tendon; pink arrow: calcaneous; green arrow: epidermis; orange arrows: damaged architecture of the subcutaneous tissue with hypoechoic areas representing edema secondary to shearing.
Evaluation for DTI would involve the use of a high-frequency, high-resolution transducer (3–17 MHz) in order to image the soft tissues and bony structures. A special transducer attachment (a "standoff pad") is typically used so the superficial soft tissue structures can be further attenuated.\(^5^7\) Results of an animal study by Moghimiet al\(^5^8\) of experimentally created full-thickness pressure damage (DTI) on 36 healthy guinea pigs that were monitored with ultrasonography for 21 days supports the observations made in animal studies by Hansen et al,\(^5^9\) Iaizzo,\(^6^0\) and Harder et al\(^6^1\) in forensic correlations made and later supported by Farid,\(^5^8,6^9\) that significant changes occur in the tissues on day 7 post injury that cause DTI to become visible on examination of the skin. The investigators in Hansen's study observed the full extent of the deep tissue necrosis day 7. Farid described the same phenomenon as "demarcated purple ulcer," as did Harder, who described the formation of the faux lunatica as the phenomenon that physically defined demarcation on day 7. Iaizzo also described "consolidation" of the full-thickness ulcers on day 7. Moghimiet al describes the changes on ultrasound in his study as a hypoechoic area under the dermis, present on the earlier scans, disappearing on day 7. Going forward, investigators of DTI will need to agree on descriptive terms concerning DTI development at various time points in order to correlate findings and develop a cohesive body of research on the subject. Nevertheless, it is the opinion of the authors that the events surrounding occurrences on day 7 post DTI warrant ongoing examination.

Ultrasound in practice. Steed et al\(^6^2\) describes an ultrasound of the leg of a patient who had arrived in the emergency room with pain and swelling of the leg that was suspicious of an acute deep vein thrombosis. However, the ultrasound detected early rhabdomyolysis related to a diabetic muscle infarction of the calf muscle. The patient's symptoms had started <24 hours before the ultrasound study. Although this case study describes a different kind of muscle injury, not DTI, it highlights the sensitivity of ultrasound to detect rhabdomyolysis in its very early stages. In a prospective study by Quintavalle et al\(^6^3\) comparing nursing home admissions to healthy volunteers, 630 ultrasound images were taken of the usual sites vulnerable to pressure ulcer formation; 53.3% of the nursing home residents' images were abnormal when compared to healthy volunteers. These were one-time ultrasound examinations of the nursing home residents without any follow-up, so no conclusions could be drawn regarding later DTI development. No examples of these abnormalities were described in the study, and none of the nursing home residents had any skin changes over the sites at the time of examination. In a prospective study of 15 pressure ulcers ranging from Stage I to Stage IV, Andersen and Karlsmark\(^6^4\) compared four different noninvasive examination methods for sensitivity and specificity in evaluating pressure ulcers severity: redness index, skin temperature, skin elasticity, and ultrasound scanning. The 15 pressure ulcers all were compared to adjacent normal skin using the same examination methodology. The authors did not find any evidence that any of the examination modalities could predict the severity of the pressure ulcers, but they remark that ultrasound had the most potential for identifying early damage from pressure.

An ultrasound was performed on a reddened area on the heel of a patient with peripheral vascular disease (PVD) in the author's practice to rule out DTI (see Figures 1 and 2). An amputation of the patient's other foot had already been recommended during the same admission. A DTI was ruled out, but the sensitivity of the ultrasound technology to changes in tissues (substructures (collagen fiber bundles in connective tissue) clearly illustrates abnormal edema between the dermis and subcutaneous layers and also infiltrating and separating bundles of connective tissue under the subcutaneous fat, dilated blood vessels, and disruption (wavy patterns) of connective tissue at the level of the Achilles tendon and the calcaneus — early damage characteristic of both friction and shear. Follow-up evaluation of the patient noted full recovery of the tissues after 2 weeks of pressure relief.

The information to date regarding use of ultrasonography in the assessment of wounds is encouraging, but, for the purposes of gathering data on DTI detection from the time of initial injury/tissue death, patterns and timing of DTI progression to the time of visual identification on the skin surface, additional prospective, controlled trials need to be performed.

Managing Shear Forces

Because shearing is mostly invisible or subtle at best, it is important to have a deliberate, clear anti-shearing patient positioning protocol in place for all high-risk patients. Although the Braden Scale is an excellent risk-evaluation tool, clinicians are aware that the Braden Score does not reflect a wide variety of disease processes and physical conditions such as abnormally high or low body mass index (BMI) that may compound patient risk for pressure ulcers.\(^5^5,6^6\) It is well-known that patients below ideal body weight are at risk for pressure ulcers, even if the serum pre-albumin and albumin levels are normal, but some clinicians also suggest obese patients are also at risk.\(^6^7\) When compounding BMI abnormalities with other comorbidities, preventing injury — e.g., an ischial DTI from sitting — becomes a major challenge.\(^6^8\)

Positioning guidelines, including the Agency for Healthcare and Quality (AHQR) guidelines,\(^6^9\) recommend changing the patient’s position every 2 hours, regardless of whether the patient is in a bed or chair. Patients who need extra assistance transferring, such as a bilateral amputee patient who is not strong enough to move his body along a sliding board for transfers, may need a lift (or a specialized bariatric lift if he is obese) to get out of bed and be lifted back into bed, hopefully in 2 hours or less. The healthcare facility will need to incorporate this need into the patient’s care planning. However, when the patient returns to his home, he will need help
greater and helps offload the ischium. A prospective study of 75 elderly nursing home patients (ages 25 to 95 years, mixed male and female) by Kemozek et al\(^6\) correlated the BMI with seat-interface pressures and found that as BMI increased, theseat-interface pressures decreased. However, wheelchair-dependent bariatric patients still need to reposition every 2 hours to prevent ischial pressure ulcers.\(^6\)

Patients with normal BMI. For paraplegic patients or patients with bilateral amputations who cannot use prostheses but have use of their upper extremities and normal-range BMIs, additional pressure relief in wheelchairs is achieved by increasing the seat “dump” — ie, tilting the seat up so the occupant is shifted back against the seat, as is done for sport wheelchairs (Invacare Crossfire, Invacare, Spring Valley, OH). This causes the patient to lean further forward onto the distal posterior thighs every time he or she wheels the chair, relieving pressure on the ischium. It also distributes the pressure back over the lumbar and thoracic region. Several patients cared for by the author who spend long periods in the wheelchair and are not comfortable in a sport wheelchair have solved the problem of pressure relief utilizing the same “dump” concept as the sports wheelchairs by using a portable ramp. The ramp is professionally constructed from a height of 0 inches to a maximum height of only 4 to 5 inches. The ramp is short enough so only the front wheels are driven up the ramp, tilting the chair backwards like a recliner. Thus, the ramp can be used while watching TV or socializing with visitors while relieving shear and pressure on the ischium.

Severely disabled patients. “Tilt-in-space” adjustable electric wheelchairs are available for severely disabled patients with multiple sclerosis, cerebral palsy, and cervical spinal cord injuries (patients with thoraco-lumbar [paraplegic] spinal cord injuries have use of their upper extremities and are encouraged to wheel their wheelchairs). Tilting the whole seat (hence “tilt in space”) much like a bucket seat in a car can help achieve an effective anti-shearing position (see Figure 4). Frequently called the anti-gravity position, it evenly distributes the weight of the person over the entire length of the body, preventing shearing on all the supporting bony prominences — ie, the shoulders, sacrum, ischium, and heels. This positioning is similar to the design used in infant carseats and carriers and involves the same technology employed in astronaut positioning on the launch pad — ie, the tipped back chair position enables the astronaut to withstand up to eight times the pressure encountered in normal gravity.\(^7\)

The labor-intensive activities of daily living procedures involved in preparing a quadriplegic patient for getting out of bed into the wheelchair requires methods to be devised to relieve ischial/sacral pressures while the patient is still in the wheelchair. Quadriplegic patients can be taught to lock their elbows to one side then the other and push against the side of the wheelchair seat to rock side-to-side and relieve the ischial tuberosities. Also, manual tilt-in-space wheelchairs are now available, so if a patient wants to spend several hours out of

---

**Figure 3.** Stage III pressure ulcer caused by edge of wheelchair seat (blue arrow).

**Figure 4.** Motorized tilt-in-space wheelchair. This quadriplegic (multiple sclerosis) patient has been lifted into the chair with a motorized lift after the chair is adjusted in tilted position to facilitate placement of the patient’s pelvis into the angle of the chair back and seat. Because the patient is nonambulatory, foam heel protectors are placed to protect the lower legs and feet from shearing, should the legs go into spasm.
bed in the wheelchair, the wheelchair is built on curved railings that enable the wheelchair to be manually tilted back to a recliner position by a caregiver (see Figure 5).72,73

Beds. The key principle for prevention of shear is pressure distribution, which can be enhanced by softness of the surface — eg, elevating the foot of the bed (distribution) and foam heel protectors (cushioning). The author has observed that many people do not know the difference. Technology has made positioning patients in bed much easier over the past decades; even the most basic beds used in inpatient care provide caregivers the ability to adjust the patient into the zero-gravity recliner position (see Figure 6). For patients at home who have shortness of breath and need to sleep with the head elevated, a recliner is frequently recommended for comfort and pressure distribution and relief.

The technique of raising (“gatching”) the foot of the bed is not a new concept. It was originally recommended in the International Association of Enterostomal Therapists (IAET, the original association for wound and ostomy nurses) positioning guidelines (1988)73 and supported by the American Association of Critical Care Nurses (AACN).75 By employing the simple maneuver of pressing a button or turning a crank to raise the foot of the bed in proportion to the head of the bed to distribute weight, the lumbar curve of the spine flattens, and the surface of the back, pelvis, and thighs becomes a smooth, fluid line from the shoulders to the posterior knees, bringing the body into maximum contact with the supporting surface, helping eliminate shear. Although many beds have a default function that automatically raises the foot of the bed slightly when the head is raised, the foot of the bed may still need to be adjusted based on the height of the head.

Raising the head of the bed higher than 45˚ will make it very difficult to prevent shear or to turn the patient side to side. Also, the higher the head of the bed is adjusted, the more upper body weight shifting onto the pelvic girdle, the greater the risk of sacral and ischial pressure ulcers.24,29,74

The principle of distribution applies to all parts of the body, including the heels, another common pressure ulcer site. If the heels are sliding down the bed along with the rest of the body, the knees may buckle, causing the heels to further push into the mattress surface, increasing the risk of severe shearing damage. Raising the foot of the bed also reduces lower extremity edema, decreasing the weight of the interstitial fluid on the heels.75 Bony prominences on the feet are vulnerable to pressure damage because, anatomically, the skin lies directly over tendons and bones with minimal cushioning from subcutaneous tissue. In the opinion of the authors, egg crate 2-inch foam protectors are usually adequate, provided they cover the entire foot, top to bottom, from above the ankles to beyond the metatarsal joints.

Recliners. Placing a patient who is generally immobilized in a recliner provides an alternative to the bed. The patient can periodically be placed in the fully reclined (anti-gravity) position to offweight the ischium and use the upright sitting

Figure 5. Manual tilt-in-space wheelchair. The leg rests elevate and the “chair” is supported on curved runners (arrow) so it can be positioned at any angle along the runners. For quadriplegics, the chair can be tilted all the way back, like a recliner, every 2 hours, substantially reducing pressure and shear on sacrum and ischium.

Figure 6. Anti-shear positioning, distribution of weight, softening of the surface (heel protectors).
position for meals. The use of a recliner is an excellent arrangement for any patient who cannot stand/transfer and is not wheelchair-independent in mobilization. The intermediate recliner position (half up/half down) also can be used for short periods (<2 hours) because the pitch or angle from head to foot is too steep with only half the backward tilt of the seat achieved with the full reclining position and, eventually results in shearing on both the sacrum and the ischium. However, the advantage of using recliners in general, is that the caregiver can reposition the patient frequently and even shift the patient side to side in the fully reclined position if it is wide enough.

### Conclusion

DTIs may occur regardless of using the best techniques and consistent repositioning, whether at home or in hospitals and nursing homes.1 The earlier a DTI can be detected, the sooner special pressure-relief measures can be instituted to help tissues to recover and possibly decrease the resulting damage. Additional studies regarding the use of laboratory screening for DTI on admission to a facility and the judical use of ultrasound to screen admission or investigate PRIDAS are warranted. Although CK drops in serum calcium and myoglobinuria are general markers of muscle death, most of the links to pressure ulcers are made retrospectively. The CK, for example, may be elevated early in the patient’s admission, and a myocardial infarction can be ruled out using tests for other markers. Only when the DTI actually appears can clinicians connect the dots. Closer examination of the use of CK, myoglobinuria, and concomitant drop in serum calcium, in association with the development of DTI, perhaps in animal experiments where the necessary controls can be established, is warranted. Further studies on lesser known biomarkers are necessary to assess for sensitivity and specificity to pressure-related tissue death.

### References


