Tissue Changes in Patients Following Spinal Cord Injury and Implications for Wheelchair Cushions and Tissue Loading: A Literature Review

Amit Gefen, PhD

Abstract
Persons using wheelchairs, especially those with spinal cord injuries (SCI), may be susceptible to tissue changes that affect their risk for pressure ulcer (PU) development. To examine the complexity of the problem of designing, selecting, and prescribing an optimal cushioning solution to help prevent PUs, a literature search was conducted examining factors that affect the biomechanical interactions of the seated buttocks with the cushion and how these factors may change over time. The majority of publications retrieved were preclinical studies and case studies, and just a small fraction was randomized clinical trials. The literature indicates that external and internal anatomy and tissue structure and function change considerably in the months and years following the loss of sensation and mobility. Specifically, these changes typically include weight and fat mass gain, skeletal muscle atrophy and fat infiltration into muscles, bone loss and bone shape adaptation at the pelvis, vascular perfusion changes, and microstructural changes in skin and muscle that are associated with disuse and affect the biomechanical behavior of these tissues. Support surfaces, particularly wheelchair cushions, should be designed to accommodate microchanges that occur for a seated person throughout the day (eg, changes in posture and position or muscle tone) as well as macrochanges in anatomy, tissue composition, and long-term tissue (patho)physiological changes. Cushions must be tailored to, and adapted for, each individual patient on a regular basis. A promising and practical bioengineering approach to fit cushions to different patient conditions is to use computer simulations (finite element [FE] modeling). As understanding about PU risk in this population increases, study designs can be refined to develop a much-needed evidence-base for the appropriate use of support surfaces in general and wheelchair cushions in particular.

Keywords: review, spinal cord injuries, pressure ulcer, tissues, durable medical equipment

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The current, most fundamental guidelines for prevention of sitting-acquired pressure ulcers (PUs) are to use a soft cushion on the wheelchair that is thick enough to allow adequate immersion to distribute buttocks-support loads while preventing undesirable bottoming-out. Patients who suffer a spinal cord injury (SCI) are routinely prescribed cushions for their wheelchair as part of their rehabilitation process, with the aim of avoiding PUs and particularly the severe type deep tissue injury (DTI), which is a PU with an internal onset, typically in skeletal muscle tissues near the ischial tuberosities (ITs). Healthcare providers agree that use of appropriate cushions is critical for protecting SCI patients from PUs and DTI, but what exactly is “appropriate” is unclear. The abundant variety of cushioning solutions includes flat foams, segmented foams, contoured foams, water/gel-filled cushions, honeycomb-like cushions, and air-cell-based cushions. Analysis of the current literature in an attempt to understand which cushion technology is superior or most cost-effective is inconclusive. This is not surprising, given the difficulties and costs involved in randomized clinical trials for isolating the efficacy of a cushion.
Perhaps the most thorough and rigorous cushion trial was conducted by Brienza et al.\(^7\) This group published extensive randomized clinical trials and secondary data analyses from trials\(^4,5\) of nursing home populations (not SCI patients). However, as they stated in their own work, a key problem in these trials is the ability to isolate the effect of the cushion from that of the wheelchair. If the cushion is ideal but the wheelchair does not fit properly or is improperly adjusted, the patient will sit in a compromised posture (eg, posterior pelvic rotation or pelvic obliquity), increasing the risk for PUs. In their 2010 study, Brienza et al\(^7\) finally were able to eliminate this problem by fitting and adjusting wheelchairs to each of their participants (180 subjects, all nursing home residents who reached the study endpoint of a 6-month follow-up), which is a remarkable achievement involving substantial cost and effort. The authors tested several segmented foam or skin protection cushions that were randomly allocated to the study participants. The skin protection cushions were assigned within that category based on clinical needs and preferences, and included air, viscous fluid plus foam, and gel plus foam cushions. Given the incidence rate of PUs was higher in the patient group prescribed segmented foam cushions than the group given skin protection cushions, the researchers concluded the segmented foam cushions were less protective against PUs. A commentary by Beeckman and Vanderwee\(^6\) notes several additional factors are extremely difficult to control in such large-scale cushion trials — eg, standardizing the method of assessing skin viability/damage, handling interobserver differences, and standardizing repositioning in the wheelchair. Taken together, these studies illustrate the challenges of conducting clinical trials in this area and underscore the need for complementary research methods, particularly bioengineering laboratory studies and computer simulations, to help prioritize cushion technologies and products. That said, current research highlights the primary biomechanical goals quality therapeutic and preventative cushions should definitely meet: minimization of interface pressures (IP),\(^4\) minimization of internal sustained tissue deformations, and particularly, minimization of localized deformations in skeletal muscles under the ITs.\(^7,10\)

To examine the complexity of the problem of designing, selecting, and prescribing an optimal cushioning solution to help prevent PUs, a literature search was conducted focusing on factors that affect the biomechanical interactions of the seated buttocks with the cushion and how these factors may change over time. The relevant English language literature from the last 20 years was searched using PubMed for terms such as pressure ulcers, sitting, buttocks tissues, support surfaces and cushions, spinal cord injury, deformation injury, and biomechanics. The majority of publications retrieved were preclinical studies and case studies, and just a small fraction was randomized clinical trials. The focus of the literature search was on SCI patients who typically undergo dramatic changes in structural anatomy and tissue physiology following their injury that evolve and progress throughout life. However, some of these structural and physiological changes, particularly adaptation of muscle and skin tissues to disuse, are also relevant to the elderly and infirm. These complex and interacting changes are reviewed and then analyzed as to how they could theoretically influence the considerations for cushion design and selection for the SCI patient population. It is expected that nurses, physical therapists, rehabilitation practitioners, and other medical professionals will find this information relevant to their day-to-day patient care and practice.

**Microchanges That Affect Tissue Loading**

As used here, *microchanges* affecting tissue loading refer to changes that relate to the daily functioning of SCI patients — eg, changes in posture or movements, the individual adherence to guidelines for PU prevention such as the frequency of postural changes and performance of push-up maneuvers, and factors that concern the pathophysiology of the SCI condition, such as the muscle tone around the regions at risk.

**Sitting posture and use of the upper extremities.** Even without considering any SCI-specific pathoanatomy or pathophysiology, the factor with the most fundamental effect on soft tissue loading during wheelchair sitting is wheelchair position and momentary posture. Shabshin et al\(^11\) was the only group to investigate in vivo buttocks tissue compressive deformations near the ITs in healthy volunteers during sitting in various body tilts utilizing weight-bearing seated magnetic resonance imaging (MRI). The tissue deformations were digitally measured in a coronal T1-weighted MRI sequence. Each image was evaluated for thickness of fat tissues between the skin and the gluteus muscles and of muscle tissues between the ITs and fat. Measurements in the weight-bearing
and non-weight-bearing positions were compared per participant for calculation of compressive tissue deformations in each trunk tilt. Mean muscle compressive deformation in their study, from highest to lowest, was 20° lateral-tilt (extent of deformation = 87%); lateral 10° (85%), anterior 20° (79%), anterior 40° (74%), and neutral (72%). Fat compressive deformation was highest (and statistically similar) with anterior tilts of 20° and 40° (42%), followed by lateral 20° tilt (41%), lateral 10° (39%), and neutral (35%). Hence, a slight tilt of the trunk to the side as in imbalanced sitting or during a laterally reaching movement in healthy persons can increase muscle deformation by at least 15% and fat deformation by at least 7%. This is a considerable added extent of deformation, particularly because soft tissue deformations are already substantial during balanced sitting.12 Any additional unilateral excessive loading that persists may push these tissues above tolerance levels and cause cell and tissue death in insensate individuals, as demonstrated in animal (rodent) and tissue-engineered in vitro models.13,14 These biomechanical data highlight the rationale for preferring cushions that support the body in a balanced, stable posture and that allow maintaining a posture that is as balanced as possible throughout the day. Maintaining a balanced posture is particularly important in individuals with SCI, because these persons typically have less-than-optimal postural alignment, as indicated in a study of four Brazilian participants with paraplegia and an additional four with tetraplegia.15 Nevertheless, this does not necessarily overlap the goal of maximizing tissue protection against PUs. In other words, even if the posture appears to be balanced in the wheelchair, it does not automatically mean the cushion also is optimizing tissue loads; theoretically, even a completely rigid support can provide balanced sitting but produce extremely large tissue deformations in both sides of the buttocks, which would compromise tissue viability.

Another critically important factor the cushion affects is the patient’s ability to use his/her upper extremities, such as when performing reaching movements. Results of a study16 that tested balance and time to reach a target in 11 individuals with SCI versus six individuals without SCI showed that patients with SCI had more difficulties reaching than healthy individuals and that it takes persons with SCI more time to accomplish activities, which has been attributed to a postural control deficit. Hence, SCI patients need special attention in terms of providing a sitting solution that will not just protect them from PUs, but also will allow adequate daily functioning. On the one hand, a stable pelvis and trunk are required as a base for steady upper extremity movements; on the other hand, functional range of motion increases by allowing the pelvis and trunk to move (up to a certain extent). Therefore, as shown in a randomized, controlled test17 designed to monitor the sitting pressures and forces in nine individuals with SCI during reaching tasks, a good cushion should provide both pelvis and trunk support and sufficient trunk mobility. Attention to these issues holds important consequences for patient quality of life.17,18 In a study examining reaching movements in 22 American patients with SCI who used different cushions, Sprigle et al19 observed sitting with increased posterior pelvic tilts enhanced body stability and permitted greater reach. In general, because patients adopt different postures when using different cushions (and backrests), posture needs to be assessed along with the functional reach capacities on the selected cushion. These factors then should be considered with respect to the cushion’s capacity to accommodate changes in position throughout the day, while keeping tissue deformation as minimal as possible.

**Individual risk and prevention behavior.** Individual awareness of PUs and cooperation with rehabilitation guidelines, which essentially also affect the biomechanical interactions with the cushion, are additional considerations. In a preclinical computational simulation study, Levy et al19 investigated buttocks tissue loading when sitting down after completing a push-up maneuver, as SCI patients often are taught to do. The research found substantial differences between the loading curves of skin and fat when sitting on flat foams. Although fat tissues were loaded at a nearly constant rate during the sitting-down process, skin loads increased nonlinearly, with a greater load/time slope around the early skin-support contact — ie, the more sensitive period with respect to pressure is at initial skin-support contact when sitting down. Inclusion of scars associated with a history of PUs in their simulations indicated that, as expected, individuals with scars are more susceptible to further tissue damage. Specifically, the study found risk for skin damage during sitting down is greater when a hypertrophic scar is present. Despite the fact that this was a theoretical modeling study with associated limitations, it points to two important issues in the context of the present paper. The first is proper guidance of how to avoid PUs, which needs to accompany the prescription of the cushion, is required — ie, patients need to be trained to reposition themselves gradually and gently rather than “fall” back into the wheelchair after finishing a push-up maneuver. In other words, educating patients to use their cushion properly is a critical component of the sitting solution; a wheelchair cushion is probably not meant to be bought off-the-shelf without fitting and guidance. The second is, as reflected in the Levy article19 and the literature reviewed within, the risk for PUs depends on the individual, his/her medical condition, and his/her history, and these need to be assessed rigorously while taking a holistic approach, tailoring the sitting solution to the individual.

**Muscle tone.** As already indicated, the SCI population needs special attention when considering sitting solutions, given the substantial tissue changes that occur from the time of injury and throughout life. One such fundamental change involves skeletal muscle tone. Spasticity, the exaggeration of normal reflexes that occurs in response to stimulation of the sensory system, is one of the most common neuromuscular...
changes seen in SCI, affecting approximately 70% of this population within a year post injury. In able-bodied persons experiencing skin stimulation, a sensory signal is sent to the reflex arch in response. This signal travels to the brain via the spinal cord and is assessed in the brain. If the signal received does not indicate danger, such as from heat, injury, and the like, a subsequent inhibitory signal will be sent by the brain back through the spinal cord to cancel the reflex of the musculature. However, in individuals with SCI, inhibitory signals are blocked by the structural damage in the spinal cord; hence, the natural reflex is allowed to continue, resulting in continuous muscle contractions (spasms). The immediate biomechanical implication is the stiffening/contraction of weight-bearing muscles (eg, the gluteal muscles) during the spastic event, inflicting greater mechanical stresses when supporting the body weight, with stress proportional to stiffness. However, Sopher et al investigated the effects of spastic events on muscle exposure to mechanical stresses by means of a biomechanical computer model and found increases in gluteal muscle stresses due to spasticity were mild. These findings correlated with a human study where no correlation was found between SCI-related-spasticity and occurrence of PUs. In fact, based on a cross-sectional study in 13 patients was found between SCI-related-spasticity and occurrence of findings correlated with a human study where no correlation in gluteal muscle stresses due to spasticity were mild. These findings correlated with a human study where no correlation was found between SCI-related-spasticity and occurrence of PUs.23 In fact, based on a cross-sectional study23 in 13 patients with an incomplete SCI from whom a spasticity score and MRI-derived muscle/fat masses were recorded, some authors suggested spasticity may be an important factor in defending skeletal muscle size and indirectly preventing intramuscular fat (IMF) accumulation early after incomplete SCI.

The other extreme of SCI-associated changes in muscle tone is flaccidity (ie, lack of muscle tone) that causes muscles to be limp and soft. This change is also theoretically hazardous because the muscles are less able to resist deformations, subsequently increasing the extent and exposure to intramuscular deformations whenever in a weight-bearing posture. As such, patients with flaccid paralysis may expose their muscles to greater deformation levels than persons with normal muscle tone or persons with spastic paralysis. Interestingly, early in recovery from an injury to the central nervous system, muscles may gain tone and move from being flaccid to spastic (which is detected in neurological examinations as a velocity-dependent increase in stretch reflex in the absence of volitional activities).28 Hence, post the acute phase of SCI, muscle tissues in the buttocks may change tonus, either in continuous processes (gradual changes from flaccidity to spasticity) or transiently (during spastic episodes). An appropriate cushioning solution needs to consider and ideally accommodate these changes in muscle tone, considering the more flaccid the muscle tissue, the greater the intramuscular deformation levels are under weight-bearing circumstances.

**Macrochanges Affecting Tissue Loading**

Macrochanges to tissues associated with the chronic phase of SCI and relevant to the risk for PUs and DTIs typically include increases in body weight and fat mass, bone shape adaptation, muscle atrophy, and increase in IMF, as well as disuse-induced skin adaptation and changes to the macro- and microvasculature. These changes and their impact on the structural and functional anatomy of the buttocks are depicted in Figure 1.

**Body weight.** The literature presents considerable evidence demonstrating a tendency for major weight gain during the first year after a SCI. A retrospective chart review study in a US Department of Veterans Affairs SCI Unit (N = 85) showed the body mass index of two out of three patients increases to overweight or obesity levels within this timeframe. It appears the majority of patients are unable to adequately decrease their caloric intake to match their lower level of activity and metabolic caloric needs. This can result in a weight gain of 1.3 to 1.8 kg per week for SCI patients undergoing rehabilitation (shown in 22 patients in their early rehabilitation phase),26 which occurs after the initial weight loss of 5.3 to 9.1 kg these patients experience shortly after the acute SCI due to hypercatabolism; nutrient deficiencies such as albumin, carotene, transferrin, ascorbate, thiamine, folate, and copper typically are documented at 2 weeks post
injury. This weight change infers the size of the buttocks and weight of the trunk are changing over time and typically start to increase after the first few weeks post acute injury, which has implications for sitting. Elsner and Gfen and So- pher et al investigated the biomechanical consequences of this gradual weight gain on deformations in the gluteal muscles during sitting. It could be hypothesized that a high body (or trunk) mass may lead to a greater risk for PUs, particularly DTIs, due to the increase in compressive forces from the ITs on overlying deep soft tissues in the buttocks. Conversely, it is possible the extra body fat associated with overweight or obesity may reduce the risk for PUs by providing enhanced subcutaneous cushioning that redistributes high IP. The Elsner and Sopher computer simulation studies clearly and consistently showed being overweight, particularly when coexisting with muscle atrophy (which is typical in SCI as well), contributes to a state of elevated deformations in the glutei, which may increase the likelihood of PUs in general and DTI in particular in the overweight SCI population. However, ongoing changes to the surface shape and size of the buttocks, as well as to internal anatomy (eg, thickness of fat tissue layers) in the SCI population in terms of the prescribed cushion also should be considered, but research addressing this specific problem still is lacking.

Skeletal changes. Bone adaptation due to disuse in SCI is clearly documented in the literature; primary adaptations include demineralization of epiphyses and thinning of the diaphyseal cortical walls below the injury level, with greater severe bone loss occurring in individuals with tetraplegia. Biomechanically, the bone loss is promoted by the absence of muscular loading via tendons, which is the primary stimulus for bone mass homeostasis in the able-bodied. In Giagregorio et al’s case study of two pairs of twins, where one of the twins suffered a SCI (at 7 years old, more than 20 years before the data collection), the authors used computed tomography to measure volumetric bone mineral density (BMD) and bone geometry and found lower BMD in the hip, distal femur, proximal tibia, and (to a lesser extent) in the spine of the SCI twins. Lower moments of inertia were evident at the mid-femur and calf of the SCI twins, indicating cortical bone loss. Studying twins makes a powerful comparison because it eliminates the potential effects of age and genetics. Small cohort prospective studies using dual X-ray absorptiometry, peripheral quantitative computerized tomography, and biomechanical test methods have demonstrated these catabolic changes in bone mass and shape occur within 2 years of the SCI; bone loss is more severe in tetraplegia than in paraplegia, large variability exists with respect to anatomical site and across individuals, and gender-dependent differences also may exist. The consequences of lower BMD and thinner cortical tissues are (after several years) more fragile bones that, much like in metabolic syndromes, tend to break as a result of non-traumatic loading, including when transferring to and from a wheelchair. Specifically with respect to sitting, Linder-Ganz et al used MRI scans of seated SCI subjects and found shape adaptations of the ITs, which tend to flatten in SCI patients, possibly due to the chronic exposure to the sitting loads coupled with the ongoing loss of cortical bone mass. The Linder-Ganz data revealed 1.8 times greater radii of curvature of the ITs in the SCI patient group with respect to the controls. Hence, ideally, a sitting solution should consider the ITs are gradually changing shape (typically flattening) in a SCI patient, which inevitably affects the load transfer from the weight-bearing ITs to the overlying soft tissues in the seated buttocks.

Disuse-induced muscle atrophy. Atrophy of skeletal muscle tissues is a well-known consequence of SCI. The atrophy occurs below the injury level and at the muscle fiber scale. It includes thinning of the fibers, reduction in the numbers of slow-twitch fibers, and an increase in fast-twitch fibers, which start as early as 4 to 6 weeks after the acute injury (inter-patient variations are considerable). The progressive process lasts at least several months and stabilizes between 1 and 6 years after the acute stage, with substantial variations across individuals. The lack of neuromuscular activity leads to microvascular changes as well, which generally cause a reduced oxidative capacity and a greater risk for deformation-induced ischemia.

At the macroscopic scale, the denervation causes rapid muscle wasting. In a group of patients with incomplete SCI (ie, where damage to the spinal cord was not absolute), muscular cross-sectional area in the thighs decreased by one third 6 weeks after the acute injury, and this was accompanied by more than doubling of the IMF contents. In Giagregorio et al’s twin study, the averages of the cross-sectional areas of the thigh and calf muscles in the SCI twins were as low as approximately 30% of the values for their non-SCI twins. As could be expected, the extent of loss of skeletal muscle tissues depends on whether the spinal cord lesion is incomplete or complete. For example, at a mean time of 13 months from an acute incomplete injury, a reduction of 24% to 31% (tibialis anterior and quadriceps femoris muscles, respectively) was reported in Shah et al’s MRI study in 17 people, with a weak correlation with respect to use of a wheelchair. In Castro et al’s study, where muscle biopsies were taken from 12 patients at 6, 11, and 24 weeks after a complete SCI, it was shown that complete spinal cord lesions can lead to muscle mass loss twice that found in the Shah et al’s study of incomplete injuries.

Based on supine pelvic computed tomography scans with contrast in 10 able-bodied and 10 participants with SCI, Wu and Bogie recently reported gluteal muscle atrophy is relatively greater at the level of the ITs, which highlights the need to especially protect that region by means of a cushion during wheelchair sitting. Lastly, it should be noted muscle atrophy also occurs with normal aging, partly due to neurological and endocrinial changes and partly due to cachexia and the reduced physical activity. Accordingly, as a SCI patient ages,
the disuse-induced muscle atrophy is superimposed with the aging-related atrophy.

**Tissue composition and mechanical behavior.**

**Muscle/fat.** IMF normally functions as dynamic adipose storage depots that are close to muscle fibers and hence accessible for muscular metabolism. These depots can expand when lipids are available, and they tend to increase in non-SCI overweight or obese individuals (the percentage of IMF typically assessed in humans using MRI).45 In able-bodied individuals, the normal IMF level is 1% to 2% of the total fat stored in the body.64 In SCI patients, IMF can increase progressively up to three to four times the healthy levels; after 8 to 10 years of denervation, the adipose tissues are nearly one third of the area in some muscle biopsies.38,45 Using computational modeling, Sopher et al21 demonstrated how internal muscle tissue loads under the ITs are elevated during sitting by the increase in IMF contents, implying the more severe the IMF, the higher the risk for a DTT. The authors explained the rise in skeletal muscle loads observed with the increasing IMF contents by the greater intramuscular shear stresses at interfaces between muscle and IMF tissues. Wu and Bogie41 reported the IMF depots are not homogeneously distributed across SCI muscles but tend to concentrate proximally (so glutal tissues near the ITs are prone to have excess IMF), and also that high IMF levels correlated with a history of severe and/or repeated PUs, which supports and complements the Sopher study. Wu and Bogie41 emphasized skeletal muscle quality and particularly IMF levels in the glutei need to be assessed in SCI individuals because they are an important measure of the risk for severe PUs, keeping in mind the IMF contents evolve and tend to accumulate with time after the acute injury.

From a biomechanical perspective, the build-up of IMF acts to decrease the effective stiffness of the glutei fat tissues (from animals) tested in vitro are less stiff than skeletal muscle.46,47 Computer simulations48 have shown this phenomenon gives rise to greater deformations in the glutei under a given body weight. In other words, the decrease in stiffness of the glutei due to the progressing increase in IMF contents causes the glutei to gradually bear greater sustained deformations, which then increases the risk for PUs and DTTs in particular, even if the patient does not become substantially heavier.

**Skin.** As previously discussed, disuse adaptation of the skin secondary to SCI is analogous to the degenerative processes in bone and muscle. In a study in Turkey employing high-frequency ultrasound, Yalcin et al49 compared skin thickness in the buttocks of 32 SCI patients to 34 controls and found the skin was substantially thinner over the ITs and sacrum in the SCI group (although the skin of individuals with SCI can thicken at other anatomical sites). Thinning of the skin at the load-bearing sites of a seated body appears to occur together with stiffening of the remaining skin tissues. In a study conducted in South Korea using a noninvasive suction testing device in 48 male participants with chronic SCI and 48 age-matched healthy controls, Park et al50 demonstrated the skin was significantly less distensible in the SCI group (P <0.05). This change in the skin’s biomechanical behavior occurs in conjunction with, and as a result of, deficient vascular reactions, decreased fibroblast activity, and primarily higher collagen catabolism. The loss of stability of intermolecular collagen cross-links, which is evident in increased urinary excretion of the biochemical residues of the disintegrated collagen in SCI, occurs months before these patients develop visible PUs (demonstrated in analyses of 24-hour urine samples from 60 men with SCI who were followed-up prospectively in the US).51 These are all changes associated with the disuse of skin tissues. The Park et al50 study also reported the duration of the SCI had a substantial impact on the changes in distensibility, elasticity, and viscoelasticity—ie, biomechanical skin properties. Taking the Yalcin and Park49 studies together, the thinner and stiffer SCI skin tissues, particularly at the weight-bearing sites of sitting, must cause the mechanical stresses (eg, when a patient moves or is being moved) in the deformed SCI skin to increase. At the same time, the strength of the SCI skin should decrease due to the loss of the collagen cross-links at the material level and the loss of thickness of the skin structure.49-51 This places SCI patients at high risk for superficial (tension- and shear-related) PUs (in addition to DTI) as a direct result of these skin changes. Interestingly, although not as widely reported as the degenerative musculoskeletal changes in SCI previously described, these skin changes are progressive and develop from the time of the acute injury,50 much like the disuse-induced musculoskeletal changes.

**Tissue perfusion.** Autonomic impairments may lead to increased prevalence of cardiovascular abnormalities in SCI patients, which manifest in the macro- and microcirculation (the impaired microvascular reactivity in SCI is largely due to the sympathetic dysfunction over the cardiovascular system). With respect to macro-circulation, which was studied in a retrospective chart review,52 hypotension was found to occur more in patients with a high level SCI. Additionally, a meta-analysis53 showed individuals with a cervical SCI exhibit a lower resting systolic blood pressure in the seated versus a supine position. Literature reviews summarizing data from muscle biopsies further report a gradual pathological decrease in numbers and sizes of capillaries that feed muscle fibers in skeletal muscle tissue of SCI patients.54,55 The fewer, narrower capillaries in SCI make the deformed skeletal muscles more prone to ischemia, particularly during episodes of low blood pressures. Although ischemic damage in weight-bearing muscles is now recognized to occur subsequent to direct deformation damage,9 it should be taken into account that muscle tissue in the SCI population becomes more susceptible to ischemic damage with the time that elapses post acute injury, which is a consequence of the disuse-induced muscle atrophy. The author’s conclusion, based on the above
findings, is that when adding the systemic cardiovascular impairments, a given level of sustained muscle deformations in the SCI patient causing obstructions of the intramuscular vasculature will trigger ischemia sooner than in non-SCI subjects. This is yet another reason why skeletal muscle deformation should be minimized, especially in seated SCI individuals.

Skin, like skeletal muscle, also pathologically adapts in structure and function to conditions of disuse. The reactive hyperemia (ie, the microvascular response to re-establishment of blood flow after a period of a reduced flow due to, for example, sustained tissue deformations) differs between SCI patients and healthy individuals. In a laser Doppler perfusion study, eight SCI patients experienced greater perfusion to the buttocks skin tissues following a period of pressure-induced obstruction of the skin vasculature than eight control subjects. This probably indicates the skin of SCI patients experiences more severe ischemic conditions when loaded compared to a healthy skin response to the same loading conditions. Hence, the above-mentioned studies suggest muscle, as well as the skin of SCI patients, undergoes micro- and macro-structural changes over time. These changes, together with systemic cardiovascular changes, eventually cause these tissues to become more sensitive to development of ischemia when sitting for prolonged periods.

Interpreting Changes in Tissue-loading Levels With Respect to Safe Sitting Times

Shabshin et al evaluated the impact of foam and gel wheelchair cushion materials on deformations in the glutei during sitting in a group of healthy persons using a seated MRI setting that employs a “double-donut” MRI configuration. This configuration allows persons to sit in the magnet between the two donut-shaped structures that facilitate the imaging. The authors found the foam and gel cushions were able to decrease muscle deformations by 10%, with rather small differences in that parameter across the tested cushion materials (two foam and two viscoelastic types). At first glance, this may lead one to conclude there is little clinical significance between these two materials. However, putting these data in the context of the cell and tissue injury thresholds obtained for skeletal muscle by Linder-Ganz et al and later by Gefen et al clarifies that decreasing internal muscle deformations, even to a mild extent, may contribute to a considerable increase in “safe” sitting time. The procedures used for experimentally obtaining these cell/tissue injury thresholds are depicted in Figure 2. Specifically, the aforementioned papers determined “safe” and cell-death-inducing deformation levels and time exposures for skeletal muscles by applying compressive deformations to muscles of anesthetized rodents and tissue-engineered muscle constructs. The existence of deformation-induced damage was determined using histological staining at the tissue scale or through fluorescent staining of the tissue-engineered constructs over time, which allowed researchers to identify and quantify damage at the cell level (see Figure 2). The latter study indicated cells in the constructs that experienced compressive deformations exceeding approximately 80% died almost immediately, but cells subjected to lower levels of deformation − eg, cells deformed to the 50% level − survived longer (ie, they died after approximately 3 hours). In the animal as well as in the tissue-engineered model systems, cell survival time emerged as considerably sensitive to the magnitude of the sustained deformation level; even a mild decrease in the level of deformation added a substantial survival time for the cells (see Figure 2;
Because hierarchical (multiscale) computer modeling has shown high deformations at the tissue (macroscopic) scale (as occurring in the soft tissues of the buttocks during sitting due to weight-bearing) are associated with high cell deformation levels, reduction in macroscopic deformation levels also will alleviate cell deformations. This is achievable using proper cushioning solutions.

To illustrate the mechanism for the function of wheelchair cushions: A completely rigid support, allowing no immersion and hence only minimal envelopment of the buttocks by the support, will cause body weight loads to transfer from the spine to the ITs and then outward to the support surface through a smaller soft tissue region with respect to a situation where the support is able to deform. The loads, concentrated in the more limited tissue volume, subsequently will cause high internal tissue deformations, as demonstrated in the computational (finite element [FE]) simulation in Figure 3, particularly in gluteal skeletal muscle tissues overlying the ITs (see Figure 3b). The greater tissue deformation levels (see Figure 3b) dictate a shorter time for normal cell viability and an earlier onset of cell and tissue damage based on the aforementioned animal and tissue-engineered model data (see Figure 2; bottom frame). This is the reason why using a completely rigid or even a relatively stiff sitting support is potentially harmful and will lead to a pressure-related injury within a relatively short period of time, particularly for SCI patients who are insensitive and cannot respond to discomfort or pain (see Figure 4; case a). A cushion placed under the buttocks that allows the buttocks to immerse and distort the unloaded shape of the cushion distributes tissue loading through deformation of the cushion. Through envelopment of the buttocks structure by the cushion, the area for transfer of body loads from the buttocks to the cushion, as well as for transfer of internal tissue loads, increases. This correspondingly decreases the levels of internal tissue deformations, because body weight forces now can spread and flow over greater tissue volumes. The outcome, in terms of tissue tolerance to the loading, would be a longer safe sitting time (see Figure 4; case b). In fact, the greater the immersion and envelopment, the longer the safe sitting time, where importantly, relatively mild reductions in internal sustained tissue deformations can increase the safe sitting time considerably due to the nonlinearity of the threshold curve, as quantitatively described by Shabshin et al (see Figure 4; case c versus b). However, this behavior holds only as long as no bottoming-out of the cushion occurs, because bottoming-out essentially
places the buttocks very near or perhaps on the surface on which the cushion is placed (e.g., the exposed wheelchair seat) which is typically stiff, causing a loading state close to the one illustrated in Figure 3b and Figure 4: case a. To summarize this point, a cushion that provides better immersion and greater envelopment without any bottoming-out effects theoretically should provide safer sitting times for patients by maintaining the shape of the body and minimizing tissue deformations. Given the pathoanatomical and pathophysiological changes in SCI (which, from a biomechanical perspective, make SCI patients more prone to PUs and DTIs by generally acting to increase sustained muscle deformations), these considerations are critical. Hence, the above considerations should be taken into account with regard to the SCI population by guiding the design, selection, and prescription of cushions for these patients.

Figure 5. A scheme depicting the problem of using a non-adapting, contour-shaped foam cushion prepared at an early stage after the spinal cord injury (SCI) for a patient whose tissues continuously respond to the disuse conditions. a) A cushion prepared early after the SCI may fit for a short time period, providing potentially adequate envelopment; but b) as the remarkable pathoanatomical and pathophysiological changes that occur at the chronic phase of SCI take place and progress over time, the cushion’s contoured design becomes irrelevant to the altered anatomy, both in terms of the adapted external buttock surfaces (see arrows in b) and the internal pathoanatomy.

Implications for the Future Design and Selection of Wheelchair Cushions

Beyond striving to utilize a cushion that 1) achieves good envelopment of the buttocks without bottoming-out at the specific time point of the fitting of the cushion and 2) demonstrates the ability of the cushion to comply to the body and maintain the shape, one needs to appreciate the SCI patient’s unstable characteristics and consider the ongoing pathoanatomical and pathophysiological changes. Any cushion solution for this population ideally should adapt to the progressive changes in the patient’s body over time. A blog regarding important reimbursement changes to consider when choosing a wheelchair cushion (http://blog.therohogroup.com/index.php/tag/medicare/) notes regular replacement of cushions over short time intervals would be a valid strategy from a medical preventive point of view, but this is probably not a cost-effective approach. According to the Medicare website (www.cms.gov), US Medicare and medical insurance companies are acting to increase, rather than decrease, the reimbursement time intervals between replacements of a cushion, which used to be 3 years until recently but is now changing to 5 years. If the cushion is not able to respond to the changes to the individual’s body throughout that timeframe, the PU risk inevitably increases. Although the intention here is not to advocate for or oppose specific, commercially available cushion technologies, the example of using contoured foam cushions for SCI patients is presented to illustrate current problems that need to be addressed by the cushion industry based on scientific knowledge and understanding of the etiology and risk factors related to PUs and DTIs.

Manufacturing a contoured-foam cushion that fits the individual’s buttocks shape appears to be initially appealing with regard to the importance of envelopment, because that will create the greatest possible buttocks-cushion contact area. The literature to date reports IP were lower on custom-contoured shapes as opposed to flat foams, which is not surprising considering the contoured cushions were fitted to the (able-bodied) subjects just before the measurements. Tasker et al’s study included 30 able-bodied participants who sat with restricted movement for 30 minutes in three sessions in order to evaluate two contoured cushion shapes against a flat support. In addition to IP recordings, the researchers evaluated discomfort levels using a visual analog scale; participants were found to be more comfortable on the contoured cushions. Nevertheless, considering that within several months to a few years, a SCI patient is expected to gain body weight and fat mass; lose gluteal muscle mass; and experience flattening of the ITs due to bone adaptation, thinning of the skin around the IT regions, and accumulation of IMF (see Figure 1), the individual’s anatomy is changing progressively and remarkably. As these changes take place and progress over time, the cushion’s contoured design becomes irrelevant to the altered anatomy, both in terms of the adapted external buttock surfaces and the internal pathoanatomy (see Figure 5).
In addition to the changes that occur in the patient’s body, the cushions may present wear-related changes, as recently noted in a study by Sprigle in which 202 cushions used for an average of 2.7 years were inspected. This research revealed more than 60% of the foam cushions showed signs of permanent deformation, and in nearly half, the foam exhibited granulation or brittleness. Even if not causing complete bottoming-out, these wear-and-tear effects can increase tissue loads considerably by giving the cushion a flatter and thinner shape and particularly by compacting the foam at the regions supporting the bony prominences.

Hence, the patient and cushion both are changing over time. In the SCI population, these changes will typically act to increase the risk for PUs and DTIs by increasing sustained subcutaneous tissue deformations, particularly in skeletal muscle tissues. One of the fundamental problems of the available cushion testing methods is their tendency to focus on new cushions (which were not yet influenced by daily use and wear) and apply surrogate body shapes or some simple artificial indentors that do not consider the changes the patient’s body may be undergoing. Analysis of IP data published more than 20 years ago notes extreme caution must be taken when using nondisabled participants as substitutes for people with SCI due to the inherent differences between the groups, illustrating how far the artificial phantoms (or indentors) are from the complexity of the biomechanical interactions of a human patient with a cushion and how they vastly oversimplify research. Hence, cushion developers, evaluators, and prescribers need new technologies for investigating, testing, and fitting cushions, particularly with regard to the high-risk SCI population and desirably based on the individual patient conditions at the time of the fitting and the projected changes that will occur to the individual over time.

The most promising and practical bioengineering approach to overcome the aforementioned problems in evaluating and fitting cushions is to use computer simulations based on FE modeling, which is very likely to be adopted as a design standard by the wheelchair cushion industry, much as it is currently used by car manufacturers for designing car seats. These computer simulations allow researchers to capture pathoanatomical and pathophysiological changes that directly reflect the biomechanics of sitting in SCI patients and the internal tissue deformations occurring in their buttocks. Moreover, the simulations allow for the influence of microclimate factors and incontinence on tissue loads and tolerances. Recent major conferences in the field, such as the US National Pressure Ulcer Advisory Panel and the European Pressure Ulcer Advisory Panel conference in 2013, clearly indicated this to be the direction of the support surface industry. However, in the more distant future, individualization of the design and fitting of cushions based on internal tissue loading conditions in a specific patient also may occur. This may take the form of subject-specific modeling that will consider the individual anatomy of a patient at a given time point, as well as changes that occur with time and captured by means of imaging in follow-ups or through a simulated prognosis for the individual.

To summarize: the most ideal situation for the health of weight-bearing tissues is to avoid localized intensified external and internal deformations relevant to activity. An example involves soft tissues during diving, where tissues are loaded by hydrostatic pressures. Such diving pressures can sometimes be extreme, but because they are uniformly distributed over the body surfaces and without any external shear, no load-related discomfort or pain occurs, and certainly no PUs. Of course, it is extremely difficult to reproduce this body-suspension-in-water effect by means of a cushion. However, if implementing this concept, it is clear a good cushion needs to adapt to posture and movement and to longer-term pathoanatomical changes to keep tissue contours of the buttocks as close as possible to their undeformed shapes, as would occur if the body were floating in a liquid medium and maintaining tissue shapes, regardless of its position. For fluid-containing cushions, this implies a mechanism needs to exist to allow the cushion to respond to movements and longer-term changes through transfer and regulation of the air or liquid pressures in-between the components of the cushion in response to changes in the weight-bearing configuration. This should eliminate build-up of localized (internal) cushion pressures acting as nearly rigid support sites and the development of corresponding concentrated tissue deformations. This underscores a need for the cushion to be responsive rather than passive and to allow these cushion responses to be rapid enough to accommodate daily microchanges as well as the long-term macrochanges without compromising upper body mobility.

Conclusion

Protecting individuals with SCI who use wheelchairs from PUs and DTIs is complex and challenging. Microchanges (eg, changes in posture and position, use of the upper extremities, performance of pushups, and changes in muscle tone) and macrochanges (mostly attributed to the disuse of tissues, which affects the body habitus and internal anatomy, the microarchitecture, composition and stiffness properties of tissues, and their pathophysiology) must be considered. Most of the literature available today, which was rigorously reviewed here, consists of retrospective and prospective cohort studies, case studies, and computer simulations, as well as a few randomized clinical trials. Together, these reports suggest important cushion design, selection, and prescription implications. Specifically, the cushion should promote a posture that is as balanced as possible but that allows adequate functional reach capacities that do not compromise the cushion’s capacity to minimize internal tissue deformations to the greatest possible extent at any transitory or sedentary posture. The cushion needs to be tailored to the individual based on a rigorous risk assessment conducted by

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expert clinician. The patient should be educated with respect to proper use of the cushion and be made aware of the life expectancy of the cushion, especially if the cushion is foam-based. Envelopment appears to be a key feature in evaluating cushion performances in laboratory settings, with greater immersion and envelopment most likely providing more protection against DTIs, as long as there is no bottoming-out or near bottoming-out; this contributes directly to maintaining the shape of the body and minimizing tissue and cellular deformations. The patient’s external and internal anatomy and tissue structure and function will change considerably in the months and years following the SCI, typically along a trend that makes tissues progressively more fragile and the risk for PUs increasingly greater. A review of medical records of 7,489 Iranian patients revealed the time that passed since the SCI had a statistically significant association with the prevalence of developing PUs (though the authors of that paper were careful to also stress SCI patients are a heterogeneous group and the risk factors associated with PUs may vary in specific subgroups). Accordingly, a cushion that cannot adapt to the individual’s conditions throughout the day and over time will soon be less able to protect the patient from PUs and DTIs, even if that cushion was initially suitable for the individual.

The most promising and practical bioengineering approach in providing and fitting cushions to different patient conditions is to use computer simulations (FE modeling), which already is being adopted by the wheelchair cushion industry. Computer simulations and individualized computer-based biomechanical risk assessments in particular are expected to play a key role in development and evaluation of current and future cushion products. FE computer modeling adds critically important information on biomechanical interactions internally in the body, which can be individualized to represent person-specific (patho)anatomical conditions and the corresponding internal tissue deformations. Such understanding, which clearly cannot be achieved by IP measurements per se, may facilitate the development of better cushion products and guidelines. Although understanding of the pathophysiology of PU risk in individuals with SCI is increasing, studies to evaluate the efficacy, effectiveness, and cost-effectiveness of cushions are needed, particularly because it appears reimbursement policies internationally will be increasingly driven by evidence of efficacy from both laboratory and clinical studies.

References


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