PART ONE OF THIS ARTICLE highlighted some fundamental problems in using the Reswick and Rogers (1976) pressure-time curve to understand the conditions under which pressure ulcers occur, or for developing guidelines and technologies to prevent pressure ulcers. It was indicated that extrapolations of the Reswick and Rogers (1976) data to short and long periods of exposure to body loads are incorrect, and that interface pressures are not a standardised measure of internal tissue loads (Gefen and Levine 2007).

Formulation of a standardised tissue injury threshold

Recently, intensive research work has been carried out (Peeters et al 2005, Linder-Ganz et al 2006, Stekelenburg et al 2007, Gefen et al 2008), including some collaborative studies, to propose a more accurate and standardised tissue injury threshold as related to pressure ulcers. The studies’ focus was on muscle tissue, which is more susceptible to damage than fat and skin (Daniel et al 1981, Salcido et al 1994), and is also the site for the onset of deep tissue injury (Ohura et al 2007). The purpose of the studies (Peeters et al 2005, Linder-Ganz et al 2006, Stekelenburg et al 2007, Gefen et al 2008) was to determine the load tolerance of muscle tissue as an inherent property of the muscle material, as opposed to the Reswick and Rogers (1976) approach of using external (interface) pressures, which may have different internal effects for different anatomies (Gefen 2009).

Determining an injury threshold that is specific for muscle tissue requires contemporary bioengineering research methods that can determine internal mechanical conditions (for example, distribution of deformations) in loaded muscle tissue in vivo, cell and tissue culture models, animal models and living humans. Such methods include small animal magnetic resonance imaging (MRI), open MRI and computational modelling, the use of which in pressure ulcer research is described elsewhere (Gefen 2008). The availability of these methods has opened up new opportunities in pressure ulcer research, and has boosted the efforts to characterise the injury tolerance of muscle tissue to mechanical loads. The key outcomes from these recent studies are reviewed later on in this article.

Stekelenburg et al (2007) attempted to distinguish between the contributions of tissue deformation and ischaemia to muscle tissue damage. They studied the separate and combined effects of the deformation and ischaemia factors, by loading the hind limb of the anaesthetised rats for two hours, during which the loaded muscles were examined internally by means of small animal MRI. Tissue deformations were induced to the tibialis anterior muscle of the animals using an indenter, a rigid 3mm diameter rod, curved at the edges, that is pressed against the skin; ischaemia...
was produced with an inflatable tourniquet. The muscles were also analysed histologically post-euthanasia. One of the most important results from this study was that two hours of sustained deformation inflicted substantial and irreversible damage to muscle tissue, which indicated that high enough deformations can induce serious tissue damage in a relatively short period of time, or even immediately (Stekelenburg et al 2008), as explained in part one of this article (Gefen 2009). This finding in the animal studies of Stekelenburg et al (2007) reconfirmed – at the scale of observing an entire living muscle – previous results obtained from observing single cells (Peeters et al 2005).

Specifically, Peeters et al (2005) used muscle (myoblast) cell cultures to measure the maximal deformations that can be sustained by individual cells attached to the surface of a culture dish. A device was developed that was able to compress the cells at controlled deformations, while measuring the forces applied to deform the cells. The entire process of deforming the muscle cells was visualised by means of a confocal laser scanning microscope. The confocal images showed that at large deformations, the cell membranes begin to bulge, and if deformation is increased further, the membrane tears completely. Deformations exceeding about 80% consistently ruptured the cell membranes, thereby indicating that cells subjected to such elevated deformations would be damaged instantaneously and irreversibly.

The Stekelenburg et al (2007) and Peeters et al (2005) studies reveal that even for short exposures to mechanical loads, living cells and tissues do have a finite tolerance, which can be viewed as their failure strength. This statement might be considered almost trivial in the context of traumatic injury to soft or hard tissues, from bruises to bone fractures, because all physical structures, including biological tissues, have an inherent failure strength which is constituted by their internal geometrical arrangement and mechanical properties. However, the failure strength characteristic of soft tissues is not reflected in the Reswick and Rogers (1976) pressure-time curve.

An alternative to the Reswick and Rogers (1976) pressure-time curve was first suggested by Linder-Ganz et al (2006). With a specific focus on deep tissue injury, Linder-Ganz et al (2006) analysed muscle histopathology from models of pressure ulcers in albino rats. Muscle tissue in the rats’ hind limbs was subjected to pressures of 86mmHg to 578mmHg for 15 minutes to six hours, by means of a rigid indenter that locally compressed the tissue. The histopathology from each animal in the experiment was used to determine whether muscle tissue was able to tolerate the applied pressure and time exposure, and remain viable. Data from these experiments were superimposed on data from a meta-analysis of all the previous histopathology reported for albino rat muscles subjected to pressure (refer to Linder-Ganz et al (2006) for relevant references to the literature). The pooled data enabled a new mathematical characterisation of the pressure-time threshold for muscle tissue damage. This took the form of a sigmoid pressure-time function, which corrects the previously discussed inaccuracies in the Reswick and Rogers (1976) hyperbola function for short and long time periods.

A sigmoid function is a mathematical function that produces an s-shaped curve. The sigmoid function of Linder-Ganz et al’s (2006) study is shown in Figure 1 (solid line), together with the Reswick and Rodgers (1976) curve (dashed line) for comparison. The sigmoid data of Linder-Ganz et al (2006) refer to pressures applied directly to muscle tissue, whereas the Reswick and Rogers (1976) data refers to interface pressures. However, the sigmoid and the Reswick and Rogers (1976) curves show similarity at the time frame between one and a half and five and a half hours. However, the curves become substantially dissimilar at the shorter and longer times because of the inaccuracies in the Reswick and Rogers (1976) pressure-time curve at these time domains (see Figure 1, Gefen 2009). The most important advantage of the sigmoid curve is that it defines a finite failure strength for muscle tissue at short times that is consistent with experimental data.
The steepest decrease in endurance of the cells to deformations occurred between one and three hours post-loading (Gefen et al 2008), which was consistent with the findings of Reswick and Rogers (1976).

Overall, the sigmoid curve in Figure 1 provides a complete description of how much internal pressure or how much deformation is allowed in muscle tissue, and for how much time, to avoid tissue damage. This is a necessary piece of information for understanding the aetiology of pressure ulcers and deep tissue injury. Moreover, when methods are available to measure internal mechanical loads in deep soft tissues in vivo (for example, deformations in muscle tissue next to a bony prominence), this tissue injury threshold could be used to assess the risk of an individual developing a deep tissue injury.

### Clinical implications

The sigmoid tissue injury threshold in Figure 1 can be interpreted in practice to indicate risk factors and perhaps interventions that are needed to protect patients from developing serious pressure ulcers and deep tissue injury. One such risk factor, as implied by analysis of the sigmoid threshold, is obesity. Paraplegia and quadriplegia are often accompanied by a significant gain in body weight (Gupta et al 2006, Weaver et al 2007). Accordingly, other than the impaired motor-sensory capacities that these patients have, the weight of the trunk increases the load on their load-bearing soft tissues, particularly on muscle tissue in contact with bony prominences.

For example, let us assume that one ischium transfers 10% of the body weight during sitting, and that the effective contact radius of the ischium with overlying gluteal muscle tissue is approximately 4cm. A seated male with a recent spinal cord injury and a non-obese body weight of 55kg would then apply a pressure of about 80mmHg on gluteal muscle tissue under the ischial prominence (pressure calculated as body weight per contact area (Figure 2, bottom left)). Let us assume that in the years following spinal cord injury, that person becomes obese so that his body weight increases to 120kg. The individual’s increased trunk load would now apply pressures of 180mmHg on the gluteal muscle tissue next to the ischial prominence (Figure 2, top left). Using the sigmoid tissue injury threshold, the obese patient is expected to develop a deep tissue injury in the gluteal muscle tissue within one and a half hours if internal tissue loads are not relieved during that time, whereas with the patient’s previous 55kg body weight, the muscle tissue was likely to sustain loads for three hours.
The risk of developing pressure ulcers and deep tissue injury is therefore expected to be much higher for obese patients, as observed in clinical reports (Gallagher 1997, Kramer 2004, Hahler 2006, Baugh et al 2007).

It should be emphasised that the risk for deep tissue injury intensifies only if obesity is combined with complete immobilisation so that no internal load relief occurs in the tissues, since ambulant patients who are obese do not seem to be at a higher risk of pressure ulcers or deep tissue injury (Compher et al 2007). This means that maintaining a normal body weight is important for patients with impaired motor-sensory capacities to avoid pressure ulcers and deep tissue injury. Efforts should therefore be made to engage such patients in regular physical exercise programmes, whenever this is possible.

Another example of why physical exercise, if feasible, can lower the risk of pressure ulcers and deep tissue injury relates to the muscle mass of patients. Let us assume again a hypothetical male patient, in whom thickness of the gluteal muscle tissue is 23mm, which is normal according to MRI studies of the buttocks (Linder-Ganz et al 2008). Given that the ischial tuberosities sag into the overlying gluteal muscle tissue about 12mm during sitting (Linder-Ganz et al 2008), the mean compressive deformation of gluteal tissue under the ischial tuberosities would be approximately 52% (12mm/23mm). Using the right vertical axis in Figure 2, this will allow the patient a three-hour ‘safe time’ during which injury can be avoided, which is similar to the case of the non-obese patient from the previous example. Now let us assume that in the years following spinal cord injury, the patient loses gluteal muscle mass, so that the thickness of his atrophied gluteus muscles reduces to 17mm. While sitting, the patient now deforms his muscle tissue to about 71% (12mm/17mm), which gives him a much shorter ‘safe time’ of only one and a half hours before injury is likely to occur (Figure 2).

It is interesting to note that in recent MRI studies of the buttock anatomy in patients with spinal cord injury, the only patient who did not develop muscle atrophy was an athlete (Linder-Ganz et al 2008). Accordingly, the benefit of maintaining muscle mass is another reason why physical activity is desirable in avoiding pressure ulcers and deep tissue injury in patients with impaired motor-sensory capacities. However, although because of individual limitations and lifestyle, it cannot be considered an overall solution.

To summarise, patients who use a wheelchair as a result of neurological injury or disease tend to gain body weight and lose muscle mass, which both contribute to substantially increased internal muscle loads (increased pressures from the bones, or higher tissue deformations). The present sigmoid tissue injury threshold (Figure 2) suggests that obesity and muscle atrophy each shorten the time for tissue damage, and therefore these factors increase the risk of the individual to pressure ulcers and deep tissue injury.
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Conclusion

This article has outlined some fundamental flaws in the commonly used hyperbola pressure-time curve of Reswick and Rogers (1976) for predicting the risk of developing pressure ulcers. It was shown that the Reswick and Rogers (1976) hyperbola is inaccurate at the extremes of the timescale. Moreover, the Reswick and Rogers (1976) pressure-time curve is not applicable for predicting deep tissue injury, or any pressure ulcer that chronologically appears first in deep tissues, because it was developed based on interface pressure measurements, which cannot be associated with a unique loading condition in deep tissues. An alternative injury threshold was presented, in the form of a sigmoid mathematical function, which provides more accurate limits on the pressure levels and exposure times.

Clinical implications of the sigmoid curve were discussed, as related to patients who use a wheelchair as a result of neurological injury or disease. The literature indicates that such patients tend to gain body weight and lose muscle mass owing to denervation, physical limitations and sedentary lifestyle. Obesity and muscle atrophy each contribute to a substantially increased internal muscle load. As a consequence of either obesity or muscle atrophy, the time for tissue damage shortens, which increases the risk of the individual to experience pressure ulcers and deep tissue injury. Therefore, the healthcare team should actively encourage patients confined to a wheelchair to be physically active, and to practise sports, if possible, so that patients will maintain a normal body weight, and retain muscle mass. For overweight wheelchair-bound patients, involvement of a dietician or a nutrition consultant is important to help achieve and maintain a normal body weight.

References


