Reswick and Rogers pressure-time curve for pressure ulcer risk. Part 1


Summary
Pressure ulcers are one of the most potentially devastating complications in individuals confined to a bed or a wheelchair for an extended period. Severe pressure ulcers may form in deep tissues overlying bony prominences, and only at a later stage become visible, or may even induce a full-thickness breakdown of the soft tissues at the affected site. A new type of pressure ulcer known as 'deep tissue injury' has therefore been defined internationally. To understand the aetiology of deep tissue injury, health professionals should be able to predict whether or not a certain state of internal mechanical loads in deep tissues, such as tissue deformations and forces per unit area of tissue, would lead to localised irreversible cell damage. Part one of this article explains the concepts of injury thresholds as related to deep tissue injury. Some serious flaws in the classical, commonly used Reswick and Rogers pressure-time curve are analysed, and an alternative contemporary tissue injury threshold, the sigmoid threshold, is suggested. Part two of this article describes recent and ongoing work aimed at defining injury thresholds that are specific for deep tissue injury, standardised and therefore suitable for use with different patients. Clinical implications of current injury thresholds are also discussed, in relation to obese patients and patients with muscle atrophy.

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Pressure ulcers and deep tissue injury
Pressure ulcer research is undergoing major developments in terms of redefining the categories of wounds, based on current scientific and clinical studies (Berlowitz and Brienza 2007). The literature indicates that, in a patient with motor and/or sensory impairments, there are basically two separate paths that can lead to lesions or wounds that were previously given the general designations 'pressure sores', 'bedsores' or 'decubitus ulcers' (Berlowitz and Brienza 2007).

One path is friction of the skin against clothing, bed sheets or cushions, which leads to shear damage of the skin surface, and may be further exacerbated in the presence of moisture, for example from sweat or incontinence. Tissue damage associated with frictional forces on the skin results in a superficial lesion that is visible, and can therefore be treated promptly, with little chance of irreversible damage.

The second path relates to sustained deformation of deep soft tissues under bony prominences, as a result of prolonged pressure of the bone on these soft tissues. Damage that initiates at deep tissue structures because of unrelieved bone pressures cannot be detected as quickly, since the initial lesion is not visible on inspection. The potential for a deep wound to deteriorate without treatment is much greater than for superficial lesions, and therefore the associated consequences are more likely to involve reconstructive surgery, aggressive treatment with medication, longer hospital stays and increased corresponding costs (Berlowitz and Brienza 2007).

Berlowitz and Brienza (2007) suggested that only an injury caused by sustained deformations...
of subcutaneous soft tissues in the vicinity of bony prominences should be called a pressure ulcer, whereas frictional damage that starts at the skin should be treated as a different wound category. This distinction between pressure ulcers that commence as a deep tissue injury, as opposed to skin lesions resulting from frictional damage, has significant implications for prevention and treatment guidelines (Berlowitz and Brienza 2007). Specifically, it imposes additions to existing definitions of pressure ulcer severity, to describe a wound that includes deep tissue necrosis under intact skin (Black 2009). It also indicates a need for development of new risk assessment tools and screening methods (Gefen 2008, Linder-Ganz and Gefen 2009), diagnostic imaging protocols (Hencey et al 1996, Quintavalle et al 2006, Ohura et al 2007), technological protective measures (Portnoy et al 2007, Agam and Gefen 2008) and minimally invasive surgical interventions (Tetsuya and Akane 2006) to deal with so-called ‘true’ pressure ulcers that start as deep tissue injuries (Berlowitz and Brienza 2007).

Taking into account these ongoing developments and the concurrently evolving terminology used in relation to tissue viability and wound care, the terms pressure ulcer and deep tissue injury are used in this article interchangeably; both refer to a lesion involving irreversible deep tissue damage, where damage chronologically started at the deep tissues. Additional research is needed to develop technologies for early diagnosis and for prevention of pressure ulcers, based on the current understanding that these wounds originate subcutaneously (Berlowitz and Brienza 2007). Considering that pressure ulcers develop internally (Berlowitz and Brienza 2007), clinicians need to be able to measure mechanical loads inside deep tissues, not just pressures or shear forces at the body-support interface (Gefen and Levine 2007). Clinicians should also be able to predict whether or not a certain state of internal mechanical loads in the tissue could lead to irreversible tissue damage deep tissue injury. For this purpose, researchers need to determine tissue injury tolerances that will allow evaluation of the risk for an individual patient to develop pressure ulcers, based on a measurement of the internal mechanical state in the patient’s deep tissues (Linder-Ganz et al 2006, Gefen et al 2008).

This article focuses on the second task; it describes the current efforts in the bioengineering community to determine, based on experimental data, threshold levels of mechanical loads that lead to irreversible damage in deep soft tissues overlying bony prominences. The availability of technological means to measure internal tissue loads, and to predict whether data of internal tissue loads identify a patient as being at risk, will eventually influence medical decision making, for example regarding risk assessment and prevention policies. Therefore these issues are also discussed. Specifically, the up-to-date injury threshold curvatures for deep tissues (Linder-Ganz et al 2006, Stekelenburg et al 2007, Gefen et al 2008) are interpreted in this article, to define some specific subject characteristics that theoretically indicate greater risk for the development of a deep tissue injury.

### Tissue injury tolerance and pressure ulcer research

**The evolution of the Reswick and Rogers pressure-time curve**

Historically, the quest for quantitative injury thresholds in relation to pressure ulcer research attracted the attention of only a few investigators. The literature therefore reflects bursts of scientific progress followed by long periods of stagnation, which allows a rather straightforward review of the development of knowledge.

Methodological research of pressure ulcers dates back to the second world war, where in Germany, Groth (1942) produced pressure ulcers in the gluteus muscles of rabbits, and analysed histological samples from the inflicted ulcers. He made the important qualitative observation that the extent of damage to muscle fibres and capillaries increases with the magnitudes and durations of the mechanical loads applied to the tissue. At about the same time, medical procedures started to emerge in an attempt to prevent pressure ulcers in wounded soldiers who were brought back from the battlefields. Defloor et al (2006) highlighted that it is commonly believed that, during the second world war, wards developed policies of turning patients every two hours, seeking a balance between minimisation of pressure ulcer risk and the availability of personnel to reposition hospitalised soldiers. This was probably the precursor to modern turning regimens.

Soon after the war, rigorous scientific work led by Husain (1953) sought to characterise the timescales along which tissues are able to bear mechanical loads while remaining viable, and to determine how much load is allowed for how much time. Husain (1953) applied pressures of 100mmHg for two hours to legs of rats and guinea pigs. Twenty-four hours later he observed myofibrillar degeneration, infiltration of macrophagic immune cells, capillary...
haemorrhages and localised oedema in muscle tissue. He believed this indicated that two hours’ exposure to 100mmHg pressure is the injury threshold for skeletal muscle. His work was followed by studies undertaken by Kosiak (1959, 1961) who used normal and paraplegic rats as well as dogs as models for pressure ulcers. Kosiak (1959, 1961) was able to show an inverse trend between the magnitude and duration of pressures causing the histological damage patterns described earlier by Husain (1953). Kosiak (1959) specifically showed that a pressure as high as 190mmHg, applied either continuously or intermittently for up to one hour in rats, did not induce microscopic changes, whereas lower pressures of 70mmHg applied for two hours inflicted irreversible damage, as mentioned above. This represented major progress over the work of Groth (1942) – in the sense that pressures and exposure times were well controlled and methodologically documented – and over the work of Husain (1953). Kosiak (1959, 1961) emphasised that the injury threshold for muscle is a continuous load-time relationship, rather than a single discrete level. It is therefore no wonder that articles describing further animal studies of pressure ulcers cited Kosiak’s (1959, 1961) work as the cornerstone of modern pressure ulcer research (Daniel et al 1981, Stekelenburg et al 2005).

The concepts proposed by Kosiak (1959, 1961) were later applied to humans. In the United States, Reswick and Rogers (1976) formulated their tissue tolerance guidelines of allowable pressure levels on tissues overlying bony prominences, against the time of the sustained pressure (Figure 1). The data used in their studies were collected from more than 980 medical cases reviewed in the Rancho Los Amigos hospital in Downey, California, where staff documented a wide span of wound severities, from early signs of potential skin breakdown to serious pressure ulcers.

Reswick and Rogers (1976) chose to present their pressure-time data as a function known in mathematics as ‘rectangular hyperbola’, which indicates that the product $[\text{Pressure (mmHg)}] \times [\text{Time (hr)}]$ must always be kept under an acceptable level, the $[\text{Critical Pressure Time Product}]$, to avoid a pressure ulcer. The $[\text{Critical Pressure Time Product}]$ is a constant quantity, approximately equal to 300mmHg x hr (Reswick and Rogers 1976). To avoid pressure ulcers, this means that if a patient does not change his or her posture for one hour, interface pressures under his or her bony prominences should be kept lower than (300mmHg x hr)/(1hr), which equals 300mmHg. Likewise, if a patient does not move for three hours, interface pressures under the bony prominences during that time should be lower than (300mmHg x hr)/(3hr), which equals 100mmHg (Figure 1).

Although these calculations make sense for time periods of a few hours, they appear flawed when applied to very short or very long periods. For example, a patient undergoing complicated surgery, for example to treat head and neck cancer, is expected to be immobilised on the operating table for 12 hours or more (Wei et al 1999). The Reswick and Rogers (1976) pressure-time curve indicates that the individual could then be injured at pressure levels lower than 25mmHg (300mmHg x hr/12hr). However, operating table mattresses currently available will, in most cases, produce peak interface pressures that are substantially higher than this (Defloor and De Schuyjmer 2000). If these calculations are accurate, avoiding pressure ulcers during complicated surgery is therefore impossible. However, only a minority of patients undergoing long periods of surgery develop pressure ulcers and the majority do not (Kemp et al 1990), so the Reswick and Rogers (1976) inverse relationship of $[\text{Pressure}] \times [\text{Time}] = [\text{Critical Pressure Time Product}]$ cannot be accurate for extremely long periods (Figure 1, legend 1).

A similar difficulty arises when interpreting the Reswick and Rogers (1976) pressure-time curve for very short time exposures to pressure.
For example, consistent with the above calculations, a ‘safe’ pressure level for tissues that undergo a five-minute exposure to loads should be 3,600mmHg (300mmHg x hr)/(5/60) hr. However, this is unlikely because compression loads of that order cause total rupture of delicate organs such as kidneys (Farshad et al 1999), and are close to the rupture strength of large blood vessels, including the aorta (Stemper et al 2007). In fact, pressures of 600mmHg (five times the normal systolic blood pressure) are immediately enough to tear layers of vascular tissue from blood vessels (Carson and Roach 1990) (Figure 1, legend 2). Hence, the Reswick and Rogers (1976) pressure-time curve must be inaccurate at the extremes of the timescale (Figure 1, legends 1, 2).

It is important and fair to note that Reswick and Rogers (1976) referred to their pressure-time curve as ‘general guidelines’, and stated that it should not be interpreted quantitatively for clinical decision making. This is because they had high variability and the patient population included in their study, with little control of the loading situation for each patient. However, there are no other comparable data, excluding the Patterson and Fisher (1986) study on only five patients, which contradict the Reswick and Rogers (1976) study. This paucity of publications may have contributed to the misinterpretation of the Reswick and Rogers (1976) pressure-time curve in later studies, outlined below, where the curve was taken as quantitative data, rather than as general guidance, which was the original intention of the authors. Specifically, in terms of basic science, modelling work was conducted using the quantitative hyperbola pressure-time curve, [Pressure] x [Time] = [Critical Pressure Time Product], to extrapolate skin oxygen tension (Chang and Seireg 1999). Other mathematical functions relating to tolerable pressure levels and exposure times, which similarly allow very high pressure levels at the short time exposures (Figure 1, legend 2), were proposed as well (Sacks et al 1985).

Misinterpretation of the Reswick and Rogers (1976) observations also emerged in the patent and engineering design literature as related to pressure ulcer prevention. The development of new inventions and the evaluation of existing technologies that attempt to minimise the risk of pressure ulcers was biased by considering the Reswick and Rogers (1976) curve as a quantitative measure, which can evaluate, for instance, how well a mattress or cushion performs (Ryan and Byrne 1989, Spano and Asada 1999, Shah and Ezra 2004, Lowne and Tarler 2005).

Other than the fact that the Reswick and Rogers (1976) ‘general guidelines’ are incorrect at the extremes of the timescale, as outlined earlier (Figure 1, legends 1, 2), the data used for developing these guidelines use interface pressures (like the majority of pressure ulcer studies), recorded between the body and a cushion or mattress, as the measure of the load to which tissues are subjected. The bioengineering work carried out in pressure ulcer research since the beginning of the 1980s, starting with simple physical models and followed by sophisticated computer models, indicated that internal tissue loads cannot be predicted by means of interface pressure measurements (Agam and Gefen 2007, Gefen 2007, Gefen and Levine 2007). Moreover, all physical models and computer simulation studies over the past 20 years have concluded that load levels in muscle tissues overlying bony prominences are higher than interface pressures. For example, seated persons’ internal gluteal muscle tissue loads near the ischial tuberosities would be about two times higher than peak buttock pressures (Gefen 2007, Linder-Ganz et al 2008). This fits with animal histology studies, which observed substantial damage in muscle tissue near bony prominences preceding damage in more superficial tissue layers (Daniel et al 1981, Salcido et al 1994).

Anatomical variations, for example in curvatures of bones, thicknesses of the soft tissue layers and body weights, and physiological differences, such as muscle tone, habitual postures and movement, cause the internal tissue loads and exposure times to differ considerably between individuals (Linder-Ganz et al 2007, Linder-Ganz et al 2008, Linder-Ganz and Gefen 2009). Consider the two computer model simulation cases in Figure 2, which show distributions of internal deformations in soft tissues under the ischial tuberosities in a simplified geometrical and biomechanical configuration. These simulations are based on mathematical modelling that approximates the relative deformations of tissues compressed by an external ‘sitting’ pressure, between the seat and a round bony prominence (Gefen 2008). The interface pressure between the buttocks and seat at the region under the ischial tuberosity was assumed to be the same in the two model cases shown (150mmHg); however, the thicknesses of the soft tissues under the ischial tuberosity were assumed to differ between the models (38mm and 22mm). These two values are realistic measurements and are based on the magnetic resonance imaging studies of Linder-Ganz et al (2008). The simulations depict remarkably different intensities of tissue.
deformations, despite the interface pressure being kept the same (Figure 2).

The result means that two patients may show a similar interface pressure under a bony prominence and at the same time, despite having substantially distinct internal tissue load distributions. This demonstrates an inherent ambiguity in the Reswick and Rogers (1976) pressure-time curve. The patient showing the more intense internal tissue deformations (Figure 2b) may develop a serious pressure ulcer or deep tissue injury much sooner than the other patient, but the Reswick and Rogers (1976) pressure-time curve – being based on interface pressure as the measure of tissue load levels (Figure 1) – is not able to distinguish between these two cases. The large variation in internal tissue loads, even for similar interface pressures, is a result of inter-subject differences in internal anatomy and tissue mechanical properties, which may explain why the Patterson and Fisher (1986) study was at variance with the Reswick and Rogers curve (1976). Accordingly, the Reswick and Rogers pressure-time curve (1976) might not be reproducible if the Berlowitz and Brienza (2007) concept that pressure ulcers develop in deep tissues is accepted. Regardless, using the definition of the US National Pressure Ulcer Advisory Panel that a deep tissue injury is ‘a pressure-related injury to subcutaneous tissues under intact skin’ (Black 2009), the Reswick

References


and Rogers (1976) pressure-time curve is not applicable to predict a deep tissue injury.

**Conclusion**

In the first part of this article, the concepts of an injury threshold were explained, and it was shown that the Reswick and Rogers (1976) injury threshold is inaccurate at the extremes of the timescale. It has further been demonstrated that the Reswick and Rogers (1976) curve is not suitable for studying deep tissue injuries, and may not be appropriate for studying most pressure ulcers (Berlowitz and Brienza 2007). Accordingly, healthcare practitioners should not establish injury criteria, risk assessments or other clinical guidelines based on the Reswick and Rogers (1976) study. The second part of this article will describe the current knowledge regarding tissue injury thresholds, particularly thresholds that are specific for deep tissue injuries NS

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**References**


