

Back to basics: understanding the aetiology of pressure ulcers

KEY WORDS

- ▶ Pressure ulcer aetiology
- ▶ Shear
- ▶ Strain
- ▶ Stress

Pressure ulcers remain a worldwide problem. As research techniques have advanced, specifically in the field of finite element computational modelling, the tissue viability community has been able to gain a deeper understanding of the aetiology of pressure ulcers. Historically, the magnitude and duration of pressure was investigated to establish a pressure duration curve, initially for animals and, subsequently, for humans. However, this was restricted in its ability to provide a definitive answer to how long and how much pressure is required for a pressure ulcer to develop. Mechanical loading of the skin gives rise to forces acting either perpendicular to the skin, i.e. pressure, or parallel to the skin, i.e. shear. A combination of perpendicular and shear forces changes the shape of soft tissues. Individuals will have a unique pressure-resistant threshold that will depend on the composition of their tissues and general health and lifestyle. Debate continues in relation to the aetiological differences in the formation of superficial and deep pressure damage and into the role of microclimate in pressure ulcer formation as the author highlights in this article.

Pressure ulcers are caused when an area of skin and the tissues below are damaged by being placed under pressure sufficient to impair its blood supply. They are more likely to occur in people who are seriously ill, have a neurological condition, impaired mobility, impaired nutrition, or poor posture or a deformity (National Institute for Health and Care Excellence [NICE], 2014). The international pressure ulcer prevention and management guidelines define a pressure ulcer as “*a localised injury to the skin and/or underlying tissue, usually over a bony prominence, resulting from sustained pressure (including pressure associated with shear)*”. They add that there are several contributing or confounding factors also associated with pressure ulcers; the primary of which are impaired mobility and impaired sensory perception (National Pressure Ulcer Advisory Panel, European Pressure Ulcer Advisory Panel and Pan Pacific Pressure Injury Alliance [NPUAP, EPUAP, PPPIA, 2014]).

Pressure ulcers are commonly found over bony prominences, however, device-related pressure damage can occur in other parts of the body (Coyer, Stotts and Blackman, 2014; Ham et al 2014).

The international guidelines provide a historical context for the terminology used. Bedsore was used following a publication (Kenedi, 1976) from the first international conference on pressure ulcer aetiology that took place in Glasgow, UK in 1975 (NPUAP, EPUAP, PPPIA, 2014). Latterly, the term changed to pressure sore and then to pressure ulcer. The American and Pan Pacific pressure ulcer organisations are now using the term ‘pressure injury’ to reflect that pressure damage can be present in intact skin (NPUAP, 2016).

PRESSURE ULCER PREVALENCE

Pressure ulcers remain a problem for hospital in-patients, with European prevalence rates ranging from 8.3% to 22.9% (Clark et al, 2002). Hahnel et al (2016) undertook a systematic review of the epidemiology of skin conditions in the aged (65 years and over) and reported pressure ulcer prevalence of 0.3%–46% and incidence 0.8%–34% across 14 countries worldwide. A recent audit of in-patients in Wales reported a pressure ulcer prevalence rate of 8.9% (Clark et al, in press).

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AETIOLOGY

Historically, two factors that were and still are thought to influence pressure ulcer formation are the intensity and duration of the applied pressure. Early experimental work on animals attempted to establish if a critical balance could be obtained between the maximum amounts of pressure tissue could withstand and the minimum time for which it could be tolerated (Brooks and Duncan, 1922; Husain, 1953; Kosiak, 1959). This resulted in a pressure-duration curve for swine (Daniel et al, 1981) and other species, including man (Reswick and Rogers, 1976). However, the Reswick and Rogers curve was based on clinical experience with relatively few controlled measurements. Subsequent work identified additional factors that gave rise to pressure ulcer formation including shearing forces, which act parallel to the surface of the skin (Bennett and Lee, 1985), capillary auto regulation (Larsen et al, 1979), tissue deformation (Bell et al, 1974), and the protective function of dermal tissue (Reddy et al, 1975).

The international guidelines provide an explanation for pressure ulcer causation based on contemporary research. For pressure damage to occur the skin must be subjected to pressure from an external mechanical load, often including chairs, beds and mattresses prosthetic and medical devices (Fletcher, 2012; NPUAP, EPUAP, PPIA, 2014). Contact with medical devices often being the prime cause in the neonatal and paediatric population (Baharestani and Ratliff, 2007; Kottner et al, 2010; Schuler, 2017). NICE (2014) guidance on safeguarding children

reminds clinicians that abuse (malnourishment in particular) can contribute to or cause pressure ulcers in children.

Mechanical loading of the skin gives rise to forces acting either perpendicular to the skin or parallel to the skin, the latter termed a shear force. A combination of perpendicular and shear forces changes the shape of soft tissues, the first step towards tissue damage. Tissue deformation arises through mechanical stresses and strains created by the forces applied to the skin and underlying soft tissues; stress tells us about the amount of force applied per unit area while strain measures the amount of deformation the tissue experiences. Frictional forces occur when there is continuous rubbing or sliding of a surface over the skin which results in a contact force that is parallel to skin. (Coleman et al, 2013) (*Figure 1*).

The tissue stresses and strains produce two different outcomes, at a lower threshold partial to total occlusion of the blood vessels will occur while at higher stresses and strains direct damage of the cells occurs as cell membranes become squeezed together. Muscle tissue deforms easier and faster than skin and is, therefore, more prone to speedier deformation and ischaemic injury (Salcido et al, 1994). Strains of sufficient magnitude have the potential to cause cell death within very short periods of time (Gefen et al, 2008).

The ischaemic-induced damage will produce a lack of oxygen and nutrient supply to the tissues and eventually cause tissue necrosis and death. The unwanted waste products arising from tissue death will perpetuate the situation and produce an oxygen deficiency that will cause further tissue damage. (Hoogendoorn et al, 2017). In addition to occlusion of the circulatory system, the lymphatic system is also affected by pressure. The pressure prohibits the formation and clearance of lymph, an accumulation of which can further damage tissue (Gray et al, 2016).

A secondary effect of prolonged occlusion of the circulation occurs when the pressure is relieved and blood can once again flow in the affected area; reperfusion injury. The key to the damage produced from a reperfusion injury is the inflammatory response and subsequent release of harmful oxygen-free radicals (Peart, 2016). The extent of the reperfusion injury is related to

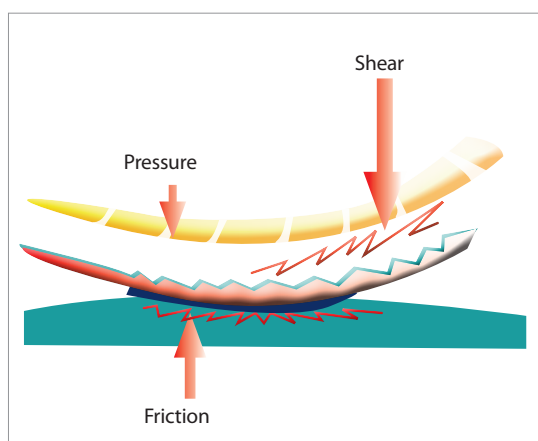


Figure 1. Forces applied to a surface (adapted from Pressure Ulcers, Urgo Medical®)

the magnitude of the external pressure that was applied to the skin (Hoogendoorn et al, 2017).

The deformation-induced damage will alter the shape and structure of the cells and interfere with their ability to function normally, e.g. osmosis and diffusion (Peart, 2016).

Regardless of the physiological pathway, all damaged tissue will have altered mechanical properties that can worsen the effect of the tissue stresses and strains (Linder-Ganz and Gefen, 2004, Gefen et al, 2005).

Pressure damage will depend on the magnitude and duration of the mechanical load along with the ability of the individual's tissues to withstand the external pressure. The auto-regulation of skin blood flow is an example of an internal mechanism that protects the circulatory system from external pressure (Hoogendoorn et al, 2017). The young, elderly, and spinal-cord injured are less able to resist the external pressure than a healthy adult (NPUAP, EPUAP, PPIA, 2014; Schuler, 2017; Levy et al, 2016). Because of the different nature of tissue at different parts of the body, an individual can react to pressure differently depending on the site of the body subjected to the pressure. The most common sites for pressure damage are those over a bony prominence, the intensity of the force increases as the force passes from the skin through the tissues and is therefore higher at the level of the bone than the surface of the skin.

Consequently, damage may be occurring at the level of the deeper tissues before it is visible on the skin surface. Therefore, the interface pressure at the surface of the skin is not equal to the internal pressure inside the tissue (Hoogendoorn et al, 2017).

An additional factor that influences the body response to the external pressure is the length of time the pressure is applied for, consequently high pressure for a short time is as dangerous as low pressure for a long time as both can cause pressure damage (Salcido et al, 1994; Linder-Ganz et al, 2006; Stekelenburg et al, 2006).

The excesses of temperature and the turgor of the skin can affect its ability to withstand external pressures. Moisture on the skin can be caused by internal mechanisms such as pyrexia or external factors such as a humid environment. An increase in humidity and temperature of the skin weakens the skin (NPUAP, EPUAP, PPIA, 2014).

Conversely, dry skin at the feet may be a risk factor for heel pressure ulcer development, again demonstrating the differences in an individual's response to pressure at different parts of the body (Lechner et al, 2017). Microclimate refers to the humidity and temperature between the skin and the support surface, its relationship to pressure damage has been investigated by various research groups and the link between the two is starting to be established (Yusuf et al, 2015; Yoshimura et al, 2015; Forriez et al, 2017) (Figure 2).

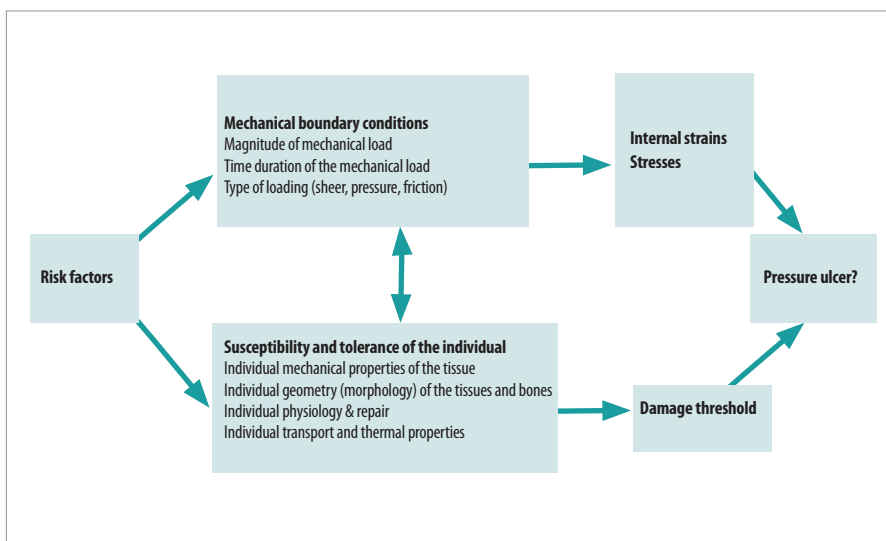


Figure 2. Factors leading to pressure ulcer development (adapted from Coleman et al, 2013)

MEDICAL-DEVICE RELATED PRESSURE DAMAGE

There is increasing awareness of medical-device related pressure damage with a reported prevalence of 3.1% to 40% in the intensive care environment with endotracheal and nasogastric tubes causing the most damage (Coyer, Stotts and Blackman, 201; Hanonu NS Karadag, 2016). Medical-device related damage does not necessarily occur over a bony prominence and one study identified such injuries on 16 locations on the front and back of the body (Ham et al, 2017). A multidisciplinary quality improvement project was able to reduce the prevalence of respiratory device-related pressure ulcers. It involved a focus on documentation of the occurrence of the injury along with a root cause analysis for each episode of damage and educational support (Padula et al, 2017).

PRESSURE MEASUREMENT

Previously research into pressure ulceration relied somewhat on interface pressures and pressure mapping which are limited to quantifying pressures on the surface of the skin. However, with the advent of finite element computational modelling it is possible to evaluate the internal mechanical loads in subcutaneous fat and skeletal muscle (Levy et al, 2016). An example of this is the work undertaken by Oomens (2003) in which computer modelling demonstrated that individuals with comparable interface pressures had significantly different internal stresses and strains.

SUPERFICIAL AND DEEP PRESSURE DAMAGE

Since the early 80s, different research groups have attempted to separate the cause of pressure injury into superficial damage (category I and II) and full-thickness pressure ulcers (category III, IV, unstageable and suspected deep tissue injury), (Barton and Barton, 1981; NPUAP, EPUAP, PPIA, 2014).

There may be a potential difference in the aetiology of superficial and deep pressure ulcers. Superficial ulcers may be primarily caused by high shear forces at the skin surface acting both to deform skin as it wrinkles under superficial shear and also to help separate the epidermis from the dermis (delamination) as the two layers deform through superficial shear at different rates, while deeper ulcers could result from high pressure at the surface over bony prominences. The caveat is that although some studies support this proposition, the current evidence is minimal and the precise response of skin to high shear deformation is not yet fully understood and continues to be debated (Lahmann and Kottner, 2011).

Takahashi et al (2016) purport that category II pressure ulcers (NPUAP, EPUAP, PPIA, 2014) are associated with superficial shear forces whereas, deep pressure ulcers correlate with pressure force that is perpendicular to the skin and high deep shear forces close to the bony prominence. They acknowledge that the correlation between the type of force and depth of pressure ulcer has not been precisely demonstrated but state that it appears to be widely accepted in practice.

CONCLUSION

Pressure ulcers continue to be a problem for individuals around the globe with their aetiology related to a combination of physiological changes namely tissue ischaemia and deformation. The application of an external load to the skin produces perpendicular and shear forces which together distort tissue shape and lead to occlusion of blood and lymphatic vessels and direct damage to cells. The effects of these forces differ between the skin, subcutaneous tissue, muscle and connective tissues.

There is a rise in interest in the role of the microclimate in pressure ulcer formation.

Computer modelling has enabled pressure ulcer research to move to the next level by facilitating the investigation of pressure responses in subcutaneous tissue and skeletal muscle.

There is still debate surrounding the different pressure damage seen in superficial and deep pressure ulcers, where shear forces rather than perpendicular forces are thought to be primarily responsible for superficial pressure ulceration.

Understanding the aetiology of pressure ulcers relies on an awareness of the internal response to mechanical load and not just what is apparent on the outside of the body or on the skin surface (NPUAP, EPUAP, PPIA, 2014). WUK

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