Development of pressure ulcers when sitting

KEY WORDS
- Adults
- Extended periods of time
- Pressure ulcers
- Sitting

Studies suggest that sedentary lifestyles are increasing with the average adult spending 50–60% of the day sitting. This change in lifestyle activities poses a risk not just to a person's general health but also for the development of a pressure ulcer, particularly in people who are forced to sit for extended periods of time due to illness or disability. The purpose of this article is to define and categorise pressure ulcers. To explore how and where pressure ulcers develop in the seated individual, the risk factors from direct and indirect forces when seated and the evidence base to explain why pressure ulcers develop when sitting.

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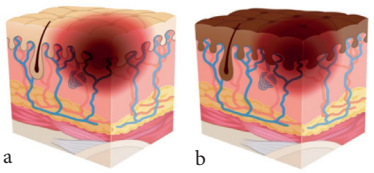
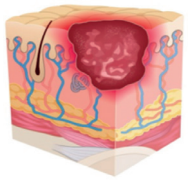
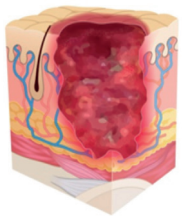
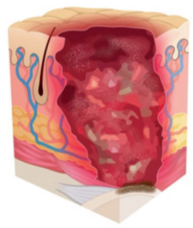
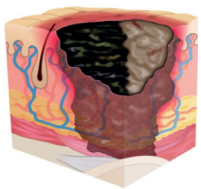
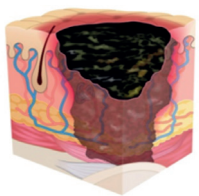
Sedentary behaviours are endemic in modern society and are considered universally as a public health risk (Harvey et al, 2013). The World Health Organization (WHO, 2010) reported that the fourth leading risk factor of global mortality is directly attributable to physical inactivity. The ramifications of physical inactivity extend to life-threatening conditions such as diabetes, ischaemic heart disease and some cancers (WHO, 2009). The word 'sedentary' originates from the Latin term sedentārius, meaning 'one that sits' (Merriam-Webster, 2018). In adults, a sedentary lifestyle can be characterised by engaging in activities that encourage prolonged sitting, for example, watching TV, using mobile devices, sitting at the computer. According to Healy et al (2011), sedentary pursuits account for 50–60% of the average adult's day. While it has been acknowledged in the literature that sedentary behaviours are linked with increased mortality due to the physiological changes that occur in the body, the evidence relating to skin damage from pressure ulcers when sitting is becoming more prevalent (Bhattacharya and Mishra, 2015; Bartley and Stephens, 2017; Stephens and Bartley, 2017, Stephens et al, 2017). This paper will explore the risk of sedentary behaviour on people who sit for extended periods of time on the development of pressure ulcers.

WHAT IS A PRESSURE ULCER?

In 2018, NHS Improvement revised the definition of a pressure ulcer in response to a lack of consistency and standardisation in defining, measuring and reporting pressure ulcers in England (Smith et al, 2016). A consensus was agreed to define a pressure ulcer as *"localised damage to the skin and/or underlying tissue, usually over a bony prominence (or related to a medical or another device), resulting from sustained pressure (including pressure associated with shear). The damage can be present as intact skin or an open ulcer and may be painful"* (NHS Improvement, 2018, p.4). This expands on the definition by the National Pressure Ulcer Advisory Panel, European Pressure Ulcer Advisory Panel and Pan Pacific Pressure Injury Panel (NPUAP, EPUAP, PPPIA, 2014) as it makes reference to medical devices, skin integrity and terminology. There are different categories of pressure ulcers depending on the level of damage and visible skin failure. Current best practice recommends the use of the NPUAP, EPUAP and PPPIA (2014) categorisation of pressure ulcers *(Table 1)*. However, an interesting observation in the NHS Improvement (2018) document is that whilst the recommendations made are to ensure *"a more consistent approach to the definition and measurement of pressure ulcers at both local and national levels across all trusts"* (p.2), there remain discrepancies across terminology. For example, if pressure ulcers are now to be defined as localised damage, the category deep tissue injury should have been redefined as deep tissue damage. Unstageable should also then be termed uncategorisable. NHS Improvement, nevertheless, clarify that minimal changes have been made to the category system as a way to lessen the disruption of changes to current practice.

Table 1. NPUAP, EPUAP and PPPIA (2014) categorisation of pressure ulcers

Categories of pressure ulcer damage	Visual representation
A Category I pressure ulcer is superficial damage and the affected area of skin appears discoloured (red in people with white skin [a], and purple/bluish in people with darker skin tones [b]) The skin is not broken, but it may be painful, itchy, and feel warm and squishy, or hard on touching.	 a b
A Category II pressure ulcer presents as *"partial thickness loss of dermis presenting as a shallow open ulcer with a red pink wound bed, without slough. May also present as an intact or open/ruptured serum filled blister. Presents as a shiny or dry shallow ulcer without slough or bruising. *This Category/Stage should not be used to describe skin tears, tape burns, perineal dermatitis, maceration or excoriation. *Bruising indicates suspected deep tissue injury."* (p.12).	
A Category III pressure ulcer presents as *"full thickness tissue loss. Subcutaneous fat may be visible, but bone, tendon or muscle are not exposed. Slough may be present but does not obscure the depth of tissue loss. May include undermining and tunnelling. The depth of a Category III pressure ulcer varies by anatomical location. The bridge of the nose, ear, occiput and malleolus do not have subcutaneous tissue and Category III ulcers can be shallow. In contrast, areas of significant adiposity can develop extremely deep Category III pressure ulcers. Bone/ tendon is not visible or directly palpable."* (p.12).	
A Category IV pressure ulcer presents with *"full thickness tissue loss with exposed bone, tendon or muscle. Slough or eschar may be present on some parts of the wound bed. Often include undermining and tunnelling. The depth of a Category IV pressure ulcer varies by anatomical location. The bridge of the nose, ear, occiput and malleolus do not have subcutaneous tissue and these ulcers can be shallow. Category IV ulcers can extend into muscle and/or supporting structures (e.g., fascia, tendon or joint capsule) making osteomyelitis possible. Exposed bone/tendon is visible or directly palpable"* (p.13).	
An unstageable pressure ulcer is one that is covered with dead tissue however, the extent of damage cannot be assessed until the dead tissue has been removed by a trained healthcare professional or via autolysis.	
A suspected deep tissue injury presents as a *"purple or maroon localized area of discoloured intact skin or blood-filled blister due to damage of underlying soft tissue from pressure and/or shear. The area may be preceded by tissue that is painful, firm, mushy, boggy, warmer or cooler as compared to adjacent tissue. Deep tissue injury may be difficult to detect in individuals with dark skin tones. Evolution may include a thin blister over a dark wound bed. The wound may further evolve and become covered by thin eschar. Evolution may be rapid exposing additional layers of tissue even with optimal treatment."* (p.13).	

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WHERE DO THEY DEVELOP IN SITTING?

Common sites for pressure ulcer development when seated in an upright position *(Figure 1a)* include the: ischial tuberosities, coccyx, greater trochanter, bony prominences of the spine, scapula, heels, elbows, back of the head and back of the knees. However, from repeated slouching or sliding in the chair damage can also occur to the sacrum *(Figure 1b)*.

Other areas include: palms of the hands from manual wheelchair propulsion, genitals from sitting, and under/against equipment such as catheters, leg bags, belts, headrests and slings that are attached to

or placed under people while seated (Stephens and Bartley 2017, Health and Safety Executive [HSE] 2011).

The development of pressure ulcers when seated is dependent on a number of components. Appropriate assessment for seating must include measurement of variables such as the person's seat width, seat height, armrest height/length, seat to ground height, backrest height, seat to back angle and headrest position. Failure to address these variables can lead to pressure damage, for example in *Figure 1b*, the person slouching in the chair. This occurs when a chair is too low or too high and leads to sliding in the chair. There is no standard position when seated that is suitable for all persons and therefore what is optimal for each person should be taken into consideration. Factors that can reduce the development of pressure ulcers in the seated person include postural stability when sat in the chair, ease of transfer in and out of the chair and maintenance of functional ability (carrying out activities of daily living).

RISK FACTORS

The risk factors for pressure ulcer development are well documented (NPUAP, EPUAP, PPPIA, 2014). However, the correlation between these factors and the actual cause of pressure ulcers is not known. A systematic review by Coleman et al (2013) concluded that the probability of pressure ulcer development relies on a multitude of factors and only applied pressure being directly attributable to pressure ulcer development. Their findings note that lack of mobility/activity, poor perfusion as a result of diabetes and the presence of an existing pressure ulcer emerged as key causes. The review also found that there are forces that increase a person's risk of developing pressure ulcers; direct and indirect forces (Coleman et al, 2013).

DIRECT FORCES

Direct forces that increase a person's risk of pressure ulcer damage can include temperature and humidity at the seating interface, posture, pressure, friction and shear forces. Shear is a mechanical force working on an area of the skin in a direction parallel to the body's surface. For example, the skeletal frame moves one way and the skin the other. This causes damage to the blood supply of the underlying tissues, leading to ischaemia, cell death and tissue necrosis (Hess, 2004). Static friction occurs by the resistance to movement of the person's skin and the surface, for example, the backrest or seat. Friction itself does not necessarily cause a pressure ulcer, however, it can lead to shear forces causing tissue deformation (Antokal et al, 2012). Chang et al (1999) stated that shear forces are much higher within the deeper tissues than at the surface and impact blood supply. This is due to a factor called a pressure gradient. For example, when a person is in a seated position pressure is transferred from the external surface through the layers of the skin, subcutaneous tissue and muscle to the bony prominence in a cone shaped gradient. As pressure is applied from sitting in the chair/wheelchair it increases in intensity and can be up to two times greater in deep tissues than that of the epidermis (Gefen, 2007). If unrelieved this

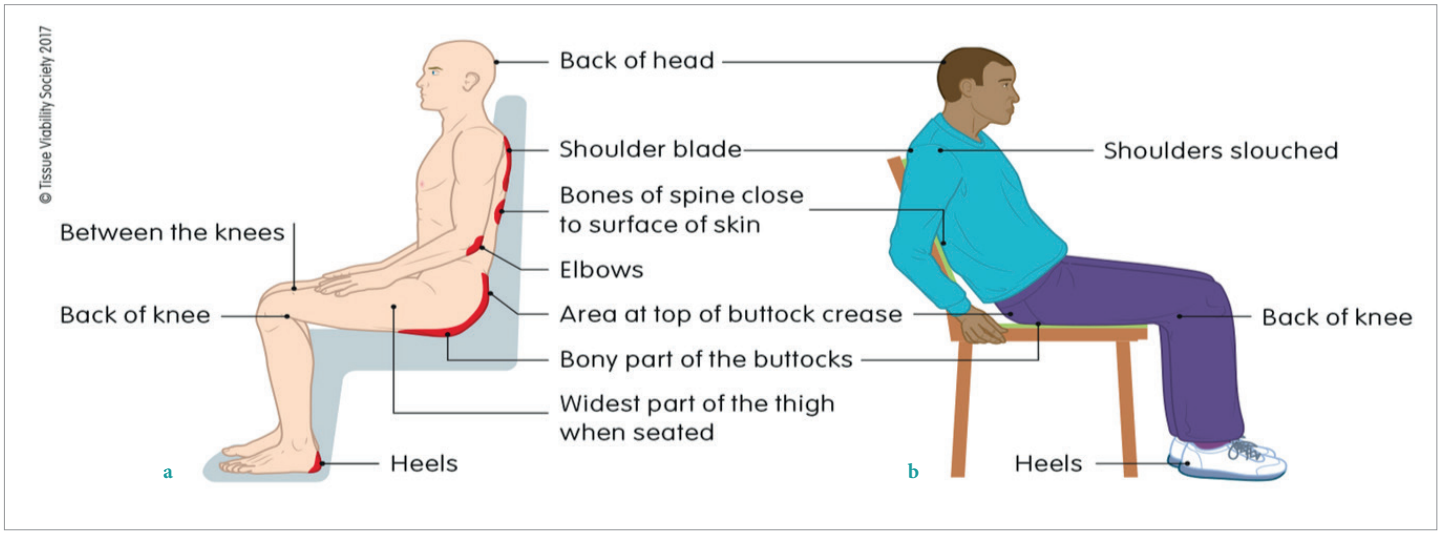

Figure 1. Common sites for pressure ulcer development when seated upright (a) or slouched (b).

can cause ischaemia, cell death and tissue damage.

Another direct force is pressure. When sitting in one position for a period of time, soft tissue is compressed between the seat surface and bony prominences of the skeleton and if unrelieved can lead to tissue damage. The amount of time required before damage occurs can be as little as 1–2 hours (Kosiak, 1959), with high pressures for short periods of time and low pressures for long periods of time being equally destructive. However, studies by Gefen (2007) and Berlowitz and Brienza (2007) suggest that there are considerable variations in tissue tolerance of people who remain seated for extended periods of time and mechanical loads can cause structural damage or tissue ischaemia especially in deep tissue injury (Oomens et al, 2015). Unrelieved pressure can also cause: lymphatic occlusion, which can cause tissue damage due to an accumulation of metabolic waste (Peart, 2016); and ischaemia-reperfusion injury which stimulates an inflammatory response and the release of oxygen free radicals and other mediators that cause tissue damage (Collard and Gelman, 2001). When moisture and humidity are present as a microclimate in the seated individual there is a risk to skin integrity and damage (Stockton and Rithalia, 2009). Moisture development may be a consequence of incontinence (Beeckman et al, 2015). However, humidity and moisture have been shown to be correlated within 5 minutes of sitting to the properties of the seat cushion and, therefore, regular movements that create ventilation are required (Liu et al, 2017).

INDIRECT FORCES

These pertain to the individual which include age, continence, mobility, pain, nutrition and fluid intake as well as comorbidities such as cardiovascular disease and diabetes and sensation (Baumgarten et al, 2003). For example, poor nutrition and fluid intake can lead to malnutrition, a deficiency of nutrients and vitamins to help tissue repair, resulting in weight loss and loss of cushioning for the bony structures such as the ischial tuberosities. Pain is a subjective phenomenon with varying degrees of severity and is influenced by the individual's experience and emotions (Wilcox et al, 2015). For people who sit for extended periods of time, pain can develop due to a lack of movement especially those whose posture is 'fixed' due to skeletal deformity. Pain will also be prevalent if the seated individual has a pressure ulcer developing.

WHY DO PRESSURE ULCERS DEVELOP WHEN SITTING? THE EVIDENCE BASE

For a pressure ulcer to develop when seated it only takes a small amount of pressure over a short period of time before damage occurs. This is due to the weight of the body being borne over a relatively small surface area, compressing the soft tissue between the interface of the seat and the bones of the pelvis (Schubert and Héraud, 1994). The science behind this comes from the work of researchers in both animal and human studies (Kosiak, 1959; Reswick and Rogers, 1976; Krouskop 1983; Barbanel, 1991; Schubert and Héraud, 1994; Defloor and Grypdonck, 1999; Gefen, 2008). Early studies concentrated on the effects of pressure in relation to intensity and duration (Kosiak, 1959; 1961; Reswick and Rogers, 1976; Daniel et al, 1981). The researchers concluded that only a small amount of pressure for one to two hours could lead to tissue damage. This extra pressure occludes the blood flow through the skin starving the area of oxygen and nutrients, and if this goes unrelieved tissue begins to break down, leading to the development of a pressure ulcer.

Pressure and shear forces may cause closure of capillaries and if left unrelieved can result in ischaemia and tissue death (Landis, 1930; Defloor, 1999). Pressure inside capillaries can differ depending on several factors: the part of the body the capillary is located, the general health of the person (Haalboom et al, 2002), self-regulation of blood flow and the type of tissue being occluded (muscle is more tolerant than skin) (Nola and Vistnes, 1980; Defloor, 1999). Reperfusion should occur once pressure is relieved; however, this can be impaired due to irreparable smooth muscle cell contraction occurring during the ischaemic phase (Michel and Gillot, 1990). It is also thought that reperfusion can be damaging due to many factors such as the increase in oxidative stress (Peirce et al, 2000) and up-regulated collagen-degrading enzymes (Roach et al, 2002) to name but a few. However, Ceelen et al (2008) questioned the reliability of these studies when frequent repositioning appears to assist with pressure ulcer prevention. Recent studies have hypothesised that deformation of muscle cells over a sustained period

of time and impaired interstitial lymph flow can contribute to pressure ulcer formation (Reddy, 1990; Oomens et al, 2015). However, this work is ongoing as the research is not inclusive of all tissues pertaining to the skin.

CONCLUSION

Sitting in the same position for extended periods of time poses a risk to the individual in relation to the development of pressure ulcers particularly across the buttock area. Recent measures such as by NHS Improvements Task and Finish Group have been taken to standardise, define and categorise pressure ulcers to aid a consistent approach to the assessment and management of skin damage from pressure and shear (NHS Improvements, 2018). The most common sites for pressure ulcer damage in the seated position are widely acknowledged, however, consideration should be given to other areas where damage can occur for example the palms of hands from self-propulsion in a wheelchair. Direct and indirect forces are known to play a large part in pressure ulcer development. An understanding of the aetiology and pathophysiology of pressure ulcers when seated can inform the healthcare practitioner in delivering key seating outcomes. WUK

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