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Review

Microclimate: A critical review in the context of pressure ulcer prevention

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ABSTRACT

Pressure ulcers are caused by sustained mechanical loading and deformation of the skin and subcutaneous layers between internal stiff anatomical structures and external surfaces or devices. In addition, the skin microclimate (temperature, humidity and airflow next to the skin surface) is an indirect pressure ulcer risk factor. Temperature and humidity affect the structure and function of the skin increasing or lowering possible damage thresholds for the skin and underlying soft tissues. From a pressure ulcer prevention research perspective, the effects of humidity and temperature next to the skin surface are inextricably linked to concurrent soft tissue deformation. Direct clinical evidence supporting the association between microclimate and pressure ulceration is sparse and of high risk of bias. Currently, it is recommended to keep the skin dry and cool and/or to allow recovery periods between phases of occlusion. The stratum corneum must be prevented from becoming overhydrated or from drying out but exact ranges of an acceptable microclimate are unknown. Therefore, vague terms like 'microclimate management' should be avoided but product and microclimate characteristics should be explicitly stated to allow an informed decision making. Pressure ulcer prevention interventions like repositioning, the use of special support surfaces, cushions, and prophylactic dressings are effective only if they reduce sustained deformations in soft tissues. This mode of action outweighs possible undesirable microclimate properties. As long as uncertainty exists efforts must be taken to use as less occlusive materials as possible. There seems to be individual intrinsic characteristics making patients more vulnerable to microclimate effects.

1. Introduction

In the context of severe acute and chronic illness, disability and high care dependency, pressure ulcers (PUs) are one of the most unwanted adverse events. Epidemiological data indicate that PUs occur in all settings and age groups (Kottner et al., 2010b; National Pressure Ulcer Advisory Panel et al., 2014; Tomova-Simitchieva et al., 2018a), but are clearly associated with increasing age (Hahnel et al., 2017b). PUs are caused by sustained mechanical loading and deformation of soft tissues such as skin, subcutaneous fat, or muscle between internal stiff anatomical structures (bone, tendon) and external surfaces or devices. If the intensity and duration of deformation exceeds the individual's physiological capacity and resistance of the deformed tissues, cells will die and necrotic regions will develop (National Pressure Ulcer Advisory Panel et al., 2014; Shoham and Gefen, 2012). In a supine lying position the lateral heel and sacral areas are most often affected by the development of PU, and are well known as PU predilection areas.

Empirical evidence suggests that there are two main pathophysiological pathways for pressure ulceration: (1) Soft tissue deformation leads to the reduction of perfusion, resulting in ischemia and all its consequences (e.g. reduced nutrients supply, accumulation of waste products, acidification). After off-loading the damaging effects may also be aggravated by a reperfusion injury. (2) Soft tissue deformation exceeding certain tolerance thresholds leads to direct deformation damage of cells through structural failure of the cytoskeleton and plasma membrane (National Pressure Ulcer Advisory Panel et al., 2014; Oomens et al., 2015; Slomka and Gefen, 2012). Experimental study results and clinical observations suggest that initial deformation-induced necrosis starts near bony prominences in the muscle or other subcutaneous tissues under intact skin, which is termed deep tissue injury (Berlowitz and Brienza, 2007; Kwan et al., 2007; National Pressure Ulcer Advisory Panel et al., 2014; Shoham and Gefen, 2012).

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Depending on the size of the necrotic area, mechanical properties of the affected and adjacent areas, off-loading, and the overall health condition of the individual, deep tissue injuries may eventually progress to full thickness pressure ulcers (Aoi et al., 2009; Kottner et al., 2010a; National Pressure Ulcer Advisory Panel et al., 2014).

In addition to the widely accepted bottom-up pathogenesis of PUs a possible top-down process starting in the epidermal and dermal layers progressing to deep PUs is also reported (Scheel-Sailer et al., 2017). It is known that deformation-caused and ischemia-inflicted damage might also originate in skin, but whether this leads to cell damage pathways that are similar to those occurring in subcutaneous tissues is currently unclear. In clinical practice, there are various other cutaneous lesions and wounds such as incontinence-associated dermatitis, intertrigo, or friction-related lesions, that a not PUs according to the current pressure ulcer definition (Berke, 2015; Garcia-Fernandez et al., 2016; Mahoney et al., 2013).

Besides cutaneous lesions, the structure and function of the skin are also considered to play a key role in PU susceptibility and development (Coleman et al., 2013; Kottner et al., 2015; National Pressure Ulcer Advisory Panel et al., 2014). Skin properties are influenced by several intrinsic (e.g. age, medications, systematic diseases) and extrinsic (e.g. temperature, humidity next to the skin surface) factors. In this context, the concept of microclimate gained increasing attention in the PU prevention literature in the last years (Chai et al., 2017; Gefen, 2011; National Pressure Ulcer Advisory Panel et al., 2014; Yoshimura et al., 2015). First mentioned by Roaf in the 1970s (Roaf, 2006), it is generally accepted today that 'microclimate management' is important in PU prevention. The empirical evidence regarding how microclimate affects skin properties and how this is related to subsequent PU development is not sufficiently developed (Clark et al., 2010). The optimal microclimate at the skin surface and how this can be achieved remains unknown at this time (National Pressure Ulcer Advisory Panel et al., 2014).

The aim of this review is to provide an up-to-date and in-depth discussion of microclimate in the context of PU prevention, to link current ideas from dermatological, biomechanical, laboratory, and clinical practice perspectives, and to discuss current and future PU prevention technologies from a microclimate perspective. The review has a clinical focus and draws on contributions in the fields of nursing, skin research, biomechanics, mechanobiology, and PU research. Technical biomechanical aspects are summarized in the online Appendix A.

2. What is microclimate?

Microclimate can be defined as the climate in a local region that differs from the climate in the surrounding region (ambient climate). It consists of temperature, humidity, and airflow (Imhof et al., 2009). This concept is ubiquitous in many scientific disciplines such as botany, zoology, architecture, and aeronautics. It was first mentioned in the late 1940s (Edstrom et al., 1948; Haddow et al., 1947; Waterhouse, 1950) and today the term microclimate is listed more than 3000 times in the MEDLINE/PubMed database of the U.S. National Library of Medicine. In context of PU prevention, the microclimate near to the skin surface is of particular relevance.

2.1. Microclimate next to the skin surface

In cold or moderate climate zones or for indoor conditions the human skin is usually warmer than the ambient temperature. This temperature gradient causes continuous convection of warm air moving away from the warmer skin surface to the cooler environment. The actual air flow is non-uniform and complex. In a simplified laminar air flow model, there is a boundary layer of still air directly at the skin surface and a laminar flow region next to this boundary layer. The boundary layer thickness has been estimated to be 6 to 12 mm indoors, depending on the body and air movements in the room (Imhof et al., 2009). The higher the air speed, the smaller the boundary layer thickness is, and the higher is the rate of convective heat loss. Natural convection of air is one major mechanism to cool the human body and to maintain an internal thermal core temperature equilibrium of approximately 37°C. The skin surface, including the uppermost skin layers, has an intermediate temperature between the environment and the body core temperature. The skin surface temperature shows large regional variations. In tightly controlled environmental room conditions, the mean temperatures of uncovered PU predilection skin areas such as heels and sacrum was measured to range between 29 and 31°C (Chai et al., 2017; Fisher et al., 1978; Kottner et al., 2015; Worsley et al., 2016a). However, skin surface temperatures vary widely when conditions change. In- or decreasing environmental temperatures cause in- or decreasing skin temperatures (Chen et al., 2011; Igaki et al., 2014) leading primarily to vasodilatation or vasoconstriction respectively (Blatteis, 2012).

In addition to heat flow, there is also a humidity gradient from the skin surface to the environment. In normal physiological conditions there are two sources of humidity on the skin surface: transepidermal water loss (TEWL) and sweat. The term TEWL was first introduced by Rothman in 1954 (Rothman, 1954) and describes the process of the passive diffusion of water molecules from the fully hydrated dermal and epidermal layers through the outermost layer of the epidermis, the stratum corneum, to the usually much drier environment (Rogiers and EEMCO Group, 2001). There are a number of factors affecting TEWL such as skin area, skin condition, and age (Kottner et al., 2013; Rogiers and EEMCO Group, 2001) but because TEWL is based on a passive diffusion process it largely depends on the skin temperature and environmental humidity. TEWL increases with increasing skin temperature (Cravello and Ferri, 2008; Mathias et al., 1981; Rogiers and EEMCO Group, 2001) and decreasing relative environmental humidity (Cravello and Ferri, 2008; Imhof et al., 2009; Liu et al., 2017; Rogiers and EEMCO Group, 2001). The average amount of TEWL per day is estimated to vary between 50 and 600g (Figliola, 2003; Reger et al., 2001) which corresponds to approximately 2 to 14 g/m^2/h over an average 1.8 m^2 body surface. This range corresponds to mean TEWL estimates in healthy volunteers over various skin areas (Kottner et al., 2013). Under standardized conditions, the mean (baseline) TEWL at the heel is much higher compared to sacral skin (Kottner et al., 2015). This is consistent with the observation that TEWL at ridged skin areas is, in general, typically higher compared to patterned skin (Mayrovitz et al., 2013).

The human skin contains approximately 100 to 200 eccrine sweat glands/cm^2 with a maximum density of 600 glands/cm^2 on the palms and soles (Pierard et al., 2003) (Figure 1). Sweat is a clear colourless

Fig. 1. Skin surface of the human heel skin with orifices of sweat ducts visible as tiny pits lining on top of the ridges