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Clinical study

Skin response to sustained loading: A clinical explorative study

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Abstract *Background:* Severe illness, disability and immobility increase the risk of pressure ulcer development. Pressure ulcers are localized injuries to the skin and/or underlying tissue as a result of long enduring pressure and shear. Little is known about the role of the stratum corneum and the upper skin layers in superficial pressure ulcer development.
Objectives: To investigate possible effects of long enduring loading on the skin barrier function under clinical conditions at two pressure ulcer predilection sites.
Methods: Under controlled conditions 20 healthy females (mean age 69.9 (3.4) years) followed a standardized immobilization protocol of 90 and 150 min in supine position wearing hospital nightshirts on a standard hospital mattress. Before and immediately after the loading periods skin surface temperature, stratum corneum hydration, transepidermal water loss and erythema were measured at the sacral and heel skin.
Results: Prolonged loading caused increases of skin surface temperature and erythema at the sacral and heel skin. Stratum corneum hydration remained stable. Transepidermal water loss increased substantially after loading at the heel but not at the sacral skin.
Conclusions: Skin functions change during prolonged loading at the sacral and heel skin in aged individuals. Accumulation of heat and hyperaemia seem to be primarily responsible for increasing skin temperature and erythema which are associated with pressure ulcer development. Increased transepidermal water loss at the heels indicate subclinical damages of the stratum corneum at the heel but not at the sacral skin during loading indicating distinct pathways of pressure ulcer development at both skin areas.

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1. Introduction

Acute severe illnesses, disability, functional limitations or advanced care dependency are often associated with mobility and activity impairments. Patients or care receivers are confined to beds or chairs and are unable to move themselves and change positions. Maintaining body positions over longer periods of time leads to sustained deformation of soft tissues in contact with the underlying support surface especially over bony prominences [1]. If the duration and intensity of the loading exceeds the structural and functional capacity of the deformed cells and tissues pressure ulcers (PUs) may develop [2]. PUs are localized injuries to the skin and/or underlying tissue as a results of long enduring pressure and shear [1].

Worldwide PU prevalence and incidence are high ranging from 1 to 12% in hospital and acute care settings [3—7] to 20% in institutional long term and geriatric care [8—10]. Although PUs do occur in all age groups it is especially a condition of the aged [11]. PUs are associated with pain, reduced quality of life [12] and PU treatment is burdensome and expensive. Among 15 selected common skin conditions in the latest Global Burden of Disease report PUs were regarded as the most severe dermatological diseases contributing to a substantial health loss [13]. Furthermore PU development is regarded as an unintended adverse event and it is therefore a widely accepted indicator for the quality and safety of care [14,15]. Consequently, efficient PU prevention and maintaining skin integrity are major goals in health care [16].

During the last years the knowledge about PU aetiology increased substantially. It could be shown that strains within deeper tissues especially near bony curvatures are much higher compared to the skin surface [17,18] and that there is a time depended increase of muscle cell death during sustained deformation [19]. Ischemia, reperfusion damage, and impaired lymphatic functions further contribute to necrosis development in muscle and/or subcutaneous fat tissues [20,21] leading to so called deep tissue injuries [10,22,23]. While this inside-out mechanism of PU development is widely accepted today the distinct role of the skin under sustained deformation has gained less attention. Above all little is known about the role of the epidermis and the stratum corneum (SC), the upper most skin layers, which are in direct contact to the support surface and which contribute to mechanical strength of the skin [24].

Results of previous experimental *ex vivo* and animal studies indicate that sustained loading or deformation alter or even damage epidermal and dermal layers [25—27] and that there are associations between temperature and moisture content and biophysical properties of the SC [28,29] and the whole skin [30]. However, little is known about the functional skin barrier characteristics *in vivo* under 'real world' clinical conditions especially in aged individuals. Based on increased transepidermal water loss (TEWL) values Angelova-Fischer et al. recently showed skin barrier impairments in early stages of chronic venous insufficiency compared to healthy controls [31]. Whether such subclinical epidermal changes also occur prior to early PU development is unknown. Therefore the aim of this investigation was to explore possible effects of long enduring loading on the skin under clinical conditions.

2. Materials and methods

2.1. Study design, setting and participants

Between March and June 2013 an explorative clinical study was conducted. The study followed a protocol to simulate immobilization while lying in bed. Healthy volunteers were invited to participate meeting the following eligibility criteria: age 60—80 years, absence of skin diseases, absence of acute diseases, ability to move independently and to maintain supine and prone positions. We included only female subjects at this stage because in aged populations females are generally overrepresented in many clinical and long-term care settings. All habitual medications were allowed and documented that the subjects took at least 6 months before the study.

At the start of the study participants were requested to wear standard hospital nightshirts. Two investigational skin areas typically prone to PU development when lying supine were marked: above the Os sacrum ('sacral area'), and on the lateral side of the Tuber calcanei ('heels'). This heel skin area was chosen because in supine position there is a foot abduction of approximately 60° [32]. A skin area over the sternum was used as control site. An acclimatization period of 30 min took place at controlled room conditions (temperature of 19—21 °C and air humidity of 40—60%). After this baseline values were recorded for all three skin areas. Then individuals were requested to stay in supine position for 90 min on a standard hospital mattress covered by cotton linen. It was