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Blood perfusion and transcutaneous oxygen level characterizations in human skin with changes in normal and shear loads — Implications for pressure ulcer formation

Abinand Anbazhagan Manorama [a], Seungik Baek [a], Joseph Vorro [b], Alla Sikorskii [c], Tamara Reid Bush [a,*]

[a] Department of Mechanical Engineering, Michigan State University, East Lansing, MI 48824, USA
[b] Department of Community and Family Medicine, Michigan State University, East Lansing, MI 48824, USA
[c] Department of Statistics & Probability, Michigan State University, East Lansing, MI 48824, USA

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ABSTRACT

Background: Decubitus ulcers (pressure ulcers) are localized areas of tissue breakdown in the skin and the underlying regions. Decubitus ulcers affect approximately 3 million people in the USA every year, including seniors, individuals with diabetes, and those who spend long periods in wheelchairs. Experimental studies demonstrate that static or dynamic normal loads cause blood occlusion in the skin, while prolonged loading conditions can result in skin damage. However, few studies report the effects of 'normal and shear' combined loading on blood perfusion. The goal of this research was to study alterations of transcutaneous oxygen levels and blood perfusion in human skin when both normal and shear loads were applied.

Methods: Fifteen human subjects were evaluated under seven different conditions for changes in transcutaneous oxygen and blood perfusion levels during applications of normal and shear loading on the forearm. Transcutaneous oxygen levels and blood perfusion were continuously measured using a Laser Doppler system, while applied forces were quantified with a multi-axis load cell.

Findings: Transcutaneous oxygen and blood perfusion levels decreased when shear loads were applied in addition to normal loads. Further, blood perfusion during recovery periods increased gradually from the first to the last test condition.

Interpretation: Results indicate that adding shear loads decreased transcutaneous oxygen and blood perfusion levels in the skin. Based on these findings, shear force may play a role in skin damage, and both shear and normal loads should be considered when trying to prevent ulcer development.

1. Introduction

Decubitus ulcers, also known as pressure sores or pressure ulcers, are localized areas of tissue breakdown in the skin and underlying regions. Ulcers are a considerable health concern, costing over a billion dollars a year (Bansal et al., 2005), with approximately 1.3 to 3 million individuals affected in the United States (Lyder, 2003; Whitney et al., 2006). Spinal cord and brain injuries, as well as chronic diseases such as diabetes and vascular diseases, have significant implications in the formation of skin ulcers. The incidence of ulcers is most prevalent in special populations, particularly the bedridden or wheelchair-bound, where reports indicate that over 50% of these individuals develop ulcers (Sugarman, 1985). Furthermore, reports of recurrence rates for patients who have had pressure ulcers range from 19 to 38% (Kierney et al., 1998; Kuwahara et al., 2005).

Decubitus ulcers are most likely to develop in areas of the body where the dermal tissues are subject to sustained mechanical loading between bony regions and an external structure such as a wheelchair or mattress. This form of mechanical loading (between a bony prominence and an external structure) causes obstruction of blood flow, leading to blockage of nutrients to cells and preventing waste product removal. This process can quickly lead to necrosis of the skin and the underlying tissues (Bouten et al., 2003; Bader et al., 2005). The areas most prone to ulcers include the hips (67% of all decubitus ulcers), portions of the lower extremities such as ankles, knees, and heels (25%), and the remaining 8% of injuries are located in parts of the head (Revis, 2008).

Decubitus ulcers are often referred to as pressure ulcers or pressure sores, which reflects the common understanding of the cause of these ulcers. However, as Parish and Witowski (2004) suggested, the term pressure ulcers, "conveys the notion that sustained pressure is the only factor and disregards friction, shear force, moisture, temperature elevation, sensory impairment, or oxygen deprivation in its pathogenesis." Shear stress has long been proposed to play an important etiological role in decubitus ulcer development, but few studies have focused specifically on the effects of shear loads on blood perfusion. For example, the histological paper by Witowski and Parish (1982) suggested that "shearing injury exerts

* Corresponding author. Michigan State University, Department of Mechanical Engineering, 2555 Engineering Building, East Lansing, MI 48824-1226, USA.
E-mail address: reidtama@msu.edu (T.R. Bush).

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its effect on the large vessels in the deep portion of the superficial fascia". Bennett et al. (1979) found that in the thenar eminence of the hand, a combination of shear with normal loading caused an enhancement in vascular occlusion. Goossens et al. (1994) reported that cut-off pressure for skin oxygen tensions decreased in the presence of shear stresses. Further, Goldstein and Sanders (1998) applied varying types of repetitive mechanical stress on porcine skin, and concluded that skin breakdown occurred earlier when shear loads were increased.

Although it has been reported that a combination of normal and shear loads on the skin can accelerate tissue breakdown, details of the loading (e.g., magnitudes and dynamic or static application), and how it affects regional blood perfusion and transcutaneous oxygen levels in human skin remains unclear. We hypothesize that the combination of normal and shear loads decreases blood perfusion (PU) and transcutaneous oxygen levels ($tcpO_2$) in the areas of loading as compared with normal loads alone. We tested this hypothesis using a quasi-experimental design with 15 human subjects under seven different test conditions for changes in transcutaneous oxygen and blood perfusion levels during applications of normal and shear forces on the forearm.

The purpose of this research was to study alterations of transcutaneous oxygen levels and blood perfusion in human skin under normal loads as well as combined (normal and shear) loads. These data will help researchers develop a better understanding of the role shear load plays in the development of pressure ulcers.

2. Methods

2.1. Subjects

A total of 15 subjects (seven male and eight female) with an average age of 23 (SD 2.4) years were tested. All subjects were free from arm injuries at the time of testing.

2.2. Testing procedure

This investigation was approved by the Biomedical and Health Institutional Review Board at Michigan State University. After a briefing regarding the test procedures and the collection of anthropometric measurements, each subject was asked to sit on an office chair and place, in a comfortable fashion, his/her right forearm (approximately normal to the torso) on the load cell apparatus (Fig. 1).

A multi-axis load cell (Advanced Mechanical Technology, Inc., Watertown, MA, USA) was used to collect the force data. The load cell was connected to an AMTI amplifier and the gain was set to 1000. The built-in low-pass filter was set at 10.5 Hz. The load cell calibrations were checked prior to the initiation of the experiments, and after completion of the experiments. A specially designed wooden support was attached directly to the load cell. This support had custom carved regions to accommodate the oxygen and blood perfusion probes, as well as a portion of the cabling, so they did not interfere with the load application. When a subject placed his/her forearm on the wooden fixture, the location of the interface support was marked on the forearm, and the probe sites were identified. A Laser Doppler Perfusion Monitoring system (LDPM, Perimed, Stockholm, Sweden) was used to collect blood perfusion and $tcpO_2$ data (Holloway and Watkins, 1977; Leahy et al., 2003; Nilsson et al., 1980). The LDPM was calibrated prior to the start of the experiments, and once every week thereafter. The LDPM uses a two-point calibration in conjunction with a special motility standard. For our tests, the blood perfusion probe was attached to the forearm with double-sided adhesive tape, while the $tcpO_2$ probe was attached to the skin of the forearm with disposable fixation rings. PU is the product of local speed and concentration of blood cells and in reported in perfusion units (Leahy et al., 1999) Fig. 2.

A total of seven test conditions were performed, each condition had a one-minute duration (Table 1). The first condition established baseline values for blood perfusion and $tcpO_2$ in the absence of loading. Between each of the conditions, a two-minute recovery period was allowed. Based on the data from our pilot work and keeping in mind the practicality of subject involvement (i.e. limiting the length of time in the laboratory to 3 h or less), we implemented a recovery time of 2 min between each test condition. During each recovery period, all loading was removed.

Fig. 1. Individual during testing.

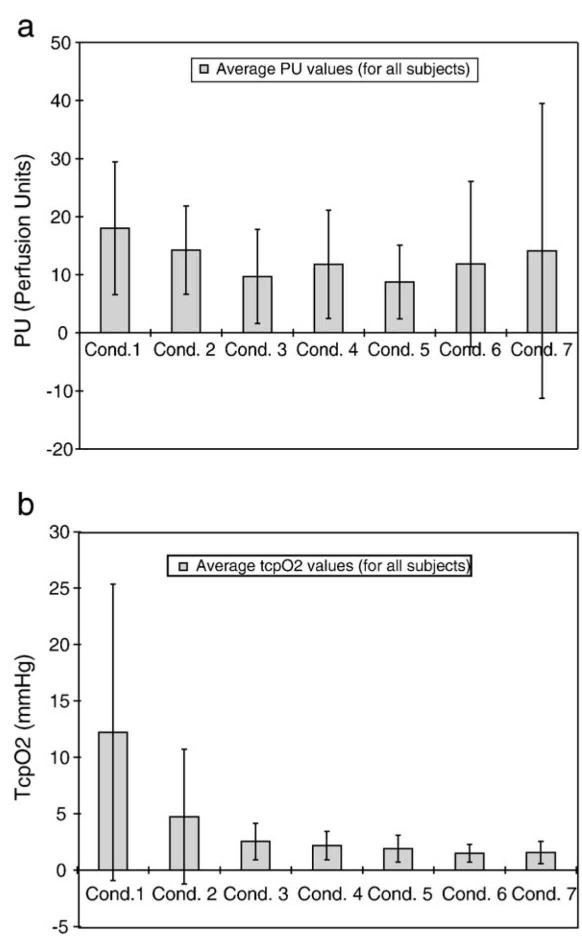

Fig. 2. (a) Average perfusion (PU) values and (b) transcutaneous oxygen ($tcpO_2$) levels obtained during testing for all subjects with the standard deviation of the entire subject pool.