Pressure ulcer research from seated posture to buttock tissue strain



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Hypotheses

- Ischemia
 - Lack of oxygen, nutrients, etc.
- Deformation
 - Cells die because of the pure deformation



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Hypotheses

- Deformation is the most important factor
- Ischemia does not give any effect before 22 hours
- Cells behave like
 water balloons
- The membrane is stretched

Annals of Biomedical Engineering, Vol. 35, No. 2, February 2007 (© 2006) pp. 273–284 DOI: 10.1007/s10439-006-9222-5

The Relative Contributions of Compression and Hypoxia to Development of Muscle Tissue Damage: An *In Vitro* Study

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Abstract—Deep pressure ulcers develop in tissues subjected to sustained mechanical loading. Though it has been hypothesized that this damage mechanism results from local tissue ischemia, it has recently been shown with a cell model that sustained compression can cause cell deformation, leading to tissue breakdown. The present study focuses on the assessment of cell viability during compression and ischemia in an sensitive to prolonged compression.^{13,23} Pressure ulcers involving muscle damage, denoted as grade III and IV ulcers, are typically observed in insensate patients with spinal cord injury.^{11,15} In addition, fiber morphology, contractile ability, and capillaries associated with muscle tissues are affected in this group of patients, which lead to

Membrane-Stretch-Induced Cell Death in Deep Tissue Injury: Computer Model Studies

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(Received 5 November 2008; accepted 2 February 2009)

Abstract—Deep tissue injury (DTI) is a serious pressure ulcer, involving a mass of necrotic soft tissue under bony prominences as a consequence of sustained tissue deformations. Though several processes are thought to participate in the onset and development of DTI (e.g., cellular deformation, ischemia, and ischemia-reperfusion), the specific mechanisms responsible for it are currently unknown. Recent work indicated that pathological processes at the cell level, which relate to cell deformation, are involved in the etiology. We hypothesized that sustained tissue deformations can lead to abauted intercablute concentration of call matchelities under intact skin.^{2,4,9,11,29,30,63} This injury initiates in skeletal muscle tissue overlying bony prominences, as a consequence of sustained tissue deformations. The DTI widens and progresses outward until becoming visible (as a purple or black spot) once it is close to the skin surface.^{2,4,9,11,29,30,63} Substantial research efforts are currently invested in understanding the exact mechanisms underlying the onset of pressure ulcers, and particularly of DTI. Existing theories suggest the

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Literature

Review

J Appl Physiol 108: 1458-1464, 2010. First published March 18, 2010; doi:10.1152/japplphysiol.01006.2009.

Missing links in pressure ulcer research—An interdisciplinary overview

Christian Gammelgaard Olesen,^{1,2} Mark de Zee,² and John Rasmussen¹

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Submitted 4 September 2009; accepted in final form 14 March 2010

Olesen CG, de Zee M, Rasmussen J. Missing links in pressure ulcer research-An interdisciplinary overview, J Appl Physiol 108: 1458-1464, 2010. First published March 18, 2010; doi:10.1152/japplphysiol.01006.2009.-This paper surveys the literature on the etiology of sitting-acquired deep tissue pressure ulcers from three different viewpoints. The first viewpoint is identification of risk factors related to seated posture. The second viewpoint focuses on the external factors that can cause necrosis to human cells, such as ischemia and compression. The third viewpoint focuses on computational models of the human buttocks to calculate where stress concentrations occur. Each viewpoint contributes to the understanding of pressure ulcer etiology, but in combination they cover the multiple scales from cell to organism, and the combined insight can provide important information toward a full understanding of the phenomenon. It is concluded that the following three questions must be answered by future research. 1) Does compressive stress alone explain cell death, or is it necessary to consider the full three-dimensional strain tensor in the tissues? 2) How does the change in posture-induced load applied on the human buttocks change the stress distribution in the deep muscle tissue? 3) Is it possible to optimize the seated posture in a computational model to reduce the deeper tissue loads?

wheelchair; biomechanics; deep tissue injury; spinal cord injury; pressure ulcer



The Seated Human

- Webcast <u>www.anybodytech.com</u>
 - Prof. John Rasmussen (development)
 - By me (Validation)
- Inverse dynamic model
- Human sitting in a generic chair
- Chair
 - Seat, Backrest, Armrest, Legrest, Footrest, and headrest
 - Adjustments:
 - Friction coefficients
 - Backrest, seat, foot, etc., can be adjusted



Validation

- Experiments
 - Reaction forces
 - Seated postures
- Model
 - Input = Seated posture
 - Reaction forces





The Seated Model

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- Setup
 - Angles and distances
- Calculating forces acting between chair and body
 - (Boundary conditions)



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FE-model of buttocks

- Wölfel Beratende
 Ingenieure GmbH
- Casimir/Automotive
- Non-linear model
- Automotive seating
- Vibration analysis



Casimir FE-model of buttocks

- Continuum model
 - Bones
 - Soft tissue
 - Muscles for posture
- Strain results depends on positioning in the chair



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Interface

Seated AnyBody Model

- Depends on posture incl. muscles
- Calculate reaction forces
- Fast calculations

Casimir/Automotive

- Detailed tissue strain
- Depend on boundary condition

• No soft tissue

• Depend on boundary conditions



Interface

Gravity only

9.189e-D1 1 D17e+D

Gravity and 80N shear force

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Interface

- Work in progress
- Geometry (Casimir)
- Material Properties (Casimir)
- Boundary conditions (Anybody)
- Mesh refinement
 - Tissue strains

Relate posture with tissue strain

Tissue strain

- Cells die due to deformation
 - What type of deformation?
 - Tissue is nearly incompressible
 - Water balloon analog



Mechanical tests of cells



Mechanical tests of cells

- Early study show similar results
- Pressure cuff around rat leg
 - Point load
 - Hydrostatic

AN EXPERIMENTAL STUDY OF SOME PRESSURE EFFECTS ON TISSUES, WITH REFERENCE TO THE BED-SORE PROBLEM

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(PLATES XCVI-C)

THE object of this investigation was to determine whether short periods of pressure might induce damage of the skin and its underlying tissues and influence their susceptibility to bacterial infection.

METHODS

Pressure effects have been studied by means of (1) a plethyamograph applied to the tail of rats and (2) a pressure oulf attached to the legs of rats and guineapigs (fig. 1). In the former experiment pressures of 100-800 mm. Hg. were applied for periods of 1-10 hours to ninety-three rats. Some of these animals were killed almost at once, others 24 hours after the release of pressure. In the second experiment pressures of 100-600 mm. Hg. were applied for 1-6 hours to the legs of 60 normal rats. Sections of the skin and underlying muscle from





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