BIOMECHANICAL MODELING TO PREVENT FOOT ULCER

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Introduction

Most foot ulcers are a consequence of a trauma (repetitive high stress, ill-fitting footwear…) associated to diabetes. They are often followed by amputation and shorten life expectancy [Reiber, 2010]. Because of somatosensory deficits, diabetic patients cannot perceive the pain announcing deep tissue injury and their natural defence is thus altered. Although several devices enhance wound healing, little has been done to prevent the ulcers, especially those starting inside the tissues at the boundary with bony prominences. We plan to assess the ulceration risk by estimating the internal strains within the patient's soft tissues through a biomechanical foot model driven by plantar pressures measured with a commercial sensor.
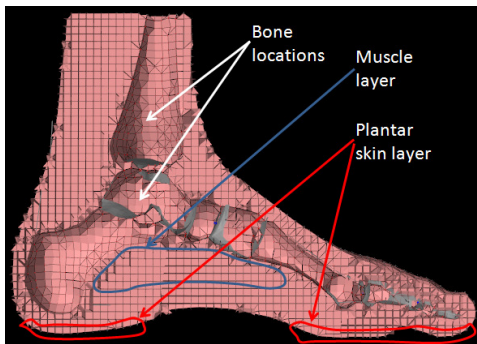

Figure 1: Cross section of the FE mesh with its different layers: skin, muscles and bones. Only the surface elements into the red areas define the plantar skin, the rest forms the soft skin layer. The other internal elements form the fat layer.

Methods

The foot model runs on the Artisynth 3D biomechanical simulation platform (artisynth.org). It integrates soft tissues, bones, joints and ligaments. The soft tissues are divided into skin, muscles and fat. They are modelled as a finite element (FE) mesh. Its outer surface is inspired by the skin surface from the zygote database (zygote.com). An automatic hexahedron-dominant FE mesh generator [Lobos, 2010] was used to mesh the soft tissues, Fig. 1, with 36,894 elements and 22,774 nodes. Muscles and fat are modelled as two Neo Hookean materials with Young moduli of 50 kPa and 4 kPa, respectively [Sofer, 2011]. The 1-element skin layer is split into two Neo Hookean materials: the plantar skin with E=6 GPa and the rest of the skin with E=200 kPa. All have a Poisson ratio of 0.495. The 26 foot bones are modelled as rigid body surfaces fixed to the nearby FE nodes. The 33 foot joints are simulated by pivots connecting each bone with its neighbours. Their angles of rotation vary from 45 degrees for the phalanx pivots to 5 degrees for the others. Three large ligaments constrain the model: the plantar fascia, the Achilles tendon, and the transversal metatarsal head ligament. They are simulated as cables with an extension stiffness of 200 MPa and a compression stiffness of 0 MPa.

Results

As pressure ulcers seem mainly induced by internal strains, we monitored their range in a given foot position. Pressures were therefore measured on a young healthy subject standing on a Zebris platform (zebris.de). While tibia and fibula remained fixed, the pressures were applied to the foot sole following a ramp. These pressures ranged from 0 to 10.5 $N.cm^{-2}$. Von Mises strains were measured below the metatarsal heads and the heel, where the maximal strains were observed ranging from 1.8 % to 4.5 % for the skin surface and from 33.4 % to 137 % for the soft tissues near the bony structures. This strain pattern realistically replicates a possible foot ulceration scenario.

Discussion

Our biomechanical model allows computing the strains on and within the foot for a given upright stance. The analysis of internal strains caused by prescribed external loads enables localization of higher deformations inside the foot and could therefore define an objective risk assessment scale. Future works will aim at linking this model with real-time data flow provided by pressure sensors to enable daily foot ulcer prevention for diabetic patient.

References

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