

NERVE EXCITATION IN THE DIABETIC FOOT: AN ANATOMICALLY BASED MODEL TO EXPLORE MECHANO-STIMULATION OF SENSORY NERVES

¹Justin Fernandez, ¹Jessica Jor, ¹Muhammad Ul Haque, ¹Marc Jacobs, ¹Peter Hunter and ¹Kumar Mithraratne
¹The Auckland Bioengineering Institute, The University of Auckland, Auckland, New Zealand.
 E-mail: j.fernandez@auckland.ac.nz

SUMMARY

The diabetic patient typically suffers from reduced foot sensation and poor circulation. This results from tissue stiffening and sensory nerve death (neuropathy). Increases in foot loading are not sensed, hence, the patient does not know to modify their foot posture to minimise foot stress. The consequences include foot ulcers, slow healing and amputation in extreme cases. External methods of stimulation can provide an alternative mechanism to stimulate the patient's foot leading to self-modification of foot posture. An anatomically-based foot model consisting of muscles, bones, soft tissue and sensory nerves was developed as a framework to explore other methods of stimulation. Data from an instrumented treadmill at mid-stance (the highest loading point during gait) was used to load the foot. The diabetic foot was modelled by stiffening the plantar soft tissue. The strain distribution that typically excites the nerves was observed throughout the tissue layers up to the bone. We hypothesized that the unloaded region under the arch could possibly be stimulated to give the subject some sensation in order to modify their movement. This model can potentially assist in the design of orthotics to aid the diabetic foot and other pathologic lower-limb conditions.

INTRODUCTION

The diabetic patient has trouble detecting increased foot pressure due to nerve death, especially under the front metatarsal region and calcaneus at the rear. We have developed a model of the foot consisting of all the bony, soft tissue and nerve structures in order to observe the actual strain patterns throughout gait to establish if there was other means of stimulating the foot. The main sensory areas are through the micro nerves of the foot sole, however, these do not perform optimally in the diabetic patient. We propose a new means of mechanically stimulating the foot by exciting the nerves under the arch. Orthotics are usually designed in order to reduce foot pressure by increasing contact area. We hypothesize that if an arch was added to stimulate the sole than this region could possibly give the sufferer some sensation of loading. Such a device would be very cost effective.

METHODS

An anatomically-based finite element model of a right foot derived from the Visible Human male [1] was created (see Figure 1). All the geometries were developed as part of the International Union of Physiological Sciences (IUPS) Physiome project, which allows for easy sharing and

dissemination throughout the scientific community [2]. The muscles, bones and soft tissue layers were digitised separately and used to fit a single continuum representation of the foot for computational efficiency. This was performed in 5 steps. (i) A foot derived from tricubic-hermite volume elements was created and fitted to the outer skin layer [3]. The foot was then morphed from a supine to a vertical position using free-form deformation. (ii) A hollow cavity in the shape of each bone was removed from the volume mesh and each individual bone mesh inserted. In this way the solid foot was coupled to each bone using contact constraints. (iii) The fibre distribution derived from each muscle was fitted to the entire foot creating a continuous fibre distribution [4]. In this way the individual muscle geometries were now replaced with a single efficient continuous mesh, which captured the muscle fibre directions. (iv) Individual fat and skin layers were defined according to diabetic tissue adapted from the work of Gefen [5]. The soft tissue was described using a hyperelastic material constitutive law [6] and the bones were treated as rigid bodies for this analysis. (v) The main neuronal branching in the foot was adapted from Grey's anatomy [7,8] from both axial and sagittal views. This was then morphed to fit the geometry of the Visible Human male. The finer nerve branching was grown using the neuronal TREES Toolbox developed for Matlab [9]. This employs the neuronal branching and organisation laws of Ramon y Cahal [10], which included conservation of nerve material and conduction time. The resulting neural network was embedded as material coordinates within the foot. The model was then solved using a static analysis and the peak ground reaction force at mid-stance (~1.1 BW) was applied through a ground plate that interacted with the sole of the foot.

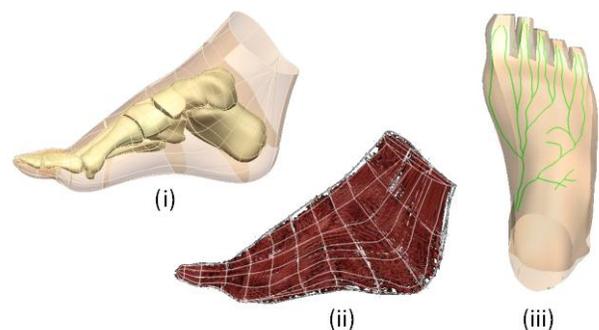


Figure 1: Geometry of foot model showing (i) skin with embedded bones; (ii) muscles fitted as a continuous fibre field; (iii) embedded medial and lateral plantar nerves.

RESULTS AND DISCUSSION

The model was first evaluated by checking the plantar pressure predicted during stance with the pressure pattern from an instrumented running shoe fitted with a pressure pad. The pressure pattern was consistent in shape and showed peaks in the correct locations.

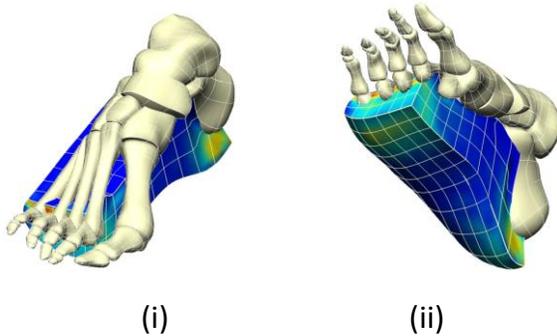


Figure 2: Von Mises strain distribution throughout the foot from the (i) oblique front and (ii) oblique sole. Dark blue is 0ϵ , light-blue is 0.15ϵ , yellow is 0.3ϵ and red is 0.45ϵ .

Figure 2 highlights two strain concentrations under the metatarsal bones and the calcaneus at the rear in line with the floor contact locations. We plotted the Von Mises (VM) strain, a scalar that accounts for all the principal components as a measure of mechano-stimulation and also a yield criteria for tissue damage. The VM strain is highest at the tissue-bone interface as expected, especially under the metatarsal region. The most important conclusion from this is that when the contact strain (and stress) is high at the sole-floor interface, the strain is up to 3 times higher within the foot at the bone interface. This means that tissue rupture and damage is likely to occur within the foot long before degeneration is noticed on the foot sole. This is consistent with the stress and strain concentrations reported by Gefen [5]. This also means that the nerves in this region would normally be stimulated in a healthy tissue, however, the diabetic sufferer would have reduced sensation. Increasing the load to stimulate the nerves would only exacerbate the problem in this region.

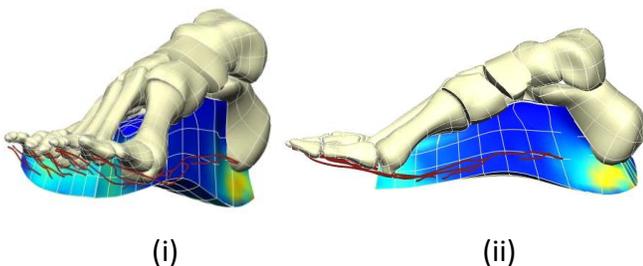


Figure 3: Von Mises strain plot from (i) oblique and (ii) side view with medial and lateral plantar nerves overlaid.

Figure 3 shows the medial and lateral plantar nerves overlaid in the foot model. Significant stress concentration exist around the nerves under the metatarsals and phalanges (toe-region), however, the region above the arch is relatively untouched. Tissue damage would not normally be felt by the diabetic patient due to neuropathy, however, any form of stimulation to indicate loading would be ideal. The arched region would be an ideal location to provide some sensation of touch to the diabetic sufferer that might trigger a response to move the foot. Nerves in this region are less likely to be damaged. This could be achieved by orthotics to stimulate the foot in this generally unloaded region. The load should not be high as the tissue in this region is generally not meant to be loaded. An orthotic with a slight convex surface could give a slight sensation when the load is high.

CONCLUSION

- An anatomically-based foot model showed that strains at the bone-cartilage interface can be up to 3 times that of the surface strains.
- The region under the arch provides a suitable region to externally induce mechano-stimulation to provide the diabetic patient with a degree of foot loading sensation.
- Foot sensation could potentially be achieved by a convex arch in an orthotic sole that does not increase the loads under the metatarsal and calcaneus regions.

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REFERENCES

1. Ackerman MJ, *Proceedings of the IEEE* **86(3)**:504-511, 1998.
2. Hunter, P.J. and T.K, Borg, *Nat Rev Mol Cell Biol* **4(3)**:237-43, 2003.
3. Fernandez JW, et al., *Biomech Model Mechanobiol.* **2(3)**:139-55, 2004.
4. Mithraratne K, et al., *IFMBE Proceedings* **31(3)**:1024-1027, 2010.
5. Gefen A, *Medical Engineering & Physics* **25**:491-499, 2003.
6. Herzog W, *Skeletal muscle mechanics: from mechanisms to function*, John Wiley & Sons. Chapter 12, 2000.
7. Gosling J. A, et al., *Lower Limb: Human anatomy: Color Atlas and Textbook fifth Edition*: pp: 28, 2008.
8. Ellis H., *The nerves of the leg and foot: Anaesthesia and Intensive Care Medicine* 8: 148-150, 2007.
9. Cuntz H, et al., *Neuroinformatics*, In press, 2010. (www.treestoolbox.org/download.html)
10. Ramon y Cajal S, *Histology of the nervous system of man and vertebrates*, Oxford University Press, 1995.