UNDERSTANDING THE BIOMECHANICS OF MEDIAL TIBIAL STRESS SYNDROME

- A SIMULATION STUDY USING A MUSCULOSKELETAL MODEL

^{1,2}Christian Gammelgaard Olesen ¹Michael Skipper Andersen, ³Michael Skovdal Rathleff, ^{1,2}Mark de Zee & ¹John Rasmussen 1 Department of Mechanical Engineering, Aalborg University, Denmark 2 Department of Health Science and Technology, Aalborg University, Denmark 3 Orthopedic Division, North Denmark Region, Aalborg Hospital - Aarhus University Hospital email: cgo@hst.aau.dk

INTRODUCTION

Foot pronation is a complex triplanar movement. Visually, it is characterized by a flattening of the Medial Longitudinal Arch (MLA) and an abduction of the calcaneus. The diagnosis Medial Tibial Stress Syndrome (MTSS) has been associated with a greater degree of foot pronation. O'Conner et al. [1] suggested that passive properties may primarily account for the increased energy absorption associated with greater foot pronation. Bouche et al. [2] have hypothesized that large foot pronation induces tension on the tibial fascia at its insertion into the medial tibial crest and this could be one of the causes of MTSS. Due to the highly complicated and invasive nature of experimental investigations of this problem, the current study suggests an analytical approach for understanding the biomechanics of the lower leg and excessive pronation of the foot.

METHODS

A musculoskeletal model of the lower extremitiy was built in the AnyBody Modeling System [3]. The model was based on cadaver data [4] and included 38 muscles that were divided into 316 muscle fascicles, based on the line-of-action. A Hill-type muscle model with passive elasticity and force-length-velocity relationships was used. The model was driven through a gait cycle with kinematic and kinetic data from a gait experiment on a healthy male (173 cm; 85 kg). For simulating different degrees of pronation, the right foot was artificially rotated about an axis (Figure 1) going from the calcaneus and through the 2nd metatarsal bone. The rotation went from 20° pronation to -5° supination, mimicking foot postures from highly pronated to slightly supinated. The simulations were run with increments of 5°. For each foot posture the muscle recruitment problem was solved and the passive force of the muscles in the deep flexor compartment was estimated.

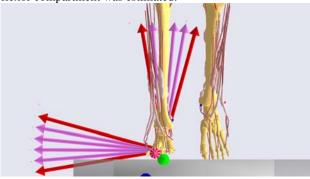


Figure 1. The foot of the AnyBody model with arrows showing the rotation around the longitudinal axis of the foot. The dark red arrows are the two outer boundaries in the simulations.

RESULTS AND DISCUSSION

When the degree of pronation is increased the m. tibialis posterior has less activity, especially between the heel strike and stance phase (Figure 2). The passive elastic forces from the muscles included in the deep flexor compartment increased as the pronation became more excessive. Figure 2 shows the passive elastic forces in the muscles as a function of the stance phase.

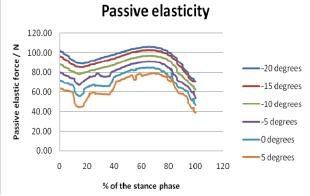


Figure 2. The passive forces of the muscles in the deep flexor compartment. -20° is a highly pronated foot and $+5^{\circ}$ is a supinated foot.

Our results corresponds well with the tibial traction theory [2]. This theory suggests that MTSS is caused by excessive traction to the tibial fascia at its insertion 2-8 cm above the medial malleolus. Our results show that excessive foot pronation causes increased forces to be transmitted to passive elastic fibers of the deep flexor compartment (tibialis posterior, flexor digitorum longus and flexor hallucis longus). We speculate that the increase seen in the passive elastic forces is because of altered moment arms of muscles controlling foot posture. This corresponds well with the model's prediction of less work being produced by tibialis posterior.

CONCLUSIONS

Based on the results found from the simulations, it can be concluded that the musculoskeletal model supports the theory suggested by Bouche et al. [2].

REFERENCES

- 1. O'Connor KM, et al. Clin Biomech (Bristol, Avon) 2004 Jan; 19(1): 71-7.
- 2. Bouche RT, et al. *J Am Podiatr Med Assoc* 2007 Jan; 97(1): 31-6.
- Damsgaard, M. et al., Simul. Model. Pract. Theory 2006 pp. 1100-1111
- Klein Horsman MD, et al. Clin BioMech (Bristol, Avon) 2007 Feb; 22(2): 239-47.