

ESTIMATION OF ANTERIOR TIBIAL TRANSLATION AND LIGAMENT LOADING IN HEALTHY AND ACL-DEFICIENT KNEES DURING WALKING

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INTRODUCTION

Knowledge of internal knee-ligament loading is important for developing better surgical procedures and rehabilitation regimens of ACL-deficient patients. A few numerical models have been used to estimate ligament loading during walking [1,2]. However these models were not driven by measured EMGs, so they might have difficulty predicting the abnormal muscle activation patterns of ACL-deficient knees.

In this paper we describe an EMG-driven model that incorporates a knee-ligament model, and we apply this approach to estimate anterior tibial translation (ATT), anterior shear forces, and ligament loading in the knee joints of an ACL-deficient subject and a healthy subject during walking. The results of the ACL-deficient gait will be compared with those of the healthy gait to explain how the ACL-deficient subject compensated for the loss of the ACL.

METHODS

One male healthy subject (mass 60.5 kg, height 1.70 m) and one male subject without an ACL (mass 74.0 kg, height 1.72 m; right leg was the affected leg) gave informed consent before participating in this study. The experimental protocol was approved by the Human Subjects Review Board of the University of Delaware. The subjects were required to finish four walking trials with right foot striking the force plate, and another four walking trials with left foot striking the force plate. EMG, joint position and force plate data were collected during the trials. EMGs were collected from nine muscles of the leg of interest using surface electrodes, including RF, VL, VM, SM, BFL, MG, LG, Sol, and TA [3]. In this study we simulated the stance

phase of both knees of the ACL-deficient subject, and the right knee of the healthy subject.

We calculated knee-ligament forces through a two-step procedure. First, an EMG-driven model [3,4] was used to estimate the muscle forces of the leg of interest to match the inverse dynamics calculated knee and ankle joint moments during stance phase of the walking trials.

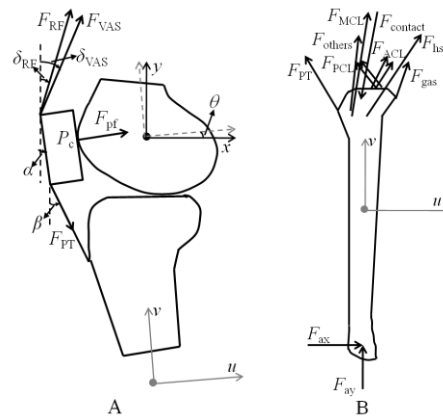


Figure 1: The knee joint model.

Second, a knee model that incorporated knee-ligaments was developed to calculate ATT and ligament loading. The model included the tibiofemoral and patellofemoral joints in the sagittal plane, and three segments: the femur, tibia and patella (Fig. 1A). The muscle forces and joint reaction forces calculated from the previous step were used as inputs. The approach used to model the patellofemoral joint was similar to that used by Liu and Maitland [2]. The contact and geometric compatibility conditions were required to be satisfied at the tibiofemoral joint. Knee ligaments were modeled as nonlinear elastic elements [1]. The ATT, knee joint contact force and ligament forces were solved through iterations until the force equilibriums of the tibia were satisfied (Fig. 1B).

RESULTS AND DISCUSSION

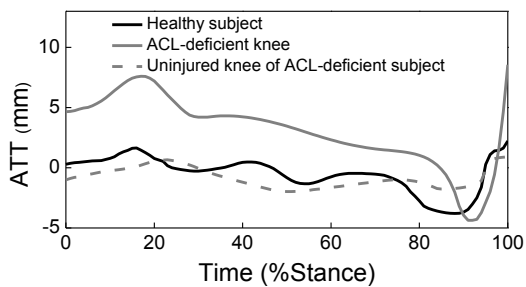


Figure 2: ATT comparison during stance phase.

The ACL-deficient knee had increased ATT compared to the contralateral uninjured side and the knee of the healthy subject (Fig. 2), and these calculated results were similar to previous findings of *in vivo* and *in vitro* experiments [5,6].

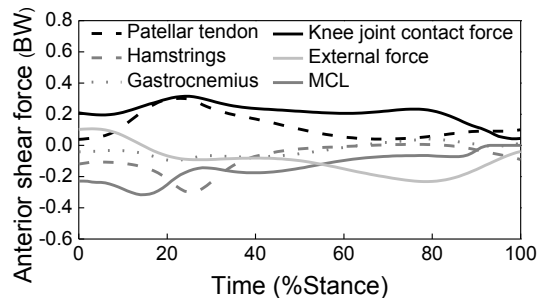


Figure 3: Anterior shear force distribution of the ACL-deficient knee.

For the ACL-deficient knee, the medial collateral ligament (MCL) was the major passive restraint of anterior shear force instead of the ruptured ACL (Fig. 3). We found strong co-contraction of the hamstrings and gastrocnemius, and these muscles functioned as active restraints to anterior shear force. However, co-contraction alone could not counteract the effect of ACL rupture, and the tibia of the ACL-deficient knee shifted more anteriorly during the stance phase.

For the healthy subject, the ACL was loaded for most of the stance phase, and the MCL, lateral collateral ligament and posterior capsule were also loaded (Fig. 4A). For the ACL-deficient subject, the MCL was loaded for most of the stance phase for the injured side (Fig. 4B), and both the ACL and MCL were loaded for the uninjured side (Fig. 4C).

CONCLUSIONS

This is the first time that an EMG-driven model has been used to estimate ligament loading in ACL-deficient patients. The approach could take into account the abnormal muscle activation strategies of ACL-deficient patients, and the results provided insights on how the patients compensated for the loss of the ACL. In future studies we will explore differences in ligament biomechanics associated with different rehabilitation protocols.

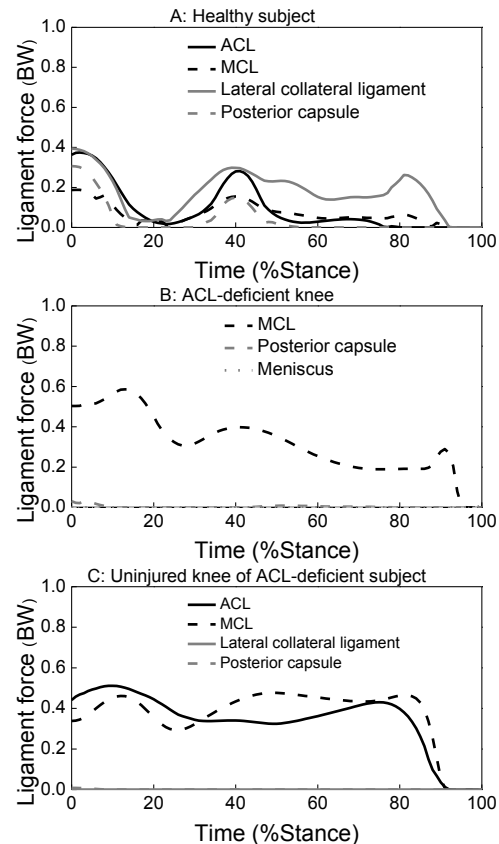


Figure 4: Knee-ligament loading comparison.

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