Mechanism of improved knee flexion after rectus femoris transfer surgery
Melanie D. Fox1, Jeffrey A. Reinbolt2, Sylvia Õunpuu3, and Scott L. Delp1,2

1Departments of Mechanical Engineering and 2Bioengineering, Stanford University, Stanford, CA, USA
3Center for Motion Analysis, Connecticut Children’s Medical Center, Hartford, CT, USA

Introduction
One of the most common gait problems in children with cerebral palsy is the inability to appropriately flex the knee during the swing phase of walking, or “stiff-knee gait” [1,2]. Rectus femoris transfer surgery is frequently performed to treat stiff-knee gait in subjects with cerebral palsy. In this surgery, the distal tendon is released from the patella and re-attached to one of several sites, such as the sartorius or the iliotibial band. Surgical outcomes vary [3,4], and the mechanisms by which the surgery improves knee flexion are unclear. It has been suggested that transferring the rectus femoris converts the muscle from a knee extensor to a knee flexor, thereby increasing knee flexion [5]. However, experimental studies have found that the muscle produces a knee extension moment after surgery [6,7], possibly due to scarring to underlying tissue [8]. The purpose of this study was to examine the mechanism by which the rectus femoris transfer surgery increases knee flexion.

Clinical Significance
It is difficult to improve surgical outcomes because the mechanism by which knee flexion increases in some patients is unknown. This study examines three types of transfer to clarify the mechanism by which the transferred muscle improves knee flexion.

Methods
Muscle-driven simulations were created of ten children diagnosed with cerebral palsy and stiff-knee gait. These simulations were altered to represent surgical transfers of the rectus femoris to the sartorius (Fig. 1b) and the iliotibial band (Fig. 1c). Rectus femoris transfers in which the muscle remained partially attached to the underlying vasti through scar tissue were also simulated by reducing the muscle’s knee extension moment (Fig. 1d).

Figure 1. Illustration of rectus femoris in each musculoskeletal model with plots of moment arms at the knee (averaged over 20 - 60 degrees of knee flexion) compared to experimental data [7,9].
Results
Simulated transfer to the sartorius, which completely converted the rectus femoris’ knee extension moment to a flexion moment, produced $32° \pm 8°$ improvement in peak knee flexion on average (Fig. 2). Simulated transfer to the iliotibial band, which eliminated the muscle’s knee extension moment but did not convert it to a flexion moment, predicted only slightly less improvement in peak knee flexion ($28° \pm 8°$). Scarred transfer simulations, which reduced the muscle’s knee extension moment, predicted significantly less ($p < 0.001$, paired t-test) improvement in peak knee flexion ($14° \pm 5°$).

![Figure 2](image)

**Figure 2.** Increases (plus one standard deviation) in peak knee flexion averages over the ten subjects for each of the simulated treatments.

Discussion
Simulated transfer to the sartorius, which converted the rectus femoris’ knee extension moment to a flexion moment, predicted only $4°$ greater knee flexion improvement on average than transfer to the iliotibial band, which eliminated the muscle’s knee extension moment. Significant improvement in knee flexion was attained even when the extension moment of the rectus femoris was reduced. These findings suggest that the primary mechanism for improvement in knee flexion after surgery is reduction of the muscle’s knee extension moment, rather than conversion to a knee flexion moment. Scarred transfer simulations resulted in an average peak knee flexion improvement ($14° \pm 5°$) comparable to the average increase in knee flexion range of motion ($13° \pm 11°$) in the subjects’ postoperative data. Simulated non-scarred transfers to the iliotibial band and the sartorius predicted greater improvements in knee flexion than the scarred transfer simulations, suggesting methods to reduce scarring may further augment knee flexion.

References

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