MUSCLE ACTIVATIONS TO STABILISE THE KNEE IN PATHOLOGICAL GAIT

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INTRODUCTION
Mechanical joint loading is believed to contribute to the development of osteoarthritis (OA) (Radin, 1982). In particular, larger than normal knee adduction moments during the stance phase of gait have been shown to accelerate progression of tibiofemoral (TF) OA (Miyazaki et al., 2002). Larger knee adduction moments may also require increased muscular activity to maintain joint stability and prevent ‘condylar lift off’ (Schipplein & Andriacchi, 1991).

Previous work has shown two main muscle activation strategies for supporting frontal plane moments at the knee. One of these strategies involves selected activation of muscles that have moment arms to support the external load (Lloyd & Buchanan, 1991). For example, an increase in m. tensor fascia latae would support the external adduction moment placed on the knee during the stance phase of gait. The quadriceps muscles can also be selectively activated to counter external adduction and abduction moments, by virtue of their insertion into the tibial tuberosity (see Figure 1). The second activation strategy is a general co-contraction of knee flexors and extensors (Lloyd & Buchanan, 2001, Schipplein & Andriacchi, 1991). This general activation pattern effectively ‘stiffens’ the joint and provides stability against any external load. Both these muscle activation strategies have been shown to occur during running and cutting manoeuvres in a young healthy population to counter large loads in the frontal and transverse plane of the knee (Besier et al., 2003). However, activation patterns to counter frontal plane knee moments during pathological gait have not previously been investigated.

The aim of this study was to investigate the contribution of muscle activation patterns to pathological gait patterns in a population at increased risk of knee OA. It was hypothesised that APM subjects would have altered muscle activation patterns compared to a group of ‘injury free’ subjects, and that these changes would be associated with larger-than-normal knee adduction moments.

METHODS
Three-dimensional gait analysis, using a 50 Hz VICON motion analysis system, was performed on 107 patients who had recently undergone APM, and 49 healthy controls (CON), walking at a freely-chosen velocity. Subjects underwent knee strength testing on a Biodex isokinetic dynamometer. Subjects were categorised with ‘Weak’ knee extension strength if they were less than one standard deviation below the mean of CON knee extension strength. During gait and strength testing, EMG data were synchronously captured at 2000 Hz from 10 major muscles crossing the knee (Lloyd and Buchanan, 2001). Muscle activations were full wave rectified then low pass filtered before normalization to a maximum voluntary contraction. Mean muscle activation for quadriceps and hamstring muscles were calculated over the stance phase of gait as well as the net muscle activity from all muscles. Data were statistically tested using two-way repeated measures ANOVA, Bonferroni corrected for multiple comparisons and a p value set at 0.05.

Our concurrent paper has shown that patients who have undergone an arthroscopic partial meniscectomy (APM) display greater knee adduction moments during the stance phase of gait than a control group. Knee flexion/extension moments, however, were found to be similar between the two groups. The APM group also displayed weaker knee extension strength compared to their age-matched controls. Furthermore, those APM subjects with weak knee extension strength had greater knee adduction moments compared to APM subjects with normal knee extension strength (Figure 2). Muscle activation patterns at the knee are expected to reflect these differences in joint moment requirements during gait.

![Figure 1: Support of both adduction AND abduction moments at the knee by the quadriceps muscle.](image)

![Figure 2: Stance phase knee adduction moments in subjects with “Normal” and “Weak” knee extension strength](image)

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Weak APM subjects walked with greater net muscle activity over the stance phase of gait compared to APM subjects with normal knee extension strength ($p<0.05$). The weak APM subject’s mean hamstrings activity was 187% of normal APM, while quadriceps activity was 124% of the normal APM group. This increased co-contraction of quadriceps and hamstring muscles during gait is believed to be a strategy for stabilising the larger-than-normal adduction moments found in this subgroup.

Subjects with weak knee extension strength also had reduced knee flexion during the weight acceptance phase of gait. This extended knee posture improves the moment arms of hamstring muscles to support adduction moments, particularly when coupled with an increase in activation. Increased activation of hamstrings in the APM group may result in increased TF joint contact forces, predisposing APM populations to degenerative joint disease. Furthermore, APM subjects with weak knee extension strength might be at elevated risk of TFOA due to increased co-contraction of quadriceps and hamstring muscles, and the subsequent increase in joint contact forces.

Testing these hypotheses requires knowledge of the articular forces during gait. Therefore, an EMG-driven musculoskeletal model of the knee is currently being used to estimate individual muscle forces and the resultant TF joint contact forces. Preliminary data from this model will be presented.

These findings suggest that APM patients with weak knee extension strength have increased co-contraction of quadriceps and hamstrings with a more extended knee posture. The increased co-contraction is hypothesised to support the larger-than-normal knee adduction moments at the detriment of increased articular load. The role of muscle activity in increasing articular load requires further investigation via computer simulation.

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**REFERENCES**


