ACTIVE AND PASSIVE KNEE STABILITY IN PATIENTS WITH SEVERE OA

1 Gregory Freisinger, 1 Erin Hutter, 1 Jackie Lewis, 1 Rachel Baker, 1 Jeffrey Granger, 2 Matthew Beal, 1 Xueliang Pan, 1 Laura Schmitt, 1 Robert Siston and 1 Ajit Chaudhari

1 The Ohio State University, Columbus, OH, USA
2 Northwestern University, Evanston, IL, USA
email: gregory.freisinger@gmail.com, web: http://u.osu.edu/osusportsbiomechanics/

INTRODUCTION

Osteoarthritis (OA) is the largest single cause of disability in the United States, and the knee joint is most frequently affected [1]. Increased knee laxity has been reported in knee OA patients compared to controls [2], and additional active knee stability, by increased muscle activation or co-contraction of antagonist muscles, has been hypothesized to assist in knee stabilization [3]. Muscle guarding may reduce the magnitude of measured laxity, and it is currently unknown how large of an effect this plays in identifying a relationship to active stability.

The primary purpose of this study was to identify the association between passive knee laxity, measured when each participant is under general anesthesia, and contributors to active knee stability in participants with severe OA. We hypothesized that increased passive knee laxity would be associated with increased quadriceps muscle activity and co-contraction indices during gait.

METHODS

Twenty individuals (22 total knees: 7 male / 15 female, age=59.0 ± 7.3 y, ht=1.68 ± 0.11 m, mass=93.5 ± 15.3 kg, BMI=33.3 ± 4.5 kg/m²) completed this study after providing IRB approved consent. Participants had predominantly medial compartment tibiofemoral OA and were awaiting total knee arthroplasty (TKA). Prior to surgery, each participant completed gait analysis in Ohio State’s Movement Analysis and Performance Lab. Participants walked along a 10 m path at a self-selected speed (0.98 ± 0.26 m/s). Marker data were collected at 150 Hz utilizing 10 motion-capture cameras (MX-F40; Vicon; Oxford, UK). A modified point-cluster technique marker set was used in conjunction with a functional hip joint center to calculate lower extremity kinematics.

Wireless surface electromyography (EMG) (Telemyo DTS; Noraxon USA, Inc; Scottsdale, AZ) was recorded at 1500 Hz for specific lower extremity muscles that cross the knee joint on the involved limb. Three separate, unweighted tasks were used as submaximal reference activities to normalize EMG signals. Seated uni-lateral knee extension was used to normalize the rectus femoris (RF), vastus medialis (VM), and vastus lateralis (VL). Standing uni-lateral knee flexion was used to normalize semimembranosus (SM) and biceps femoris (BF). Standing bi-lateral ankle plantarflexion was used to normalize medial and lateral gastrocnemius (MG & LG). The highest 500ms running average of EMG for each muscle was used for normalization.

The average quadriceps activation (avgQUAD) and average co-contraction indices (avgCCI) were used as active stability measures for their potential to stabilize the knee joint during gait. avgQUAD was found by calculating the mean of RF, VM, and VL muscle activity and avgCCIs were calculated using Equation 1 [4] for the following antagonist muscle pairs: quadriceps and hamstring (QH); quadriceps and gastrocnemii (QG); VM and SM (MQH); VL and BF (LQH); VM and MG (MQG); and LV and LG (LQG).

Equation 1. Average co-contraction index (avgCCI) was calculated using the relatively lower and higher EMG signals for antagonistic muscles.
avgQUAD and avgCCIs were calculated over the weight acceptance phase of gait (WA), which was defined as the time period from initial heel contact to peak knee flexion. Four trials of gait were used to calculate ensemble averages for the muscle activations during WA. Selected muscle activation values from specific participants were dropped from further analysis due to poor signal quality, so the number of participants used are shown in Table 1.

Passive varus-valgus knee laxity was measured intra-operatively using a custom navigation system and knee stability device [5] in the osteoarthritic knee before TKA. Laxity was calculated as the difference in varus-valgus knee angle when the knee was loaded with 10 Nm of varus and valgus moment, respectively. Each participant’s overall varus-valgus laxity was the average value found during three trials in the operating room.

Non-parametric statistics were chosen after an initial inspection of the data revealed 4 of the 7 EMG variables were not normally distributed (Anderson-Darling test; p<0.05). Spearman’s rank order correlations were calculated for varus-valgus laxity and EMG variables of interest. A p-value <0.05 was used to indicate statistical significance.

RESULTS AND DISCUSSION

No association was observed between passive varus-valgus laxity and any active stability measure during the WA phase of gait. The distribution of passive varus-valgus laxity, avgQUAD activation and avgCCIs are reported in Table 1.

Quadriceps force can create a moment to counteract varus-valgus rotation, as a result of the bicondylar nature of the knee joint and attachment point of the quadriceps on the tibia; however this was not related to varus-valgus laxity in this cohort. While no CCI was related to laxity, LQH was significantly larger than MQH (Mann-Whitney U test, p=0.005). This may be a neuromuscular strategy to stabilize the adduction moment during weight acceptance, and prevent the lateral side of the femur from lifting off the tibia [3].

CONCLUSIONS

The primary purpose of this study was to identify the relationship between passive knee laxity and contributors to active knee stability in participants with severe OA. Contrary to our hypothesis, we found no associations between passive varus-valgus laxity and average quadriceps activity or any of the co-contraction indices during gait. Stability of the knee joint necessitates a complex control strategy; further study is needed to understand the contributions of passive and active structures in osteoarthritic populations.

REFERENCES


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