Introduction: The principal objectives of performing a total knee replacement (TKR) are to relieve pain and to restore normal knee joint function. TKRs consistently reduce knee pain [1]; however abnormalities in functional performance often remain postoperatively [2]. These abnormalities have been attributed to compensatory biomechanics due to pain experienced pre-operatively [3], absence of the anterior cruciate ligament [4], and muscle co-contraction of the knee joint musculature [2]. Particularly, increased antagonistic muscle co-contraction, the simultaneous recruitment of muscles that produce moments in opposite directions around the joint [5], has a substantial influence on the movement patterns of the knee during gait. Information on TKR lower limb muscle activation and quantified co-contraction during gait is limited.

The purposes of the present study were to investigate lower limb muscle activation and quantify co-contraction differences among TKR patients and asymptomatic controls. It was postulated that both muscle activity and time domains would differ between the subject groups and higher indices of co-contraction between the agonistic and antagonistic musculature in the TKR patients would be present.

Methods: Nine males and 10 females (mean age 61±7 years; mean height 1.7±0m; mean weight 78±10kg; mean BMI 30±7kg/m²) were recruited based on original diagnosis of primary degenerative osteoarthritis and no history of neurological disorders or significant lumbar spine disease. All TKR patients received a posterior cruciate retaining model (NexGen, Zimmer, Warsaw, IN) and had an average time in situ of 38±27mo. A group of healthy age-, height-, and weight-matched control subjects, with asymptomatic radiographic knees, were enrolled for comparison. Nine males and 10 females (mean age 56±7 years; mean height 1.7±0m; mean weight 78±10kg; mean BMI 28±5kg/m²) participated. Subjects were prepared utilizing standardized protocols for motion capture and surface electromyography (sEMG) analysis. A multi-component force plate, embedded in a level walkway, identified heel strike and determined walking velocity events for time normalization. Muscle activity was collected using a transmitter and receiver (Noraxon Inc, Scottsdale, AZ) at a rate of 1200 Hz from eight major muscles: tensor fasciae lata (TFL), vastus medialis (VM), rectus femoris (RF), vastus lateralis (VL), biceps femoris (BF), semimembranosus / semitendinosus (SM/ST), medial gastrocnemius (MG), and lateral gastrocnemius (LG). Self-adhesive dual electrodes were placed along the muscle fibers over the bellies of the muscles. The skin was cleaned using antimicrobial wipes to reduce inter-electrode impedance. One trial from each TKR subject was selected for analysis based on the self-selected walking velocity which provided the closest match to the antagonist control group. The raw sEMG signal for each subject and each muscle was filtered, rectified, and smoothed. Signal normalization was accomplished by using the dynamic reference voluntary contraction (RVC) method where the normative value represented the highest attainable magnitude of the muscle signal (100% RVC activation). Muscle “on” and “off” timing was obtained from the burst durations and dynamic %RVC values. The muscle co-contraction index (CCI) was computed using the equation below for pairs of agonistic and antagonistic muscles [6]. The CCI provided a measure of the relative activation of the muscle pairs at each instance of the gait cycle. The CCI was averaged over the entire gait cycle for each muscle combination and independent sample t-tests were applied to test for the relative activation of the muscle pairs at each instance of the gait cycle agonistic and antagonistic muscles [6]. The CCI pro vided a measure of index (CCI) was computed using the equation below for pairs of burst durations and dynamic %RVC values. The muscle co-contraction RVC activation). Muscle “on” and “off” timing was obtained from the voluntary contraction (RVC) method where the normative value based on the self-selected walking velocity which provided the closest time

Results: Muscle activation patterns of asymptomatic control subjects were in agreement with the literature [2]. Muscle patterns of the TKR patients, however, showed prolonged activity (Figure 1). In particular, the quadriceps and hamstring muscles activity was extended 1.2 and 1.4 times longer, respectively, compared to the matched control subjects. The TKR patients had greater CCI compared to the control subjects, particularly during 30-70% of the gait cycle where prolonged muscle activation was observed (Figure 2). There were significant differences for all muscle combinations when comparing the mean CCI for the TKR and control subjects (P < 0.001, Figure 3).

Discussion: The results demonstrated that the TKR patients utilized different muscle activation patterns during gait compared to matched control subjects. Since neither pain nor severe quadriceps weakness were present in the tested TKR population, it is suggested that increased muscle co-contraction around the knee joint accounted for a compensatory mechanism aimed at providing greater control of knee kinematics during gait. With increased muscle co-contraction, stiffening of the knee joint occurs together with increased contact forces [7]. This could be potentially harmful for the polyethylene inlay of the prosthesis device. Numerical contact force modeling can profit from this new insight. For example, high CCI were present during mid-stance, a time with relatively low external knee moments. Numerical models using equilibrium equations may underestimate contact forces at this time if they do not account for increased antagonist muscle activity.


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