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Dynamic joint stiffness and co-contraction in subjects after total knee arthroplasty

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ABSTRACT

Background: Although total knee arthroplasty reduces pain and improves function, patients continue to walk with asymmetrical movement patterns, that may affect muscle activation and joint loading patterns. The purpose of this study was to evaluate the specific biomechanical abnormalities that persist after total knee arthroplasty and examine the neuromuscular mechanisms that may contribute to these asymmetries.
Methods: Dynamic joint stiffness at the hip, knee and ankle, as well as co-contraction at the knee and ankle, were compared between the operated and non-operated limbs of 32 subjects who underwent total knee arthroplasty and 21 subjects without lower extremity impairment.
Findings: Subjects after total knee arthroplasty demonstrated higher dynamic joint stiffness in the operated knee compared to the non-operated knee (0.056 (0.023) Nm/kg/m/deg vs. 0.043 (0.016) Nm/kg/m/deg, $P=0.003$) and the knees from a control group without lower extremity pathology (controls: 0.042 (0.015) Nm/kg/m/deg, $P=0.017$). No differences were found between limbs or groups for dynamic joint stiffness at the hip or ankle. There was no relationship between dynamic joint stiffness at the knee and ankle and the amount of co-contraction between antagonistic muscles at those joints.
Interpretation: Patients after total knee arthroplasty walk with less knee joint excursion and greater knee stiffness, although no differences were found between groups for stiffness at the hip or ankle. Mechanisms other than co-contraction are likely the underlying cause of the altered knee mechanics. These findings are clinically relevant because the goal should be to create interventions to reduce these abnormalities and increase function.

1. Introduction

Osteoarthritis (OA) is a degenerative disease that most often occurs in the knee and causes substantial pain, decreased range of motion and reduced functional performance (Jacobs et al., 2009). Although OA is classically described as degeneration of the articular cartilage within the joint, knee OA is also associated with muscle weakness (Becker et al., 2004), joint instability (Fitzgerald et al., 2004), loss of proprioception (Hassan et al., 2002), altered muscle coordination patterns (Zeni et al., 2009) and abnormal kinetics and kinematics in the affected and adjacent joints (Briem and Snyder-Mackler, 2009; Zeni and Higginson, 2009; Zeni and Higginson, 2011). Knee replacements are the most common surgical treatment for end-stage OA. Although patients typically report reduced pain and improved functional performance after surgery (Petterson et al., 2009), biomechanical asymmetries (Farquhar et al., 2008) and muscle weakness (Valtonen et al., 2009) are not concomitantly resolved.

Asymmetrical movement patterns adopted in the presence of pain and weakness associated with OA persist one year after total knee arthroplasty (TKA) (Farquhar et al., 2008). These gait patterns are characterized by reduced stance time, reduced knee joint excursions and reduced use of the quadriceps to attenuate the rate of force development during loading response (Yoshida et al., 2008). These gait patterns are typically associated with the 'stiff-legged' or 'quadriceps avoidance' gait patterns that are seen in patients with OA who present with increased joint laxity and increased co-contraction of antagonistic muscles (Rudolph et al., 2007; Schmitt and Rudolph, 2007). In a study of patients with knee OA, Fitzgerald et al. (2004) found that 63% of their subjects reported knee instability (the knee buckling or 'giving way') and that 44% of these subjects reported that it affected their ability to function. This perception of joint instability persists after TKA, which can have negative implications for overall function and joint health (Barsoum et al., 2011; Lo et al., 2010). In patients with OA, increased antagonistic muscle activity may be used to increase the stability of the joint (Zeni et al., 2009), some studies suggest that patients after TKA may use a similar strategy (Benedetti et al., 2003). Although this increase in co-contraction may help alleviate the perception of instability, it can also increase the compressive load experienced by the lower extremity (Lu et al., 1997). These gait patterns may be especially detrimental to the prosthesis as higher rates of force development and higher peak forces may play a role in the breakdown of the prosthetic components.

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Although gait asymmetries and biomechanical alterations exist after TKA, the mechanisms underlying these abnormalities are not well understood and the timecourse of their development and resolution is not known. Therefore the purpose of this study was to evaluate the specific biomechanical abnormalities that persist in the knee, ankle, and hip after TKA and examine the mechanisms that contribute to these persistent asymmetries. We hypothesized that subjects 6 months after TKA would demonstrate greater stiffness in the operated knee and this would be related to greater co-contraction of the muscles surrounding the joint.

2. Methods

2.1. Subjects

A total of 53 subjects participated in this cross-sectional study. Thirty-two subjects 6 months after unilateral TKA for osteoarthritis and 21 healthy adults with no reported knee pain were included (Table 1). Subjects were excluded if they had a self-reported pain greater than or equal to 4 out of 10 in the non-operated limb, neurological or vascular problems that interfered with their ability to perform the ascribed tasks, diabetes that impaired lower extremity sensation, or were currently receiving treatment for cancer. All subjects in the TKA group underwent rehabilitation at one of our community clinics where the standard of care is a progressive rehabilitation paradigm that focuses on normalizing strength, range of motion and functional ability. Modalities are used to control pain and swelling. This work was approved by the appropriate institutional review board, and all subjects signed an informed consent prior to participation.

2.2. Motion analysis

Joint kinematics and kinetics during gait were measured using an 8-camera motion capture system (VICON, Oxford Metrics Ltd., London, UK) synchronized with two force plates (Bertec Corporation, Worthington, OH, USA). Spherical retro-reflective markers were placed bilaterally on iliac crest, greater trochanter, lateral femoral condyle, lateral malleolus, head of the 5th metatarsal, and 2 markers on the heel. Rigid thermoplastic shells with 4 markers were secured on the lower leg and thigh bilaterally. The pelvic motion was tracked using a rigid thermoplastic shell with 3 markers. Motion data was collected at 120 Hz and analog data from the force plate was sampled at 1080 Hz. Subjects walked at a self-selected pace. Five walking trials were collected and the average of these trials was used in the analysis. Marker trajectories were low pass filtered at 6 Hz, and force platform data were filtered at 40 Hz using a second-order phase-corrected butterworth filter. Joint angles were calculated using Euler X–Y–Z sequence corresponding to flexion/extension, abduction/adduction, and then rotation sequences. Joint moments were calculated using 3D inverse dynamics and were normalized to subject height and weight using Visual 3D software (C-motion, Germantown, MD, USA). The time points between consecutive heel strikes were normalized to 101 points. Heel strike events were determined when the vertical ground reaction force crossed a threshold of 20 N. Joint excursions and peak joint moments were calculated for the operated and non-operated limbs for all subjects. "Operated" limbs in the control subjects were randomly selected between left and right limbs, and matched to the percentage left and right knee replacements in our TKA groups.

Table 1
Subject characteristics, mean (SD). * P<0.05.

	TKA	Control	*P*-value
Sex (M/F)	20/12	12/11	
Age (years)	69.9 (7.9)	62.7 (6.6)	
Knee Outcome Survey (%)	87 (9)	99 (1)	<0.001*
Walking speed (m/s)	1.26 (0.17)	1.44 (0.15)	<0.001*
Height (m)	1.72 (0.10)	1.73 (0.11)	0.818
Weight (kg)	92.7 (21.4)	85.8 (12.5)	0.177
Body mass index (kg/m^2)	31.3 (6.1)	29.0 (4.8)	0.147
Extension ROM Op. Limb (°)	0 (5)	−1 (3)	0.427
Flexion ROM Non-Op. Limb (°)	119 (8)	134 (6)	<0.001*
MVIC Op. Limb (N/BMI)	18.3 (6.6)	30.0 (10.4)	<0.001*
MVIC Non-Op. Limb (N/BMI)	21.9 (8.8)	27.9 (8.8)	0.015*

2.3. Dynamic joint stiffness

To evaluate the biomechanical stiffness of the limb during gait, we calculated dynamic joint stiffness (DJS) of each limb and joint. Dynamic joint stiffness was defined as the change in moment (M) divided by the change in angle (θ):

$$\mathrm{DJS} = \frac{\Delta M}{\Delta \theta}. \tag{2.1}$$

The joint moment was plotted against the knee angle and a linear fit of the slope was determined to be the joint stiffness. Knee stiffness was calculated during weight acceptance, which was determined to be the linear region in which the average external knee flexion moment started to increase and ended with peak knee flexion (Zeni and Higginson, 2009). Ankle stiffness was calculated during stance in the linear region that began with maximum plantarflexion and ended at maximum dorsiflexion. Hip stiffness was calculated during stance from minimum to maximum hip flexion. These phases encompass important components of the stance phase: weight acceptance for the knee and the end of weight acceptance to push-off for the hip and ankle. Excursions and joint moments for the hip, knee and ankle were also analyzed during the same periods of time used to calculate hip, knee and ankle stiffness, respectively.

2.4. Electromyography

Electromyographic (EMG) data was collected bilaterally for each subject using active surface electrodes (Motion Lab Systems, Baton Rouge, LA, USA). The skin was cleaned with alcohol prior to electrode placement. Electrodes were placed on the following eight muscles on each limb: gluteus medius, lateral hamstring, vastus lateralis, vastus medialis, tibialis anterior, soleus, and the medial and lateral heads of the gastrocnemius. Prior to walking, maximum volitional isometric contractions (MVIC) were performed to determine the maximum levels of voluntary contraction. For knee extension (vastus lateralis), the subject sat with his or her leg flexed to 75° and performed an isometric contraction with a leg cuff that was secured to the table with metal chains. For hamstring testing, the subjects stood supported at a table and were asked to flex their knee while the investigator applied opposing force to resist knee flexion. MVICs for different muscle groups were recorded in separate trials, with each trial containing 1–2 s in which the muscle is not active for normalization to resting levels.

The EMG signal was pre-amplified at the skin and sampled at a rate of 1080 Hz. Visual 3D software (C-motion, Germantown, MD, USA) was used to filter the signals using a low pass filter at 350 Hz. A linear envelope was created on the absolute value of the raw EMG signal using a phase-corrected low-pass Butterworth filter with a cut-off of 20 Hz. Data were normalized to the maximum signal obtained during MVIC. All EMG data were visually inspected prior to analysis. EMG signals that were not usable were excluded from the analysis. EMG data were excluded if the signal was excessively noisy, demonstrated excessive motion artifact (large periods of low frequency signal), or the signal was clipped during the dynamic trials.