Journal of Biomechanics ∎ (∎∎∎∎) ∎∎∎–∎∎∎

Contents lists available at ScienceDirect

Journal of Biomechanics

journal homepage: www.elsevier.com/locate/jbiomech
www.JBiomech.com

How human gait responds to muscle impairment in total knee arthroplasty patients: Muscular compensations and articular perturbations

Marzieh M. Ardestani [a,b,*], Mehran Moazen [c]

[a] Department of Orthopedic Surgery, Rush University Medical Center,1611 W. Harrison St., Suite 204, Chicago, IL 60612,USA
[b] State Key Laboratory for Manufacturing System Engineering, School of Mechanical Engineering, Xi'an Jiaotong University, Xi'an, China
[c] Department of Mechanical Engineering, University College London, Torrington Place, London WC1E 7JE, UK

ARTICLE INFO

Article history:
Accepted 25 March 2016

Keywords:
Human gait
Total knee arthroplasty
Rehabilitation
Muscle weakness
Joint force
Multi-body dynamics

ABSTRACT

Post-surgical muscle weakness is prevalent among patients who undergo total knee arthroplasty (TKA). We conducted a probabilistic multi-body dynamics (MBD) to determine whether and to what extent habitual gait patterns of TKA patients may accommodate strength deficits in lower extremity muscles. We analyzed muscular and articular compensations in response to various muscle impairments, and the minimum muscle strength requirements needed to preserve TKA gait patterns in its habitual status.

Muscle weakness was simulated by reducing the strength parameter of muscle models in MBD analysis. Using impaired models, muscle and joint forces were calculated and compared versus those from baseline gait i.e. TKA habitual gait before simulating muscle weakness. Comparisons were conducted using a relatively new statistical approach for the evaluation of gait waveforms, i.e. Spatial Parameter Mapping (SPM). Principal component analysis was then conducted on the MBD results to quantify the sensitivity of every joint force component to individual muscle impairment.

The results of this study contain clinically important, although preliminary, suggestions. Our findings suggested that: (1) hip flexor and ankle plantar flexor muscles compensated for hip extensor weakness; (2) hip extensor, hip adductor and ankle plantar flexor muscles compensated for hip flexor weakness; (3) hip and knee flexor muscles responded to hip abductor weakness; (4) knee flexor and hip abductor balanced hip adductor impairment; and (5) knee extensor and knee flexor weakness were compensated by hip extensor and hip flexor muscles. Future clinical studies are required to validate the results of this computational study.

1. Introduction

Remarkable functional improvement and pain relief have been reported following total knee arthroplasty (TKA-(Da Silva et al., 2014)). However, various factors such as joint instability (Yercan et al., 2005), muscle impairments (Schache et al., 2014; Yoshida et al., 2013) and pre-surgical gait adaptations (Ouellet and Moffet, 2002) often prevent patients to restore a "normal" gait pattern after surgery. Muscular impairment (i.e. strength decline) occurs frequently following TKA and may persist long after surgery (Bjerke et al., 2014; Davidson et al., 2013; Thomas et al., 2014; Yoshida et al., 2013; Farquhar et al., 2009). Recent studies have reported 50–60% strength decline in hamstring and quadriceps (Judd et al., 2012; Stevens-Lapsley et al., 2010) that may persist up to three years after surgery (Schache et al., 2014).

A subtle weakness in an individual muscle can be compensated by additional contribution of other muscles (Goldberg and Neptune, 2007). However, severe muscle impairments, such as post-operative muscle deficits in TKA patients, may not be easily addressed by other muscles. As a matter of fact, patients will adapt to "*kinematic*" compensations so as to offload the impaired muscles. Quadriceps avoidance (Andriacchi, 1993) or knee stiffening (Benedetti et al., 2003) strategies are examples of such kinematic adaptations. The existent body of literature is rich with studies describing the abnormal gait characteristics of TKA patients compared to non-injured population (Alnahdi et al., 2011; Hatfield et al., 2011; Mcclelland et al., 2010; Yoshida et al., 2012). However, there are still various questions remaining on TKA patient gait patterns; e.g. how vulnerable the TKA habitual gait pattern is to any muscle impairment before kinematic adaptation may be demanded? and how muscle impairment may influence muscle

* Corresponding author at: Department of Orthopedic Surgery, Rush University Medical Center, 1611 W. Harrison St., Suite 204, Chicago, IL 60612, USA.
E-mail address: mostafavizadeh@yahoo.com (M.M. Ardestani).

http://dx.doi.org/10.1016/j.jbiomech.2016.03.047
0021-9290/

and joint forces? While such questions have been investigated for non-injured subjects (Goldberg and Neptune, 2007; Thompson et al., 2013; Valente et al., 2013; Der Krogt et al., 2012), previous findings cannot be easily extrapolated to TKA subjects.

Beside, comprehensive investigation of all potential muscle impairments and their consequences on muscle and joint forces are currently lacking from literature as most previous studies simulated the weakness of only one (Thompson et al., 2013; Valente et al., 2013) or a few muscles (Knarr et al., 2013; Steele et al., 2012; Der Krogt et al., 2012). Also, from a technical point of view, previous studies documented muscular compensations in terms of scalar gait features (defined at discrete time points); e.g. "magnitudes" of muscle forces. Such an abstraction can oversimplify the complex gait waveforms and the underlying dynamic information. Therefore, a more holistic understanding of the muscular compensations throughout the entire gait cycle is required.

The overall aim of this study was to understand how TKA gait responds to muscle weakness. In particular, this study aimed to (1) quantify the minimum muscle strength requirements to execute habitual gait strategy (i.e. baseline gait), (2) identify the muscular compensations and joint force perturbations in response to an impaired muscle group and (3) quantify the sensitivity of joint forces due to weakness of various individual muscles. A probabilistic multi-body dynamic (MBD) approach was combined with statistical parameter mapping (SPM) and Principal component analysis (PCA) to address the aforementioned technical shortcomings of previous studies. It should be pointed out that although TKA gait strategies contain some adaptations compared to non-injured counterpart; TKA habitual gait status is referred to "baseline" gait for the present study to imply the gait pattern *before* simulating muscle weakness in the musculoskeletal model.

2. Methodology

Experimental gait measurements of six TKA patients were adopted from a published repository (Section 2.1). Three sets of MBD simulations were conducted: The first set of MBD simulations was called "baseline simulation" calculating the habitual muscle and joint forces for every subject (Section 2.2). Second, individual muscles were systematically weakened until the baseline gait could no longer be executed by the musculoskeletal model unless by means of remarkable kinematic changes. From this set of simulations, the "minimum strength requirements" were determined (Section 2.3). Third, muscles were impaired randomly by sampling their strength parameters in muscle models between the "minimum requirements" and their "nominal" values from the baseline simulation. Once again, muscle and joint forces were calculated using the impaired musculoskeletal models (Section 2.3). Using SPM analysis, muscle and joint forces from impaired simulations were compared versus those obtained from baseline simulations (Section 2.4). PCA was then used to quantify the sensitivity of joint forces due to the weakness of each individual muscle (Section 2.5).

2.1. Experimental gait data

Gait data, i.e. ground reaction forces (GRF) and marker trajectories, from six TKA patients (five males, one female; height: 170.8 ± 5.2 cm; mass: 69.7 ± 4.4 kg), walking at self-selected pace, were adopted from a published repository (⟨https://simtk.org/home/kneeloads⟩, accessed Sept 2014). These patients were implanted with sensor-based knee prostheses that could measure in vivo knee forces. GRFs were measured at a frequency of 1000 Hz (Force plate, AMTI Corp., Watertown, MA, USA) and marker trajectory data were recorded at a frequency of 200 Hz (10-camera motion capture system, Motion Analysis Corp., Santa Rosa, CA, USA) using a modified Cleveland Clinic marker set with extra markers on the feet and trunk. Electromyography (EMG) signals were recorded at a frequency of 1000 Hz (Surface electrodes, Delsys Corp., Boston, MA, USA) for several muscle groups including: semimembranosus, biceps femoris long head, vastus medialis, vastus lateralis, rectus femoris, medial gastrocnemius, lateral gastrocnemius, and tensor fascia latae. For a complete description of this database see (Fregly et al., 2012; Kinney et al., 2013). Experimental EMG measurements were band-pass filtered with a 6th order Butterworth within the frequency of 20–420 Hz. Root mean square (RMS) was computed within 30 msec intervals with 15 msec overlap. The magnitudes of EMG measurements for every subject were normalized to the corresponding maximum values over all his/her gait trials. The average of normalized RMS computations were then compared versus those computed by MBD analysis for validation purposes.

2.2. Multi-body dynamic analysis

2.2.1. Musculoskeletal model

A 3D musculoskeletal model, based on the University of Twente Lower Extremity Model (TLEM -(Klein Horsman, 2007), was recruited in the multi-body simulation software, AnyBody Modeling System (version 5.2, AnyBody Technology, Aalborg, Denmark). In brief, the model included trunk, pelvis, thigh, shank and foot segments (Fig. 1). Hip joint was modeled with three degrees of freedom (DOF) while knee joint was modeled as a hinge joint with only one DOF for flexion-extension and universal joint was considered for ankle-subtalar complex. TLEM model had 160 Hill-type muscle-tendon actuators and the strength of each muscle was modeled as follows (AnyBody Modeling System, User's Guide):

$$\text{Strength} = F_0\left(2\frac{Lm}{\overline{Lf}}-1\right)\left(1-\frac{Lm'}{V0}\right) \quad (1)$$

Where F_0 is the strength of the muscle at neutral fiber length ($\overline{Lf}$) and contraction velocity (L_m') equals to zero. L_m is the current length of the contractile element and V_0 is the contraction velocity at maximum voluntary contraction. F_0 is related to muscle isometric strength and has been estimated from cadaveric studies (Klein Horsman, 2007). Muscle groups and corresponding individual muscles are described in Table 1. The generic musculoskeletal model was scaled to each patient based on a Length–Mass–Fat scaling law in which body mass, body height and segment length were taken into account (Ali et al., 2013; Worsley et al., 2011). Body segment lengths were calculated based on the markers' coordination data in an optimization routine in which the model was scaled such that the differences between "model marker" and the "experimental marker" trajectories were minimized. For every subject, isometric muscle strengths (F_0) were also scaled based on a Height-Squared law (Jaric, 2002) and were considered as "nominal" strengths corresponding to "baseline" simulations. Muscle weakness was then simulated by reducing the F_0 values.

2.2.2. Baseline simulation

The scaled musculoskeletal model was recruited in an inverse dynamic analysis to calculate muscle and joint forces based on marker trajectories and GRFs. Joint forces were calculated from equilibrium equations whilst muscle forces were calculated in an optimization framework (Damsgaard et al., 2006):

$$\text{Minimize}_f\ G\left(f^{(M)}\right)\ ,\quad G(f^{(M)}) = \text{Max}\left(\frac{f_i^{(M)}}{N_i}\right)$$

$$\text{Subjectto}:\ C\times f = d \text{and} 0 \le f_i^{(M)} \le N_i \quad i=\{1,\ldots,n^{(M)}\} \quad (2)$$

where G is the the objective function, f=[$f(M)$, $f(R)$] refers to all unknown forces including muscle forces ($f^{(M)}$) and joint reaction

Please cite this article as: Ardestani, M.M., Moazen, M., How human gait responds to muscle impairment in total knee arthroplasty patients: Muscular compensations and articular.... Journal of Biomechanics (2016), http://dx.doi.org/10.1016/j.jbiomech.2016.03.047