Journal of Biomechanics 42 (2009) 1236–1240

Contents lists available at ScienceDirect

Journal of Biomechanics

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

Experimentally reduced hip abductor function during walking: Implications for knee joint loads

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ARTICLE INFO

Article history:
Accepted 11 March 2009

Keywords:
Movement analysis
Pain
Knee
Hip
Biomechanics

ABSTRACT

Hip and knee functions are intimately connected and reduced hip abductor function might play a role in development of knee osteoarthritis (OA) by increasing the external knee adduction moment during walking. The purpose of this study was to test the hypothesis that reduced function of the gluteus medius (GM) muscle would lead to increased external knee adduction moment during level walking in healthy subjects. Reduced GM muscle function was induced experimentally, by means of intramuscular injections of hypertonic saline that produced an intense short-term muscle pain and reduced muscle function. Isotonic saline injections were used as non-painful control. Fifteen healthy subjects performed walking trials at their self-selected walking speed before and immediately after injections, and again after 20 min of rest, to ensure pain recovery. Standard gait analyses were used to calculate three-dimensional trunk and lower extremity joint kinematics and kinetics. Surface electromyography (EMG) of the glutei, quadriceps, and hamstring muscles were also measured. The peak GM EMG activity had temporal concurrence with peaks in frontal plane moments at both hip and knee joints. The EMG activity in the GM muscle was significantly reduced by pain (−39.6%). All other muscles were unaffected. Peaks in the frontal plane hip and knee joint moments were significantly reduced during pain (−6.4% and −4.2%, respectively). Lateral trunk lean angles and midstance hip joint adduction and knee joint extension angles were reduced by $\leqslant 1^{\circ}$. Thus, the gait changes were primarily caused by reduced GM function. Walking with impaired GM muscle function due to pain significantly reduced the external knee adduction moment. This study challenge the notion that reduced GM function due to pain would lead to increased loads at the knee joint during level walking.

1. Introduction

Knee joint loadings are largest, and thus potentially harmful to the knee, during weight bearing activities. Particularly, the knee joint loadings during walking are of interest in this context, because walking is the most natural way of human locomotion and causes repetitive joint loadings. There is increasing agreement that knee osteoarthritis (OA) is biomechanically driven (Wilson et al., 2008; Dieppe, 2004; Andriacchi et al., 2004) and caused by aberrations in the biomechanics of the knee (Hunter and Felson, 2006; Andriacchi and Mündermann, 2006). The fulcrum of the biomechanical factors in initiation and progression of the disease is joint loadings and it is generally accepted that joint loadings are associated with the pathogenesis of knee OA (Miyazaki et al., 2002; Andriacchi et al., 2004; Mündermann et al., 2005a, b; Sharma et al., 1998; Felson et al., 2000; Andriacchi and Mündermann, 2006).

Gait differences between knee OA patients with medial knee OA and control subjects are mainly observed in the frontal plane. The focus has been on the external knee adduction moment, which is determinative of medial knee joint loads (Schipplein and Andriacchi, 1991), while other gait changes have also been observed in patients with severe medial compartment knee OA (Mündermann et al., 2005a). These included decreased internal hip abduction moments throughout the stance phase in patients with severe knee OA, which could be due to reduced hip abductor muscle strength, leading to a *Trendelenburg* gait, which presumably would result in higher peak external knee adduction moments (Mündermann et al., 2005a). Indeed, greater internal hip abductor moments during walking have been shown to decrease the risk of knee OA progression (Chang et al., 2005), and consequently it has been speculated that decreased hip abductor activity might lead to increased load on the medial compartment of the knee (Chang et al., 2005) with the ultimate consequence being knee OA initiation or progression.

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doi:10.1016/j.jbiomech.2009.03.021

However, such causal relationship between decreased internal hip abductor and increased external knee adduction moments is not fully established. Bearing the observations made by (Mündermann et al., 2005a) and (Chang et al., 2005) in mind, we hypothesized that reduced function of the hip abductor muscles would result in increased knee adduction moments during walking.

We have previously shown that induction of experimental muscle pain, by means of intramuscular hypertonic saline injections, effectively results in reduced muscle function and joint moments during walking and forward lunging (Henriksen et al., 2007, 2008). The experimental muscle pain causes reduced electromyographic (EMG) activity during pain, and the reduced muscle function can be observed up to 20 min after cessation of the conscious sensation of pain (Henriksen et al., 2007, 2008). The saline-induced experimental pain causes a centrally (neural) mediated reduction in muscle activation, while leaving the contractile elements of the muscle unaffected (Graven-Nielsen et al., 2002).

Thus, intramuscular injections of hypertonic saline not only allow for evaluation of nociceptor–motor interactions, but also provide possibilities for evaluation of the effects of reduced function of a single muscle on movement dynamics under well-controlled conditions.

The hip muscles that produce and control hip abduction (balancing the external hip adduction moments) are gluteus medius and minimus, of which gluteus medius is the main abductor. Accordingly, the purpose of this study was to test the hypothesis that reduced function of the gluteus medius muscle would lead to increased external knee adduction moment during level walking in healthy subjects. In this study, we used healthy subjects to account for the possibility that in patients with knee OA the effects of experimentally reduced hip abductor muscle function could be masked by other disease-related changes, e.g. already existing reduced hip abductor function.

2. Materials and methods

2.1. Study subjects

Fifteen healthy subjects (9 females and 6 males) gave voluntary written consent to participate in the study. Their mean age was 24.8 years (SD 6.1), mean height was 1.74 m (SD .08), and mean body mass was 65.8 kg (SD 11.1). The study was approved by the ethics committee for the Capitol Region of Denmark (j. no. H-C-2007-0053).

2.2. Study design

The study was designed as a cross-over study with each subject tested on two days separated by at least one week. During each test day, 3 series of walking trials were performed. Series 1 and 2 were separated by a 5 min break, during which the subjects rested in a chair. Immediately before the second series, an intramuscular saline injection to the gluteus medius muscle was given. The saline solution was either hypertonic (5.8%) or isotonic (0.9%). On a single test day only one type of saline solution was injected according to the randomization process: the order of the saline solution injections was allocated using an envelope-based randomization technique. Between series 2 and 3 a 20 min break was held to ensure that any pain had vanished before the 3rd series of walking trials. Thus, 3 series of walking trials were performed on each test day at the following time points: Baseline (time 0 min), T5 (time: 5 min after last baseline trial), and T25 (20 min after last T5 trial).

2.3. Instrumentation

Kinematic data were acquired using a 3D motion analysis system (Vicon MX, Vicon Motion Systems, Oxford, UK) with 6 cameras (MX-F20, Vicon Motion Systems, Oxford, UK) operating at 100 Hz. Two force platforms (AMTI OR 6-5-1000, Watertown, MA, USA) embedded in the laboratory floor captured ground reaction forces at 1500 Hz synchronised with the kinematic data. Electromyography of 6 leg muscles was recorded using a wireless EMG recording system (Telemyo 2400T G2, Noraxon, USA) operating at 1500 Hz synchronized with the kinematic and force platform data.

The 3D orientation of 8 body segments of interest (trunk; pelvis; left and right thighs; left and right shanks; both feet) was obtained by tracking marker trajectories according to a common commercially available kinematic model (Plug-In-Gait, Vicon Peak®, Oxford, UK), with markers placed on the spinous process of the 7th cervical vertebra and the 10th thoracic vertebra, the jugular notch, the xiphoid process, the right scapula, bilaterally on the anterior and posterior iliac spines, lateral aspect of the thighs, lateral femoral epicondyles, lateral aspects of the shanks, lateral malleoli, calcanea, and 2nd metatarsal heads.

Surface electromyography were recorded from 6 muscles of the right leg: gluteus medius (GM), gluteus maximus (GMa), vastus medialis (VM), vastus lateralis (VL), semitendinosus (ST), and biceps femoris (BF). The EMG recordings were obtained by a bipolar electrode configuration (Blue-Sensor N-00-S, AMBU, Ballerup, Denmark) with a 2 cm inter-electrode distance. Before mounting of the electrodes, the skin was carefully shaved, abraded, and rinsed with alcohol. The EMG signals were sampled at 1500 Hz using a wireless EMG system with 16 bit analogue-to-digital resolution (Telemyo 2400T G2, Noraxon, USA). After placement of the electrodes but before the first baseline walking trials, a measurement of maximal EMG activity was measured during maximal voluntary isometric contractions (MVC).

2.4. Experimental procedures

Anthropometric parameters required for estimating the location of join centres were first measured. EMG electrodes were then placed and resting activity and MVC activities were recorded. Subsequently, the markers were placed at the anatomical landmarks. Each test day commenced with a capture of a static anatomical landmark calibration trial. After the static trial the subjects practiced their desired walking speed until they could walk comfortably within $\pm$0.1 km/h. The walking speed was measured by photocells and a digital display provided the subjects with immediate visual feedback of the walking speed. The starting point was adjusted for each subject so that both feet successfully struck the force platforms, without obvious targeting. Once walking speed and starting points were determined, the subjects performed 3 series of walking trials according to the study design. Each series consisted of 5 acceptable trials defined as being within $\pm$0.1 km/h of the predetermined self-selected speed with successful force platform hits without targeting. Intramuscular saline injections were given immediately before the 2nd series of walking trials (T5).

2.5. Experimental muscle pain

GM muscle pain was induced by intramuscular bolus injections of 1 ml sterile hypertonic saline (5.8%) into the right GM. The injections were given at the midpoint of the line between the most cranial and lateral point of the iliac crest and the greater trochantor. Injections of isotonic saline (0.9%) were used as a pain-free control situation. The pain intensity was scored on a 100 mm visual analogue scale (VAS) where 0 mm indicated "no pain" and 100 mm indicated "worst imaginable pain". Pain intensities were acquired after each walking trial.

2.6. Data analysis

All analyses were performed on the subjects' right leg. Marker coordinate data were filtered using Woltring's generalized cross-validation quintic smoothing spline with a predicted mean-square error of 15 mm. The analyses focused on the stance phase of the gait cycle. Joint kinematics and kinetics were calculated for the trunk (kinematics only), hip, knee, and ankle joints using a common commercially available biomechanical model (Plug-In-Gait, Vicon Motion Systems, Oxford, UK).

Local maxima in frontal plane hip, knee, and ankle joint moments were extracted for statistical analyses and normalized to body mass (Nm/kg). Local maxima in frontal and sagittal plane hip joint angels, sagittal plane knee and ankle joint angles, and frontal plane trunk lean angles were extracted for statistical analyses. Trunk lean angles were defined in two ways as (a) the angle between the trunk and pelvis segments ($Trunk_{local}$), and (b) the angle between the local trunk segment vertical axis and the global vertical axis ($Trunk_{global}$). Step lengths were extracted from the biomechanical model outputs.

The raw EMG signals were digitally high- and low-pass filtered (Butterworth fourth-order zero-lag filter, cut-off frequencies 20 and 450 Hz, respectively), full-wave rectified and low-pass filtered (cut-off frequency 15 Hz) to construct linear envelopes. The linear envelopes were normalized in amplitude to the peak EMG linear envelope signal during the MVC (expressed as %MVC). From the normalized linear envelopes, the peak amplitudes were extracted for each muscle during the stance phase of walking.