Applied Ergonomics 46 (2015) 224–230

Contents lists available at ScienceDirect

Applied Ergonomics

journal homepage: www.elsevier.com/locate/apergo

The effect of sustained static kneeling on kinetic and kinematic knee joint gait parameters

Tara Kajaks [a, *], Patrick Costigan [b, 1]

[a] Department of Kinesiology, McMaster University, 1280 Main Street West, Hamilton, Ontario, Canada L8S 4K1
[b] School of Kinesiology and Health Studies, Queen's University, 28 Division Street, Kingston, Ontario, Canada K7L 3N6

ARTICLE INFO

Article history:
Received 6 September 2011
Accepted 7 August 2014
Available online 27 August 2014

Keywords:
Gait
Knee osteoarthritis
Kneeling

ABSTRACT

Despite epidemiological evidence for kneeling as an occupational risk factor for knee osteoarthritis, biomechanical evidence is lacking. Gait knee joint mechanics, a common measure used to study knee osteoarthritis initiation, were used in the present study to investigate the effect of sustained static kneeling on the knee. Ten healthy male subjects (24.1 years ± 3.5) performed ten baseline walking trials, followed by a 30-min kneeling protocol and a second set of walking trials. Knee joint moments and angles were calculated during the stance phase. Within-subject root mean squared differences were compared within and between the pre- and post-kneeling gait trials. Differences were observed between the pre-kneeling and post-kneeling walking trails for flexion and adduction knee moments (0.12 Nm/kg ± 0.03, 0.07 Nm/kg ± 0.02) and angles (3.18° ± 1.22 and 1.64° ± 1.15), indicating that sustained static deep-knee flexion kneeling does acutely alter knee joint gait parameters.

1. Introduction

Osteoarthritis is considered the most common joint disorder (Arden and Nevitt, 2006), with the weight-bearing joints of the ankle, knee, hip, and back being the most frequently affected. Knee joint osteoarthritis (KOA), in particular, affects a large portion of the population and is the leading cause of chronic disability in the middle aged and elderly (Felson et al., 2000).

While KOA can result from acute traumatic injuries to the knee joint it also develops as the result of years of chronic, cumulative, mechanical insults to the cartilage (Baker et al., 2002; Ezzat et al., 2013). One hypothesized cause of KOA onset and progression is that the constant abnormal loading of the knee joint during activities of daily living, such as one's occupation, results in cumulative stress to the cartilage that are beyond the cartilage's tolerance limits (Thambyah et al., 2005) such that the body cannot repair any damaged cartilage (Andriacchi et al., 2004). Over time, the repeated insults reduce cartilage integrity and degeneration occurs. However, there is little information concerning both the modifiable risk factors and the biomechanical mechanisms responsible for KOA initiation (Felson, 1993; Rossignol, 2004).

Occupational postures have been identified as risk factors for KOA (e.g. Baker et al., 2002; Coggon et al., 2000; Miranda et al., 2002; Fransen et al., 2011), particularly for manual laborers, such as farmers, construction workers, carpenters, floor layers, and general blue collar workers (Cooper et al., 1994). These workers have twice the incidence rate of KOA as their non-manual worker counterparts (Rossignol et al., 2003). These higher incidence rates may be because manual workers typically have low task diversity resulting in more repetitive tasks (Rossignol et al., 2005). Manual workers are also more likely to endure greater joint stress due to their need to adopt awkward working postures and their exposure to high physical demands, such as manually handling heavy loads (Felson et al., 1991; Jensen, 2008). Kneeling, squatting, and stair climbing have also been identified as high risk activities for the development of KOA (Baker et al., 2002; Cooper et al., 1994; Jensen, 2008). Not surprisingly, years of work in knee straining occupations (Anderson et al., 2012) and measures of occupational cumulative knee joint loading (Ezzat et al., 2013) have been linked to higher incidents of KOA. Despite the growing body of epidemiological evidence that certain occupations place workers at a greater risk of KOA, biomechanical support for the link between occupational postures and KOA is lacking (Vignon et al., 2006). Occupational biomechanists now have the responsibility of examining these

Abbreviations: KOA, knee osteoarthritis; IRED, infrared-emitting diode; RMSD, root mean squared difference; ACL, anterior cruciate ligament; PCL, posterior cruciate ligament; MCL, medial collateral ligament; LCL, lateral collateral ligament.

* Corresponding author. Tel.: +1 905 512 2603.
E-mail addresses: kajakst@mcmaster.ca (T. Kajaks), pat.costigan@queensu.ca (P. Costigan).
[1] Tel.: +1 613 533 6000x79037.

http://dx.doi.org/10.1016/j.apergo.2014.08.011
0003-6870/

tasks to uncover the mechanisms responsible for the onset of KOA in these workers.

Two potential mechanisms are hypothesized for the relationship between kneeling and the onset of KOA (Fig. 1). First, deep-knee flexion kneeling alters the location of tibio-femoral contact location and increases the contact pressure on the cartilage (Wang et al., 2014). The direct loading at a single non-conditioned location during kneeling may over time initiate the biological processes associated with cartilage degeneration (Andriacchi et al., 2004). Second, the posterior displacement and external rotation of the tibia relative to the femur during deep-knee flexion kneeling places additional stress on the knee joint ligaments, particularly the PCL and MCL (Hofer et al., 2011). Based on these kneeling mechanics, we propose that sustained static deep-knee flexion kneeling alters joint control that can be seen in changes in the joint kinematics and kinetics and that these changes alter the expected joint loading pattern thereby adversely affecting joint integrity during subsequent activities.

This hypothesized mechanism is supported by research showing that a constant tibial load (200 N for men and 150 N for women) for a period of 10 min, at knee flexion angles of 35 and 90°, induced ligament creep in the human ACL and associated neuromuscular disorders, as demonstrated by 3–5 mm of tibial displacement and quadriceps and hamstring muscle spasms immediately following the protocol (Chu et al., 2003). Although the duration of residual creep was not reported in the study by Chu et al. (2003), Courville et al. (2005) showed that three cycles of work-to-rest ratio of 2:1, with 10 min of static lumbar flexion in felines, resulted in signs of a neuromuscular disorder and ligament creep lasting over 7 h. Additionally, prolonged static loads on ligaments induce acute inflammatory responses that lead to acute neuromuscular disorders and the associated injury risks (as reviewed by Solomonow, 2006). Additionally, the proposed pathway connecting occupational kneeling to the onset of KOA via knee instability (Fig. 1) is supported by Sharma (2001) and Andriacchi et al. (2004), who reported that decreased joint stability, as caused by ligament laxity, proprioceptive deficits, or neuromuscular impairments, is a risk factor for KOA.

Therefore, the anticipated post-kneeling change in joint motion, seen in the altered kinematics and kinetics during walking, although temporary after a single bout of prolonged kneeling but becoming more chronic with daily bouts of prolonged kneeling such as that experienced by workers in trades with frequent kneeling (Gaudreault et al., 2013), may reduce the joint's adaptive mechanisms or cause abnormal tibiofemoral joint loading patterns. Either of these outcomes may lead to joint damage during load-bearing tasks performed within a window of time after rising from a sustained kneeling posture. Since altered loading patterns at the knee joint during ambulation are a known risk factor for KOA (e.g. Andriacchi et al., 2004; Andriacchi and Mundermann, 2006; Chaudhari et al., 2008), the present exploratory investigation was designed to test for an acute effect of static kneeling on changes in knee kinematics and kinetics. This was achieved by examining changes in knee kinematics and kinetics during gait after three 10 min bouts of kneeling. It was expected that the stance-phase kinetic (knee adduction and flexion moments) and kinematic waveforms (knee adduction and flexion angles) would be altered as a result of the kneeling protocol. Such a finding would support the proposed pathway (Fig. 1), thus providing biomechanical evidence to support the pathogenesis of KOA through deep-knee flexion postures.

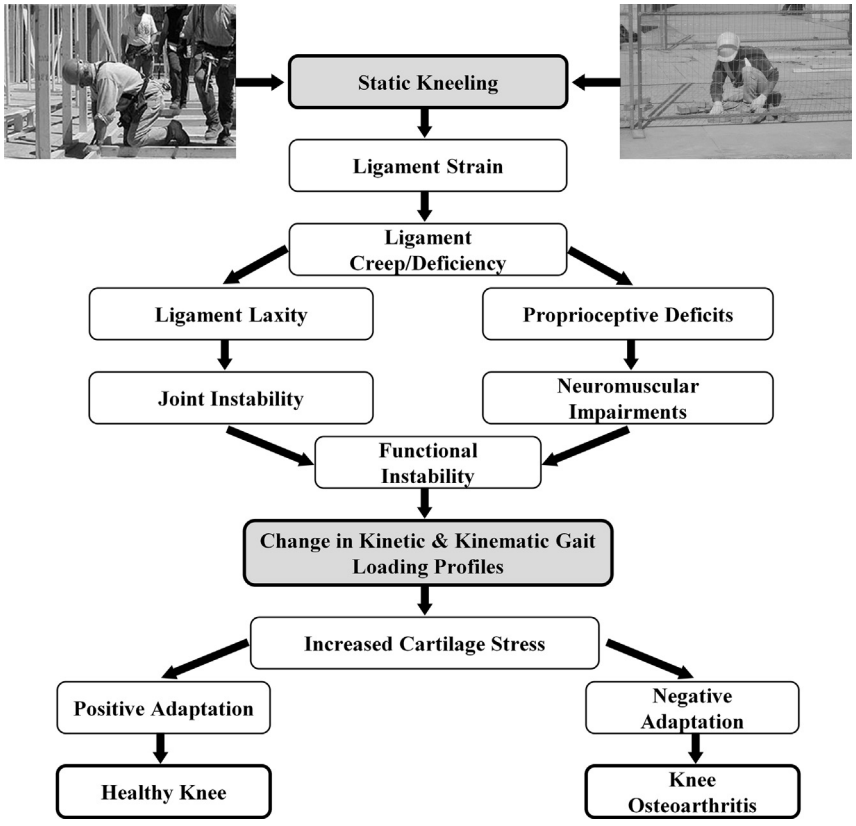

Fig. 1. A hypothesized model of the relationship between static kneeling and KOA, which suggests that static kneeling may lead to KOA through a pathway that begins with ligament strain leading to functional joint instability causing abnormal joint loading patterns. Highlighted are the current study's dependent (static kneeling) and independent (change in kinetic and kinematic gait loading profiles) variables.