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ScienceDirect
Gait & Posture 27 (2008) 616–621
GAIT POSTURE
www.elsevier.com/locate/gaitpost

The influence of gluteus maximus on transverse plane tibial rotation

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Received 14 March 2007; received in revised form 14 August 2007; accepted 21 August 2007

Abstract

There is a common clinical belief that transverse plane tibial rotation is controlled by the rearfoot. Although distal structures may influence the motion of the tibia, transverse plane tibial rotation could be determined by the proximal hip musculature. Cadaver studies have identified gluteus maximus as having the largest capacity for external rotation of the hip. This study was therefore undertaken to investigate the effect of gluteus maximus on tibial motion. Kinematic data were collected from the foot and tibia along with EMG data from gluteus maximus for 17 male subjects during normal walking. A number of kinematic parameters were derived to characterise early stance phase. Gluteus maximus function was characterised using RMS EMG and EMG on/off times. No differences in muscle timing were found to be associated with any of the kinematic parameters. In addition, no differences in gluteal activation levels were found between groups of subjects who had different amounts of tibial rotation. However, there was a significant difference ($p < 0.001$) in gluteus maximus activation when groups were defined by the time taken to decelerate the tibia (time to peak internal velocity). Specifically, subjects with greater gluteus maximus activity had a lower time to decelerate the tibia. We suggest that a high level of gluteus maximus activity results in a larger external torque being applied to the femur, which ultimately leads to a more rapid deceleration of the tibia.

Keywords: Tibial rotation; Rearfoot pronation; Gluteus maximus; EMG

1. Introduction

A coupling mechanism exists within the ankle joint complex which enables the transfer of pronation/supination to axial tibial rotation [1,2]. This coupling mechanism arises from the articulations within the ankle, subtalar and midtarsal joints [3]. As the subtalar joint is inclined approximately 45° from the horizontal, calcaneal eversion transfers into a similar amount of internal tibial rotation [1,4]. It is commonly believed that, through this mechanism, the foot motion controls the transverse plane rotation of the tibia and subsequently the entire lower limb. Abnormal magnitude or timing of foot pronation is believed to result in abnormal internal tibial rotation and to be associated with a number of musculoskeletal pathologies, including patello-femoral pain [5,6], plantar fascitis [7] and Achilles tendonitis [8].

Transverse plane tibial motion is determined by torques applied both proximally and distally. These torques are generated by muscle-tendon forces, ligamentous constraints and external forces, such as the ground reaction force. If the torques acting at the proximal end of the tibia are larger than those acting at the distal end, the tibia is considered to be driven proximally. Conversely, larger distal forces would mean more distal control. By using a 'power-flow' analysis, Bellchamber and van den Bogert [9] found evidence for proximal control during walking in late stance phase. This result demonstrates that tibial motion is not always controlled by the structures surrounding the ankle. As the muscles surrounding the knee joint have little capacity for transverse plane tibial rotation, proximal control may

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doi:10.1016/j.gaitpost.2007.08.007

originate from the hip musculature. Although there have been some studies attempting to relate foot structure to tibial kinematics [2], there is a lack of published studies investigating the effect of hip biomechanics and specifically the potential role of the hip musculature. Therefore, further research is required to establish whether specific hip muscles could have a significant influence on transverse plane tibial rotation. Any research which could demonstrate a link between hip muscle function and tibial rotation has the potential to influence clinical practice. Although, the traditional view is that abnormal rotation should be corrected at the ankle, it may be possible to change transverse plane tibial rotation by focusing on muscle activity at the hip.

At heel strike the ground reaction force, acting on the plantar aspect of the foot, causes the rearfoot to pronate and the tibia to internally rotate [10,11]. This motion continues to the point of peak internal rotation, which occurs between 20% and 25% stance during normal walking [10] (Fig. 1a). During this period it is possible that specific hip muscles could act eccentrically to apply an external rotation moment to the femur and thus decelerate the internal rotation of the tibia. During the initial 20–25% of stance, the hip is flexed approximately 25° [12]. In this position, activation across all compartments of either gluteus medius or minimus would produce limited transverse plane femoral rotation [13]. The same is true for the combined effect of the hamstrings or the adductors [14]. The situation is different for gluteus maximus, which, due to is anatomical position, has a large capacity for external rotation [13]. Given the relative strength of this muscle, the fact it is active during early stance [15] and its large capacity for external rotation [13], it has significant potential to affect tibial kinematics.

Soft tissue motion artefact is known to significantly influence derived kinematics for transverse plane lower extremity motion [16]. In particular a number of studies have shown that errors from skin-mounted marker-based estimates of thigh rotation are similar in magnitude to the true motions [17–19]. As such it has been concluded that skin-mounted marker systems are not appropriate for representing the transverse plane motion of the femur or the knee joint [16,19]. Soft tissue artefact has been found to be considerably less for the tibia [17,18]. Moreover, a recent study demonstrated that soft tissue artefact can be reduced by using a constrained marker cluster, placed distally on the shank [20]. Using this suggested marker protocol, it may be

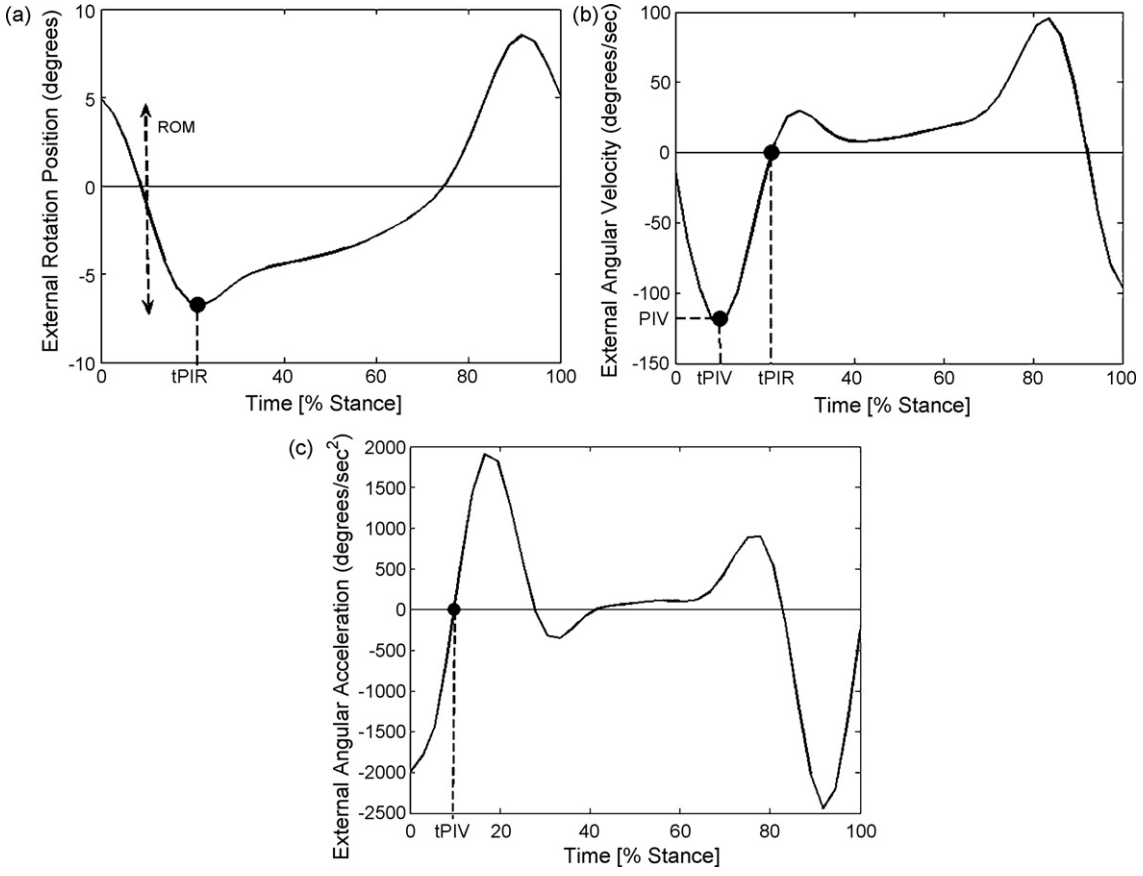

Fig. 1. (a) Typical transverse plane tibial rotation during the stance phase of walking (0% represents heel strike and 100% toe off). ROM refers to tibial range of motion over 0–50% stance phase and tPIR to the time taken to reach the point of maximal tibial internal rotation. Adapted from [10]. (b) Typical transverse plane tibial velocity during the stance phase of walking (0% represents heel strike and 100% toe off). PIV refers to the maximal internal rotation velocity of the tibia and tPIV to the time taken to reach this point. Adapted from [10]. (c) Typical transverse plane tibial acceleration during the stance phase of walking (0% represents heel strike and 100% toe off). Adapted from [10].