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Activation of the gluteus maximus and hamstring muscles during prone hip extension with knee flexion in three hip abduction positions

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ABSTRACT

The direction of fiber alignment within a muscle is known to influence the effectiveness of muscle contraction. However, most of the commonly used clinical gluteus maximus (GM) exercises do not consider the direction of fiber alignment within the muscle. Therefore, the purpose of this study was to investigate the influence of hip abduction position on the EMG (electromyography) amplitude and onset time of the GM and hamstrings (HAM) during prone hip extension with knee flexion (PHEKF) exercise. Surface EMG signals were recorded from the GM and HAM during PHEKF exercise in three hip abduction positions: 0°, 15°, and 30°. Thirty healthy subjects voluntarily participated in this study.

The results show that GM EMG amplitude was greatest in the 30° hip abduction position, followed by 15° and then 0° hip abduction during PHEKF exercise. On the other hand, the HAM EMG amplitude at 0° hip abduction was significantly greater than at 15° and 30° hip abduction. Additionally, GM EMG onset firing was delayed relative to that of the HAM at 0° hip abduction. On the contrary, the GM EMG onset occurred earlier than the HAM in the 15° and 30° hip abduction positions.

These findings indicate that performing PHEKF exercise in the 30° hip abduction position may be recommended as an effective way to facilitate the GM muscle activity and advance the firing time of the GM muscle in asymptomatic individuals. This finding provides preliminary evidence that GM EMG amplitude and onset time can be modified by the degree of hip abduction.

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1. Introduction

The group of muscles in the gluteal region consists of the gluteus maximus, medius, and minimus. The gluteus maximus (GM) is the largest and most superficial muscle in the area. It is a broad, thick, fleshy mass of a quadrilateral shape and its fibers are directed obliquely downward and laterally (Frank and Netter, 1987; McAndrew et al., 2006). The muscle primarily acts as a powerful extensor of the hip. Because the GM muscle fibers are aligned perpendicular to the sacroiliac (SI) joint, GM contraction produces compression of the SI joint, and also contributes to the force transmission mechanism from the lower extremity to the pelvis through the SI joint during functional activities such as ambulation (Lyons et al., 2006).

Inappropriate timing of GM activation during gait is thought to be one of the causes of low back pain (LBP), resulting in a deficiency in the shock absorption mechanism at the sacroiliac joint. Earlier onset

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1356-689X/S - see front matter © 2012 Elsevier Ltd. All rights reserved. http://dx.doi.org/10.1016/j.mafh.2012.11.006 of hamstrings (HAM) activation has been noted in patients with LBP as compensation for delayed firing of the GM (Hungerford et al., 2003; Hossain and Nokes, 2005). In addition, weakness and imbalanced strength in the GM are associated with lower extremity injuries, including patellofemoral pain syndrome, anterior cruciate ligament sprains, and chronic ankle instability (Powers, 2003; Friel et al., 2006; Hewett et al., 2006; Cichanowski et al., 2007; Yang et al., 2011). Weakness of the GM also leads to slouched posture, makes walking extremely difficult, and necessitates substitution by synergists (Kisner and Colby, 2005). Therefore, neuromuscular reeducation and/ or specific GM strengthening exercises are clinically necessary in rehabilitation for low back pain and lower extremity injuries.

Many studies have demonstrated various methods to reduce delayed firing of the GM. During prone hip extension exercise, lower abdominal hollowing and the abdominal drawing-in maneuver (ADIM) using a pressure biofeedback unit reduced the delay of GM firing relative to that of the HAM (Oh et al., 2006, Chance-Larsen et al., 2010). In addition, gluteal verbal cues during prone hip extension resulted in nearly simultaneous electromyography (EMG) onset of the HAM and GM, which means delayed HAM onset and advanced GM onset based on the no-cues condition (Lewis and Sahrmann, 2009).

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