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Arthrogenic neuromusculature inhibition: A foundational investigation of existence in the hip joint

Stephanie Freeman [a], Anthony Mascia [b], Stuart McGill [a,*]

[a] Department of Kinesiology, Spine Biomechanics Laboratory, University of Waterloo, Waterloo, ON, Canada
[b] Department of Radiology, Humber River Regional Hospital, Toronto, ON, Canada

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ABSTRACT

Background: Patients and athletes with diminished gluteal muscle activation are thought to have 'gluteal inhibition'. This may be a component of arthrogenic neuromuscular inhibition, which has been well documented in the knee and generalized to all joints. While clinical evidence surrounding gluteal inhibition increases, supportive research is non-existent. This study investigated whether arthrogenic neuromuscular inhibition occurred about the hip following instillation of intra-articular fluid during functional hip extension tasks.
Methods: Data was collected in a biomechanics laboratory (control) and hospital setting (intervention). Nine healthy individuals (4M/5F) comprised the control group. The intervention group contained twelve patients (4M/8F) with hip pathology requiring a magnetic resonance arthrogram (capsular distension via intra-articular fluid injection) procedure. The participants performed a pelvic bridge (PB) and active hip extension (EXT) before and after the control time or injection. Peak EMG from the gluteus maximus (GM) was collected bilaterally.
Findings: The findings of this study provide substantial support for arthrogenic inhibition following hip intra-articular fluid instillation during functional tasks. Two-way repeated measures ANOVA revealed a significant group by session interaction effect (PB,EXT: affected/unaffected = 0.0192/0.9654 $P = 0.05$, <0.0001/0.0826 $P = 0.05$). Tukey post hoc revealed decreases in ipsilateral peak GM EMG following intervention were significant (0.0238/<0.0001 $P = 0.025$). No changes were observed in the control group.
Interpretation: These concepts are of clinical importance to both patient and athletic populations. Understanding the role of gluteal inhibition in the injury process is essential to the development of rehabilitation and prevention protocols. Restoration and promotion of optimal recruitment patterns are crucial to enhancing athletic performance.

1. Introduction

Patients with hip and/or low back pain are often described to present with clinical findings of diminished activation in the gluteal musculature. This is often termed 'gluteal inhibition' and/or 'gluteal amnesia' and may be one component of the collective findings that comprise 'arthrogenic muscle inhibition'. Arthrogenic or neuromuscular inhibition is defined as continued reflex inhibition of musculature surrounding a joint following injury or joint effusion (Hopkins and Ingersoll, 2000). Characteristically, selective inhibition of the extensor musculature is accompanied by facilitation of the flexor musculature surrounding the affected joint (Hopkins and Ingersoll, 2000; Palmieri et al., 2005). While increasing clinical observations and awareness make it difficult to deny the presence of this phenomenon, what remains unrealized is the absence of research to support its existence about the hip joint. Perhaps this concept of arthrogenic inhibition has been accepted as a broad neurological truism due to the fact that this pattern of inhibition–facilitation has been documented extensively following intra-articular injuries, surgical intervention and/or fluid administration of some peripheral joints, primarily the knee (Palmieri et al., 2003; Palmieri-Smith et al., 2007). Although it has conceptually been generalized to occur at all joints, its apparent occurrence at the hip joint has never been quantitatively validated. For example, extensive research by the Palmieri group demonstrated selective inhibition of the knee extensors associated with increased intra-articular fluid, induced by injury or experimentally (Palmieri et al., 2003, 2004, 2005; Palmieri-Smith et al., 2007). This 'inhibition' was displayed during both experimentally-stimulated contractions and functional tasks. This pattern of inhibition in peripheral joints is supported to a much lesser degree by a few studies investigating its presence in the elbow and ankle joint (Hopkins and Palmieri, 2004; McVey et al., 2005).

The evidence supporting the presence of arthrogenic inhibition in the knee is convincing, but the specific mechanisms remain speculative. Irrespective of the joint involved, an injury- or experimentally-induced joint effusion is thought to interfere with joint afferents, causing interruption of afferent integration, neurological feedback mechanisms and

* Corresponding author at: Department of Kinesiology, University of Waterloo, 200 University Ave W, Waterloo, ON, Canada N2L 3G1.
E-mail address: mcgill@healthy.uwaterloo.ca (S. McGill).

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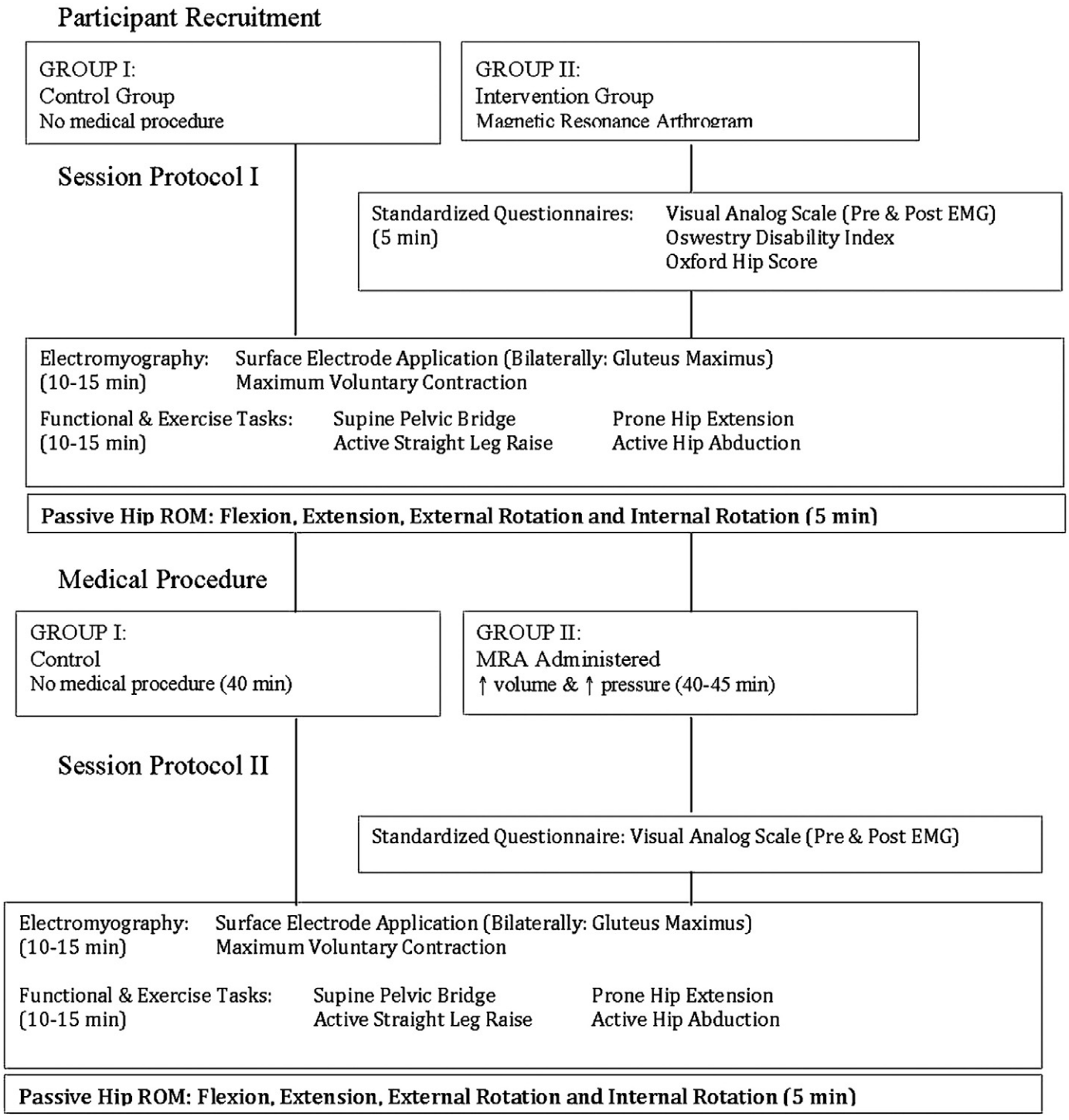
Table 1
Overview of research design.



ultimately muscle inhibition (Hopkins and Ingersoll, 2000; McVey et al., 2005; Palmieri et al., 2004). The measures commonly used in previous research to characterize inhibition have been quantification of H-reflex, M-wave, the H:M ratio and/or electromyography (EMG). Lasting implications of inhibition include compromise of strength development and restoration of normal proprioceptive function, limiting healing capabilities and the rehabilitation process restoring normal function (Hopkins and Ingersoll, 2000; Hurley, 1997). The consequence is an increased risk of early degenerative changes in the bony and cartilaginous structures, findings commonly associated with hip joint injuries (Palmieri et al., 2005; Suter and Herzog, 2000). Confirming the presence of arthrogenic inhibition about the hip joint would contribute some insight into the neuropathological mechanisms surrounding muscle inhibition, a requisite to enhancement of treatment protocols for prevention and management of hip pathology.

The objective of this study was to investigate whether hip extensor inhibition was elicited following instillation of intra-articular fluid. We hypothesized that a unilateral reduction in peak gluteus maximus