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An Electromyographic Comparison of the Functional Performance of the Gluteus Maximus Muscle in Prolonged Sitting Versus Standing Populations

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THE CITY UNIVERSITY OF NEW YORK

Abstract

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PURPOSE: A common clinical concern of Physical Therapists is the inexplicably weak gluteus maximus (GM) muscle; we hypothesized that this may be linked to the popular habit of prolonged sitting. The purpose of this study was to determine if surface electromyography (sEMG) output and timing of the GM and hamstrings muscles differed between people who sit for prolonged periods of time and people who stand for prolonged periods of time. METHODS: The design of our study was a single session case-control study. Subjects were 22 healthy adults (23-36 years old) who either sat or stood for 8-10 hours a day at least 5 days per week. There were 11 subjects in each group. Written informed consent and questionnaires were obtained at the start of the session, with all forms and procedures approved

by the CUNY Human Research Protection Program. The BioNomadix MP150 EMG system collected sEMG during: a manual muscle test (Maximum Voluntary Isometric Contraction) of both the GM and hamstrings muscles; the functional activity of sit-to-stand; and, during a repeated forward step-up. The maximum sEMG signal amplitude recorded during the Maximum Voluntary Isometric Contraction (MVIC) of each muscle represents 100% muscle activity, and the sEMG activity recorded during the functional activities was expressed as a percentage of the MVIC. Relative timing of muscle onset was recorded. DATA ANALYSIS: Repeated measures Multivariate Analysis of Variance (MANOVA) was used to compare normalized mean signal amplitude levels, expressed as a percent of a MVIC, across functional tasks. Friedman tests were used to analyze muscle onset. Post hoc testing with pairwise comparisons were used to find further significance. Bonferonni correction was applied to eliminate false positives. RESULTS: Repeated measures MANOVA did not reveal any main effects or interactions for muscle activity. Friedman tests showed similar results with timing data. Post hoc tests failed to meet the criteria of the Bonferroni correction. The data showed the similarity of muscle timing and activation specifically during the sit-to-stand task in both the sitting group and standing group. CONCLUSIONS: When comparing the sEMG muscle activity of the GM and hamstring muscles during sit-to-stand and a repeated forward-step up in subjects that sit for 8 hours or more per weekday to those who stand for 8 hours or more, no statistical significance differences were found. However, this could be due to limitations of the study such as small sample size, a sample that did not accurately represent the general public, and absence of kinematic data. Due to these limitations further research in this area is needed to determine whether or not prolonged sitting can be linked to decreased sEMG output and timing of the GM.

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Introduction

Our society has become increasingly sedentary due to advances in work- and chore-related technologies, suburban commuter lifestyles, and the popularity of television and computer games. The average American adult spends more than half of his or her day in a seated position (Matthews et al., 2008). Recently, many articles in established publications, such as the New York Times (Reynolds, 2012), the Annals of Internal Medicine (Biwas et al., 2015), and the Journal of the American Medical Association (Grøntved & Hu, 2011), have turned the public eye towards the myriad health risks incurred by this habit of spending excessive amounts of time sitting. While diabetes, cardiovascular disease, and premature death have been correlated with prolonged sitting (Biwas et al., 2015; Patel et al., 2010), the musculoskeletal implications have not yet been explored.

Prolonged Sitting

Researchers have defined sedentary behavior as engaging in activities that have a low level of energy expenditure, around 1.0 - 1.5 METs (Owen, Healy, Matthews, & Dunstan, 2010), which would include most seated activities. Patel et al. (2010) used a marker of greater than or equal to six hours of leisure time spent sitting for their study, which looked at the relationship between sitting time and total mortality. They did not include work-related sitting time, but the majority of their subjects were homemakers and retirees. For our study, we defined prolonged sitting as sitting for at least 8 hours per day. If a person sat for his or her job and for meals, he or she would probably have met meet our criteria.

Owen et al. proposed that "too much sitting is distinct from too little exercise" (2010, p. 105). They defined the *Active Couch Potato* phenomenon as people who meet public health

guidelines for exercise and yet have health risks, apparently due to their prolonged sitting (Owen et al., 2010). Biwas et al. (2015) performed a meta-analysis that agreed with the main findings of Owen et al., stating that prolonged sitting time is associated with increased mortality, cardiovascular disease, cancer, and diabetes; however the literature review of Biwas et al. (2015) actually found that a high level of physical activity somewhat mitigates the deleterious effects of sitting.

Television has been used as one way to look at how much time people spend sitting. Healy et al. (2008) looked at data from the Australian Diabetes, Obesity and Lifestyle study and found that the more television these *Active Couch Potatoes* watched, the more they experienced increased waist circumference, systolic blood pressure, plasma glucose, triglycerides, and cholesterol. Of course, people sit during other activities, too -- most sit for commuting, for work, for school, and for meals. Over the past fifty years, the percentage of Americans with moderate-intensity occupations fell from 50% to 20%, and the number of sedentary and light-intensity jobs in the U.S. grew to about 70% (Church et al., 2011).

A study comparing biopsies from the muscles of young people, active older adults, and sedentary older adults showed that a sedentary lifestyle is correlated with not only the expected sarcopenia, which leads to a loss of strength and functional abilities, but is also correlated with low-grade chronic inflammation and a loss of oxidative capacity in muscle tissue (Safdar et al., 2010). Patel et al. found that "women who reported sitting more than six hours a day outside of work had a 34 percent higher risk of death than those who sat fewer than three hours daily" (as cited in Pope, 2012, p. 28). Health, longevity, and quality of life seem to depend heavily upon our ability to stay active, a real challenge for a population that tends to sit most of the day.

A study by Peddie et al. (2013) demonstrated the benefits of taking breaks from prolonged sitting. They implemented three randomized intervention groups that included prolonged sitting (sitting for nine hours), prolonged sitting with a single round of walking for thirty minutes, and lastly, prolonged sitting with rounds of walking for one minute and forty seconds every thirty minutes. This randomized, crossover study included seventy healthy adults who completed all three interventions. Various tests and outcome measures specifically testing insulin, glucose, and triglyceride levels were conducted. Peddie et al. (2013) concluded that the healthiest intervention was prolonged sitting with interrupted activity every thirty minutes. This study informed society of the effect prolonged sitting had on our endocrine system, but did not address the musculoskeletal system.

Sitting and Pelvic Tilt

In humans, upper body weight rests on the ischial tuberosities during sitting (Moore, Dalley, & Agur, 2010), but clinically one may observe that many people who sit for prolonged periods, such as wheelchair users, tend to sit in a posterior pelvic tilt (Kemmoku, Furumachi, & Shimamura, 2012). This population has been studied extensively to discover preventative measures for pressure ulcers in the sacral and ischial areas (Kemmoku et al., 2012; Peterson & Adkins, 1982), but some of this information may actually pertain to the otherwise healthy population that engages in prolonged sitting. Peterson and Adkins (1982) noted that a posterior pelvic tilt in the sitting position causes the sacrococcygeal area, which is relatively unpadded compared to weight-bearing surfaces such as the soles of the feet, to bear excessive weight. The gluteus maximus (GM) has several attachments in this area, attaching posteriorly to the sacrum, ilium, and coccyx, as well as the sacrotuberous and sacroiliac ligaments (Neumann, 2002), and is likely being adversely affected by prolonged sitting in a posterior pelvic tilt.

In the past, an anterior pelvic tilt was described as a common clinical finding due to tightness in the low back, but this was before the age of prolonged sitting (Peterson-Kendall, McCreary, Geise-Provance, McIntyre-Rodgers, & Romani, 2005). Our new habits of sitting may have changed this since most people sit with a flexed spine, causing a host of new biomechanical problems (Peterson-Kendall et al., 2005). A study that looked at the intervertebral joint (IVJ) angles of 27 healthy subjects in standing, standing flexion, upright sitting, and "slouched" sitting found that the lower IVJs neared their maximum range of flexion when the subjects sat in the "slouched" position and were between 35% and 60% of maximum flexion when subjects sat in an upright position (Dunk, Kedgley, Jenkyn, & Callaghan, 2008). These researchers expressed their concern that "sitting imposes a flexed posture that may have detrimental effects on the tissues of the spine" (Dunk et al., 2008, p.167), but we believe that prolonged sitting and poor sitting posture may be linked to more musculoskeletal problems than just back pain.

According to Lambert, "we were never designed to actually sit" (2007, p. 22). He went on to explain that by sitting on contractile tissue, which is not meant for weight-bearing, we are damaging the muscles' ability to do work (Lambert, 2007), and unsurprisingly, numerous physical therapists over the past decade have made the clinical observation that a vast number of people of all ages and occupations are presenting with gluteal performance problems (Boyle, 2005; Lambert, 2007).

Tissue Damage from Prolonged Sitting

Exactly what damage is occurring when people sit for long periods of time? A study by Linder-Ganz, Shabshin, Itzchak, and Gefen (2007) demonstrated the distribution of stress and strain in deep muscle and fat while in a sitting posture with six healthy adults. The authors compared magnetic resonance imaging of the gluteal muscles in a weight bearing (sitting)

position versus a non-weight bearing (standing) position to determine the distribution of stress and strain. They found that when people sit on their ischial tuberosities, the gluteal muscles undergo maximal tissue strain and stresses as compared to fat and other tissues of that region (Linder-Ganz et al., 2007).

According to another study, this type of stress, or "mechanical loading," over a long period of time will lead to ischemia (Gawlitta et al., 2007). This lack of blood supply to an area eventually results in the formation of pressure ulcers in compromised patients. Engineered skeletal muscle tissue was tested under various compressions in order to evaluate whether pressure ulcers result from cell deformation or lack of oxygen. In either case, the time it takes for tissue to become apoptotic and/or necrotic was also investigated (Gawlitta et al., 2007). Apoptosis is defined as "programmed cell death" after it recognizes its incompetence (Guyton & Hall, 2010, p. 40). Necrosis, on the other hand, is cell death as a result of bursting and loss of the cell membrane (Guyton & Hall, 2010). Gawlitta et al. (2007) showed that under 40% of compression, 43% of the tissue had become apoptotic after 22 hours.

Pressure distributions during sitting showed that in 15 healthy subjects, 18% of body weight is borne over each ischial tuberosity, 21% is borne over each thigh, and 5% is borne over the sacrum, but this study did not note the posture of their subjects (Drummond, Narechania, Rosenthal, & Breed, 1982). Kemmoku, Furumachi, & Tadashi (2012) found almost identical values in their study where pressures were measured with subjects in a 10-degree posterior pelvic tilt, so that one may wonder if the subjects in Kemmoku et al.'s study were also sitting with a posterior pelvic tilt. Researchers who looked at sitting biomechanics warned that with a posterior pelvic tilt, the feet are not able to bear their fair share of the body weight since the body's center of gravity is behind the ischial tuberosities (Harrison, Harrison, Croft, & Harrison,

1999). While research shows that when sitting in a posterior pelvic tilt, we are putting excess weight on the sacrococcygeal area (Peterson & Adkins, 1982), it has not yet been shown that when sitting in a posterior pelvic tilt, we are in fact sitting on the adjacent gluteal muscles. However, part of our hypothesis implies that sitting 8-10 hours a day with little or no interruption or activity may lead to apoptosis of cells in the GM muscle.

Gluteus Maximus: The Primary Hip Extensor

The hip extensors, the GM and the hamstring muscles, have different phasic patterns and moments of peak activity depending on the activity performed (Lyons, Perry, Gronley, Barnes, & Antonelli, 1983). The GM is the largest of the hip muscles and accounts for 16% of the total cross-sectional area of the hip region (Reiman, Bolgla, & Loudon, 2012). The GM can be functionally divided into an upper and lower half. The upper half acts as a hip abductor, while the lower half acts as a hip extensor (Perry, 1992). The GM is known as the primary hip extensor and external rotator, but shows the most muscle activity during activities that require more powerful hip extension (Neumann, 2002). It has a modest level of activity during steady-speed level walking, and increases in activity as gait speed increases. Bartlett, Sumner, Ellis, and Kram (2014), found that the GM had similar activation during running and climbing, which was greater than during level walking. They also found the GM to be much more active during sprinting than during running, supporting the idea that the GM is used in activities that require increased speed and power (Bartlett, Sumner, Ellis, & Kram, 2014). When comparing the percent MVIC during five weight-bearing activities, the GM had the most activity during a unilateral wall squat and a forward step-up. The unilateral wall squat required 86 percent MVIC (standard deviation = 43), whereas the forward step-up required 74 percent MVIC (standard deviation = 43; Ayotte et al., 2007).

As discussed earlier, prolonged sitting may cause compression to the GM muscle tissue, which may lead to apoptosis. In addition to the effects compression may have on the GM, our sedentary lifestyle also creates repetitive movements and sustained postures that can cause a change in the strength, length, and stiffness of various muscles. Therefore, individuals who regularly participate in daily activities and even exercise frequently can still present with muscle weakness (Sahrmann, 2002). The musculoskeletal system's ability to adapt to demands is usually thought to be advantageous, but in this case the strengthening of some muscles changes the relationship between synergistic and antagonistic muscles and can alter the precision of joint motion. This alteration causes muscles that are prone to weakness, such as the GM, to fire sub-optimally for actions where it should be the primary mover (Sahrmann, 2002**).**

Significance of the GM to lower extremity function. GM weakness is of clinical concern because reviews of current research have concluded that hip muscle weakness and altered kinematics may contribute to pathologies such as low back pain, patellofemoral pain syndrome, anterior cruciate ligament injury, iliotibial band syndrome, and osteoarthritis, as well as increase the risk of lower extremity injury (Powers, 2010; Reiman, Bolgla, & Lorenz, 2009). For example, patellofemoral pain (PFP), a prevalent lower-extremity disorder, is thought to be caused by laterally directed forces on the patella caused by excessive hip internal rotation and adduction, a hip position that is permitted by weak gluteal muscles (Prins & van der Wurff, 2009; Souza & Powers, 2009). This altered position changes the alignment of the lower limb, affecting the loading of the knee joint (Powers, 2010). Supporting this correlation between weak gluteal muscles and PFP, a case report showed that increasing the force production of the GM and gluteus medius muscles results in reduced pain, improved lower-extremity kinematics, and increased function (Mascal, Landel, & Powers, 2003).

Furthermore, as a strong hip extensor and the most powerful external rotator, it has been argued that the GM is best suited to provide the three-dimensional stability needed to protect the knee joint against the hips' tendency to collapse into adduction and internal rotation during weight bearing (Powers, 2010). Despite this theory, and the fact that that females with PFP were found to have weak hip external rotation, abduction and extension (Prins & van der Wurff, 2009), one cannot demonstrate a definite cause-and-effect relationship between decreased hip-muscle strength and PFP or other lower-extremity disorders. Because these studies were retrospective, one cannot determine whether the gluteal weakness was a cause or a result of the pathologies (Powers, 2010). This can also be said for studies that have found the GM to have less endurance, to fatigue quicker, and to activate less in subjects with chronic lower-back pain than in pain-free individuals (Kankaanpaa, Taimela, Laaksonen, Hänninen, & Airaksinen, 1998; Leinonen, Kankaanpää, Airaksinen, & Hänninen, 2000).

Leetun, Ireland, Willson, Ballantyne, and Davis (2004) demonstrated a causal relationship in a prospective study which examined whether or not decreased lumbo-pelvic stability predisposed an individual to lower-extremity injuries. By testing male and female athletes' lumbo-pelvic muscle strength and endurance at the beginning of the season, prior to sustaining any injuries, they were able to assess whether or not the weakness contributed to subsequent injury. Those who had not sustained an injury during the season had tested significantly stronger in hip abduction and external rotation at the start of the season. Logistic regression analysis showed that the strength of hip external rotation was the only useful predictor of injury (Leetun et al., 2004).

A weak GM muscle also directly affects the kinematics of the hip joint itself. Lewis, Sahrmann, and Moran (2007) found that weakness, or a decreased force contribution from the

gluteal muscles, during hip extension causes an increase in the anterior hip joint force. Sahrmann (2002) believes that an anteriorly directed hip force is likely the cause of femoral anterior glide syndrome, a condition caused by the femoral head exerting pressure on the anterior joint structures, due to improper posterior gliding. She argues that over time this can lead to hip pain, instability, and a tear of the acetabular labrum (Sahrmann, 2002).

Hip muscle imbalances. In addition to weakness, muscle imbalances of the hip have also been thought to cause dysfunction. Muscle imbalance can be defined as an altered relationship between muscles that are prone to inhibition and those that are prone to tightness. This altered relationship can affect joint mechanics and may lead to pain, dysfunction, and eventually degeneration. For example, during prone hip extension, it is considered a faulty muscle activation pattern when the hamstrings or erector spinae are activated first and the activation of the GM muscle is delayed or if there is little to no force contribution (Janda & Liebenson, 2007). Sahrmann (2002) believes that this specific faulty pattern causes an anterior shear of the trochanter. This imbalance between the GM and the hamstrings, where the hamstrings become dominant is also seen in individuals with sacroiliac joint dysfunction and in runners (Hossian & Nokes, 2005; Wagner et al., 2010; Sahrmann, 2002). Wagner et al. (2010) argues that, "Given that the hamstrings and gluteus maximus are agonists, it is conceivable that weakness of the gluteus maximus could increase the relative effort of the hamstring muscles, leading to overuse, premature fatigue, and cramping" (p. 113). This unbalanced relationship can result in hamstring strain and a variety of hip problems (Sahrmann, 2002).

Comparing Prolonged Sitting and Standing Groups

Is the hamstring now acting as the primary hip extensor in those people who lead sedentary lifestyles (Lambert, 2007)? In order to answer this question, our study compared the

strength and timing of the GM and the hamstring muscles between a group of individuals who engage in prolonged sitting versus a group who engage in prolonged standing. This was assessed through surface electromyographic (sEMG) analysis in two functional activities where the GM should be the primary mover: a forward step-up and going from a seated to standing position. In order to compare the sEMG activity between subjects, the sEMG was normalized by using a Maximum Voluntary Isometric Contraction (MVIC). The MVIC serves as a reference point, in which the muscle activity during the two functional tests is compared to the muscle's activity with maximal force production capabilities. The sEMG activity is recorded as a percentage of the MVIC (Ayotte, Stetts, Keenan, & Greenway, 2007; Boren et al., 2011; Distefano, Blackburn, Marshall, & Padua, 2009; Ekstrom, Donatelli, & Carp 2007; Halaki & Ginn, 2012).

Prolonged Sitting and a Lack of GM Activation

Prolonged inactivity has already been proven to cause an increase in insulin and glucose levels (Peddie et al., 2013), as well as poor sitting in a posterior pelvic tilt (Kemmoku et al., 2012) leading to compression of the sacrococcygeal area (Peterson & Adkins, 1982). Prolonged sitting and disuse of muscles have been proven to be significantly more important factors than aging when it comes to strength and cellular function (Safdar et al., 2010). However, the gap in knowledge is the effect of prolonged sitting on the gluteal muscles. Yet to be determined is whether or not the stress placed on the GM will lead to ischemia and apoptosis, thereby causing muscle weakness, even though a pressure ulcer may not be present. Although we will not be able to determine this factor directly by testing the actual cells of the muscle, finding differences in sEMG activity for those with prolonged sitting versus those with prolonged standing may justify a study to examine GM cellular structure.

In summary, GM activation, or the lack thereof, has recently been a common topic of research. Studies of interest have included lack of GM recruitment linked with low back pain (Kankaanpää, Taimela, Laaksonen, Hanninen & Olavi, 1998; Himmelreich, Vogt, & Banzer, 2008), sacroiliac instability (Hossain & Nokes, 2005), patellofemoral pain syndrome (Prins et al., 2009) (Souza et al., 2009), hip pain (Sahrmann, 2002), and muscle imbalance leading to hip instability (Sahrmann, 2002) to name a few. This study may connect the lack of GM activation with all the above pathologies associated with prolonged sitting.

Purpose

The purpose of this study was to determine if sEMG output and timing of the GM and hamstrings differs between people who sit for prolonged periods of time and people who stand for prolonged periods of time. Our study hopefully begins to answer part of the larger question of how the popular habit of prolonged sitting may affect the musculoskeletal system, as well as beginning to address a common clinical concern of physical therapists -- the inexplicably weak GM.

In functional activities where the GM should be the primary mover, we hypothesized there will be delay and decreased percent MVIC for GM firing in individuals who sit for 8 hours or more when compared to those who stand for 8 hours or more. While more research is needed in this area of inquiry, we believe that the compression of the GM during prolonged sitting, as well as general disuse, may be causing GM muscle weakness in populations that sit for prolonged periods. Alternately, it is possible that individuals who sit for 8 hours or more could have a normal activation pattern of GM followed by the hamstrings and will also have a regular or increased percent MVIC for GM firing. Possible explanations for this alternative hypothesis could be that the hamstrings and the GM are both weak, or that prolonged sitting simply does not

have an effect on hip extensor activation and strength. There is also the possibility of a null hypothesis in which there is no link between degree of sitting and muscle activation.

If prolonged sitting status appears to influence GM performance, then follow-up research may provide additional information that can be used to help educate the public about this additional health risk associated with prolonged sitting. This is important since the GM is key to the optimal function of the lower extremity (Lambert, 2007). Physical Therapists and other health professionals will be able to use our findings, not only to assist in treating various lower extremity injuries and conditions, but also as a means to encourage people to sit less, take breaks from sitting, and sit with good posture, all of which will have myriad health benefits extending far beyond the fitness of one muscle.

Methods

Research Design

The design of our study was a single session case-control study.

Subjects

Subjects were recruited through flyer postings in the Brookdale community (City University of New York schools, local hospitals and local businesses). We were able to obtain 22 healthy subjects, 11 of which were females and 11 were males. All subjects were able to read and speak English. The subjects were divided into two groups, a sitting group and a standing group, based on their reported habits. Subjects in the sitting group reported sitting for 8-10 hours a day at least 5 times per week. Subjects in the standing group reported standing for 8-10 hours a day at least 5 times per week. We excluded interested participants who did not meet the previous criteria, who were under 18 or over 50 years of age, and who had a past or current injury, surgery, or pain of the lower back, hip, knee, ankle, or foot. Participants were also excluded if they were pregnant or had a history of or currently had any neuromuscular or central nervous system diseases. We obtained 11 eligible participants for each of the sitting and standing groups, as other studies have shown that this sample size provides consistent results (Lewis et al., 2007; O'Sullivan et al., 2010). Written informed consent and questionnaires were obtained at the start of the laboratory test session with forms and procedures approved by the Hunter College Institutional Review Board Human Research Protection Program. The questionnaires were used to self-report each participant's height, weight, and age, as well as lifestyle habits. All forms were coded by one of the investigators in order to ensure the subjects' privacy and anonymity during the data reduction and analysis phases. The testing was completed in a one hour session.

Materials

Muscle activity of the GM and hamstrings was recorded using BioPac Systems, Inc EL500 wireless electrodes (Ag/AgCl 11 mm diameter foam, self-adhesive, disposable) . The same EMG machine and electrodes were used for the MVIC testing and the functional activities protocols. The BioNomadix MP150 EMG system (BioPac, Aero Camino Goleta, CA) with a 4-channel remote amplifier with a transmission of 5-500 Hz and an amplifier of +/- 10 Volts was used for data collection with a sampling frequency of 4,000 samples/sec. Raw data were band pass filtered at 50-500 Hz using MP150 data acquisition software, *AcqKnowledge*®. The maximum EMG signal amplitude during the MVIC of each muscle was recorded and represented 100% muscle activity. The muscle activity recorded during the functional activities was then expressed as a percentage of the MVIC. (Ekstrom, Donatelli, & Carp, 2007).

Procedures

All testing was performed on the participant's dominant leg: the leg with which the person would kick a ball (Ayotte, Stetts, Keenan, & Greenway, 2007; Distefano, Blackburn, Marschall & Padua, 2009). The protocol was as follows: warm up, instructions, electrode placement, MVIC, and functional testing protocol.

The warm up included walking the hallway at a leisurely, submaximal speed for 5 minutes. Participants were given brief verbal instructions and watched a demonstration given by one of the investigators about how to perform the two functional activities. The same investigator gave instructions to each participant to ensure consistency among subjects. Participants practiced the two functional activities along with the metronome until they were able to correctly perform each activity and keep pace with the metronome (Ayotte et al., 2007; Distefano et al., 2009; Mercer, Gross, Sharma, & Weeks, 2009).

Once a participant had accurate performance of the functional activities, he or she was prepared for electrode placement. Electrode sites were prepared by shaving any hair from the immediate vicinity of the muscle belly and the skin was cleansed with 70% isopropyl alcohol applied with a sterile gauze pad, in order to reduce impedance to the EMG signal and to allow for proper electrode fixation (Distefano et al., 2009; Ekstrom, Donatelli, & Carp, 2007). Electrodes were placed over the midsection of the muscle bellies as in other research evaluating muscles of the lower limb and detailed by Rainoldi et al. (2004). The placement for the electrodes for the GM was 33% of the distance between the second sacral vertebrae and the greater trochanter, starting at the second sacral vertebrae (Distefano et al., 2009). The placement for the electrodes for the biceps femoris was 35% of the distance from the ischial tuberosity and the lateral side of the popliteal fossa, starting from the ischial tuberosity (Ayotte et al., 2007). The placement for the electrodes for the semimembranosus and semitendinosus was 35% of the distance between the ischial tuberosity and the medial side of the popliteal fossa, starting from the ischial tuberosity. All electrodes were positioned well within the borders of the muscles and applied parallel to the muscle fibers in such a way that this alignment was maintained throughout the entire arc of movement (Isear, Erickson, & Worrell, 1997; Rainoldi et al., 2003; Cram et al., 1993; Ekstrom et al., 2007). Center-to-center interelectrode distance was 20 mm in order to reduce EMG crosstalk between muscles (Ekstrom et al., 2007). To ensure consistency with electrode placement, the same investigator placed all electrodes on all participants.

MVIC Testing

Maximal voluntary isometric contractions (MVICs) were performed for the GM and hamstrings to normalize muscle activation data recorded during the exercises (Ekstrom et al., 2007; Distefano et al., 2009). All subjects were securely strapped with a stabilizing strap around

the area of the posterior superior spine of the ilium, to prevent lumbar compensatory motion and to maintain pelvis alignment (Hislop & Montgomery, 2007, p. 190). Subjects performed three trials of each MVIC to ensure they understood the verbal cues and holding time. All subjects were given standard verbal cues to help them produce maximal contractions (Distefano et al., 2007). The standard manual muscle testing start position for the gluteus maximus was used: subject was lying prone with knee flexed to 90°and hip at 0° (Ekstrom et al., 2007). The subject was asked to extend their hip, moving their foot toward the ceiling, while the investigator applied an anteriorly-directed force at the distal thigh immediately above the femoral condyles. Each subject was asked to hold their strongest contraction when the hip was at 5° of extension and the knee at 90° of flexion (Distefano et al., 2007; Ekstrom et al., 2007; Sahrmann et al., 2009). For the MVIC on the hamstrings, subjects were positioned prone with 0° of hip and knee flexion and toes hanging over the edge of the table. The investigator then flexed the knee to 45° and applied an anteriorly-directed force to the distal tibia immediately above the malleoli (Hislop & Montgomery, 2002) at 0° hip flexion, knee at 45° of flexion and their trunk neutral on the table (Ekstrom et al., 2007). The third manual muscle test position was used to analyze the strength of the hamstring as a hip extensor, rather than a knee flexor. Subjects remained prone with hip at 0° of flexion and the foot resting on a pillow underneath the ankle. The subject was asked to raise the limb slightly off the pillow and hold while an anteriorly directed force was applied directly above the medial and lateral malleoli of the ankle. The subject was to keep the knee at 0° of flexion, completely extended the entire time. The same pillow was used with all subjects to ensure the same start position. All MVICs were tested with 3 separate trials holding each for 5 seconds (Distefano et al., 2009; Ekstrom et al., 2007), the highest peak value from each of the three MVIC trials were averaged to obtain one MVIC value for each muscle (Ayotte et al., 2007;

Distefano et al., 2009), and this was used as a reference value to represent the maximum force producing capabilities of that muscle. Subjects were given a 30-second rest between each repetition and a 1-minute rest between muscle groups (Ekstrom et al., 2007).

Functional Activities Testing Protocol

After MVIC testing, subjects were given a 5-minute rest break (Ayotte et al., 2007; Distefano et al., 2009; Isear et al., 1997) with electrodes still in place. Muscle activity was measured while completing two functional tests: sit-to-stand and forward step-up. Both functional tests were performed with a metronome set to 60 beats per minute to standardize repetition speed (Distefano et al., 2009). Each exercise was started with a simple verbal cue of "ready, set, go" with no increase in pitch or tone in any of the words (Ayotte et al., 2007; Lewis et al., 2009). The sit-to-stand was completed 3 times (Ayotte et al., 2009) and the forward step-up consisted of 3 sets of 5 repetitions (Ayotte et al., 2007; Mercer et al., 2009). A 1- minute rest break was given between the two functional activities (Ekstrom et al, 2007).

Sit-to-stand. Subjects were asked to sit on the high/low therapeutic table present in the testing room. The table height was adjusted so that each participants' hips and knees were both at 90° while their feet were resting flat on the floor. Feet were positioned directly below the knee at hip width apart. Participants were asked to cross their arms across their body, and the instructions given were to stand up as they normally would rise from a chair. Participants were asked to stand after hearing the beep of the metronome and then at the next beep to return to the starting position (Ayotte et al., 2009). Participants then would take a 30-second rest (Ekstrom et al., 2007), trying to be as relaxed as possible before beginning their next repetition. Repetition speeds were standardized by the use of the metronome, as described in the functional activities protocol (Ayotte et al., 2007).

Forward step-up. The height of the step used was 15.24 cm. Subjects stood in front of the step with hands on their hips, feet parallel and shoulder width apart (Ayotte et al., 2007) at whatever distance was comfortable for them (Mercer et al, 2009). After the verbal cue to start, the participant would step onto the step with their dominant leg, keeping the nondominant knee extended with the foot dorsiflexed and hip slightly extended (Ayotte et al., 2007). Subjects were instructed to move up and down the steps at one foot per beat (dominant up, non-dominant up, non-dominant down, dominant down) until 5 consecutive step-ups were completed (Ayotte et al., 2007; Mercer et al., 2009). The subjects were instructed to maintain an upright and vertical position of the head and trunk with the pelvis level throughout the exercise. Participants were given a 30- second rest between sets (Ekstrom et al., 2007). Repetitions were standardized by use of the metronome, as described in the functional activities protocol (Ayotte et al., 2007).

Data Reduction

Data were recorded and exported using *AcqKnowledge*® software and MP150 system. Raw EMG data were full-wave rectified, processed using a root-mean square algorithm. The amplitude of MVIC was calculated from a 1-sec window centered about the peak activity of all 3 trials. The three peaks were then averaged to find one sEMG MVIC value. This process was conducted for the GM, lateral hamstring (LH), and medial hamstring (MH) (Eksrom et al., 2007; Distefano et al., 2009).

For the sit-to-stand activity, the second or most representative trial was selected for analysis. Starting with the hot key marker which signified the start of the task, 2 seconds of the rectified sEMG data were averaged for each muscle and recorded in the database. For the forward step-up, the second, third and fourth trials were selected for analysis. Twelve seconds at the mid-point of the tracing of rectified sEMG data were averaged for each trial for each

muscle. The averages for the second, third and fourth trials were themselves averaged, producing one mean value. The mean amplitude of the muscle activity during the functional tasks was divided by the respective MVIC to yield a percent MVIC for each functional task, and allowed for comparisons to be made amongst subjects (Ayotte et al., 2007; Distefano et al., 2009; Boren, Conrey, & Robinson, 2011).

For order of muscle activation during the sit-to-stand task data were rectified for muscle activation then a preset algorithm determined when muscle activity was above baseline activity by at least 2.0 standard deviations. The software generated a new tracing. The time of deviation from baseline of the new tracings designated muscle onset for each sit-to-stand task. To normalize data regarding muscle timing amongst subjects a percent onset was calculated for each muscle:

percent onset = (muscle onset time-task onset time)/task duration

A hot key was placed during data collection when the person initiated movement to stand, delineating task onset. Muscle onset was obtained from sEMG tracings and the metronome was used to pace and normalize the task duration at 2 seconds. These data markers combined yielded us a percent onset for sit to stand. This was calculated for all muscles (GM, LH, MH) for all subjects.

For order of muscle activation in the forward step-up, full wave rectified data were used in combination with the hot key delineating task onset. The metronome paced the stairs task itself to be 4 seconds and a hot key was placed to identify every time the subjects dominant leg contacted the step. In order to focus on muscle onset, we used the first 1-second interval of the task and considered this phase one, or concentric activation of the GM and HS muscles. Muscle onset of the third step of the second trial of the stairs task was visually selected and change in

time (delta T) was identified with MP150 data acquisition system relative to the hot key indicator. The difference between muscle onset and task onset was divided by the task duration of 1 second to yield the time to onset for the GM, LH, and MH muscles for all subjects:

time to onset = (muscle onset-task onset)/task duration

Time to peak was also calculated for the forward step-up task. In the second trial of the repeated forward step-up, the third step was used. In the full wave rectified data the first peak after the hot key was visually found and delta T was calculated with MP150 data acquisition system, to yield a time to peak for all three muscles (GM, LH, MH) for each subject.

Statistical Analysis

Muscle activation. Normalized sEMG signal amplitudes were compared amongst subjects using a repeated measures MANOVA (Multivariate Analysis of Variance) which consisted of a group to group between-subject analysis (sitting group to standing group) and muscle to muscle (GM vs. LH vs. MG) within-subject analysis. The Wilks' Lambda statistic was used to judge significance level. The group by muscle interaction was the statistic of interest for this study. During post hoc analysis, the Bonferonni correction was applied, due to the number of comparisons made, in order to reduce chances of false positives. Dividing the standard $p < .05$ level of significance by the number of comparisons being made, which was 6, established our significance level at .008. When the MANOVA showed significance, post hoc analyses for pairwise comparisons (paired *t*-tests) were applied to find where the significant main effect was found (Ekstrom et al., 2007; Distefano et al., 2009).

Timing of muscle activation. To analyze the order of muscle activation we used non-parametric statistics, Friedman Two-Way Analysis of Variance (Friedman Test). The Friedman Test assigns a rank to the muscle activation time for raw data of each group (Lewis et al,

2009). The sit-to-stand activity and the forward step-up activity were analyzed separately. Groups were analyzed separately. We then applied a pairwise comparison, the Wilcoxon Signed Ranks test, to find if any pair of muscles differed significantly (Lewis et al., 2009).

Results

Demographics

Twenty-two healthy subjects volunteered to participate in our study. Subjects in the sitting group (mean height, 67.3 inches ± 3.7, mean body mass, 22.5 ± 1.8) had a mean age of 29.3 years ± 4.1, 8 were female, 3 were male. Of the 11 subjects in the sitting group, 8 subjects reported meeting the exercise recommendation of exercising for 4 or more days a week for at least 30 minutes, where 3 did not. Subjects in the standing group (mean height, 67.3 inches ± 4.0, mean body mass, 25.7 ± 4.2) had a mean age of 28.8 years ± 3.7, 3 were female and 8 were male. Of the 11 subjects in the standing group, 6 subjects reported meeting the exercise recommendations, where 5 did not. Post hoc analysis with the Fischers Exact test found this difference in exercise habits to be statistically insignificant with $p = .659$. Post hoc analysis regarding gender distribution was conducted with the Fischers Exact test and found that there was no significant difference regarding the distribution of gender between groups, $p = .086$.

Maximum Isometric Voluntary Contraction

The repeated measures MANOVA showed a main effect for muscles, regardless of group, between the average MVIC values. The repeated measures MANOVA showed that there was no main effect for group and no group by muscle interaction. We were mostly interested in whether the groups differed for the respective muscles. Group by muscle interactions were statistically significant ($F(2,19)=5.26$, $p=.015$). Group main effect was not statistically significant ($F(1) =1.26$, $p=.275$). However, when analyzed separately, main effect for muscle regardless of group, the Wilks' Lambda showed statistical significance ($F(2,19) = 7.77$, $p=.000$). Post hoc analysis showed that the sitting group and standing group had unique patterns of how muscles differed

from each other. Standers showed that all average MVIC values for muscles differed from each other. These results are highlighted in Table 2.

Muscle activation during functional tasks. As shown in Tables 3 and 4, repeated measures MANOVA compared percent of MVIC used during functional tasks. During sit-to-stand, the standing group had an average activity of 7.4% MVIC (standard deviation = .03) of the GM compared to 6.9% MVIC (standard deviation = .04) by the sitting group. During the forward step-up task, the sitting group GM average activation was 8.7% MVIC (standard deviation = .04) while the standing group had a muscle activation of 8.2% MVIC (standard deviation = .03). There was no significant difference for the muscle main effect, no significant muscle by group interaction and no significant differences for the group main effect between the sitting and standing groups.

Table 1

Average BMI (kg/m^2) Amongst Sitters and Standers

Gender	Group	Mean	SD
Male	Sitter	23.23	0.64
	Stander	27.03	3.93
Female	Sitter	22.29	2.02
	Stander	22.90	3.82

Note: BMI= Body Mass Index, SD = Standard deviation

Table 2

Paired Sample Test Comparing Muscle MVIC Average in Millivolts (mV)

Muscle comparison	Group	Mean	SD	*T*	*df*	*p*
GM - LH						
	Sitters	0.00	0.01	0.56	10.0	.590
	Standers	-0.01	0.01	-3.52	10.0	.006**
LH - MH						
	Sitters	-0.01	0.00	-4.76	10.0	.001*
	Standers	-0.02	0.01	-3.91	10.0	.003**
GM - MH						
	Sitters	-0.01	0.01	-3.33	10.0	.008*
	Standers	-0.03	0.01	-6.98	10.0	< .001**

Note: SD = Standard Deviation, GM= Gluteus Maximus, LH= Lateral Hamstring, MH= Medial Hamstring

***** MH is statistically different from the LH and from the GM in the sitters group

**The MVIC average for each muscle in the standers group are statistically different

Table 3

Percent MVIC Used During the Forward Step-Up Task for the Gluteus Maximus, Lateral Hamstring, and Medial Hamstring Muscles

	GM		LH		MH	
Group	% Stairs	SD	% Stairs	SD	% Stairs	SD
Sitters	8.7	0.04	7.3	0.02	.6.1	4.3
Standers	8.2	0.03	6.4	0.03	6.9	7.1

Note: SD = Standard deviation, GM= Gluteus Maximus, LH= Lateral Hamstring, MH= Medial Hamstring

Table 4

Percent MVIC Used During the Sit-to-Stand Task for the Gluteus Maximus, Lateral Hamstring, and Medial Hamstring Muscles

	GM		LH		MH	
Group	% STS	SD	% STS	SD	% STS	SD
Sitters	6.9	0.04	3.3	0.14	7.5	0.05
Standers	7.4	0.03	9.1	0.07	6.2	0.05

Note: SD = Standard deviation, GM= Gluteus Maximus, LH= Lateral Hamstring, MH= Medial Hamstring

Timing Results for Sit-to-stand Task

The repeated measures MANOVA compared muscle onset activity during the sit-to-stand task. Group by muscle interactions were not statistically significant ($F(2,17)=3.49$, $p=.054$). Group main effect as measured by Wilks' Lambda was not statistically significant ($F(2,17) =2.65$, $p=.100$). We also conducted a Two-Way ANOVA for a between group analysis which also proved no statistical differences ($F(1, 18) =.77$, $p=.391$).

The Friedman Test yielded no significant difference with a mean rank of 1.75 for the GM, 1.90 for the LH, and 2.35 for the MH in the sitting group ($\chi^2=2.17$, $p=.338$). The standing group had a mean rank of 1.60 for the GM, 2.40 for the LM, and 2.00 for the MH ($\chi^2=3.20$, $p=.202$).

Post hoc Wilcoxon tests were also conducted to compare timing during the sit-to-stand task (Table 5) between the muscles for each group. These results did not demonstrate a clear pattern.

Timing Results for Forward Step-up Task

The repeated measures MANOVA compared muscle onset of phase 1 during the forward step-up task. Group by muscle interactions were not statistically significant ($F(2,19)=.38$, $p=.691$). Group main effect as measured by Wilks' Lambda was not statistically significant ($F(2,19) =5.52$, $p=.013$). We also conducted a Two-Way ANOVA for a between group analysis which also proved insignificant statistical differences ($F(1, 20) =.01$, $p=.944$).

The Friedman Test yielded a mean rank of 2.45 for the GM, 1.59 for the LH, and 1.95 for the MH in the sitting group ($\chi2=4.23$, $p=.120$). In the standing group the Friedman Test demonstrated a statistical difference between the muscles with a mean rank of 2.73 for the GM, 1.45 for the LH, and 1.82 for the MH ($\chi2=9.91$, $p=.007$). The descriptive statistics are illustrated below (Table 6).

Table 5

Wilcoxon Tests Comparing Muscle Percent Onset During the Sit-to-Stand Task in Sitters and Standers

Muscle	N	LH < GM	LH > GM	LH = GM	MH < LH	MH > LH	MH = LH	MH < GM	MH > GM	MH = GM
Sitters	10	4	5	1	2	5	3	3	7	0
Standers	10	2	8	0	6	4	0	4	6	0

Note: N= Number of subjects, GM= Gluteus Maximus, LH= Lateral Hamstring, MH= Medial Hamstring

Table 6

Descriptive Statistics for the Gluteus Maximus, Lateral Hamstrings, and Medial Hamstrings Mean Time to Onset in Standers During the Forward Step-up Task

Muscle	N	Mean	SD	Minimum	Maximum
GM	11	0.194	0.109	0.070	0.390
LH	11	0.135	0.094	0.023	0.280
MH	11	0.172	0.119	0.23	0.410

Note: N= Number of subjects, GM= Gluteus Maximus, LH= Lateral Hamstring, MH= Medial Hamstring

Post hoc Wilcoxon tests were also conducted to compare timing (Table 7) between the muscles for each group during the forward step-up task. These results did not demonstrate a clear pattern.

A second analysis was conducted comparing time to peak rather than time to onset during the forward step-up task. The repeated measures MANOVA compared muscle time to peak of phase 1. Group by muscle interactions were not statistically significant ($F(2,19)=.44$, $p=.650$). Group main effect as measured by Wilks' Lambda was not statistically significant ($F(2,19) =3.13$, $p=.067$). We also conducted a Two-Way ANOVA for a between group analysis which also proved insignificant statistical differences ($F(1) =2.78$, $p=.111$).

In the time to peak analysis, the Friedman Test yielded a mean rank of 2.36 for the GM, 1.86 for the LH, and 1.77 for the MH in the sitting group ($\chi2=2.51$, $p=.285$). In the standing group the Friedman Test demonstrated a mean rank of 2.59 for the GM, 1.64 for the LH, and 1.77 for the MH ($\chi2=6.29$, $p=.043$) which demonstrated the similarities between the groups and no significant differences.

Table 7

Wilcoxon Test Comparing Muscle Percent Onset During Phase One of the Forward Step-Up Task

Muscle	N	LH < GM	LH > GM	LH = GM	MH < LH	MH > LH	MH = LH	MH < GM	MH > GM	MH = GM
Sitters	11	8	3	0	3	7	1	8	3	0
Standers	11	10	1	0	3	6	2	9	2	0

Note: N= Number of subjects, GM= Gluteus Maximus, LH= Lateral Hamstring, MH= Medial Hamstring

Discussion

This study examined the sEMG output and timing of the gluteus maximus and hamstring muscles to uncover any differences among people who sit for prolonged periods of time and people who stand for prolonged periods of time. We hypothesized that we would find a connection between prolonged sitting and a common clinical finding of weak gluteus maximus muscles.

Muscle Activation

When comparing the percentage of MVIC used by each muscle during our two functional tasks, the repeated measures MANOVA showed no significant difference for the Muscle Main Effect, no significant difference for Muscle by Group Interaction, and no significant difference for the Group Main Effect between the sitting group and the standing group. This means that, for the most part, people who sit all day and people who stand all day use their GM and HS similarly when compared to each other, in terms of what percentage of maximal muscle output they use during our two functional tasks. This goes against our hypothesis that the sitting group's GM would have a decreased output during functional tasks, but perhaps our findings are affected by the overall fitness and youth of our subjects.

Our sample was one of convenience, and most of our sitters were physical therapy students, who were 36 years old or younger. In response to the exercise queries in our questionnaire, the sitting group revealed that 8 out of 11 met the weekly exercise recommendations, and also reported a greater variety of types of exercise. If we had looked at sedentary office workers who could report a decade or more of prolonged sitting, perhaps the results may have supported our hypothesis. It is quite possible that the Active Couch Potato phenomenon does not occur in the musculoskeletal system in the same way that it does in

metabolic, cardiovascular, and hormonal systems of the body, as noted by Owens et al. (2010), Biwas et al. (2015), and Healy et al. (2008).

It is also interesting to note that in the research on muscle activity in stair climbing performed by Zimmerman et al. (1994), it was found that the percentage of MVIC used by the GM increased significantly with a quicker cadence on the stairs. The speed we chose for our forward step-up task was close to the slowest speed (35 steps per minute) in the Zimmerman, et al. study. They demonstrated 19.8% of MVIC in the GM for the slow speed stepping, where Worrell et al. (1998), used a metronome set to 30 beats per minute and found subjects used 15-23% of GM MIVC. Ayotte et al. (2007) found that subjects used 56-84% of MVIC in the GM during a forward step-up with the metronome paced to 40 beats per minute, where our subjects used between 8.2-8.7% of GM MVIC, with a metronome paced to 60 beats per minute. Methodological differences in step height, step-up cadence and MVIC testing positions likely account for reported differences.

Another discrepancy to draw attention to is the study by Ayotte et al. (2007). Their results from the mean normalized EMG signal amplitude for the GM during the forward step-up was 74% with a standard deviation of 43%. We found that the sitting and standing group used less than 10% of their MVIC during functional tasks. The variations in the studies may have contributed to the significant difference in muscle activity. We attempted to distinguish one factor; sitting or standing. It is important to realize many factors play a role in individual muscle activity. Although our study did not prove our hypothesis, it is a reason further research should consider other factors (exercise activity, years spent sitting, age).

Timing of the Muscle Activation

A Friedman 2-way analysis of variance (ANOVA) was used to look at the rank order of activation of the muscles, revealing a statistical difference between the muscles within the respected groups. The GM and LH in the standing group demonstrated a significant difference during the forward step-up task. Contradictory to our hypothesis, the LH activated first and the GM activated last. We also used the Wilcoxon test to compare the timing of the onset of muscle activity in the forward step-up task. Unfortunately, there was no clear pattern of activation.

We did observe, when visualizing the timing data for the forward step-up task, that there was an overall pattern of greater HS activation compared to GM activation in both the sitting and the standing group, as expressed by the percent of the task during which the muscle was firing. This might be explained again in part by the slow speed (one second per step) at which we asked the subjects to perform the task, since according to Zimmerman et al. (1994), Neumann (2002), and Bartlett et al. (2014), the GM is known to activate most strongly during fast and forceful hip extension. Janda and Liebenson (2007), as well as Lewis and Sahrman (2009), stated that during prone hip extension, an activation of the HS before the GM is indicative of a muscle imbalance. However, overall research results of this timing have been conflicting, and prone hip extension does not have the exact same biomechanics and forces as exist in our functional tests. Wagner et al. (2010) also argued that the HS will take over for the GM when the GM is weak, but from our results, this would mean that most of our subjects, both sitters and standers, had weak GM muscles, which from clinical observation seemed unlikely.

Another consideration to be taken into account for our forward step-up findings was the placement of our electrodes, which we derived from the detailed research on the GM performed by Distefano et al. (2009). According to Lyons, Perry, Gronley, Barnes, and Antonelli (1983),

the lower portion of the GM extends the hip during stair ascension, whereas the upper portion of the GM acts more like the Gluteus Medius, stabilizing the hip. Our electrodes were placed more on the upper portion of the GM, being 33% of the distance between the second sacral vertebrae and the greater trochanter, starting from the second sacral vertebrae.

We also observed that the GM appeared to typically activate first in both groups during the sit-to-stand task. This did not support our hypothesis that the sitting group would have a delayed activation of the GM. This result actually indicated that both groups were using their GM properly to initiate hip extension during the sit-to-stand task. Again, our young and mostly fit sample of convenience may be the cause of this finding of normal, unremarkable activation of the GM. Our sitting group subjects were mostly physical therapy students who tend to be aware of their posture and know how to sit with proper alignment. Perhaps good alignment, frequent postural adjustments, and regular breaks/ or regular exercise mitigated the effects of compression that we expected to see in sitters, based on clinical observations and the work of Sahrmann (2002), Linder-Ganz et al. (2007), and Gawlitta et al. (2007).

Although not statistically significant, there was a difference in time to onset for the MH during the sit-to-stand task in the standing group. If we had a larger sample, these results would have proven to be statistically significant.

Limitations

Although the sample size of eleven subjects per group was determined based on previously conducted research such as Lewis and Sahrmann (2009), and Ekstrom and Donatelli (2007), it may not have been enough subjects to find statistical significance between the two groups. For example, the Wilks' lambda group by muscle interaction had a *p*-value of 0.054 which is not statistically significant; however, the MH had a lower value in the standing group

compared to the sitting group and may have shown statistical significance if a larger sample was used. As mentioned earlier, the sample used was a sample of convenience and was not a randomized sample. The sitting group consisted mainly of physical therapy students and subjects in both groups had a very narrow age range. Physical therapy students have learned about correct sitting posture, the need to take frequent breaks from sitting, the benefits of exercise, and how to properly perform sit-to-stand. This knowledge base may set them apart from typical sedentary individuals. The mean average age of sitters was 29.7 years with a standard deviation of 4.05 and the standers had a mean of 28.82 years with a standard deviation of 3.73. According to Mercer at al. (2009), the level of maximal muscle strength is lower in older adults than in younger adults and older adults who utilize a larger percentage of their neuromuscular capacity for daily tasks. This could account for the low percentages of MVIC that were utilized during functional tasks in our study as our sample was all younger adults.

In reference to our method, there are several areas of limitation. Information gathered from subjects such as hours spent sitting or standing, whether or not the exercise requirement was met, height, weight, and whether they currently, or in the last five years experienced significant pain or lower extremity injury, were all self-reported and thus are subject to error. Sitting and standing posture were not assessed, which would have provided additional insight into our results. Whether an individual sits with a posterior pelvic tilt or on their ischial tuberosities may influence the amount of compression applied to the GM. Kemmoku, Furumachi, and Shimamura (2012) found that shearing force on the ischial tuberosity and the sacrococcygeal areas increased with increased posterior pelvic tilt. For example, a 20-degree posterior pelvic tilt resulted in an 11% increase in pressure (Kemmoku, Furumachi, & Shimamura, 2012). SEMG of the two functional activities was collected without the use of

kinematic data. In order to help link the data correctly to the activity, a metronome was used to standardize the timing and a hot key was used as a marker. This system allowed for human error during data collection and the use of subjectivity when analyzing the data. Both of these sources of error could be greatly reduced with the use of kinematic data.

There are also inherent limitations with sEMG and normalizing the data by using the % MVIC. SEMG recordings can contain electrical artifacts, mechanical artifact, and cross-talk which can contaminate the data sample. Electrical artifacts are electrical noise that occurs when the electrodes are not firmly attached to the skin, a monopolar recording is used, or a ground electrode is not used (Turker, 1993). Our study utilized a bipolar reading technique with ground electrodes, the skin was shaven if hair was present, and the skin was cleansed with an alcohol soaked gauze. In the absence of hair, friction was applied to the skin with the gauze in attempts to abrade the skin to increase conductivity. However, despite these measures it is possible that electrical artifact was present and that mechanical artifact occurred due to movement of the electrodes. Although standard electrode placement was followed for each muscle, it is possible that cross-talk occurred. Cross-talk is defined as recorded electrical activity from muscles other than the muscle of interest, which can include agonists, antagonists, the heart, and the brain. It has also been debated whether or not the onset of muscle activity can accurately be determined by the use of sEMG because it is decided subjectively and the investigator must be able to determine the difference between genuine muscle activity and artifact (Turker, 1993).

Although it is agreed upon that sEMG needs to be normalized in order to be compared across subjects, which method is best and how reliable the method of normalization by MVIC has been debated by researchers (Burden, 2010). Factors that influence MVIC include the individuals understanding of maximal contraction, different joint angles, the rate of contraction

force, and the verbal cues given. Lewis and Sahrmann (2009) found that muscle activation can be influenced by verbal cues. Although the manual muscle tests during MVIC calculation were performed by the same investigator each time, variation in the investigator's tone and level of encouragement could have influenced the subject's performance. According to Ekstrom et al. (2007), there is the potential that subjects did not generate a true MVIC for each muscle. This could be due to lack of effort or the muscle testing position may not be optimal for producing a maximum sEMG signal. It has also been suggested that without training, the MVIC obtained can be 20-30% less than that obtained after appropriate training (Merletti, 1999).

Further Research

Due to these limitations further research is warranted in order to determine if sEMG output and timing of the GM and hamstrings differs between people who sit for prolonged periods of time and people who stand for prolonged periods of time. Suggestions for further studies include gathering a larger sample size with equal numbers in each group of males and females with the same body type, using a randomized sample that better represents the general public (with better variety of ages and occupations), increasing the inclusion criteria to require a minimum number of years spent sitting or standing for prolonged periods, and utilizing kinematic motion analysis during the functional activities. To obtain more accurate information, sitting and standing postures should be assessed, and subjects that stand statically or dynamically should be differentiated. Within both groups, subjects who exercise should be separated from non-exercisers in order to obtain more correct results.

Despite our results, we continue to believe that the habit of prolonged sitting may have negative effects on the musculoskeletal system. With further research, the relationship may be discovered. Once that occurs, physical therapists and other health professionals may educate the

public on the deleterious musculoskeletal effects of prolonged sitting and the best strategies to avoid these effects, similar to those suggested by Peddie et al. (2013) for prevention of the metabolic effects of prolonged sitting.

Conclusion

Although there are a wide range of studies that have assessed the effects of prolonged sitting on one's health, to our knowledge there are currently no studies that have tried to determine the effect that sitting has on the muscle activity and recruitment of the GM during functional activities. We attempted to evaluate this by comparing the sEMG muscle activity and the timing of muscle activation for the GM, LH, and MH during sit-to-stand task and forward step-up task in subjects that sit for 8 hours or more a weekday to those who stand for 8 hours or more a weekday. During the sit-to-stand task and the forward step-up task the muscle activation had no significant statistical difference for the muscle main effect, muscle by group interaction, or group main effect in either group. For timing during the sit-to-stand task group by muscle interaction, group main effect, and between groups were not statistically significant. For timing during the forward step-up task time to onset of phase 1 and time to peak were not statistically significant. No statistical significance was found for mean rank when looking at the sitters or standers during sit-to-stand task or time to peak of forward step-up, but a statistical difference was found between the muscles in the standing group for the time to onset of phase 1 during forward step-up task. No clear muscle activating pattern was found within either group, in either functional task. The results of this study indicate that the GM, LH, and MH in sitters and standers perform similarly during sit-to-stand task and the forward step-up task. This supports a null hypothesis- that there is no link between prolonged sitting and the muscle activity and timing of the GM. However, this could be due to a small sample size, a narrow age range, and a prolonged sitting sample that exercised regularly and were educated on sitting posture, and thus did not accurately represent the typical sedentary individual. Due to these limitations, further research is required to determine whether or not prolonged sitting can be linked to decreased

sEMG muscle activity and timing of the GM. Determining whether or not there is a link is of societal and clinical concern because it would help explain the clinically observed weak GM and would further support the importance of sitting less, taking frequent breaks from sitting, and sitting with good posture.

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