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Naoko Aminaka

Brian G. Pietrosimone

Charles W. Armstrong

Andrew Mezsaros
George Fox University, amezsaros@georgefox.edu

Phillip A. Gribble

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Naoko Aminaka *, Brian G. Pietrosimone, Charles W. Armstrong, Andrew Meszaros, Phillip A. Gribble

The University of Toledo, Toledo, OH, United States

ABSTRACT

The aim of the study was to investigate differences in frontal plane knee kinetics, onset timing and duration of the gluteus medius (GMed), adductor longus (AL), and vastus medialis oblique (VMO) during stair ambulation between those with and without patellofemoral pain syndrome (PFPS). Twenty PFPS patients and twenty healthy participants completed stair ambulation while surface electromyography (EMG), video, and ground reaction forces were collected. PFPS patients had a higher peak internal knee abduction moment during stair ascent, and a higher internal knee abduction impulse for both ascent and descent. During stair ascent, PFPS patients displayed earlier onset of the AL and later onset of GMed, compared to the healthy individuals. Also, PFPS patients had longer activation duration of the AL and shorter activation durations of the VMO and GMed during stair ascent. During stair descent, PFPS patients displayed delayed GMed onset and shorter activation duration of GMed and VMO. The results of the study suggest that altered neuromuscular control of the medial thigh musculature may be an important contributor to PFPS.

1. Introduction

Recently, researchers have started to investigate the association between patellofemoral pain syndrome (PFPS) and proximal muscular function (Brindle et al., 2003; Robinson and Nee, 2007; Bolgla et al., 2008; Dierks et al., 2008; Cowan et al., 2009). While much of the current research focuses on the association between lateral hip muscle function and PFPS, little is known about how hip adductor muscle function may be affected by PFPS. The VMO and the hip adductor group, mainly adductor magnus and longus, are connected through a thin membrane known as the vastoadductor membrane (Bose et al., 1980; Checroun et al., 1996; Tubbs et al., 2007). Because of this anatomical connection, the hip adductor group is theorized to act as a pulley to increase the mechanical efficiency of the VMO. While this function of the hip adductor group may provide some mechanical advantage, it could also be a compensatory mechanism for altered VMO function, contributing to weakening or inhibition of the VMO in those with PFPS. However, this theory has not been substantiated, as previous studies investigating hip adductor strength have yielded inconsistent results (Niemuth et al., 2005; Cichanowski et al., 2007).

With excessive joint loading, surrounding muscles may respond by producing an opposing internal joint moment. Joint impulse is a product of joint moment and time, and therefore it provides information regarding the total joint load during a given period of time. Because PFPS is theorized to be associated with increased knee abduction (valgus) (Powers, 2003), one can speculate that internal knee adduction moment and knee adduction impulse are increased in these patients in an attempt to resist the external knee abduction moment. It could also be possible that imbalance among the hip adductor group, VMO, and gluteal muscles, may increase internal knee adduction moment in those with PFPS. However, to our knowledge, no current research has investigated the frontal plane knee joint kinetics and EMG activation onset and duration of the lower extremity muscles simultaneously during a functional task, such as stair ambulation. Combining these variables would provide more insight into muscular contributions to altered knee moment and impulse.

Therefore, the aims of the study were to (1) investigate group differences in knee adduction moment and impulse, and (2) examine group differences in muscle activation onset timing and activation duration in the VMO, adductor longus (AL), and gluteus medius (GMed) muscles, during stair ascent and descent. We hypothesized that (1) internal knee adduction moment and impulse would be greater in those with PFPS during both stair ascent and descent; (2) PFPS participants would display earlier AL onset and longer activation duration during both stair ascent and descent, compared with the healthy participants; and (3) PFPS participants would display later onset and shorter activation duration of the VMO and GMed during both stair ascent and descent, than the healthy participants.

* Corresponding author. Address: 2801 W. Bancroft St., Mailstop 119, Toledo, OH 43606, United States. Tel.: +1 419 530 4351; fax: +1 419 530 2477.

E-mail address: Naoko.Aminaka2@utoledo.edu (N. Aminaka).

2. Methods

2.1. Participants

This case control study included 20 PFPS and 20 healthy individuals (Table 1). Participants were recruited from the university and the surrounding community. The PFPS group (1) were diagnosed with PFPS by a licensed health care professional, which may have included an athletic trainer, physical therapist, or physician, and, as confirmed with presence of pain and/or tenderness upon palpation of the patella; (2) experienced diffuse anterior knee pain for at least 8 weeks; and (3) had increased knee pain when ascending and descending stairs, and during at least one of the following activities: going up or down hills, after sitting for a prolonged period of time, walking, running, and squatting (Aminaka and Gribble, 2008). Participants were excluded from the PFPS group if they had previous history of lower extremity injury (other than PFPS) or surgery. We excluded those who were currently receiving or had received lower extremity rehabilitation within the last year.

Healthy participants had no history of lower extremity injury or knee pain. Healthy participants were matched to the PFPS participants by gender, age, height and mass, and were assigned "symptomatic" and "asymptomatic" legs according to the matched PFPS participant's symptomatic and asymptomatic legs.

All participants were free from any neurocognitive deficits that affect postural control and ability to ascend/descend the stairs without ambulatory assistance. Each participant provided written consent, which was approved by the Biomedical Institutional Review Board prior to the participation in the study.

2.2. Procedures

The participant warmed up on a stationary bicycle (Monark Ergomedic 828E Exercise Test Cycle, Monark Exercise AB, Vansbro, Sweden), at a rate of 50–60 rpm and self-selected resistance for 5 min. During a 5-min rest, the participant's skin was cleaned, lightly debrided with sand paper, and shaved if necessary. Disposable 0.8 cm-diameter Ag/AgCl surface electrodes with a center-to-center inter-electrode distance of 1.5 cm (Noraxon U.S.A. Inc., Scottsdale, AZ) were placed for surface electromyography (EMG) recording of the GMed, AL, and VMO. For the GMed, the electrodes were placed half-way between the highest point of the iliac crest and the femoral greater trochanter (Delagi, 1981; Hermens et al., 2000). The electrodes for the AL were applied on the anteromedial thigh at the proximal one third of the distance between the pubic symphysis and the adductor tubercle. For the VMO, the electrodes were placed 4 cm proximal to the superior-medial angle of the patella at a 55° angle from the line of the femur (Delagi, 1981; Hertel et al., 2005).

Kinematic, kinetic, and surface EMG data were collected synchronously using Cortex 1.0.0.198 motion capture software (Motion Analysis Corporation, Santa Rosa, CA). A 12-Eagle digital camera (Motion Analysis Corporation) passive marker system recorded the participant's lower limb movement at 100 Hz. Retroflective marker placements are shown in Fig. 1. An 8-channel telemeterized surface EMG system (Noraxon U.S.A. Inc.) recorded muscle activity at 1000 Hz. Unit specifications for the EMG system

included the baseline noise of >1 μV, input impedance of >100 mOhms, and a common-mode rejection ratio of >100 dB. The participant's static data were collected while quietly standing on the force plate (AMTI OR-7, Advanced Mechanical Technology Inc., Watertown, MA, 1000 Hz sampling rate) for 5 s with their arms crossed in front of the chest. The middle 2 s were used to obtain the baseline measures of the EMG for each muscle, since we determined that the participants achieved the quietest stance with minimum movement during that period, and therefore we would be able to obtain baseline activities of the muscles with minimum noise and motion artifacts. Then, the participant was asked to walk up and down the 4-step stairs at a self-selected pace. We utilized a custom built stair case with a standard step height and depth (Fig. 2). For both stair ascent and descent, the second step was used for data analysis. The second step of the stairs consisted of an 80-lb box, which was placed directly on the force plate. The pre-loading of the box was done to minimize any motion artifacts when the participant stepped on it. The stair case had an opening to receive the box to allow it serve as the second step, but without contacting the rest of the staircase and thus avoiding noise artifact when in contact with the other stairs.

Each stair ambulation task was performed 5 times starting with each leg, for the total of 10 trials. The order of the starting leg was randomized to minimize the effect of fatigue across participant. Trials were recollected if the participant placed more than one foot on the step, or missed the second step. Up to 1 min of rest was allowed between trials to avoid fatigue.

2.3. Data processing

Visual 3D Basic/RT software (C-Motion Inc., Germantown, MD) was used for data processing. Stance phase was defined as the period when the foot was in contact with the second step. Foot strike and toe off were determined as the time at which the ground reaction force exceeds above or fell below 10 N, respectively. Knee kinetics were calculated using inverse dynamics, and normalized to the participant's height and mass. Peak knee moments were identified as the maximum value during the stance phase. A positive value was determined as knee adduction moment, and a negative value as knee abduction moment. Knee adduction/abduction impulses were calculated as the area under the curve of the knee moment over the entire stance period.

The EMG signals for quiet standing and stair ambulation trials were full-wave rectified, band-pass filtered at 20–500 Hz, and processed using the root mean square calculation over the 55-ms window. Activation onset (milliseconds) for GMed and AL during the stair ambulation trials was defined as the time when the EMG amplitude exceeded 3 standard deviations (SDs) of baseline for a minimum of 25 ms prior to or after the initial foot contact (Cowan et al., 2002; Brindle et al., 2003; McClinton et al., 2007). For the VMO, many participants exceeded this original threshold value for the entire data collection period. Therefore, a modified threshold value for the VMO was set as 10% of the mean peak amplitude across the ascending or descending trials for each participant. Since the objective of this study was to analyze activation onset and duration of individual muscles across groups, we determined that setting a different onset threshold for the VMO would not negatively affect the outcome of the study. A negative onset value

Table 1
Participant demographics.

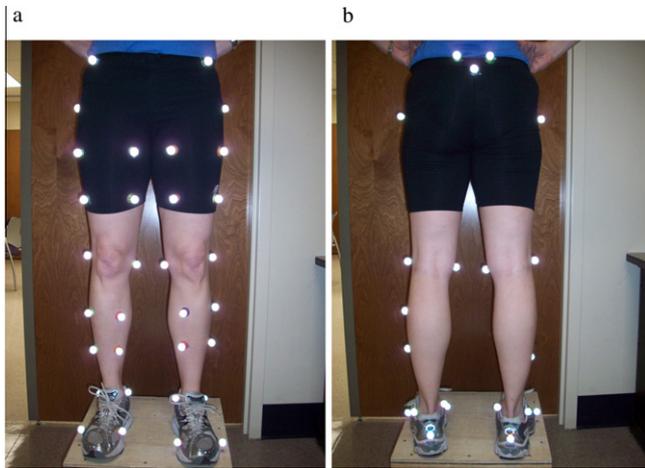|	N	Age (years)	Height (cm)	Weight (kg)	Body mass index
PFPS	20 (13F/7M)	21.45 ± 3.90	169.96 ± 10.47	71.30 ± 14.50	24.59 ± 4.13
Healthy	20 (13F/7M)	21.35 ± 3.76	172.21 ± 9.24	69.68 ± 9.78	23.48 ± 2.68

Fig. 1. Retroflective marker placement for the lower extremity. (a) Anterior view. (b) Posterior view.
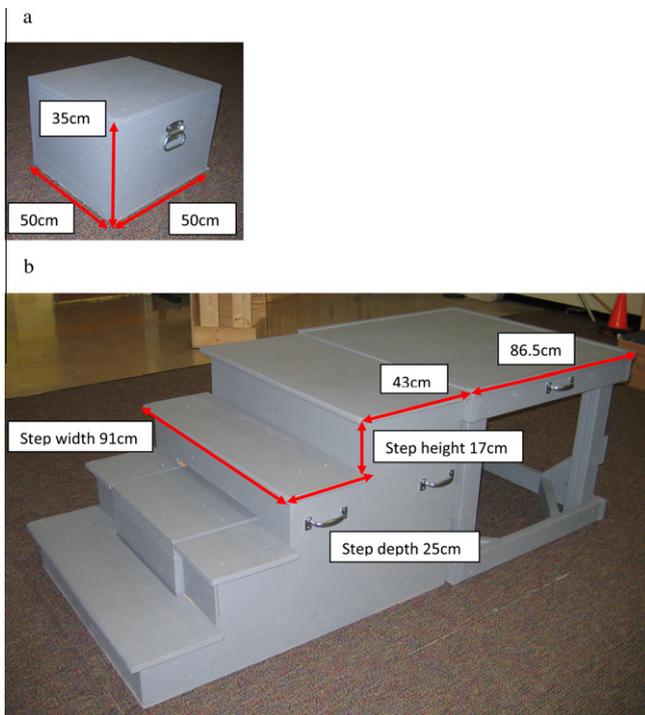

Fig. 2. Stair configuration on the force plate. (a) Box specification on the force plate. (b) Stair configuration.

indicated that the activation onset occurred prior to foot contact, and a positive value indicated that the activation onset occurred after foot contact. Muscle activation duration (milliseconds) was defined as the time between the activation onset and when the EMG amplitude fell below 3 SDs of baseline for a minimum of 25 ms for the AL and GMed, and below 10% of the mean peak amplitude for the VMO, after the initial foot contact.

2.4. Statistical analysis

Independent variables included group (PFPS and healthy), and side (symptomatic and asymptomatic). Dependent variables for each of the stair ambulation tasks (ascent and descent) included peak knee adduction moment, knee adduction impulse, activation onset and duration of the VMO, AL, and GMed. For our purposes,

stair ascent and descent were being considered as unique tasks that did not have direct statistical comparison. For each dependent variable, a separate two-way (group × side) analysis of variance was utilized to detect between- and within-group differences. Statistical Package for Social Science version 15.0 (SPSS Inc., Chicago, IL) was used for data analysis. Post hoc univariate analyses were performed in the event of statistically significant interactions. Additionally, Cohen's d was used to indicate effect sizes, along with the associated 95% confidence intervals (CI).

3. Results

3.1. Knee kinetics

3.1.1. Peak knee frontal plane moment

There was no side-by-group interaction for both stair ascent ($p = 0.707$) and descent ($p = 0.786$, Table 2). Similarly, there was no effect of side for the peak knee frontal plane moment for stair ascent ($p = 0.817$) and descent ($p = 0.927$). During stair descent, no group difference was observed for the peak knee frontal plane moment ($p = 0.198$). During stair ascent, the PFPS group displayed significantly higher knee abduction moment compared to the healthy group ($p = 0.008$, $d = -0.76$ [95% CI = -1.41, -0.13]).

3.1.2. Knee frontal plane impulse

During both stair ascent and descent, no significant side-by-group interaction was observed (ascent: $p = 0.355$; descent: $p = 0.464$, Table 2). Similarly, no effect of side was observed for either task (ascent: $p = 0.805$; descent: $p = 0.478$). The PFPS group, for both stair ascent and descent, demonstrated more knee abduction impulse, as compared to the healthy group (ascent: $p = 0.006$, $d = -0.78$ [-1.13, -0.14]; descent: $p = 0.044$, $d = 0.54$ [-1.17, 0.09]).

3.2. Muscle onset and duration

3.2.1. Ascent

3.2.1.1. Vastus medialis oblique. There was no side-by-group interaction for VMO onset during stair ascent ($p = 0.197$, Table 3). Similarly, there was no significant main effect of side ($p = 0.224$) or group ($p = 0.197$).

For the VMO activation duration during stair ascent, no side-by-group interaction was observed ($p = 0.662$). There was no main effect of side ($p = 0.353$). The PFPS participants demonstrated significantly shorter activity duration of the VMO, compared to the healthy participants ($p < 0.001$, $d = 1.00$ [0.34, 1.65]).

3.2.1.2. Adductor longus. For the AL onset during stair ascent, no significant side-by-group interaction was observed ($p = 0.165$, Table 3). While no significant group differences were observed ($p = 0.961$), a significant side main effect was observed ($p = 0.038$, $d = -0.46$ [-0.05, 0.96]). Post hoc test revealed that the asymptomatic leg in the PFPS group displayed a significantly earlier onset of the AL prior to the initial contact, compared with the symptomatic side ($p = 0.012$).

As for the AL activation duration, no side-by-group interaction was observed ($p = 0.662$). The side main effect for the AL activation duration was statistically significant ($p = 0.041$, $d = -0.41$ [-0.91, 0.10]), although there was no group difference for the AL activation duration ($p = 0.164$). The AL duration was significantly longer on the asymptomatic leg than the symptomatic leg in the PFPS group ($p = 0.035$).

3.2.1.3. Gluteus medius. A significant side-by-group interaction was observed for the activation onset of the GMed during stair ascent ($p = 0.028$, Table 3). Post hoc pairwise comparison with SIDAK

Table 2
Knee frontal plane peak moment (N m/kg m) and impulse (N m/kg m s) during stair ambulation.

	Healthy		PFPS	
	Symp	Asymp	Symp	Asymp
Peak moment				
Ascent[a]	0.0081 ± 0.019	0.0098 ± 0.022	−0.0050 ± 0.017	−0.0054 ± 0.014
Descent	−0.011 ± 0.017	−0.0098 ± 0.024	−0.0033 ± 0.018	−0.0039 ± 0.017
Impulse				
Ascent[a]	0.11 ± 0.21	0.14 ± 0.28	−0.041 ± 0.24	−0.085 ± 0.21
Descent[a]	0.078 ± 0.20	0.079 ± 0.25	−0.023 ± 0.23	−0.081 ± 0.26

[a] A significant group main effect.

Table 3
Lower extremity muscle onset and duration during stair ascent.

	Healthy		PFPS	
	Symp	Asymp	Symp	Asymp
Onset (ms)				
VMO	25.83 ± 67.89	23.69 ± 56.45	27.50 ± 75.37	59.72 ± 25.74
AL[a]	−100.88 ± 134.45	−124.53 ± 129.18	−58.36 ± 171.87	−171.91 ± 196.23
GMed[b,c]	69.50 ± 37.78	57.30 ± 63.09	92.38 ± 42.40	109.82 ± 46.47
Duration (ms)				
VMO[b]	899.07 ± 357.35	839.49 ± 243.00	634.72 ± 168.26	613.14 ± 154.68
AL[a]	521.11 ± 318.51	590.91 ± 278.85	596.55 ± 254.48	755.00 ± 254.25
GMed[b]	706.55 ± 122.59	680.75 ± 182.65	633.64 ± 146.91	552.70 ± 215.12

Symp = symptomatic leg; Asymp = asymptomatic leg.
[a] A significant side difference.
[b] A significant group difference.
[c] A significant side-by-group interaction (the asymptomatic leg of the PFPS displaying later GMed onset than the asymptomatic leg of the healthy group).

adjustment revealed that the PFPS group demonstrated with significantly later onset of the GMed than the health group on the asymptomatic leg ($p = 0.005$, $d = −0.28$ [−0.90, 0.34]); however, the group difference in the GMed activation onset on the symptomatic leg was not statistically significant ($p = 0.08$). There was no main effect of side ($p = 0.689$); however, a significant group main effect was observed ($p = 0.01$, $d = 0.52$ [−0.12, 1.14]). The PFPS group had significantly later activation onset of the GMed compared to the healthy group.

For the GMed activation duration during stair ascent, no side-by-group interaction was observed ($p = 0.352$). No significant main effect of side was observed ($p = 0.076$), although the symptomatic side, regardless of the groups, seemed to show shorter activation duration compared to the asymptomatic side. The GMed activation duration was significantly shorter in the PFPS participants than in the healthy participants ($p = 0.033$, $d = −0.60$ [−1.22, 0.04]).

3.2.2. Stair descent
3.2.2.1. Vastus medialis oblique.
There was no side-by-group interaction ($p = 0.219$, Table 4). No group difference was observed for the activation onset of the VMO during stair descent ($p = 0.758$). Similarly, there was no effect of side on the VMO onset ($p = 0.216$). For the activation duration of the VMO, there was no side-by-group interaction ($p = 0.418$). Similarly, there was no statistically significant group difference ($p = 0.947$). Regardless of group, the symptomatic leg demonstrated significantly shorter activation duration of the VMO compared to the asymptomatic leg, during the stair descending task ($p = 0.017$, $d = −0.48$ [−0.94, −0.02]).

3.2.2.2. Adductor longus.
For the AL activation onset during stair descent, there was no side-by-group interaction ($p = 0.609$, Table 4). Similarly, no statistically significant side ($p = 0.539$) or group ($p = 0.906$) difference was observed for the activation onset during stair descent.

For the AL activation duration, no side-by-group interaction ($p = 0.542$) or side difference ($p = 0.785$) was observed. The group difference for the AL activation duration did not reach statistical significance ($p = 0.083$), although the PFPS group seemed to display a longer AL activation duration compared to the healthy group.

3.2.2.3. Gluteus medius.
For the GMed activation onset during stair descent, no side-by-group interaction was observed ($p = 0.819$, Table 4). A significant main effect of group was observed ($p < 0.001$, $d = 1.12$ [0.43, 1.76]), while no effect of side was observed ($p = 0.431$). The PFPS participants demonstrated significantly later onset of the GMed as compared with the healthy group.

As for the GMed activation duration during stair descent, there was no side-by-group interaction ($p = 0.396$). Significant group difference was observed for the GMed activation duration ($p < 0.001$, $d = −1.19$ [−1.84, −0.49]), while there was no significant side difference across the groups ($p = 0.694$). The PFPS group demonstrated with significantly shorter GMed activation duration, compared to the healthy group.

4. Discussion

The aims of the current study were to simultaneously assess knee kinetics and EMG activation patterns of the lower extremity muscles between those with and without PFPS. The results of the study yielded higher peak knee abduction moment and impulse in PFPS participants during stair ascent and descent. Furthermore, the PFPS participants demonstrated altered activation onset and duration of the VMO, AL, and GMed.

4.1. Frontal plane knee moment and impulse

Our hypothesis regarding knee adduction moments and impulses during stair ambulation was rejected. In fact, our participants with PFPS demonstrated higher knee abduction moment

Table 4
Lower extremity muscle onset and duration during stair descent.

| | Healthy | | PFPS | |
	Symp	Asymp	Symp	Asymp
Onset (ms)				
VMO	−75.19 ± 117.33	−75.47 ± 117.90	−32.57 ± 133.17	−100.07 ± 111.72
AL	−1.93 ± 85.35	0.77 ± 78.50	−11.30 ± 172.96	17.94 ± 106.18
GMed[a]	−50.91 ± 93.12	−69.95 ± 88.20	38.38 ± 49.05	27.88 ± 104.64
Duration (ms)				
VMO[b]	810.88 ± 239.60	913.95 ± 246.14	754.91 ± 319.34	958.59 ± 434.82
AL	331.96 ± 270.46	283.76 ± 173.77	422.69 ± 274.89	441.13 ± 244.91
GMed[a]	526.33 ± 227.55	510.74 ± 231.26	274.43 ± 115.00	316.79 ± 151.41

Symp = symptomatic leg; Asymp = asymptomatic leg.
[a] A significant group difference.
[b] A significant side difference.

during the stance phase of stair ascent, and higher knee abduction impulse during both stair ascent and descent. Our findings are consistent with the findings from previous studies which found that the patients with PFPS had higher knee abduction moments and impulses during walking and running (McClay and Manal, 1999; Stefanyshyn et al., 2006; Paoloni et al., 2010). As suggested by Paoloni et al. (2010) our PFPS participants may have tried to adduct their knee and hip to reduce patellofemoral joint loading or to minimize pain. The increased knee abduction moment and impulse may be a compensatory strategy which was utilized by the PFPS participants to provide stability of the lower extremity during dynamic tasks. Internal frontal plane knee moment can also be affected by the location of the ground reaction force vector in relation to the center of the knee joint. Perhaps the PFPS participants adapted their stair ambulation strategies so that the line of the ground reaction force would pass medial to the knee joint center, thereby increasing the knee abduction moment and impulse. An increase in internal knee abduction moment and impulse may indicate that the knee is placed in more varus or adduction during the stance phase. Whether this strategy is a result or a cause of painful symptoms remains unknown, and further investigation is needed.

4.2. Lower extremity muscle onset and duration

Our hypotheses regarding the muscle activation onset and duration for the AL, VMO, and GMed were partially supported. Contrary to previous results (Cowan et al., 2002), our current study did not find significantly different onset timing for the VMO between groups during stair ascent and descent. However, the activation duration of the VMO was significantly shorter in the PFPS participants compared to the healthy participants during both tasks. Similar results were observed by Brindle et al. (2003), which examined the lower extremity muscle onset and duration during stair ambulation tasks, and found that the participants with PFPS had a shorter duration of the VMO during stair descent. Our results also revealed that the asymptomatic legs of the PFPS participants demonstrated a significantly earlier activation onset and longer duration of the AL compared to the matched "asymptomatic" legs of the healthy participants, while the symptomatic legs showed no statistically significant differences. The results of the increased knee abduction moment and impulse may explain the longer activation duration of the AL. The AL, because of the pulley function as suggested previously (Checroun et al., 1996), may be activated sooner and longer in compensation for the diminished activation of the VMO. However, the longer activation of the AL may have contributed to the increase in the internal knee abduction moment throughout the stance phase, which increased the knee abduction impulse. Our study may provide some evidence to the theory of the functional connection between the VMO and AL, as stated by the

authors of previous cadaver studies which found the anatomical connection between the two muscles (Bose et al., 1980; Checroun et al., 1996).

However, it is curious that the group differences were more pronounced in the asymptomatic leg, rather than in the symptomatic leg. This finding may support the theory of bilateral neuromuscular alterations due to pain or pathology (Bullock-Saxton et al., 1994). The current theory is that altered afferent input from one joint due to an injury may cause altered motor output at joints away from the source of pain or dysfunction. Also, the central nervous system may send inhibitory efferent signals to both sides despite the unilateral source of pain. Although the experiment was performed on the upper extremity, Falla et al. (2007) found that induced pain on the right upper trapezius muscle affected activation of multiple divisions of trapezius muscles on both the right and left sides. Other studies discuss that arthrogenic muscle inhibition, a measure of decreased motor neuron pool excitability, has been observed not only unilaterally (Palmieri-Smith et al., 2008), but also bilaterally (Sedory et al., 2007). If the motor neuron pool excitability is diminished due to injury, muscle activation patterns may be disrupted, further contributing to movement dysfunction. While a direct comparison cannot be made between the current study which examined individuals with PFPS and the aforementioned studies (Falla et al., 2007; Sedory et al., 2007; Palmieri-Smith et al., 2008), it is possible that our PFPS individuals displayed bilateral deficits on the muscle activation patterns regardless of the source of painful symptoms. Further research is warranted to establish central effects of pain on muscle activation during functional tasks in this population.

Our study provides evidence that during both stair ascent and descent, the participants with PFPS displayed significantly delayed activation onset and shorter activation duration of the GMed compared to the healthy participants. These findings are similar to those reported by Brindle et al. (2003); however, our study is the first to have measured the activities of medial and lateral hip musculature during stair ambulation tasks using both healthy and pathological groups. Due to the threshold methods being different between the VMO and the rest of the LE muscles, we cannot make comparisons of activation onset and duration between muscles, especially between the VMO and AL, and VMO and GMed. However, as a general conclusion, GMed activation during stair ascent was markedly later than AL activation, and these differences are more pronounced in the PFPS participants. Since statistical comparisons between muscle activations was not appropriate and was not performed, the direct relationship between these two muscles cannot be established in this study. Regardless, because of the earlier onset of the AL during stair ascent and the longer duration of the AL during stair ascent and descent, along with our findings regarding the GMed onset and duration, this may be a new insight to the development of rehabilitation exercise

programs. Specifically, our results may urge the clinicians to consider implementing exercises that will reestablish balance in onset timing and duration among all three muscles.

For example, many clinicians utilize "ball squeezes" or knee extension exercises with hip internal rotation or adduction in the exercise program for PFPS patients, under the idea that adding hip internal rotation or adduction may enhance VMO activation or strength. However, some researchers have found no increase in the VMO activity with those exercises (Boling et al., 2006). Our findings may suggest that the AL in the PFPS patients are turned on longer already, which may bring the knee into more abduction, causing further maltracking of the patella and subsequent pain. Therefore, the exercises that have been used to help PFPS patients may be creating more pain and dysfunction. On the other hand, our results further confirmed that the lateral hip musculature such as GMed is not activating properly, likely further contributing to the knee abduction due to increased hip adduction. Our findings support the recent ideas of incorporating exercise programs that would reestablish proper neuromuscular control of the lateral hip musculature. Clinicians should be cognizant when selecting appropriate exercise regiments for patients with PFPS to ensure proper restoration of neuromuscular control.

For the future study, it may be beneficial to assess kinetics and EMG activity according to the specific stages of the gait cycle and their relationship to the spatiotemporal parameters such as cadence, velocity, and single support time. While the intent of this current study was to assess muscle activation timing and duration, as well as frontal plane knee kinetics, it may be of help for the clinicians to understand whether altered activation may affect spatiotemporal measures of stair ambulation, and identify whether muscle activity is altered at different stages of the gait cycle.

5. Conclusion

To our knowledge, this study is the first to investigate the differences between those with and without PFPS on the muscle activation onset and duration of the GMed, AL, and VMO, as well as the frontal plane knee kinetics, during stair ambulation. Our results revealed that the VMO and GMed activation duration were shorter and GMed onset was delayed in the PFPS participants, while the AL displayed earlier onset and longer duration during both stair ascending and descending tasks, compared to the muscle activation patterns of the healthy participants. Furthermore, the knee abduction impulse was higher in the PFPS participants than the healthy participants during both stair ascent and descent. These findings may provide additional insights to neuromuscular control alterations in those with chronic knee pain, and suggest implementation of therapeutic exercises which enhance the GMed activity relative to the AL.

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Naoko Aminaka is a visiting assistant professor in the Department of Kinesiology at the University of Toledo, Toledo, OH. She received her B.S. in Athletic Training from Central Michigan University, Mt. Pleasant, MI, and her M.S. and Ph.D. in Exercise Science from the University of Toledo. Her research interests include biomechanical and neuromuscular alterations associated with chronic knee conditions (patellofemoral pain syndrome and osteoarthritis).

Brian G. Pietrosimone is an assistant professor in the Department of Kinesiology and the Director of the Joint Injury and Muscle Activation Laboratory at the University of Toledo. He received his B.S. degree in 2005 from Springfield College in Springfield, MA (Athletic Training). He then obtained his M.Ed. in 2006 (Athletic Training) and his Ph.D. in 2009 (Sports Medicine) from the University of Virginia in Charlottesville, VA. His line of research is developed around understanding and treating the neural origins of disability following lower extremity joint injury.

Charles W. Armstrong is a professor in the Department of Kinesiology and the director of the Motion Analysis Laboratory at the University of Toledo. He received his B.Ed. degree in Health and Physical Education from Slippery Rock State College, Slippery Rock, PA, and his M.Ed. and Ph.D. in Exercise Science from the University of Pittsburgh, Pittsburgh, PA. His research interests involve the influence of pathology on functional movements, biomechanics of sport injury, and biomechanics of golf.

Phillip A. Gribble is an associate professor in the Department of Kinesiology, the director of the graduate athletic training program, and the director of Athletic Training Research Laboratory at the University of Toledo. His research interests involve the mechanisms of neuromuscular and functional deficits related to the lower extremity musculoskeletal injuries, specifically chronic ankle instability. He received his B.A. (Physical Education/Exercise and Sport Science) and M.A. (Exercise and Sport Science) from University of North Carolina, Chapel Hill, NC, and his Ph.D. (Kinesiology with concentration in Athletic Training) from Pennsylvania State University, State College, PA.

Andrew Meszaros is an assistant professor in the Department of Neuroscience at the University of Toledo. He obtained his B.S. from Bowling Green State University, Bowling Green, OH, and his Ph.D. from the University of Iowa, Iowa City, IA. His research interests involve the neural control of human skeletal muscle as it relates to pre-programmed or involuntary motor unit recruitment.