

Physical therapy alters recruitment of the vasti in patellofemoral pain syndrome

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ABSTRACT

COWAN, S. M., K. L. BENNELL, K. M. CROSSLEY, P. W. HODGES, and J. MCCONNELL. Physical therapy alters recruitment of the vasti in patellofemoral pain syndrome. *Med. Sci. Sports Exerc.*, Vol. 34, No. 12, pp. 1879–1885, 2002. **Purpose:** To investigate the effect of physical therapy treatment on the timing of electromyographic (EMG) activity of the vasti in individuals with patellofemoral pain syndrome (PFPS). **Methods:** Sixty-five (42 female, 23 male) participants aged 40 yr or less (29.2 ± 7.8 yr) diagnosed with PFPS. Participants were randomly allocated into physical therapy treatment (McConnell-based) or placebo groups. Treatment programs were standardized and consisted of six-treatment sessions over 6 wk. Vastus medialis oblique (VMO) and vastus lateralis (VL) EMG activity was recorded with surface electrodes during a stair-stepping task and onsets of EMG activity were measured pre- and post-treatment. **Results:** Before treatment, the EMG onset of VL occurred before that of VMO in both participant groups. After physical therapy intervention, there was a reduction in symptoms, and this improvement was associated with a significant change in the time of onset of VMO EMG compared with that of VL in both phases of the stair-stepping task. After physical therapy treatment, the onset of VMO preceded VL in the eccentric phase and occurred at the same time in the concentric phase of the stair-stepping task. There was no change in time of EMG onset in the placebo group. **Conclusion:** This study demonstrates that a “McConnell”-based physical therapy treatment regime for PFPS alters the motor control of VMO relative to VL in a functional task and this is associated with a positive clinical outcome. **Key Words:** VASTUS MEDIALIS OBLIQUE, ELECTROMYOGRAPHY, STAIR STEPPING, TREATMENT

Patellofemoral pain syndrome (PFPS) is a common complaint in the sporting and general populations, especially when repetitive lower-limb loading is involved (10). PFPS is defined as anterior or retropatellar pain in the absence of other pathology. Clinically the condition presents as diffuse pain, exacerbated by activities such as stair climbing, prolonged sitting, squatting, and kneeling. Despite the prevalence of PFPS, its etiology is not well understood. The most commonly accepted hypothesis is abnormal lateral tracking of the patella (29). Commonly, treatment aims to restore the equilibrium of the patellar tracking system (29). Routine clinical practice addresses this aim by attempts to selectively activate vastus medialis oblique (VMO; often using biofeedback) in functional positions, aided by taping of the patella, improvement of pelvic control, and reduction of tightness in lateral soft tissue structures, hamstrings, and the anterior hip musculature.

Patellar tracking is the outcome of an interaction between passive structures, muscle, and the neuromotor control systems. Previous research has focused on the effects of the muscular and osseoligamentous contributions to patellar tracking (e.g., 11,16). More recently, the importance of the neural control system has been highlighted. Recent studies have provided evidence of an imbalance in the activation of the timing of VMO and vastus lateralis (VL) in people with PFPS (5,6,30). This is consistent with a change in motor control rather than a simple change in strength. Furthermore, a recent study by Neptune et al. (25) demonstrated the functional significance of such a timing difference with the finding that a 5-ms VMO timing delay was associated with a significant increase in lateral patellofemoral joint (PFJ) loading.

Despite evidence that demonstrates a difference in the timing of EMG onsets of VMO and VL in PFPS, it is unclear whether this difference can be changed by physical therapy management and, if so, whether such a change is associated with a positive clinical outcome. Although several studies have failed to find changes in the ratio of VMO:VL amplitude with maneuvers that involve rotation of the hip and terminal knee extension (1,2,28), these studies have not investigated the timing of VMO:VL onset of activation nor the effect of specific muscle reeducation. From a biomechanical perspective, it is possible that the efficacy of physical therapy treatment of PFPS may be due to changes in the motor control the vasti. Thus, the aim of this study was to investigate the efficacy of a comprehensive

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TABLE 1. Inclusion and exclusion criteria.

Inclusion criteria

- Anterior or retropatellar knee pain on at least two of the following activities: prolonged sitting, stairs, squatting, running, kneeling, and hopping/jumping
- Pain on patellar palpation
- Pain while stepping down from a 25-cm step or during a double leg squat
- Symptoms for at least 1 month
- Average pain level of 3 cm or more on a 10-cm VAS
- An insidious onset of symptoms unrelated to a traumatic incident.

All participants were aged 40 yr or less to reduce the likelihood of osteoarthritic changes in the patellofemoral joint

Exclusion criteria

- Signs or symptoms of other pathology including coexisting pathology
- A recent history (within 3 months) of knee surgery, a history of patellar dislocation/subluxation, or clinical evidence of meniscal lesion, ligamentous instability, traction apophysitis around the patellofemoral complex, patellar tendon pathology, chondral damage, osteoarthritis, or referred pain from the spine
- Features that could affect the implementation of the trial
- Previous experience with patellar taping, an inability to attend a physical therapy clinic for a 6-wk treatment program, allergic reaction to adhesive tape, pregnancy, and an inability to understand written and spoken English

McConnell-based program (including specific VMO re-training), using a double-blind, randomized, placebo-controlled trial. To address this aim, the present study investigated the timing of EMG onset of VMO relative to VL in a stair-stepping task before and after physical therapy intervention. The pain and disability outcome measures have been reported elsewhere (9).

METHODS

Participants. Sixty-five (42 female, 23 male) of the 71 participants taking part in the randomized controlled trial were included for EMG measurement. The smaller participant population was due to equipment failure (*N* = 1) and participant availability at testing times (*N* = 5). All participants were diagnosed with PFPS on the basis of clinical examination by an experienced physiotherapist. The inclusion and exclusion criteria were based on those used in other PFPS studies (20,22) and are detailed in Table 1.

Participants were randomly allocated into physical therapy treatment (*N* = 35) or placebo groups (*N* = 30) using block randomization stratified to treatment center. The mean (SD) age, height, weight, body mass index, and duration of symptoms are detailed in Table 2. There were no significant differences between groups for these variables assessed using an independent *t*-test. Both participants and the examiner (SC) were blind to intervention allocation.

The study was approved by the University of Melbourne Human Research Ethics Committee. All participants provided written informed consent.

Interventions. Standardized treatment protocols were based on interventions and techniques used by the Australian physical therapy profession (24). Participants were

treated once a week for 6 wk for a similar length of time in each treatment group. Ten physiotherapists experienced in the treatment of PFPS performed both treatments. The physical therapy and placebo intervention protocols are detailed in Table 3.

Pain and disability measures. Patients completed a number of self-administered questionnaires at the baseline assessment and at completion of the 6-wk treatment program. Patients rated their worst and average pain in the last week on a 10-cm visual analog scale (VAS) (14), their worst pain in the last week on a 10-cm VAS for stair negotiation (12), and completed an anterior knee-pain-specific self-administered questionnaire (AKP) (21). Patients also completed a 5-point scale to determine the global rating of change compared with baseline (1, marked worsening; 2, moderate worsening; 3, same; 4, moderate improvement; 5, marked improvement).

Electromyographic recordings. At baseline and after the completion of the trial the electromyographic (EMG) activity of VMO and VL was recorded using surface electrodes. Ag/AgCl electrodes (Graphics Control Corp, c/o Medical Equipment Services PTY.LTD, Richmond, Australia) were placed over the muscle bellies of VMO and VL with an interelectrode distance of 22 mm. The electrode for VMO was placed over the muscle belly 4 cm superior to and 3 cm medial to the superomedial patella border and orientated 55° to the vertical. The electrode for VL was placed 10 cm superior and 6–8 cm lateral to the superior border of the patella and orientated 15° to the vertical (13). The ground electrode was placed over the tibial tubercle. Before electrode placement the skin was shaved, swabbed with alcohol, and gently abraded with sandpaper to reduce the electrical

TABLE 2. Description of mean (SD) or frequency characteristics at baseline for the physical therapy and placebo groups.

	Active (<i>N</i> = 35)	Placebo (<i>N</i> = 30)	<i>t</i> -Test/chi	<i>P</i>
Age	29.1 (7.5)	25.6 (8.3)	-1.78	.08
Gender	22 F, 13 M	20 F, 10 M	.10	.75
Height	1.7 (.09)	1.7 (.1)	-.19	.85
Weight	68.7 (13.9)	72.0 (14.1)	.96	.34
BMI	23.4 (3.9)	24.6 (3.9)	1.24	.22
Leg dominance	5 L, 30 R	2 L, 28 R	.34	.56
Durtotal	39.6 (42.7)	29.3 (33.3)	-1.07	.29
Durcurrent	16.7 (29.4)	8.9 (21.2)	-1.21	.23

Durtotal, total time since first onset of symptoms; durcurrent, duration of current episode of symptoms; leg dominance, leg used to kick a football.

TABLE 3. Physical therapy and placebo intervention protocols.

Physical therapy intervention protocol

Patellar taping

Progressive functional retraining of VMO using dual channel, surface EMG biofeedback*

- (i) Weeks 1–2: isometric VMO contractions in sitting with the knee at 90° flexion and inner range squats to 40° knee flexion combined with isometric gluteal contraction
- (ii) Weeks 3–6: when the participant could complete five step downs from a 20cm step with out pain, the step down exercise and VMO retraining with a isometric hip abduction in standing were included

Gluteal strengthening exercises and stretching of soft tissue structures

Placebo intervention protocols

Placebo taping (vertical taping)

Inoperative ultrasound†

Light application of a nontherapeutic gel (ultrasonic gel + hospital hand cream)

* Pathway MR-20, The Prometheus Group, Dover, DE.

† Custom-made devices, Metron Australia.

impedance to less than 5 k Ω . The procedure has previously been found to be reliable in our laboratory (4).

Stair apparatus. Custom-built stairs with a 60-cm platform and two steps of 20-cm height on both sides (4,5,13) were placed in the center of a 5-m walkway.

Procedure. Participants stood 1.8 m from the lower step apparatus and walked over the stairs at a rate of 96 step·min⁻¹ (paced by an external metronome). This rate approximates usual stair-stepping pace, has been used previously (13), and may increase the repeatability of the stair-stepping task. Participants completed at least five practice trials to ensure that they were able to step in time with the metronome and were able to contact the middle step with the test leg.

Recordings of EMG activity of VMO and VL were made for five consecutive trials during the stance phase on the first stair during ascent (concentric contraction) and descent (eccentric contraction). Participants completed a 10-cm VAS to indicate the amount of pain present while completing the stair-stepping task.

EMG data were preamplified ($\times 10$), band-pass filtered between 20 and 500 Hz, sampled at 1000 Hz, and 12-bit A-D converted (Associative Measurement Pty, Ltd., North Ryde, NSW, Australia).

Data analysis. The EMG data were full-wave rectified and low-pass filtered at 50 Hz (6th-order Butterworth filter). A computer algorithm was used to identify the onset of EMG activity of each of the muscles. The algorithm identified the point at which the EMG signal deviated by more than three standard deviations, for a minimum of 25 ms, above the baseline level (averaged over 200 ms before the commencement of the trial). The rectified unfiltered EMG data were visually checked to verify the EMG onsets identified by the computer.

EMG onsets were identified from individual trials and averaged over the five repetitions. The latency between the onset of EMG of VMO and VL was quantified during concentric (concentric EMG onset timing difference) and eccentric (eccentric EMG onset timing difference) contraction by subtracting the onset of VMO EMG from that of VL.

Statistical analysis. All data were analyzed using Statistical Package for Social Sciences (Norusis/SPSS, Inc., Chicago, IL).

EMG onset timing difference. Difference scores were calculated by subtracting the baseline scores from the follow-up scores for (i) the concentric and eccentric EMG

onset timing difference, (ii) the VAS scores, and (iii) the AKP. A two-way analysis of variance was used to compare difference scores for muscle contraction between groups. Independent *t*-tests were used to compare the difference scores for the VAS and AKP scores between groups. At baseline and follow-up, independent one-group *t*-tests were used to determine whether the concentric and eccentric EMG onset timing differences differed significantly from zero (that is, did VMO and VL onset occur simultaneously?). The alpha level was set at 0.05.

Predictors of change in EMG onset timing difference. Pearson's univariate correlations were sought between age, gender, height, duration of symptoms, baseline EMG onset timing difference, baseline VAS and AKP scores, and the changes in VAS and AKP scores. Significant variables located during univariate analyses were subjected to forward stepwise multiple linear regression.

To determine whether the patient's perceived response to the treatment program was related to a change in EMG onset timing, the data were categorized. The difference scores for the concentric and eccentric EMG onset timing data were divided into three groups: those that changed in a positive direction (greater than +10-ms change), those that did not change (less than 10-ms change), and those that changed in a negative direction (greater than -10-ms change). The participants' response to treatment was dichotomized: those that improved either moderately or significantly and those who remained the same or became moderately or significantly worse. The χ^2 statistic was used to compare the response to treatment in these three groups.

RESULTS

Eight participants did not to return for reassessment, three from the placebo group and five from the physical therapy group. Three participants were lost to follow-up, one was excluded due to pregnancy, one was unable to be tested due to equipment failure, and four were unavailable to return to be tested. There were however no differences between the pretreatment scores of these eight participants and the group means.

Baseline comparability. At baseline, the EMG onset timing difference was similar between groups in the concentric and eccentric phases of stair stepping ($P = 0.06$ and $P = 0.87$, respectively). In both groups at baseline, the onset of VL occurred before VMO in the concentric and eccentric

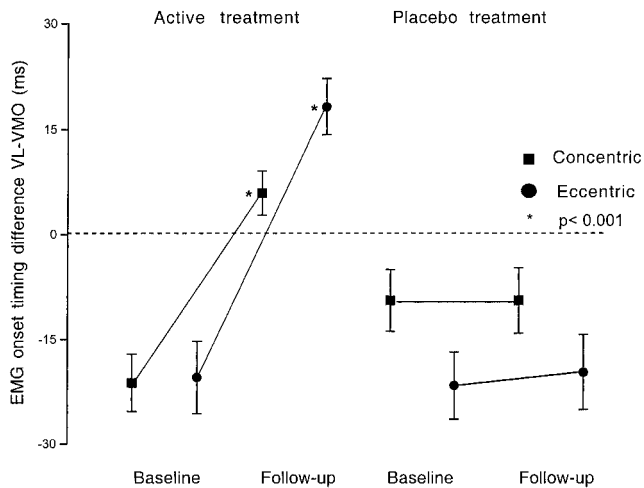


FIGURE 1—Means and standard errors for EMG onset timing difference at baseline and after the 6-wk treatment program for the physical therapy and placebo groups. Although there was no difference in EMG onsets between groups before treatment, only the physical therapy group had a significant improvement in VMO timing relative to VL after treatment.

phases of the stair-stepping task ($P < 0.001$ and $P < 0.001$, respectively).

EMG onset timing difference. After the 6-wk treatment program, the physical therapy treatment group demonstrated a change in the latency between the onsets of VMO and VL EMG. This change was greater than any change in the placebo group ($P < 0.0001$; Fig. 1). There was no difference in the size of the change between the eccentric and concentric tasks for either group ($P = 0.14$) and no interaction effect between the tasks or groups ($P = 0.69$). In the physical therapy group, this change resulted in simultaneous onset of VMO and VL EMG during the concentric phase ($P = 0.07$). In the eccentric phase, the onset of VMO EMG actually preceded that of VL ($P < 0.001$). However, in the placebo group, the onset of VL EMG still occurred before that of VMO in both phases of the stair-stepping task at follow-up ($P < 0.05$ concentric $P < 0.05$ eccentric, respectively).

Changes in pain and function. The data for the large participant group are reported in full in Crossley et al. (9). The results for this cohort ($N = 65$) are summarized here to place the EMG results in context. The physical therapy treatment group improved in pain and function to a greater extent than the placebo group. The physical therapy treatment group demonstrated a significantly greater change in both their average pain and worst pain in the last week ($P < 0.01$ and $P < 0.01$, respectively). They also improved significantly in their worst pain in the last week on ascending stairs ($P < 0.05$), but there was no difference between groups for the worst pain in the last week on descending stairs ($P = 0.12$). The AKP also showed greater improvement in the physical therapy group ($P < 0.005$). There was however no difference between the groups in the change demonstrated on the pain experienced during the stair-stepping task ($P = 0.31$).

Predictors of change in the EMG onset timing difference. The change in the concentric EMG onset timing difference was found to be univariately correlated with the baseline concentric EMG onset timing difference, the change in the worst pain on ascending stairs in the last week, the change in pain experienced on the stair-stepping task, and the change in AKP. When these variables were subject to a forward stepwise multiple regression analysis, both the baseline concentric EMG onset timing difference ($P < 0.001$; Fig. 2) and the change in pain on ascending stairs ($P < 0.001$; Fig. 3) were found to be independent predictors of the change in concentric EMG onset timing difference. The baseline concentric EMG onset timing difference accounted for 46% and the change in pain on ascending stairs accounted for an additional 10% of variability in the change in EMG onset timing.

The change in eccentric EMG onset timing difference was found to be univariately correlated with the baseline eccentric EMG onset timing difference and the change in pain experienced while participants completed the stair-stepping task. However, when these variables were subjected to a forward stepwise multiple regression analysis, only the baseline EMG onset timing difference was found to predict the change in eccentric EMG onset timing ($P < 0.001$; Fig. 2) accounting for 23% of the variability.

Correlation of change in EMG onset timing difference and treatment outcome. Figures 4 and 5 detail the number of participants in each group who changed in a positive or negative direction or had no change in their EMG onset timing difference and their perceived response to treatment. In the physical therapy group, the χ^2 statistic demonstrated that in the concentric phase of the stair-stepping task those who reported moderate or significant improvement were more likely to have a positive change in their EMG onset timing difference ($P < 0.02$), whereas in the eccentric phase of the stair-stepping task there were no

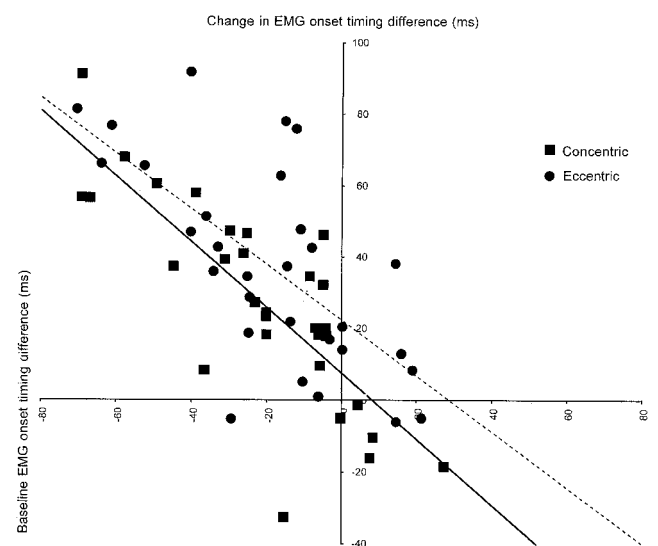


FIGURE 2—Correlations between the baseline EMG onset timing difference and the change in EMG onset timing difference with physical therapy treatment in the concentric and eccentric phases of the stair-stepping task. The line of best fit is represented for the concentric data with a solid line and for the eccentric data with a broken line.

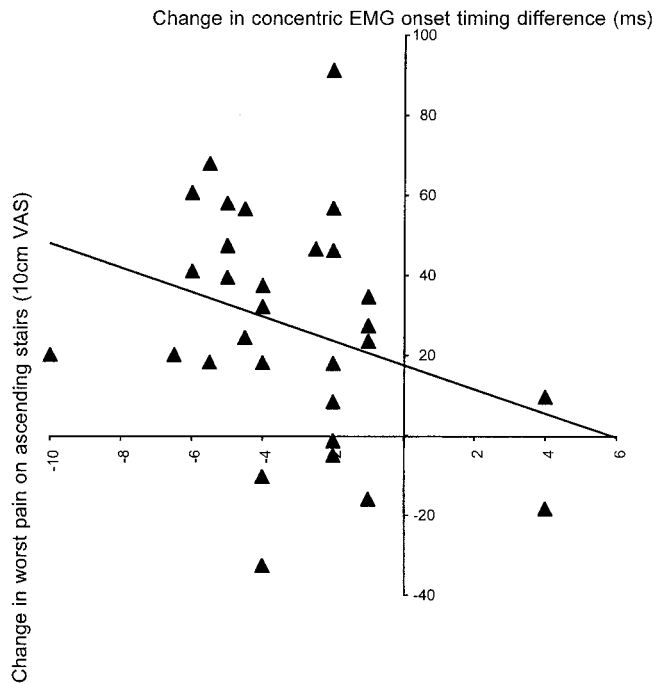


FIGURE 3—Correlation between the change in worst pain on ascending pain in the last week and the change in concentric EMG onset timing difference with physical therapy treatment. The line of best fit is represented by the solid line.

differences ($P = 0.49$). There were no differences in the placebo group in either phase of the stair-stepping task.

DISCUSSION

Contemporary rehabilitation strategies assume that physical therapy treatment is able to change motor control and that this is necessary for improvement of symptoms; however, this assumption has not previously been tested. This study provides novel evidence that a comprehensive physical therapy program incorporating VMO retraining with biofeedback results in a change in the onset of VMO EMG relative to that of VL in participants with PFPS. No such

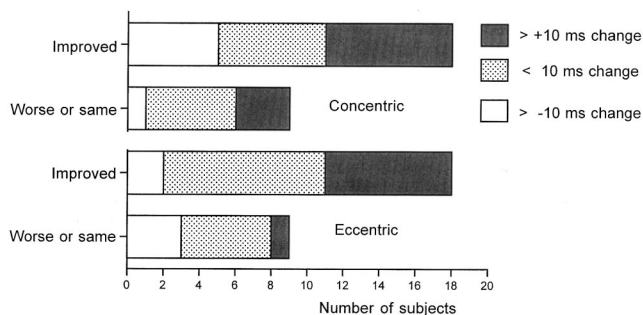


FIGURE 4—Number of participants in the physical therapy group who had a change in a positive or negative direction or no change in their EMG onset timing difference and their perceived response to treatment. Note that in the concentric phase of the stair-stepping task those who reported moderate or significant improvement were more likely to record a positive change in their EMG onset timing difference; this difference was not significant in the eccentric phase of the stair-stepping task.

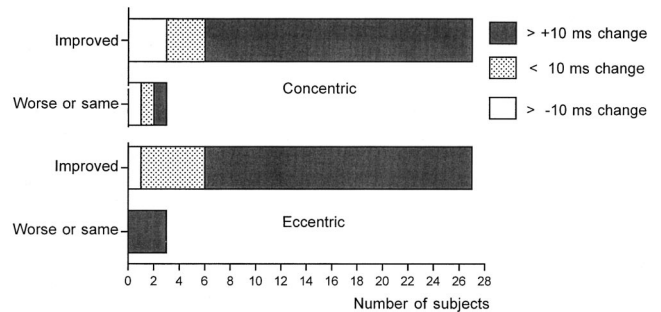


FIGURE 5—Number of participants in the placebo group who had a change in a positive or negative direction or no change in their EMG onset timing difference and their perceived response to treatment. Note that there were no differences in the placebo group in either phase of the stair-stepping task.

change in EMG onsets was identified after the placebo treatment. When participants were assessed at baseline, the EMG onset of VL occurred before the onset of VMO in both groups, and this concurs with our previous findings (5,6). After the 6-wk treatment program, pain and function improved in the physical therapy treatment group more so than in the placebo group, and these improvements were associated with alterations in the latency between EMG onsets of VL and VMO. At follow-up, the specific findings were (i) in the physical therapy treatment group, the EMG onset of VMO occurred at the same time as VL as participants ascended the stairs; (ii) in the physical therapy treatment group, the EMG onset of VMO preceded VL as participants descended the stairs; and (iii) in the placebo group, the EMG onset of VL preceded VMO in both phases of the stair-stepping task.

Physical therapy treatment alters VMO/VL EMG onset timing. The finding of an alteration in the relative timing of VMO compared with VL in the physical therapy treatment group provides a mechanism, which may explain the efficacy of physical therapy treatment of PFPS. Additionally, it provides support for frequently used physical therapy treatment programs that aim to improve VMO timing through specific retraining of this muscle. A number of clinical trials have been published that have evaluated the efficacy of physical therapy treatment (refer to Crossley et al. (8) for review). Of the trials published, only two investigated a McConnell-based program similar to that used in our study (3,15). Although the results of all the published trials generally favor physical therapy intervention, ours is the first study to measure recruitment of the vasti and compare the intervention with a placebo intervention.

Theoretical mechanisms for the efficacy of physical therapy treatment on the latency between VMO and VL EMG onset. The specific component of the physical therapy treatment program that altered VMO/VL EMG onset timing cannot be determined from this study, nor can the mechanisms as to how this occurred or whether the change was responsible for the change in symptoms. Due to the multifactorial nature of the physical therapy intervention studied, it is not possible to isolate the factor responsible for the change in vasti timing. A number of possibilities exist

including part or all of the treatment program (VMO retraining with biofeedback, home exercise program, PFJ taping, mobilization of PFJ, gluteus medius retraining, or stretching of hamstrings), or the reduction in pain or improvement in function experienced by participants in the physical therapy treatment group.

EMG biofeedback of contraction of the vasti was used in our trial when the patient attended physical therapy treatment once per week (six treatments in total). The home exercise program was then completed without the use of biofeedback. The efficacy of the use of biofeedback to aid in muscle training has been investigated in two uncontrolled case series (23,29). VMO retraining using EMG biofeedback with surface electrodes was found to improve the EMG amplitude of VMO relative to VL activity in normal participants in both studies; however, as the data were not normalized or controlled, the results should be viewed cautiously. Of the controlled trials, only Harrison et al. (15) included EMG biofeedback for specific muscle training as part of their treatment program; however, they did not include EMG assessment as an outcome measure. In low back pain, there is preliminary evidence to suggest that specific muscle training can alter the timing of muscle contraction (19).

The effect of patellar taping in PFPS has been the subject of much investigation, with taping found to decrease pain, change patellar position, and improve quadriceps torque (refer to Crossley et al. (7) for review). In terms of EMG activation levels, preliminary reports demonstrate that there is no difference in the activation levels of VMO/VL with the application of tape (2,17). Only one study has investigated the effect of patellar taping on the EMG onset timing of VMO and VL in symptomatic individuals. Gilleard et al. (13) found the EMG onset of VMO to occur earlier in both phases of the stair-stepping task with taping. However, all of these studies investigated the immediate effect of taping, and thus it is not known whether the effect is maintained. Similarly, the mechanism by which patellar taping induces pain relief is not known. It may be that the pain relief associated with the patellar taping is a cause of the change in EMG onset timing, or alternatively the change in EMG onset timing may have resulted in the decrease in pain.

The remaining components of the treatment program have been less extensively investigated, and thus the mechanisms behind how they may have contributed to the change in EMG onset timing are less clear. No study has investigated the individual effects of hamstring stretching, gluteal strengthening, or PFJ mobilization, although a number of studies have included stretching as part of their physical therapy regime. Possible theories to link these factors with the change in EMG onset timing include an alteration of joint biomechanics, loading, or improved motor control of gluteus medius; however, at present, these theories are not supported by evidence.

An improvement in function may also have contributed to the change in EMG onset timing. The change in function may have resulted from biomechanical changes such as improved pelvic control, which may have reduced forces at

the knee or provided a more stable attachment for the vasti, which was reflected by the change in motor control. It is not surprising that no correlations were identified between the AKP and timing change as changes in biomechanics are likely to be subtle and not detected in our gross measurements of function (i.e., AKP). In the present study, we did not attempt to relate the temporal measures of vasti activity with other kinematic measures.

Another possible mechanism for the change in EMG onset timing is the decrease in pain that occurred in the treatment group. Although it is unclear whether changes in motor control occur as a result of pain or precede the onset of pain, there is increasing evidence that motor output may be affected by pain directly. Pain has been found to influence motor output at differing levels of the motor system (26,27). The finding of an initial delay in the EMG activation of VMO contraction associated with pain and the changes in pain and EMG onset timing that occurred in the physical therapy treatment group provide some evidence that pain does interfere with VMO function. Similar changes in the timing of EMG onset have also been found in participants with low back pain (18). It is possible that the contraction of VMO is changed to prevent pain provocation; however, as patients did not experience pain with VMO contraction in training, this seems unlikely.

Relationship between the initial EMG onset timing and the magnitude of change with rehabilitation. Participants who presented with a long latency between the onsets of VL and VMO EMG were more likely to record a positive change in their EMG onset timing difference with treatment. Correspondingly, if the EMG onsets of VMO and VL occurred simultaneously or with VMO EMG onset before VL, then these participants were less likely to record a positive change in their EMG onset timing difference with treatment. These results have clinical applications and strengthen the argument for further research to focus on the identification of subgroups of PFPS. PFPS is a multifactorial problem, and it is apparent that some participants present with timing differences and others do not (5). If a participant presents without a difference in the EMG onset of VL relative to VMO, it follows that a program based on improving the timing of VL relative to VMO may not be the most efficacious treatment. However, clinically accurate assessment of EMG onset timing of VMO and VL is difficult as commercial biofeedback units do not provide an adequate sampling rate, and the specificity of palpation as a measuring tool has not been assessed.

Methodological considerations. There are several methodological issues that warrant consideration. As noted above, the multifaceted treatment program (chosen as it reflects current clinical practice) used makes it impossible to determine which component (or combination of components) was responsible for the change in EMG onset timing difference. Additionally, the study lacked a controlled follow up period and thus no conclusions can be drawn about the long-term effects of physical therapy. Eight participants were lost to follow-up (five in the physical therapy group and three in the placebo), but analyses of best case (partic-

ipants received the largest EMG onset timing change) and worst case (participants received the smallest EMG onset timing change) scenarios did not alter the results.

CONCLUSION

This study provides novel evidence that a comprehensive physical therapy program incorporating VMO retraining with biofeedback results in a change in the onset of VMO EMG relative to that of VL in participants with PFPS. No such change in EMG onsets was identified after the placebo treatment. As such, this study provides the first evidence

that physical therapy treatment is able to change motor control.

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