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Altered hip and trunk muscle function in individuals with patellofemoral pain

S M Cowan,1,2 K M Crossley,2,3 K L Bennell4

ABSTRACT

Objective: The purpose of this study was to investigate the role of hip muscles in patellofemoral pain (PFP), specifically by investigating neuromotor control, strength and range of motion of the hip muscles.

Design: Cross-sectional.

Setting: University laboratory.

Patients: Ten participants diagnosed with PFP and 27 asymptomatic controls were recruited, using standard inclusion and exclusion criteria.

Main outcome measures: Electromyographic activity of the vasti and anterior gluteus medius (GM) were recorded using surface electrodes and posterior GM using fine wire electrodes while the participant completed a stair-stepping task. Hip strength and trunk side flexion strength was assessed using a hand-held dynamometer and hip range of motion with an inclinometer. Pain and disability measures were recorded for the PFP participants.

Results: When individuals with PFP completed the stair-stepping task there was a delay in activation of both anterior and posterior GM (p < 0.02) and an alteration in range of motion with an inclinometer. Pain and disability measures were recorded for the PFP participants.

Conclusion: This study provides evidence that trunk side flexion strength and neuromotor control of the GM are affected in people with PFP. This study also confirms the presence of a delayed vastus medialis obliquus relative to vastus lateralis, providing further evidence underpinning the importance of retraining the vasti function in patients with PFP.

Patellofemoral pain (PFP) is a common complaint in both the sporting and general populations especially when repetitive lower limb loading is involved.1–4 Despite its prevalence, the aetiology of PFP is unknown. A commonly accepted hypothesis is that abnormal lateral tracking of the patella contributes to the development of PFP. Patellar tracking is the outcome of an interaction between the local and remote passive structures, muscles and the neuromotor control systems. Of these factors, neuromotor control is thought to be of clinical importance, due to its potential for change with physiotherapy intervention.

Neuromotor control of the vasti may play a role in PFP, with some studies showing a delay in the onset of vastus medialis oblique (VMO) relative to vastus lateralis (VL) in cohorts with PFP compared with asymptomatic controls.5,6 However, this is not a consistent finding in all PFP patients. In our cohort we found that approximately 30–40% of individuals with PFP presented with no difference in their vasti onset timing.5 This finding highlights the multifactorial nature of PFP, indicating that altered neuromotor control of the vasti (as measured by electromyographic (EMG) onset timing) may be only one component.

Remote factors, including altered neuromotor control of the hip and trunk muscles have been proposed to contribute to the development of PFP.7–10 Reduced hip and trunk strength (or core stability) has been associated with an increased risk of knee injury.11 Grelsamer and McConnell10 suggest that dysfunction of the gluteus medius (GM) may result in contralateral dropping of the pelvis, in association with increased internal rotation at the hip. These proximal kinematic patterns can result in genu valgus, increased dynamic Q angle and thus greater lateral forces acting on the patella.12–14 In support of the biomechanical links between the hip and trunk complex and patellar tracking, contemporary physiotherapy treatment of PFP commonly aims to optimise muscle function to control hip adduction and internal rotation during weight-bearing activities. Indeed, a randomised controlled trial by our group demonstrating that physiotherapy management for PFP was efficacious included a component of hip muscle control training.15

Despite common clinical practice there are limited studies investigating the control of the hip and trunk muscles in individuals with PFP. Ireland et al16 found that women with PFP had weaker hip abductors and external rotators than female controls, but did not investigate the EMG activity of the muscles. Two studies that have investigated the EMG activity of the GM in individuals with PFP provide conflicting results. Brindle et al17 found that individuals with PFP had delayed onset of GM during a stair-stepping task compared with asymptomatic controls. However, the authors did not specify the portion of GM evaluated. Boling et al18 also investigated the EMG onset of GM (middle portion) during stair stepping but reported no differences in GM onset or duration between individuals with PFP and controls. The GM is a large triangular muscle with three distinct bands, with the anterior and posterior sections postulated to have different actions.16 The posterior portion is proposed to be of particular importance to PFP, with increased posterior GM activity thought to be linked with decreased activity of tensor fascia latae and thus decreased pull on the lateral retinaculum leading to an enhancement of VMO activity.19

Given the limited studies of the hip muscle function in PFP and the conflicting results to date, the purpose of this study was to investigate further the role of the hip muscles in PFP. Specifically by examining: (1) the neuromotor control of the hip
muscles (temporally via EMG onsets of anterior and posterior portions of GM and spatially by measuring hip abduction and external rotation strength); (2) trunk side flexion strength (measure of trunk strength/core stability) and (3) hip rotation (due to the proposed role of the GM in the control of rotation).

METHODS
 Participants
 Ten participants (seven women and three men) diagnosed with PFP and 27 asymptomatic controls (15 women and 12 men) were recruited. The inclusion and exclusion criteria were based on previous studies.19 20 21 Individuals with PFP were included if they had anterior or retropatellar knee pain reported on at least two of the following activities: prolonged sitting, stairs, squatting, running, kneeling and hopping/jumping. In addition, they were to have pain on patella palpation, have had symptoms for at least 1 month, have an average pain level of 5 cm on a 10 cm visual analogue scale and have an insidious onset of symptoms unrelated to a traumatic incident. All participants were aged 40 years or less to reduce the likelihood of osteoarthritic changes in the patellofemoral joint. Participants were excluded if they had signs or symptoms of other pathology including co-existing pathology. The exclusion criteria were a recent history (within 5 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 spinal referred pain.

Asymptomatic participants were recruited from the staff and student body of the University of Melbourne. Participants were excluded if they had any history of lower limb pathology or other disorder, which might interfere with the kinetics or kinematics of hip or knee motion.

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

Electromyographic recordings
 EMG activity of VMO, VL and anterior GM were recorded using surface electrodes. Pairs of Ag/AgCl electrodes (Graphics Control Corporation, c/o Medical Equipment Services Pty Ltd, Richmond, Australia) were placed over the muscle bellies of the VMO, VL and anterior GM with an interelectrode distance of 22 mm. The electrode for the VMO was placed approximately 4 cm superior to and 3 cm medial to the superomedial patella border and orientated 55° to the vertical. The electrode for the VL was placed 10 cm superior and 6–8 cm lateral to the superior border of the patella and orientated 15° to the vertical.22 The electrodes for the anterior GM were placed 5 cm posterior to the anterior superior iliac spine and 3–4 cm below the iliac crest. Before placement the skin was shaved, swabbed with alcohol and gently abraded with sandpaper to reduce the electrical impedance to less than 5 kΩ.

EMG activity of the posterior portion of the GM was recorded using bipolar intramuscular electrodes fabricated from Teflon-coated stainless steel wire 75 μm in diameter (AM Systems, Carlsborg, Western Australia). One millimetre of insulation was removed from the cut ends of the wire, and the tips were bent back approximately 1–2 mm from the end to form a hook. The electrodes were threaded into a hypodermic needle (0.7 × 38 mm) and sterilised. The electrodes were inserted into the posterior portion of the GM under the guidance of real-time ultrasound imaging (7.5 MHz curved linear array transducer; Dornier Performa, Acoustic Imaging Technologies Corp, Phoenix, Arizona, USA).23 The ground electrode was placed over the iliac crest of the non-tested leg.

EMG data were sampled at 2000 Hz, bandpass filtered between 20 and 1000 Hz using a Power1401 data acquisition system and Spike5 software (Cambridge Electronic Design, Cambridge, UK) and analysed using IGOR Pro (Igor Pro 5, Wavemetrics, Inc, Lake Oswego, Oregon, USA).

Pain and disability measures
 Participants completed a 10 cm numeric rating scale for their usual pain, worst pain and to indicate the amount of pain occurring during testing under each of the conditions.24 The duration of symptoms (months) was also recorded.

Procedure
 Electromyography
 Participants completed a visual choice reaction time stair-stepping task. Participants stood facing a force plate (Kistler, Switzerland, model 9286AA (400 × 600 mm) software: Bioware version 3.21) placed on a step (combined height 22 cm). They were instructed to step up as quickly as possible in response to a light, with either their left or right leg (indicated by a different coloured light in a choice reaction time task). Data were collected for five stepping repetitions on each leg; however, only the data for stepping with the test leg were analysed. EMG data were expressed relative to foot contact (Fz).

Hip external rotation, abduction and trunk side flexion strength
 A hand-held dynamometer (Nicholas manual muscle tester; Lafayette Instrument, Lafayette, Indiana, USA) was used to assess the isometric force. After two warm-up trials, the maximum of three trials was recorded. Hip external rotation strength was measured sitting. The dynamometer was placed over the tibia, 10 cm above the medial malleolus. The distance from the knee joint line and the dynamometer placement was measured (mm) and used to convert the force (N) data to a torque (N.m). Hip abduction strength was measured supine. The dynamometer was placed 10 cm above the lateral joint line and the participant abducted their hip maximally. The distance from the greater trochanter and the dynamometer placement was recorded (m) and used to convert the force (N) data to a torque (N.m).

The trunk side flexion test, based on that described by McGill et al25 was used to provide an indication of lateral core stability.26 27 In this test, the participant lies on their side, supported on their elbow with their opposite hand on their shoulder, the dynamometer is placed just proximal to the greater trochanter. Due to the difficulties in calculating the moment arm for this measure, force (N) was used.

Hip rotation range of motion
 The range of active hip rotation was measured lying supine, with neutral hip extension, participants had their thighs strapped together (rolled towel maintaining neutral hip abduction/adduction). Participants were asked actively to internally rotate then externally rotate their hips as far as possible. A gravity inclinometer (Acuangle, Isomed, Portland, Oregon, USA) placed on the lower fibula recorded the tibial inclination relative to the vertical. Internal rotation range of motion was recorded as the final internal rotation position added to the resting hip external rotation. The external rotation range was recorded as the difference between the final external rotation...
position and the resting external rotation position. An average of three trials was calculated.

Data analysis
The onset of EMG was identified visually from the raw data as the point at which EMG increased above the baseline activity. To remove the possibility of observer bias, traces were displayed individually with no reference to group, muscle or trial number, to any parameter of movement, or to EMG of another muscle. Visual inspection has been shown to be reliable and is preferred to computer-based methods as it is less affected by factors such as the amplitude of background EMG or the rate of increase in EMG activity.26 Onset times for each muscle were assessed by two separate examiners using data from 15 trials in two participants. There were no significant differences between the two examiners (p = 0.62) and the two scores were closely matched with a mean absolute difference of 1.2 ms with standard deviation (SD) of 2.4 ms.

Peak muscle activity after muscle onset was identified after rectifying and low pass filtering the EMG data at 60 Hz. Muscle onset was represented relative to footstrike (derived from the force plate) and the timing of peak muscle activity relative to muscle onset. The onset of VMO relative to VL was expressed by subtracting the onset of VMO from that of VL.

Muscle strength data were divided by the participant’s body weight, to enable comparisons between individuals.

Statistical analysis
Data were analysed for normality and homogeneity of variance. Unpaired t tests were used to compare muscle onset, muscle peak, reaction time (footstrike relative to visual stimulus to commence task), force plate variables, hip range of motion and strength between the groups. Independent one group t tests were used to determine if muscle onset differed significantly from zero (ie, did muscle onset occur significantly before or after heel strike). The alpha level was set at 0.05.

RESULTS
Descriptive characteristics
Participants in the PFP group had an average pain of 4 (1) and a worst pain of 7 (2) in the past week, and had experienced symptoms for an average of 6 (5) months. The painful leg in the PFP group was the dominant leg in 50% of cases. Participants were evenly matched in terms of age, height and weight (table 1).

Neuromotor control of the anterior and posterior sections of GM and the vasti
When individuals with no history of PFP completed the stair-stepping task the onsets of the anterior and posterior GM occurred just before heel strike; however, only the onset of anterior GM occurred significantly before heel strike (p = 0.01; fig 1). The onsets of the vasti occurred before stair contact (p = 0.00) and virtually simultaneously (fig 1).

When individuals with PFP completed the same stair-stepping task there was an alteration in the control of both the gluteal and vasti muscles (fig 1). Both the anterior and posterior portions of the GM were delayed in the PFP group relative to the control group (p = 0.01 and p = 0.012, respectively). In addition, the onsets of anterior and posterior GM occurred after heel strike in individuals with PFP (~20 ms); however, the difference was not significant. There was a delay in the onset of VMO in the PFP group leading to an alteration in the EMG onset timing difference (VL–VMO) between groups (p = 0.001).

Hip muscle and trunk side flexion strength
Participants with PFP experienced no pain during strength testing. There were no differences in the strength of the hip abductors (p = 0.59) and external rotators (p = 0.77) between individuals with and without PFP. However, individuals with PFP had significantly less (29%) resisted trunk side flexion strength (p = 0.035; fig 2).

Hip internal rotation and external rotation range of motion
There were no differences in range of motion of hip internal rotation (p = 0.88) and external rotation (p = 0.96) between individuals with and without PFP (fig 3).

DISCUSSION
The results of this study demonstrate an alteration of hip and trunk muscle function in individuals with PFP. In particular, there was a delay in the onset of the anterior and posterior portions of the GM in individuals with PFP compared with controls and a reduction of trunk side flexion strength associated with PFP.

Neuromotor control of the anterior and posterior GM in the asymptomatic population
In the asymptomatic group the onset of both portions of the GM occurred before heel strike; however, there were no significant differences between the onsets of the two portions of the muscles. These results compare favourably with previous research.17 27 28 Only one of these studies presented specific results for EMG onset timing of the GM and the vasti. Brindle et al17 demonstrated a similar pattern of GM (although the exact portion assessed was not stated) onset delay relative to vasti activation during a stair-climbing task.
As a result of the proposed differences in action between the anterior and posterior portions of the GM we investigated the actions of both portions during the stair-stepping task. The finding that there was no difference in the activation of the anterior and posterior portions of the GM in the asymptomatic population is novel and unexpected. Only one previous study investigated the EMG actions of the different heads of the gluteals. Although this study did not specifically measure EMG onset timing, the histograms present indicate similar onsets for the different heads of the gluteals. Based on our results it appears that the differing heads of the GM do not function independently during a stair-stepping task, but work in synchrony.

Neuromotor control of the anterior and posterior GM and the vasti is altered in the presence of PFP

When individuals with a history of PFP completed the stair-stepping task the onset of both the anterior and posterior portions of the GM was delayed compared with controls. This is the first study to investigate both portions of the gluteals using EMG. This finding of an alteration in GM activation concurs with previous research by Brindle and coworkers, but differs from the findings of Boling et al. Boling et al investigated only one portion of the GM and did report similar later EMG onsets in the FFP group, but found no significant differences between groups due to larger variability in their data. Individuals with FFP also displayed a delay in the onset of VMO relative to VL, which concurs with previous research.

The finding of a delay in the anterior and posterior portions of the GM has important clinical implications. It provides support for the common clinical assertion that GM control can be an important factor in FFP. In addition, the findings also provide theoretical rationale for physiotherapy treatment programmes that commonly include retraining of the GM muscle. However, the question as to whether specific retraining of the gluteals is effective in altering gluteal control remains to be investigated. Boling et al recently reported that a generalised weight-bearing rehabilitation programme does not change GM activation in FFP; however, the programme was not tailored to alter GM activation. Our previous studies in FFP confirmed that specific retraining of the vasti improved vasti neuromotor control.

As a result of the cross-sectional design of this study, the temporal relationship between pain and the changes in neuromotor control of the gluteals or vasti is not known. Although an altered latency of VMO has been found to be a risk factor for the development of FFP and decreased hip muscle strength has been associated with an increased risk of knee injury, there are multiple mechanisms by which pain may affect motor control. Knowledge of this temporal sequence has implications for the prevention and treatment of FFP.

The question as to the mechanism of the delay in hip muscle activation in these individuals with FFP is intriguing. At the knee, motor neuron inhibition has been shown to occur as a result of effusion, pain, ligament stretch and capsular compression. However, in the current study although the knee was the site of pathology in these individuals, a delay in muscle activation was found at the hip. It is possible that this delay may be related to the change in neuromotor control also found in the vasti, or due to proximal factors associated with FFP. As a result of the design of the study the mechanism of delay cannot be established.

No difference in hip muscle strength in individuals with and without PFP

There were no differences in the strength of the hip abductors and external rotators when individuals with FFP were compared with asymptomatic individuals. This finding is in contrast with previous research indicating decreased strength of the hip muscles in women with FFP. Our study is underpowered to perform a gender-specific analysis. Therefore, although it appears that a difference in hip strength may be important in women, in our study hip strength was not a feature of FFP in a mixed gender cohort.

Reduced trunk side flexion strength in the presence of PFP

Individuals with FFP were found to have less strength on the resisted trunk side flexion test than asymptomatic individuals. The trunk side flexion test has been reported by McGill et al as a measure of lateral core stability. Core stability is a product of both motor control and muscular capacity of the lumbo/pelvic/hip complex, thus pure hip strength tests only one element of core stability, it is possible that this test is a simple way to measure core control of the lumbo/pelvic/hip complex.

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Competing interests: None.

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Patient consent: Obtained.
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