Patellar Tilt Correlates with Vastus Lateralis:Vastus Medialis Activation Ratio in Maltracking Patellofemoral Pain Patients

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ABSTRACT: Patellofemoral (PF) pain is a common ailment of the lower extremity. A theorized cause for pain is patellar maltracking due to vasti muscle activation imbalance, represented as large vastus lateralis:vastus medialis (VL:VM) activation ratios. However, evidence relating vasti muscle activation imbalance to patellar maltracking is limited. The purpose of this study was to investigate the relationship between VL:VM activation ratio and patellar tracking measures, patellar tilt and bisect offset, in PF pain subjects and pain-free controls. We evaluated VL:VM activation ratio and VM activation delay relative to VL activation in 39 PF pain subjects and 15 pain-free controls during walking. We classified the PF pain subjects into normal tracking and maltracking groups based on patellar tilt and bisect offset measured from weight-bearing magnetic resonance imaging. Patellar tilt correlated with VL:VM activation ratio only in PF pain subjects classified as maltrackers. This suggests that a clinical intervention targeting vasti muscle activation imbalance may be effective only in PF pain subjects classified as maltrackers. © 2011 Orthopaedic Research Society. Published by Wiley Periodicals, Inc. J Orthop Res 30:927–933, 2012

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Patellofemoral (PF) pain is a common ailment of the lower extremity. It accounts for approximately 1 in 4 knee injuries diagnosed in sports medicine clinics,[1] with even higher incidence rates reported in females.[2] In 2008, an estimated 971,000 patients were diagnosed with PF pain in the U.S. with an associated cost of $8.3 billion (www.pearldiverinc.com, accessed 07/12/2011). These data were based on medical records from 20 million patients and represent conservative estimates of the prevalence and burden of PF pain on public health in the U.S.

While several factors are associated with PF pain, patellar maltracking is considered an important contributor.[3] Maltracking is typically characterized by excessive lateral alignment of the patella within the trochlear groove. Maltracking may be due to joint conformity,[4] a large quadriceps angle,[5] PF ligament properties,[6] altered muscle recruitment at the hip with increased femoral rotation,[7] and excessive foot pronation.[8] However, vasti muscle activation imbalance has garnered substantial attention from clinicians and researchers as a cause for patellar maltracking.[3,9] Evidence relating vasti muscle activation imbalance to patellar maltracking is limited. Vasti muscle activation imbalance is generally measured in two ways - 1) the delay in activation of the vastus medialis (VM) muscle compared to the vastus lateralis (VL) muscle,[10] and 2) the ratio of the magnitudes of normalized activations (VL:VM) of the VL and VM muscles.[9–12] We recently reported relationships between VM activation delay and patellar maltracking in PF pain subjects.[13] It is unclear, however, if patellar tracking measures are also related to vasti activation ratio. A previous study evaluated the relationship between patellar tracking measures and vasti activation ratio,[11] and concluded that maltracking was not related to large vasti activation ratios. However, this study evaluated patellar tracking measures under minimal weight-bearing conditions, and all PF pain subjects were grouped together. Studies highlighted the importance of evaluating patellar maltracking under weight-bearing conditions,[14,15] and demonstrated the need for classifying PF pain subjects into subgroups for accurate diagnosis[13] and effective treatment.[16] We developed a method for classifying PF pain subjects using patellar tracking measures obtained under weight-bearing conditions.[13] Accordingly, the first aim of this study was to investigate the relationships between VL:VM activation ratio and patellar tracking in PF pain subjects and pain-free controls. The second aim of this study was to investigate the relationship between VL:VM activation ratio and VM activation delay in PF pain and pain-free control subjects.

METHODS

Subject Population

We recruited 54 subjects: 15 active, pain-free controls and 39 PF pain subjects (Table 1). No statistical differences were found in age, height, or weight between male control and male PF pain subjects or between female control and female PF pain subjects. The PF pain subjects were diagnosed by a sports medicine physician (MF) with 20 years experience. A subject was included if he/she reported consistent PF pain for $\geq$3 months (range: 3 months–11 years), and if he/she experienced reproducible anterior knee pain during $\geq$2 of the following activities: stair ascent/descent, kneeling, squatting,

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Table 1. Population Characteristics of the Pain-Free Controls and PF Pain Subjects

	Patellofemoral pain ($n = 39$)			Pain-free control ($n = 15$)		
	Mean	SD	Range	Mean	SD	Range
Age (years)	30.9	5.8	19.0–50.0	28.4	3.8	22.0–35.0
Height (m)	1.73	0.09	1.57–1.93	1.72	0.09	1.58–1.91
Weight (kg)	67.7	11.4	46.5–90.7	65.3	8.8	52.1–77.3
Anterior Knee Pain Score	72	14	42–97	NA	NA	NA

Anterior Knee Pain Score[18] was used to evaluate subjective symptoms and functional limitations in the PF pain subjects.

prolonged sitting, or isometric quadriceps contraction.[17] For subjects with bilateral pain, the more painful knee was included. A subject was excluded if he/she had demonstrated knee ligament instability, patellar tendinopathy, joint line tenderness or knee effusion, previous knee trauma or surgery, patellar dislocation, or if signs of osteoarthritis were detected from MRI of the knee. We used the Anterior Knee Pain Score[18] to evaluate subjective symptoms and functional limitations in the PF pain subjects (Table 1); a score of 100 indicated no pain or disability. Subjects were informed on all aspects of the study and provided prior consent according to the policies of our Institutional Review Board.

Classification of Subjects Based on Patellar Tracking Measures

Subjects were classified into normal tracking and maltracking groups based on patellar tracking measures obtained from weight-bearing MRI (Fig. 1).[13] A subject's PF joint was imaged in an upright, weight-bearing posture using an open-configuration MRI scanner (0.5T SP/i; GE Healthcare, Milwaukee, WI). The subjects were requested to maintain the upright pose (at ~5° knee flexion without locking their knees) with both legs evenly loaded during scanning, and were assisted by a custom-built low-friction backrest that required a subject to support ~90% of his/her weight (Fig. 1A). The scan parameters were: repetition time, 33 ms; echo time, 9 ms; flip angle, 45°; matrix, 256 × 160 interpolated to 256 × 256; field of view, 20 cm × 20 cm; slice thickness, 2 mm. The scan time was ~2 min; all subjects could maintain the upright position during the scan.

Two-dimensional patellar tracking measures, patellar tilt and bisect offset, were measured from the weight-bearing MRI data (Fig. 1A). An oblique-axial plane image was

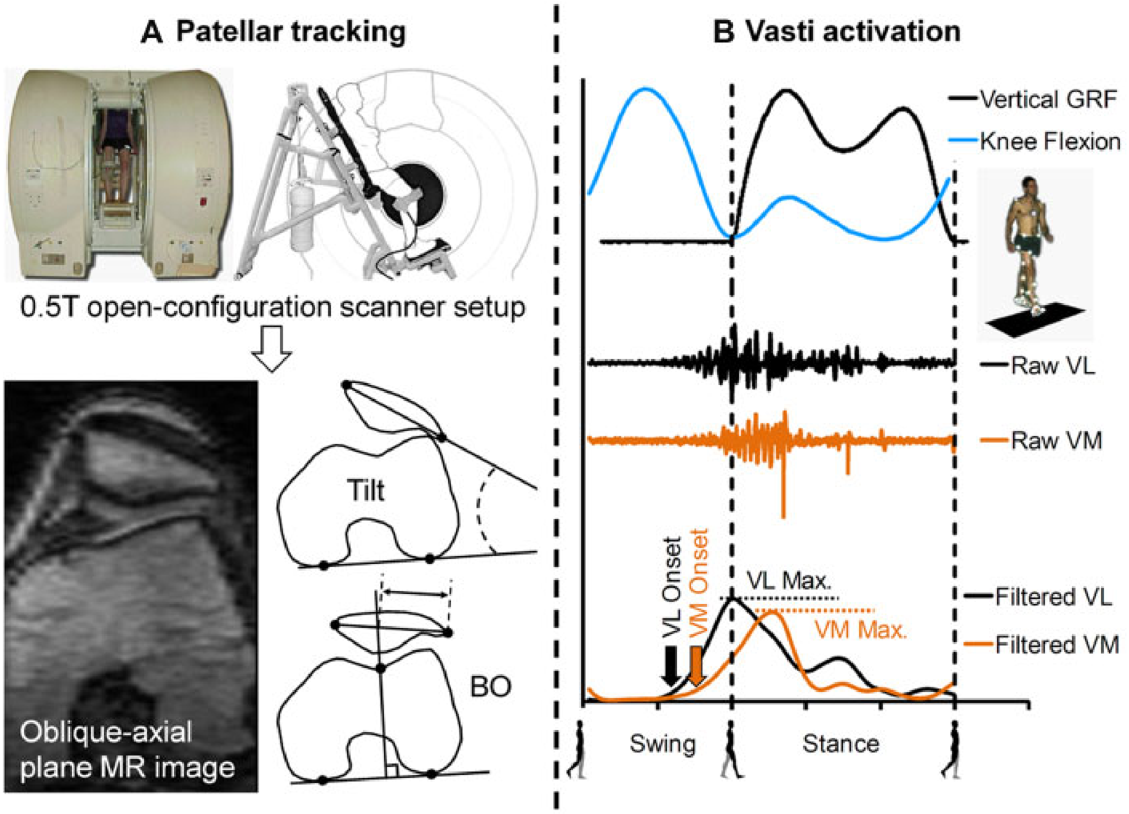

Figure 1. Measurement of (A) patellar tracking and (B) muscle EMG activity. (A) Subjects were scanned in an open-configuration MRI scanner, and oblique-axial plane images were used to calculate patellar tilt and bisect offset (BO). (B) Vertical ground reaction force (GRF), knee flexion, and raw and filtered EMG activity for vastus lateralis (VL) and vastus medialis (VM) muscles were measured during walking. Raw and filtered EMG data were synchronized with GRF and knee flexion data.

identified from the 3D MRI volume; this was done to maintain consistency with previous studies.[13,14,16] The oblique-axial plane was prescribed to intersect the center of the patella and the most posterior points on the femoral condyles. Anatomical landmarks were identified on this image; the landmarks included the most lateral and most medial points on the patella, the most posterior points on the condyles, and the deepest point of the trochlear groove (Fig. 1A).[13,16] Patellar tilt, defined as the angle between the patella and the posterior condyles, was used to measure patellar internal–external rotation relative to the femur. A more positive angle indicated greater external rotation of the patella relative to the femur. Bisect offset, defined as the percentage of the patella lateral to the midline of the femur,[16] was used to measure medial-lateral position of the patella relative to the femur. A greater bisect offset percentage indicated a more lateral position of the patella. All MRI measurements were performed by a single investigator; the average intraobserver variance between measurements due to the selection of the oblique-axial plane and anatomical landmarks was 2° for patellar tilt and 4% for bisect offset.[13]

A sex-specific classification technique was developed to categorize the PF pain and pain-free subjects into normal tracking and maltracking groups (Fig. 2). A sex-specific classification was needed because of differences in patellar tracking measures between sexes.[13] Due to skewness in their probability density functions, the measured patellar tilts and bisect offsets were best fit with a two-parameter Weibull model; the coefficients of determinations (R^2) were ≥ 0.84 for all cases (Fig. 2). The model includes a scale parameter $k(\beta)$ and a shape parameter, $w(\eta)$.[19] The shape parameter gives the distribution the flexibility to model probability distribution functions having either symmetric or skewed shapes. Because of this feature, two-parameter Weibull distributions provided substantially better fits to our tracking measures, which we previously showed to have skewed distributions.[13] The scale and shape parameter values corresponding to our distribution fits were 9.74 and 1.37 (males tilt), 13.76 and 2.14 (females tilt), 66.11 and 6.89 (males bisect offset), and 70.31 and 6.02 (females bisect offset). The Weibull distribution is commonly used to model biological and engineering phenomena.[20–22] This classification is a robust method to model observed population variability and to incorporate patellar tracking data from new subjects as we recruit them in our on-going study. Sex-specific maltracking thresholds were defined as the tilt and offset values corresponding to the 75th percentile of the distributions; a subject was classified as a maltracker if his/her patellar tilt or bisect offset value was in the highest quartile of measured population data.[13]

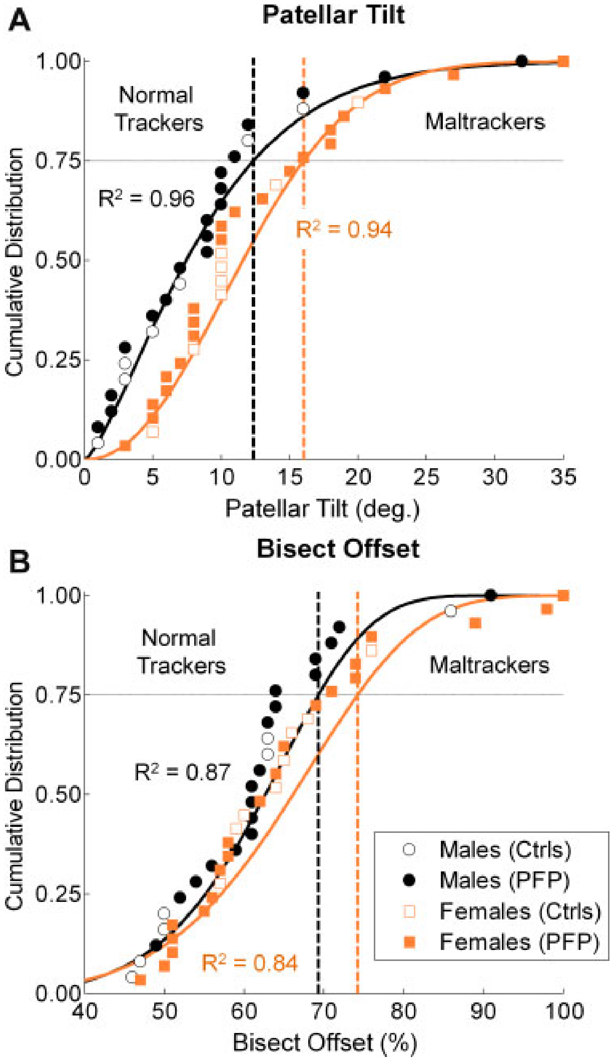

Figure 2. Sex-specific classification of pain-free controls (Ctrls) and PF pain (PFP) subjects into normal tracking and maltracking groups based on (A) patellar tilt and (B) bisect offset. The tilt and offset data were best fit with a two-parameter Weibull model (solid lines, $R^2 \geq 0.84$). The values corresponding to the 75th percentile (horizontal lines) were defined as the maltracking thresholds; a subject was classified as a maltracker if his/her tilt or offset value was greater than the sex-specific thresholds (vertical dashed lines).

Measurement of VL:VM Activation Ratio and VM Activation Delay

We measured VL:VM activation ratio and VM activation delay in all subjects during walking at self-selected speeds. We analyzed the symptomatic knee for the PF pain subjects or selected a knee at random for the pain-free controls. All subjects had a minimum of 3 trials during which the entire foot of the measured limb contacted a force plate. Retro-reflective markers were placed on lower limb landmarks,[23] and 3D marker trajectories were measured at 60 Hz using a 6-camera motion capture system (Motion Analysis Corp., Santa Rosa, CA). Ground reaction forces were measured at 2,400 Hz from a force plate (Bertec Corp., Columbus, OH). Marker trajectories were low-pass filtered using a zero-lag 4th-order Butterworth filter with a cut-off frequency of 15 Hz, and lower limb joint kinematics were calculated.[24] EMG signals were recorded using a multi-channel system (Motion Lab Systems, Baton Rouge, LA), and surface electrodes were placed on the VM and VL muscles.[25] Subjects performed five trials of maximum isometric muscle contractions to elicit maximum activation of the quadriceps muscles; while seated on a chair with the knees at ~80° flexion, a subject was instructed to extend his/her symptomatic knee against the resistance of the tester. The peak EMG value from all five trials was assigned as a muscle's maximum activation. Resting EMG signals were recorded with the subjects seated and relaxed. Raw EMG signals from the walking trials were high-pass filtered using a zero-lag 4th-order recursive Butterworth filter at 30 Hz, and then full-wave rectified and filtered using a Butterworth low-pass filter at 6 Hz.

Muscle activations collected during walking were normalized to the maximum activation values collected during the maximum contraction trials for each muscle.

Vasti EMG activations were analyzed with toe-off, the initiation of swing phase, defined as the beginning of a gait cycle (Fig. 1B). EMG data for the VM and VL muscles were synchronized with vertical ground reaction force and knee flexion angle to determine muscle activation onset times relative to heel strike. Muscle activations were detected using a threshold function: a muscle was determined to be "on" if its EMG signal exceeded the greater of 3 SD of its resting EMG value or 2% of the larger peak activation between the VM and VL muscles.[13] The 3 SD from rest threshold alone produced multiple spurious EMG onset times prior to heel strike in some subjects, while in some subjects with weak VM activation, neither the 3 SD from rest nor the 2% of VM activation identified the burst of activity prior to heel strike. We found the combination provided a reliable method to identify the clear burst of activity of each muscle prior to heel strike.

VL:VM activation ratio was determined from peak normalized activations of the muscles over the entire gait cycle.[11] A VL:VM activation ratio >1.0 represents a larger normalized activation magnitude of the VL muscle. VM activation delay was calculated as the difference between the VL and VM activation onset times. A positive VM delay indicated activation of the VL prior to VM activation. The VL:VM activation ratio and VM activation delay measurements from all gait trials of a subject were averaged.

We evaluated the relationship between VL:VM activation ratio and patellar tracking measures, and VM activation delay, in pain-free controls and all PF pain subjects grouped together, and in PF pain subjects classified as normal trackers and maltrackers. Linear regression models were used to test for the significance of a relationship ($p < 0.05$). Average VL:VM activation ratios were compared between the pain-free controls and all PF pain subjects grouped together, and between the pain-free controls and PF pain subjects classified into maltracking and normal tracking groups. Significant differences between the groups were assessed with two-tailed, unpaired *t*-tests ($p < 0.05$ for the pain-free controls and all PF pain subjects grouped together; $p < 0.017$ post-Bonferroni correction for the pain-free controls and PF pain subjects classified into maltracking and normal tracking groups).

RESULTS

Sex-specific maltracking thresholds corresponding to the 75th percentiles were 12.4° (males) and 16.0° (females) for patellar tilt (Fig. 2A), and 69.3% (males) and 74.2% (females) for bisect offset (Fig. 2B). Ten of 39 PF pain subjects were classified as maltrackers using these thresholds (4 males, 6 females). Among the 10, 6 had both abnormal tilt and abnormal bisect offset, 3 had only abnormal tilt, and 1 had only abnormal bisect offset.

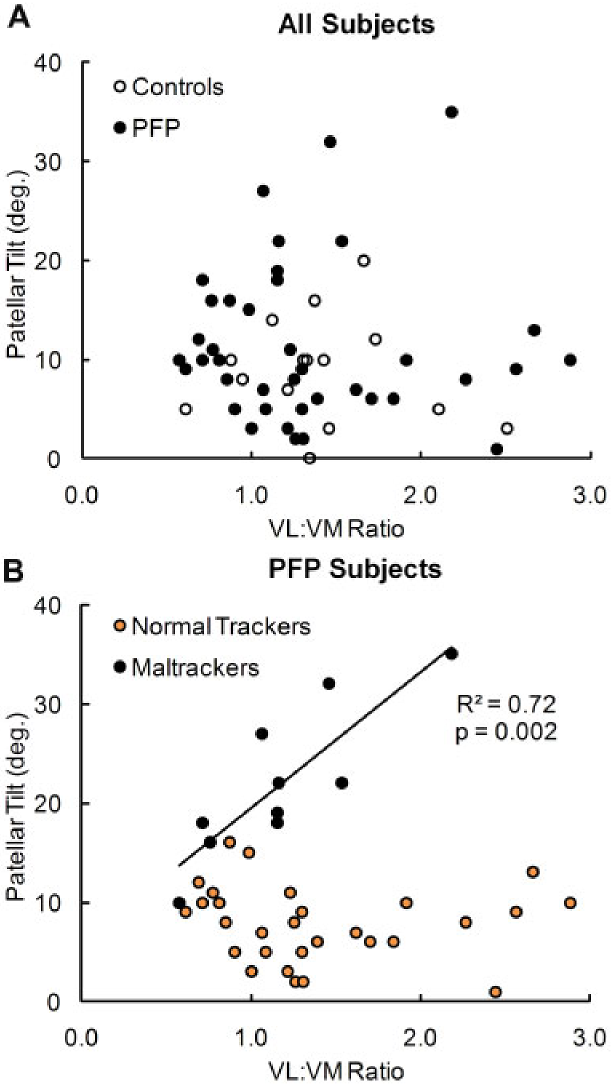

Figure 3. Relationship between patellar tilt and VL:VM activation ratio for (A) pain-free controls and all PF pain (PFP) subjects, and (B) PFP subjects classified into normal tracking and maltracking groups. The regression line represents a significant relationship ($R^2 = 0.72$, $p = 0.002$) in maltracking PFP subjects. No significant relationship existed between tilt and VL:VM activation ratio in pain-free controls ($R^2 = 0.01$, $p = 0.705$), all PFP subjects ($R^2 < 0.01$, $p = 0.949$), or normal tracking PFP subjects ($R^2 = 0.01$, $p = 0.652$).

Patellar tilt was significantly correlated with VL:VM activation ratio only in maltracking PF pain subjects ($R^2 = 0.72$, $p = 0.002$; Fig. 3). No significant correlations occurred between patellar tilt and VL:VM activation ratio in the pain-free controls, all PF pain subjects grouped together, and normal tracking PF pain subjects. There were no significant correlations between bisect offset and VL:VM activation ratio for any of the groups evaluated (Fig. 4).

VL:VM activation ratio was significantly correlated with VM activation delay only in maltracking PF pain subjects ($R^2 = 0.76$, $p = 0.001$; Fig. 5). No significant correlations occurred between VL:VM activation ratio and VM activation delay in the pain-free controls, all PF pain subjects grouped together, and PF pain subjects classified as normal trackers.

No differences were found in the means of VL:VM activation ratios between the pain-free controls and all PF pain subjects ($p = 0.724$). Average ± SD VL:VM activation ratios for pain-free controls and all PF pain

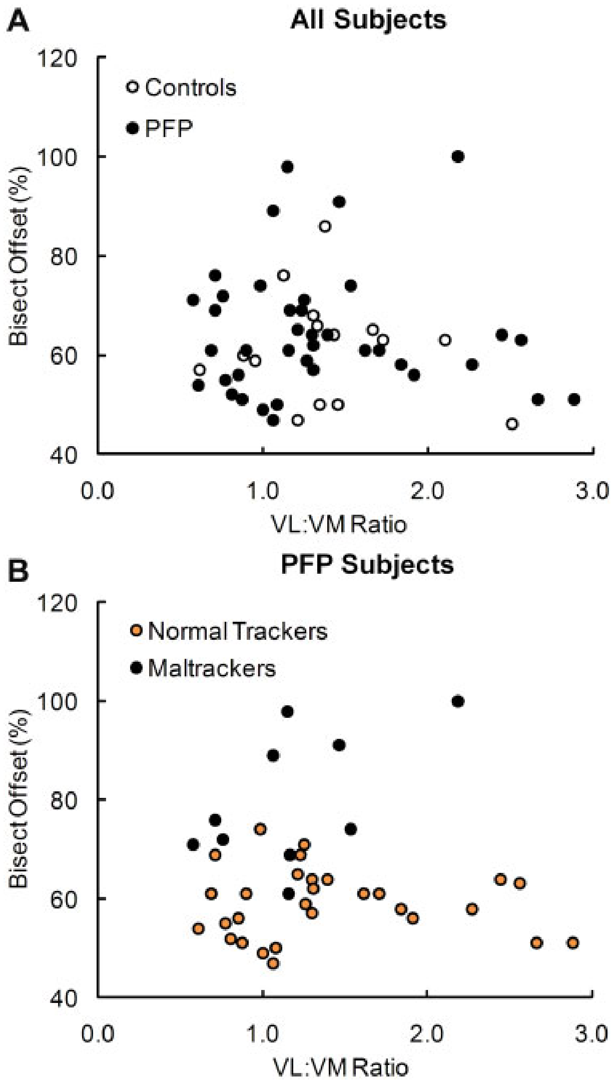

Figure 4. Relationship between bisect offset and VL:VM activation ratio for (A) pain-free controls and all PF pain (PFP) subjects, and (B) PFP subjects classified into normal tracking and maltracking groups. No significant relationship existed between bisect offset and VL:VM activation ratio in pain-free controls ($R^2 = 0.03$, $p = 0.551$), all PFP subjects ($R^2 < 0.01$, $p = 0.821$), normal tracking PFP subjects ($R^2 < 0.01$, $p = 0.761$), or maltracking PFP subjects ($R^2 = 0.30$, $p = 0.102$).

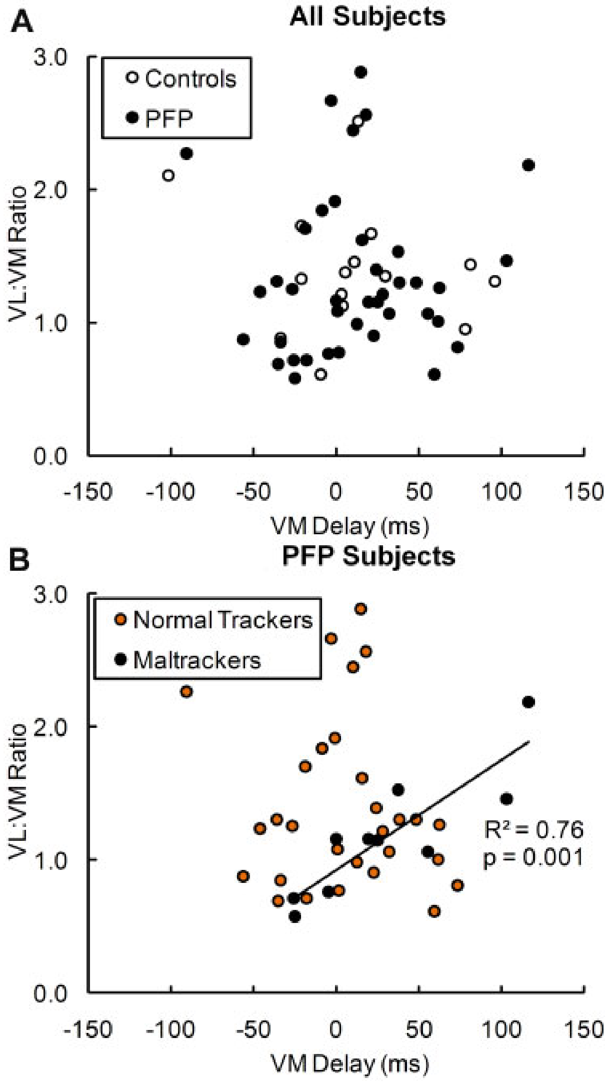

Figure 5. Relationship between VL:VM activation ratio and VM activation delay in (A) pain-free controls and all PF pain (PFP) subjects, and (B) PFP subjects classified into normal tracking and maltracking groups. The regression line represents a significant relationship ($R^2 = 0.76$, $p = 0.001$) in maltracking PFP subjects. No significant relationship existed between VL:VM activation ratio and VM activation delay in pain-free controls ($R^2 = 0.06$, $p = 0.399$), all PFP subjects ($R^2 = 0.01$, $p = 0.548$), or normal tracking PFP subjects ($R^2 = 0.01$, $p = 0.659$).

subjects were 1.40 ± 0.47 and 1.34 ± 0.60, respectively. No significant differences were found in the means of VL:VM activation ratios between the pain-free controls and maltracking PF pain subjects ($p = 0.254$), between the pain-free controls and normal tracking PF pain subjects ($p = 0.979$), and between the maltracking and normal tracking PF pain subjects ($p = 0.325$).

DISCUSSION

The purpose of this study was to investigate whether patellar maltracking is related to vasti muscle activation imbalance in PF pain and pain-free control subjects. Our results demonstrate a relationship between patellar tilt and VL:VM activation ratio only in PF pain subjects classified as maltrackers (Fig. 3). This study provides new evidence relating maltracking to vasti activation imbalance using a combination of weight-bearing MRI and gait analysis. Although studies have reported large VL:VM activation ratio[9] in PF pain subjects compared to pain-free controls, evidence relating VL:VM activation ratio to patellar maltracking is limited. One previous study[11] concluded that maltracking was not related to VM activation imbalance, and reported an inverse relationship between vasti activation ratio and patellar tracking.[11] Our conclusions differ from this previous study. There are likely two reasons for these differences - 1) the previous study evaluated patellar tracking during supine MRI under minimal (~15% body weight) weight-bearing conditions, while we evaluated patellar tracking during upright MRI under weight-bearing (~45% body weight supported by the measured limb) conditions; and 2) the previous study grouped all PF pain subjects together, while we classified our PF pain subjects based on patellar tracking. Indeed, when we lump all the pain-free controls and PF pain subjects together, we find an inverse relationship between vasti activation ratio and patellar tracking, consistent with the previous study.[11]

Our study clarifies the prevalence of vasti activation imbalance in PF pain subjects. No consensus exists regarding altered VL:VM activation ratio in PF pain subjects compared to pain-free controls. Previous studies tested the differences between the means of VL:VM activation ratios for pain-free controls and all PF pain subjects grouped together. Using this test, Santos et al.[10] and Souza and Gross[9] concluded that PF pain subjects exhibit abnormal VL:VM activation ratios compared to pain-free controls, while Powers et al.[12] reported no difference between the two groups. We found no difference between VL:VM activation ratios for pain-free controls and all PF pain subjects grouped together. Powers et al.[12] attributed the conflicting findings to methodological differences among studies, such as EMG normalization. We theorize that the conflicting findings may be due to the varying percentage of maltracking PF pain subjects recruited in these studies. In our study, only ~26% (10 of 39) PF pain subjects were maltrackers. Santos et al.[10] and Souza and Gross[9] studies might have included a greater percentage of maltracking PF pain subjects than our or the Powers et al.[12] study, resulting in significant differences in means of VL:VM activation ratios between the two groups.

Although normal tracking PF pain subjects displayed a large range of VL:VM activation ratios (Fig. 3), no correlations exitsed between tracking measures and activation ratios in normal trackers. A possible explanation is differences in PF joint geometry,[26] patella alta alignment,[4] or abnormal tensioning in the lateral retinacula[27] and/or the medial PF ligament[6] between subjects classified as normal trackers and maltrackers. As a result, tracking measures may be insensitive to VM activation imbalance in normal tracking PF pain subjects. Our study complements previous work done in understanding the role of the vasti muscles in PF pain subjects, including contributions to knee extension torque,[28] relative activations during open and closed kinetic chain exercises,[29] and relationship between muscle cross section area, insertion location, and fiber orientation to patellar tracking.[30–32]

We also investigated potential relationship between VL:VM activation ratio and VM activation delay. We found a relationship between the two only in maltracking PF pain subjects (Fig. 5). Measurement of VM activation delay during functional tasks requires synchronization of EMG data with joint kinematics and ground reaction forces.[13] However, VL:VM activation ratio can be obtained by attaching surface EMG electrodes on a patient without the need to measure joint motion or ground reaction force and is easier to perform in clinical settings.

A limitation of our study is that patellar tracking measures and vasti activations were measured during separate activities. It is difficult to acquire simultaneous VM activation delay and VL:VM activation ratio measurements during a backrest-assisted weight-bearing squat; the vasti muscle are active as soon as a subject positions himself/herself against the backrest. Reproducing a walking activity during MRI is not feasible. Another potential limitation is that we measured the activity of the entire VM muscle and not the isolated vastus medialis oblique (VMO) fibers.[10–12] It is difficult to distinguish between the VMO and VM longus activations using surface electrodes, and a recent article concluded that there is insufficient evidence to isolate the VM into separate bundles.[33] A third potential limitation is that our results were based on data acquired from an open-configuration MRI scanner, an expensive device available in few clinics around the world. A clinically feasible method for accurate classification of subjects based on patellar tracking measured under weight-bearing conditions remains a challenge. Another potential limitation is our definition of the 75th percentile of the Weibull model as maltracking thresholds; although this choice is subjective, small changes to the maltracking thresholds (e.g., ±5% points) do not change the significant relationships reported in our study. In addition, we cannot rule out the influence of increased medial femoral rotation due to altered muscle recruitment at the hip[7] or excessive foot pronation[8] on patellar maltracking in our subjects.

We showed a direct relationship between patellar tracking and VL:VM activation ratio in a subset of the PF pain population, those classified as maltrackers. Our results imply that clinical interventions targeting vasti muscle imbalance may improve patellar tracking only in maltracking subjects, highlighting the importance of accurate classification of PF pain subjects prior to the selection of a clinical intervention.

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