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Patellar Maltracking Correlates With Vastus Medialis Activation Delay in Patellofemoral Pain Patients

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Abstract

Background—Delayed onset of vastus medialis (VM) activity compared with vastus lateralis activity is a reported cause for patellofemoral pain. The delayed onset of VM activity in patellofemoral pain patients likely causes an imbalance in muscle forces and lateral maltracking of the patella; however, evidence relating VM activation delay to patellar maltracking is sparse. The aim of this study was to investigate the relationship between VM activation delay and patellar maltracking measures in pain-free controls and patellofemoral pain patients.

Hypothesis—Patellar tilt and bisect offset, measures of patellar tracking, correlate with VM activation delay in patellofemoral pain patients classified as maltrackers.

Study Design—Case control study; Level of evidence, 3.

Methods—Vasti muscle activations were recorded in pain-free (n = 15) and patellofemoral pain (n = 40) participants during walking and jogging. All participants were scanned in an open-configuration magnetic resonance scanner in an upright weightbearing position to acquire the position of the patella with respect to the femur. Patellar tilt and bisect offset were measured, and patellofemoral pain participants were classified into normal tracking and maltracking groups.

Results—Correlations between VM activation delay and patellar maltracking measures were statistically significant in only the patellofemoral pain participants classified as maltrackers with both abnormal tilt and abnormal bisect offset (R^2 = .89, P < .001, with patellar tilt during walking; R^2 = .75, P = .012, with bisect offset during jogging). There were no differences between the

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means of activation delays in pain-free and all patellofemoral pain participants during walking (P = .516) or jogging (P = .731).

Conclusion—There was a relationship between VM activation delay and patellar maltracking in the subgroup of patellofemoral pain participants classified as maltrackers with both abnormal tilt and abnormal bisect offset.

Clinical Relevance—A clinical intervention such as VM retraining may be effective in only a subset of patellofemoral pain participants—namely, those with excessive tilt and excessive bisect offset measures. The results highlight the importance of appropriate classification of patellofemoral pain patients before selection of a clinical intervention.

Keywords

patellofemoral pain; vastus medialis activation delay; surface electromyography; patellar maltracking; lateral patellar maltracking; anterior knee pain

Patellofemoral pain syndrome is a common ailment, accounting for 18% to 33% of reported knee disorders in sports medicine clinics.[21,22,32] There are likely several causes of patellofemoral pain,[5,25,47,60] and the origins of this condition remain unclear. One possible mechanism of patellofemoral pain is elevated stress at the cartilage-bone interface.[31] This hypothesis is based on the premise that localized stresses that are transmitted through the cartilage have the potential to excite nociceptors in the subchondral bone. Studies have demonstrated rich sensory innervation of mineralized bone[49] and the presence of substance-P pain receptor fibers in the subchondral plate of the human patellae.[69] Sanchis-Alfonso et al[61] reported a greater proportion of innervated soft tissues in patients with knee pain, but a direct relationship between tissue stress and pain is difficult to establish. In cases where soft tissue abnormality is not present, clinicians and researchers have accepted elevated cartilage-bone stress as a potential cause for pain in the patellofemoral joint.

One potential mechanism of elevated cartilage-bone stress is excessive lateral tracking of the patella within the trochlear groove.[29,32] Several factors may contribute to excessive lateral tracking of the patella. A large quadriceps angle—defined as the angle between the lines connecting the tibial tubercle to the middle of the patella to the anterior superior iliac spine of the pelvis—can direct the line of action of the extensor mechanism laterally, resulting in lateral displacement of the patella, reduced patellofemoral contact area, and elevated joint stress.[26,36,44] Measures of joint congruity, such as sulcus angle and depth index, are related to joint dysplasia[19] and can affect patellar alignment.[27,40] Passive soft tissue structures provide stability to the patellofemoral joint; furthermore, tightness in the lateral retinaculum or insufficient tension in the medial patellofemoral ligament can result in lateral displacement of the patella.[35,51,54] Altered muscle functioning and kinematics of the hip[7,9,15,65] and foot[24,46,50] joints are also reported to cause abnormal alignment of the patella within the trochlear groove. Another potential cause of patellar maltracking is imbalance of the quadriceps muscle forces; this may be a function of differences in muscle volume, insertion locations, fiber angles,[41] or altered activity of the muscles.[43]

Altered activity in the form of delayed onset of vastus medialis (VM) activity in comparison with vastus lateralis (VL) activity is believed to cause patellar maltracking in some

patellofemoral pain patients. A delayed onset of the VM activity may result in a medial-lateral force imbalance at the patella during the initial phase of knee extensor activity.[43] Several studies have reported delays in VM activity in patellofemoral pain patients, compared with pain-free controls,[14,16,62,68] whereas other studies have reported no differences in VL and VM activation timing between the 2 groups.[4,43,55,59,64] In a recent review article, Chester et al[12] reported substantial and unexplained heterogeneity among studies, making it difficult to extract clinical or therapeutic relevance from existing literature.

We have developed an approach for classifying patellofemoral pain patients based on measurements of patellar tracking obtained using weightbearing magnetic resonance (MR) imaging.[23] The purpose of this study was to determine if classifying patellofemoral pain patients into subgroups would lend insight into the large variation in VM activation delay. We evaluated potential relationships between VM activation delay and patellar tracking measures in the different subgroups. We hypothesized that measures of patellar tracking, patellar tilt and bisect offset, correlate with VM activation delay in patellofemoral pain patients classified as lateral maltrackers.

METHODS

Participant Recruitment

Fifty-five participants were recruited for this study: 15 active pain-free controls (28.2 ± 3.9 years, 1.72 ± 0.09 m, 65.2 ± 8.8 kg) and 40 with patellofemoral pain (28.9 ± 4.6 years, 1.71 ± 0.09 m, 66.5 ± 12.3 kg). The control group consisted of 7 men and 8 women, whereas the patellofemoral pain group included 21 men and 19 women. Participants with consistent patellofemoral pain for longer than 3 months (range, 3 months to 11 years) were recruited from the university's Orthopaedic Clinics and Sports Medicine Center and were diagnosed by a sports medicine physician. Inclusion criteria included reproducible anterior knee pain during at least 2 of the following activities: stair ascent/descent, kneeling, squatting, prolonged sitting, and isometric quadriceps contraction.[8] Exclusion criteria included knee ligament instability, patellar tendinitis, joint line tenderness or knee effusion, previous knee trauma or surgery, patellar dislocation, or signs of osteoarthritis. The severity of pain ranged from 42 to 97 on the Kujala questionnaire[45] (with 100 indicating no pain or disability). The pain-free controls were recruited from within the local community and screened to ensure that no one had previous traumatic injury or knee pathologic abnormality. All participants were notified about the nature of the study and provided prior consent consistent with the policies of the Institutional Review Board.

Gait and Electromyography Measurements

Each participant was analyzed while walking and jogging at self-selected speeds in a motion analysis laboratory. Average ± standard deviation (SD) speeds were 1.50 ± 0.16 m/s during walking and 2.65 ± 0.27 m/s during jogging. A minimum of 3 valid trials, with foot placement entirely on a force plate during an activity, was a criterion for a participant to be included in the study; all 55 participants met this criterion for walking, whereas jogging trials for 3 participants were excluded on the basis of this criterion.

Electromyography (EMG) signals were recorded with a 16-channel system (Motion Lab Systems, Baton Rouge, Louisiana). Surface electrodes were placed on the VM and VL muscles.[57] For the patellofemoral pain participants, EMG data were recorded from the symptomatic or more painful knee. For the controls, the selected knee for EMG data was chosen at random. Participants performed 5 trials of isometric muscle contractions to elicit maximum activation of the quadriceps muscles; they sat on a chair with the knee at approximately 80° of knee flexion and were instructed to extend the knee against the resistance of the tester. Verbal encouragement was given to the participant to try to improve the effort with each trial. The peak EMG value from all 5 trials was assigned as a muscle's maximum activation. Resting EMG signals were recorded with the participant seated and relaxed. Raw EMG signals were high-pass filtered with a zero-lag fourth-order recursive Butterworth filter (30 Hz) to remove motion artifact and then full-wave rectified and filtered with a Butterworth low-pass filter (6 Hz). Muscle activity was normalized to the maximum contraction values for each muscle.

Retro-reflective markers were placed on lower limb landmarks,[42] and 3-dimensional marker trajectories were measured at 60 Hz using a 6-camera motion capture system (Motion Analysis Corporation, Santa Rosa, California). Ground-reaction forces were simultaneously measured with EMG signals at 2400 Hz from a force plate (Bertec Corporation, Columbus, Ohio). Marker trajectories were low-pass filtered with a zero-lag fourth-order Butterworth filter with a cutoff frequency of 15 Hz. Spatiotemporal gait parameters—including walking and running speed, stride length, and cadence—were calculated from marker trajectories. Standard Newton-Euler inverse dynamics calculations were performed[17,18] to calculate lower limb joint kinematics and kinetics.

Anticipatory quadriceps EMG activations during leg swing phase before heel strike were evaluated (Figure 1). The EMG signals were synchronized with knee flexion angle and the vertical ground-reaction force. Toe-off, the initiation of swing phase, marked the beginning of data collection. Trial time was shifted to represent heel strike as time zero, and quadriceps activation onset times were measured relative to heel strike. To detect activation onset, a threshold function based on a muscle's resting and maximum activation values was chosen. The function assigned a muscle's onset threshold to be the greater of 3 standard deviations of its resting value[14,16] or 2% of the larger peak activation between the VM and VL muscles. We added the 2% criterion because 3 standard deviations alone produced multiple spurious EMG onset times before heel strike in some participants. For some participants with weak VM activation, neither 3 standard deviations from rest threshold nor 2% of VM activation identified the burst of activity before heel strike. We found that the larger peak activation between VM/VL muscles was a reliable method that identified the clear burst of activity of each muscle before heel strike in all participants. We measured VM activation delay relative to VL activation in all participants.

MR Imaging

Weightbearing scans of participants' knees were acquired in an open-configuration MR scanner (0.5-T SP/i MR, GE Healthcare Medical Systems, Milwaukee, Wisconsin). The participants were asked to maintain an upright posture without locking their knees (about 5°

of knee flexion, quadriceps engaged) with the support of a custom-built low-friction backrest.[3] The backrest was designed to require a participant to support about 90% of his or her own body weight. A 3-dimensional fast spoiled gradient-echo sequence was used to obtain 2-mm contiguous sagittal plane images of the patellofemoral joint. The scan time was approximately 2 minutes per participant using the following parameters: repetition time, 33 milliseconds; echo time, 9 milliseconds; flip angle, 45°; matrix, 256 × 160 interpolated to 256 × 256; field of view, 20 × 20 cm. All participants were able to maintain a static upright posture within the scanner for the duration of the scan.

Classification of Patellofemoral Pain Participants

Weightbearing MR images were analyzed to obtain the relative position of the patella with respect to the femur. To maintain consistency in methodology with a previous study,[23] 2-dimensional measurements were acquired from an oblique-axial plane of the 3-dimensional MR volume. The oblique-axial plane intersected the center of the patella and the most posterior points of the femoral condyles. The relative position of the patella with respect to the femur was calculated with anatomical landmarks (Figure 2). The landmarks were the deepest point of the trochlea, the most lateral and most medial points on the patella, and the most posterior points on the femoral condyles.[23] Medial-lateral translation of the patella relative to the femur was described with the bisect offset index, the percentage of the patella lateral to the midline of the femur.[10,66] A greater bisect offset percentage indicated that the patella was more lateral relative to the femur. Oblique-axial plane patellar rotation was measured with the patellar tilt angle, the angle between the patella and the posterior femoral condyles.[30] A more positive patellar tilt angle indicated greater external rotation relative to the femur. The average variance between measurements was 2° and 4% for patellar tilt and bisect offset, respectively.

The patellofemoral pain participants were classified into normal tracking and maltracking groups. Gender-based histograms of measured patellar tilt and bisect offset values were created, including both pain and control participants (Figure 3). A non-Gaussian 2-parameter Weibull distribution was the best fit to the measured patellar tilt and bisect offset data, with coefficients of determinations (R^2) greater than .90 in all 4 cases (male tilt, male bisect offset, female tilt, and female bisect offset). The Weibull distribution has been applied to model biological phenomena[6,33,37] and described in Haldar and Mahadevan.[34] Maltracking thresholds were defined as the 75% confidence intervals acquired from the Weibull distributions; a participant was classified as a maltracker if his or her patellar tilt or bisect offset values were in the highest quartiles of the measured patellar tilt and bisect offset values.

Relationships between VM activation delay and maltracking measures during walking and jogging were evaluated for 5 groups: pain-free controls, all patellofemoral pain participants together, patellofemoral pain participants classified as normal trackers, patellofemoral pain participants classified as maltrackers with either abnormal tilt or abnormal bisect offset, and patellofemoral pain participants classified as maltrackers with abnormal tilt and abnormal bisect offset. Linear regression models were used to test the significance of a relationship. Average VM activation delays were compared between the pain-free controls and all

patellofemoral pain participants. Significant differences between the groups were assessed with 2-tailed, unpaired *t* tests.

RESULTS

For all participants, average patellar tilt was 4° greater in women than in men ($P = .022$). Within controls, average patellar tilt was 6° greater in women than in men ($P = .014$), whereas average bisect offset was 11% greater in women than in men ($P = .005$). Maltracking thresholds for abnormal patellar tracking were 11.0° (tilt) and 68.1% (bisect offset) for men and 15.3° (tilt) and 72.3% (bisect offset) for women. Patellar tilt and bisect offset varied substantially among participants with patellofemoral pain, with several such participants having tracking measurements well below the maltracking thresholds (Figure 4). Of the 40 patellofemoral pain participants, 15 were classified as maltrackers with either abnormal tilt or abnormal bisect offset or both. Among maltracking patellofemoral pain participants, 7 were maltrackers (4 men, 3 women) with either abnormal tilt or abnormal bisect offset, and 8 were maltrackers (4 men, 4 women) with both abnormal tilt and abnormal bisect offset (Figure 4).

Maltracking patellofemoral pain participants with both abnormal tilt and abnormal bisect offset displayed significant relationships between patellar maltracking and VM activation delay ($R^2 = .89$, $P < .001$, between VM activation delay and patellar tilt during walking; $R^2 = .75$, $P = .012$, between VM activation delay and bisect offset during jogging) (Figure 5; Tables 1 and 2). There was large variation in measured VM activation delay and patellar tracking measures (Figures 5 and 6). There was no correlation between VM activation delay and patellar tracking measures in the control participants or in the patellofemoral pain group as a whole (Figures 5A and 6A; Tables 1 and 2).

Broad classification of the controls and patellofemoral pain participants resulted in average ± SD VM activation delays of 18 ± 57 milliseconds and 9 ± 39 milliseconds for the control and patellofemoral pain groups, respectively, during walking. Average ± SD VM activation delays were 28 ± 49 milliseconds and 21 ± 67 milliseconds for the controls and patellofemoral pain groups, respectively, during jogging. There were no differences between the means of VM activation delays between the control and patellofemoral pain groups during walking ($P = .516$) or jogging ($P = .731$).

DISCUSSION

A delay in VM activation relative to VL activation has been described in patellofemoral pain participants compared to pain-free controls.[14,16,62,68] This delay in VM activation has been theorized to cause an imbalance in quadriceps forces, resulting in lateral maltracking of the patella. The results of this study demonstrate a relationship between VM activation delay and patellar maltracking in a subset of patellofemoral pain patients. Broad classification of participants into pain-free and patellofemoral pain groups yielded no relationship between patellar tracking and activation delay, highlighting the importance of their classification based on maltracking measures.

Correlations between VM activation delay and patellar tracking were significant only in participants with both abnormal tilt and abnormal bisect offset (Figures 5 and 6; Tables 1 and 2). The other groups demonstrated large variation in activation delays and patellar tracking values but exhibited no clear relationship between the 2 measures. This may occur because VM activation delay is one of several factors affecting patellar tracking; the alignment of the patella is also influenced by its alta position, trochlear geometry, and tension in the surrounding passive structures. Participants with both abnormal tilt and abnormal bisect offset may represent extreme cases of maltracking and may be symptomatic of patella alta position,[48] trochlear dysplasia,[19] and abnormal tensioning in the lateral retinacula[13,39] and/or the medial patellofemoral ligament.[1,11,56] We are in the process of evaluating the effects of these anatomic conditions on patellar kinematics in our participant population.

Comparison of VM activation delay with patellar tracking measures provides additional insight into the controversial question of altered VM activity in patellofemoral pain participants. Previous studies consistently reported substantial variability in VM activation timings in control and patellofemoral pain groups and quantified the differences between the groups by testing the means of VM delays for statistical significance.[¶] Using this method, some studies reported differences,[14,16,62,68] whereas others[4,43,59,64] reported no difference between the pain-free and patellofemoral pain groups. In our study, we found no difference between the means of VM activation delays in pain-free controls and all patellofemoral pain patients grouped together. Wong[70] hypothesized that the discrepancies among studies are due to a lack of standardized methods in recognizing vasti activation times. An analysis of the different onset thresholds reported in the literature on our data set suggested minimal sensitivity of VM activation delay to onset threshold, so long as the algorithm was able to consistently detect the anticipatory activations before heel strike. We theorize that the primary reason for the discrepancies among studies may be the selection of patellofemoral pain participants. A study with a large number of maltracking patellofemoral pain participants with high tilt and bisect offset values would likely report significant delay in VM activation, compared with pain-free participants. In our study, only 20% of patellofemoral pain participants (8 of 40) were classified as lateral maltrackers with both abnormal tilt and abnormal bisect offset, which may explain the lack of significant differences between the means of VM activation delays in the pain-free controls and all patellofemoral pain participants. Furthermore, evidence relating VM activation delay to lateral tracking of the patella is sparse. Powers[58] evaluated relationships between vasti activation ratio and patellar maltracking measures acquired from supine MR imaging. Ingersoll and Knight[38] reported changes in patella locations after EMG biofeedback intervention on pain-free participants; Owings and Grabiner[55] evaluated the effects of patellar medial-lateral mobility by measuring vasti muscle activation onsets at flexed and extended knee positions; and Neptune et al[52] predicted reduced patellofemoral joint loads by advancing the onset of VM in a computational simulation. The present study utilizes a novel combination of weightbearing MR imaging and gait analysis to directly compare VM activation delay with patellar tracking measures in patellofemoral pain participants.

[¶]References 4, 14, 16, 43, 59, 62, 64, 68.

One challenge in understanding the mechanism of patellar maltracking is appropriate classification of participants. Fredericson and Yoon[28] noted that studies have not consistently demonstrated biomechanical differences between pain-free and patellofemoral pain participants, likely because of the difficulty in defining where the range of normal alignment ends and malalignment begins. In this study, gender-specific thresholds based on population measurements (28 men, 27 women) were introduced, and the female participants demonstrated greater patellar tilt ($P = .022$). The patellofemoral pain participants with tilt and bisect offset values in the highest quartiles were classified as maltrackers. This approach resulted in almost 40% of patellofemoral pain participants being classified as maltrackers with either abnormal tilt or abnormal bisect offset or both; the percentage of patellofemoral pain participants classified as maltrackers may vary among studies[20,63] owing to differences in methodology and participant population. The definition of the 75% confidence interval as a maltracking threshold is subjective; however, small changes to our maltracking threshold have minimal influence on the significant relationships presented in this study. This technique provides a robust method to represent the existing variability in patellar tilt and bisect offset measurements. Another potential cause for debate is the use of all participants (pain-free and patellofemoral pain) in determination of maltracking thresholds. Maltracking thresholds should arguably be based on pain-free participants, as previously reported.[23] This is ideal in research settings, with access to both pain-free and patellofemoral pain participants. In clinical settings, however, only symptomatic participants are evaluated. In the absence of a large research data set of pain-free participants, this method needs to be applicable to a clinician's data set to classify his or her patients into maltracking subgroups. In this study, we included pain-free and patellofemoral pain participants in our classification because of accessibility to both participant groups. Furthermore, the current results are based on 55 participants (15 pain-free, 40 patellofemoral pain), with 8 patellofemoral pain participants (4 men and 4 women) classified as maltrackers with both abnormal tilt and abnormal bisect offset. Analysis of additional participants from other centers may help test the generality of our findings. Also, there remains a need for obtaining patellar tracking data on a large population of healthy controls to provide a baseline for quantifying maltracking thresholds.

A potential limitation of this study is that we measured activity of the entire VM muscle, as opposed to activity of the isolated VMO fibers previously reported.[#] It was difficult to clearly distinguish between VMO and VML activations using surface electrodes; accordingly, we used one electrode to measure the activation of the entire VM muscle. It is unclear what influence characterizing delay of the isolated VMO fibers would have on the results of this study. Another limitation is that patellar alignment and activation timing were measured during separate activities. It is difficult to acquire quadriceps activation onset data during a backrest-assisted weightbearing squat because the quadriceps muscles are active as soon as a participant positions himself or herself. Also, reproducing a walking or jogging activity under MR surveillance is not feasible.

[#]References 4, 14, 16, 43, 55, 59, 62, 64, 68.

Patellofemoral pain syndrome has been described as a motor control problem.[32,52,67] Delayed activation of the VM relative to the VL muscle is theorized to cause a temporary imbalance in muscle forces, resulting in excessive lateral tracking of the patella. Several intervention studies using biofeedback and VM retraining have reported reductions in VM delays in patellofemoral pain participants[2,53]; however, there is little evidence relating reduced VM activation delay to improved patellar tracking. This study demonstrated a significant relationship between VM activation delay and patellar maltracking in one subset of patellofemoral pain participants, suggesting that clinical interventions to improve VM activation may improve patellar tracking only in participants with high tilt and bisect offset values. This finding underscores the importance of appropriate classification of patellofemoral pain participants before selection of a clinical intervention. An intervention study incorporating VM retraining and patellar tracking measurements would provide the much-needed evidence for relating reduced VM activation delay to improved patellar tracking in patellofemoral pain participants.

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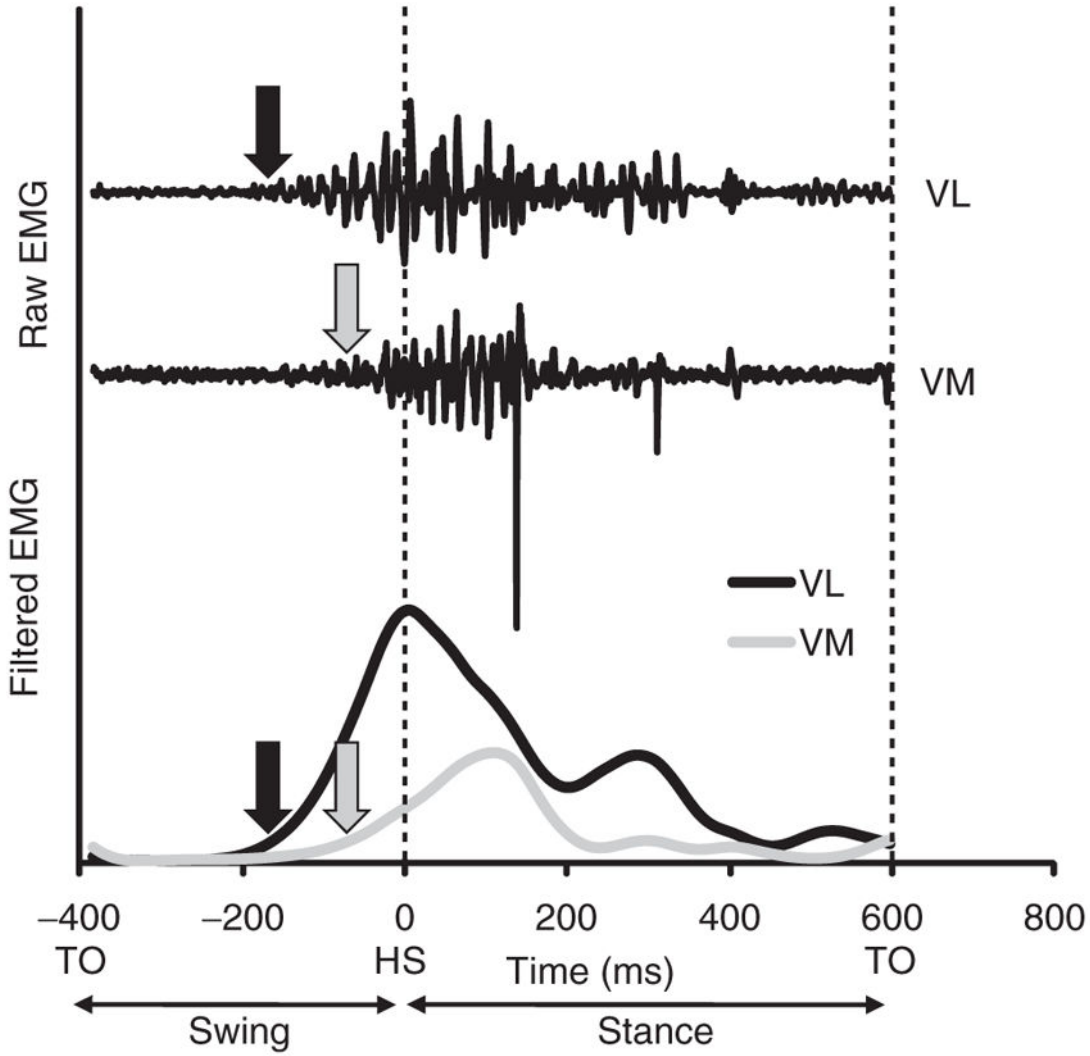

Figure 1.

Raw and filtered electromyography (EMG) activations of the vastus lateralis (VL) and vastus medialis (VM) muscles during a representative walk trial. Toe-off (TO; at left) marks the initiation of swing phase, which was the beginning of data collection. Trial time was adjusted to represent heel strike (HS) as time zero. The arrows indicate measured EMG onset times determined when activations crossed onset threshold, the greater of 3 standard deviations of resting EMG[14,16] and 2% of the larger peak activation between the VM and VL muscles.

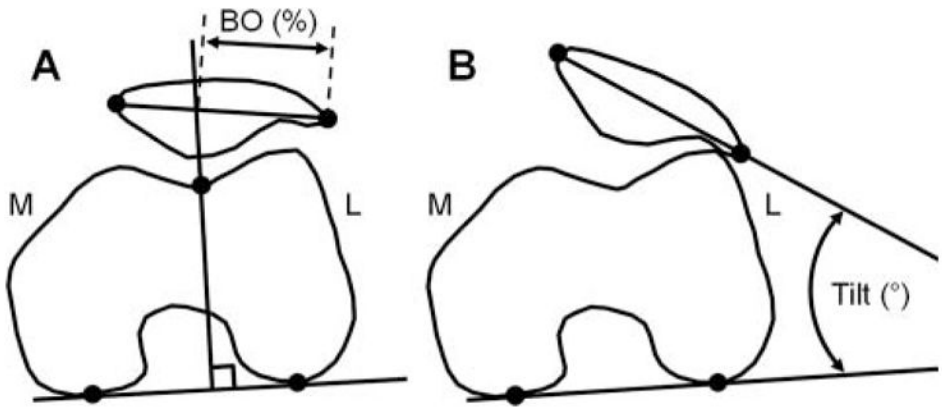

Figure 2.

Axial-plane patellofemoral joint kinematics illustrating (A) bisect offset (BO) index, a measure of the percentage of the patella lateral to the midline of the femur, and (B) patellar tilt (°), the angle formed by lines joining the posterior femoral condyles and the maximum width of the patella. Anatomical landmarks used to compute each measurement are indicated by the black dots. M, medial; L, lateral.

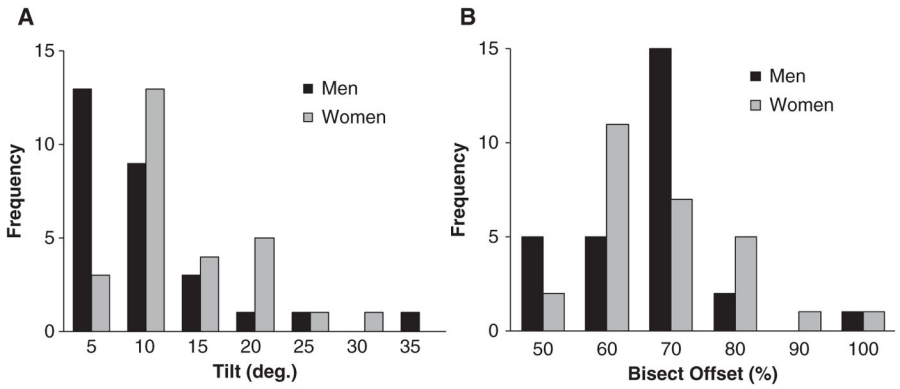

Figure 3.

Distributions of (A) patellar tilt and (B) bisect offset values for men (controls and patellofemoral pain, n = 28) and women (controls and patellofemoral pain, n = 27) measured during weightbearing at full extension.

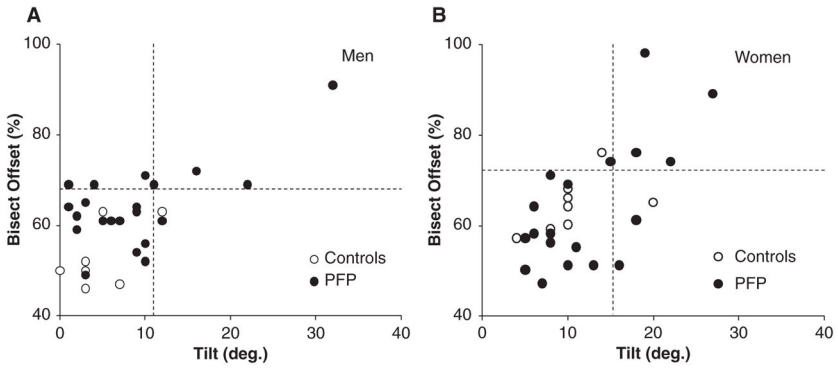

Figure 4.

Relationship between patellar tilt and bisect offset in (A) male and (B) female pain-free controls and patellofemoral pain (PFP) participants measured during weightbearing at full extension. The dashed lines represent gender-specific thresholds for classification of maltrackers based on abnormal tilt and abnormal bisect offset values.

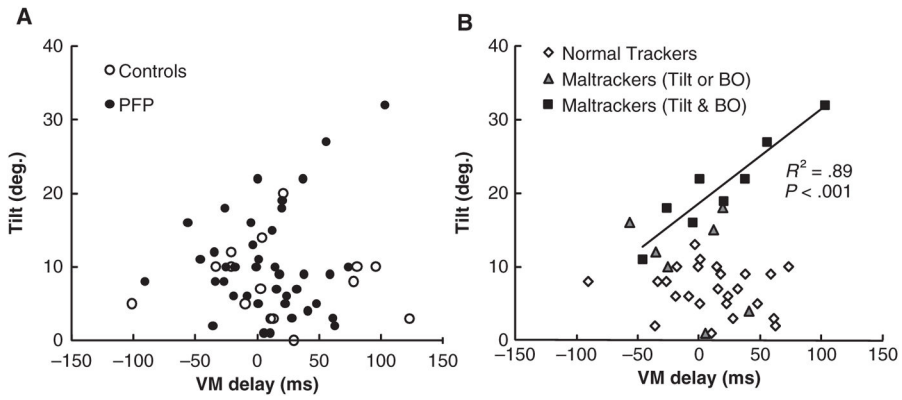

Figure 5.

Relationship between vastus medialis (VM) activation onset delay and patellar tilt during walking using two classifications: A, pain-free controls and patellofemoral pain (PFP) participants; B, PFP participants classified according to maltracking measures. Negative VM delay indicates activation of VM before vastus lateralis. The regression line represents a significant relationship (R^2 = .89, P <.001) in patients classified as maltrackers with both abnormal tilt and abnormal bisect offset (BO).

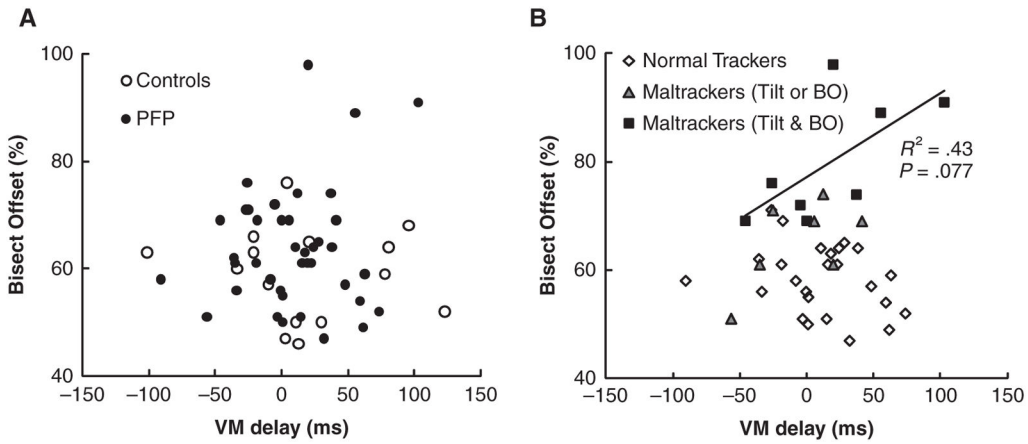

Figure 6.

Relationship between vastus medialis (VM) activation onset delay and patella bisect offset (BO) during walking using two classifications: A, pain-free controls and patellofemoral pain (PFP) participants; B, PFP participants classified according to maltracking measures. Negative VM delay indicates activation of VM before vastus lateralis.

TABLE 1

Relationship Between Vastus Medialis Activation Onset Delay and Patellar Maltracking Measures During Walking[a]

		Tilt		Bisect Offset	
Group	**n**	R^2	*P*	R^2	*P*
Controls	15	<.01	.860	.01	.744
Patellofemoral pain	40	.02	.330	.02	.358
Normal trackers	25	.02	.497	.08	.171
Maltrackers: tilt or bisect offset	7	.12	.446	.37	.144
Maltrackers: tilt and bisect offset	8	.89	<.001	.43	.077

[a]Expressed as coefficients of determination (R^2) and regression significance values.

TABLE 2

Relationship Between Vastus Medialis Activation Delay and Patellar Maltracking Measures During Jogging[a]

		Tilt		Bisect Offset	
Group	**n**	R^2	***P***	R^2	***P***
Controls	15	<.01	.878	<.01	.999
Patellofemoral pain	37	.07	.117	.05	.202
Normal trackers	24	.08	.181	.06	.268
Maltrackers: tilt or bisect offset	6	.06	.629	.54	.094
Maltrackers: tilt and bisect offset	7	.42	.113	.75	.012

[a]Expressed as coefficients of determination (R^2) and regression significance values.