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In vivo patellar tracking induced by individual quadriceps components in individuals with patellofemoral pain

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Abstract

Patellofemoral pain is a common knee disorders with a multi-factorial etiology related to abnormal patellar tracking. Our hypothesis was that the pattern of 3-dimensional rotation and translation of the patella induced by selective activation of individual quadriceps components would differ between subjects with patellofemoral pain and healthy subjects. Nine female subjects with patellofemoral pain and seven healthy female subjects underwent electrical stimulation to selectively activate individual quadriceps components (vastus medialis obliquus, VMO; vastus medialis lateralis, VML; vastus lateralis, VL) with the knee at 0° and 20° flexion, while three-dimensional patellar tracking was recorded. Normalized direction of rotation and direction of translation characterized the relative amplitudes of each component of patellar movement. VMO activation in patellofemoral pain caused greater medial patellar rotation (distal patellar pole rotates medially in frontal plane) at both knee positions ($p<0.01$), and both VMO and VML activation caused increased anterior patellar translation ($p<0.001$) in patellofemoral pain compared to healthy subjects at 20° knee flexion. VL activation caused more lateral patellar translation ($p<0.001$) in patellofemoral pain compared to healthy subjects. In healthy subjects the 3-D mechanical action of the VMO is actively modulated with knee flexion angle while such modulation was not observed in PFP subjects. This could be due to anatomical differences in the VMO insertion on the patella and medial quadriceps weakness. Quantitative evaluation of the influence of individual quadriceps components on patellar tracking

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CONFLICT OF INTEREST

The authors report no conflicts of interest associated with this study.

will aid understanding of the knee extensor mechanism and provide insight into the etiology of patellofemoral pain.

Keywords

Patella; Tracking; *In vivo*; Kinematics; Patellofemoral Pain; Malalignment

INTRODUCTION

Patellofemoral pain (PFP) is one of the most common knee disorders seen in an orthopaedic practice (Fulkerson, 1997; Powers *et al.*, 2004), with a reported incidence as high as 25% in the general population, and even higher among athletes (Devereaux and Lachmann, 1984; Insall, 1995). PFP syndrome is a serious problem because participation in sports and daily activities may be substantially affected by the pain, and chronic PFP may lead to joint degeneration (Fulkerson, 2002). Despite the high incidence, the pathophysiology of PFP is not clearly understood (Dye, 2005; Fulkerson, 2002).

Current literature suggests the etiology of PFP is multi-factorial (Csintalan *et al.*, 2002; Schepsis and Watson, 2005), and closely related to static patellar malalignment and abnormal patellar tracking (Fitzgerald and McClure, 1995; Fulkerson, 2002; Grelsamer and Stein, 2005; Sheehan *et al.*, 2009; Wilson *et al.*, 2009). Patellar alignment and tracking have been evaluated using techniques such as x-ray, computed tomography, magnetic resonance imaging, and motion analysis at different knee flexion angles (Guzzanti *et al.*, 1994; Hirokawa, 1992; McNally *et al.*, 2000; Sheehan *et al.*, 2009; Wilson *et al.*, 2009). However, none of these methods provide quantitative information on the actions of the individual quadriceps on the patella.

Patellar tracking relies on the bony architecture of the knee, but is also dependent on the magnitudes and directions of forces from the soft tissue structures surrounding the patellofemoral joint (Amis *et al.*, 2006). Reduced action of the medial stabilizers, especially the vastus medialis obliquus (VMO), is thought to be an important factor in patellofemoral kinematics (Goh *et al.*, 1995; Koskinen and Kujala, 1992; Lieb and Perry, 1968; Sakai *et al.*, 2000). The action of the VMO is believed to limit lateral shift and tilt due to its anatomical location (Goh *et al.*, 1995; Heegaard *et al.*, 1994; Koh *et al.*, 1992; Lin *et al.*, 2004; Sakai *et al.*, 2000). Therefore, it is important to understand how patellar tracking is influenced by the mechanical actions of each individual quadriceps component.

Our purpose was to compare, *in vivo* and noninvasively, patellar tracking patterns induced by selective activation of individual quadriceps components in subjects with PFP syndrome to healthy subjects. More specifically, this study focused on the function of the VMO and VL in controlling patellar tracking. We hypothesized that PFP is associated with altered six degree-of-freedom (DOF) mechanical actions of the individual quadriceps components. Quantitative evaluation of the role individual quadriceps play in patellar tracking will aid understanding of the knee extensor mechanism and provide insight into the mechanisms underlying PFP.

MATERIALS AND METHODS

Nine female subjects with clinically-diagnosed idiopathic PFP syndrome participated in this study. None of these subjects had PFP initiated by a traumatic episode or arthritis. In general, patients did not indicate any improvement from physical therapy. Seven female healthy subjects with no prior history of knee injuries were used as a control group. Each subject's activity level was documented and similar variation was seen in both groups, ranging from

inactive to active athletes. The study protocol was approved by the Institutional Review Board, and all subjects provided informed consent prior to participation. There were no significant differences between the two groups for gender, age, height, or weight (Table 1).

Before testing, patellar medial-lateral glide was manually tested to examine patellar mobility. Subjects sat upright with the back and thigh supported (Figure 1) (Lin *et al.*, 2003). The femoral condyles were held tightly by a clamp which was fixed to the seat; the clamp forced the femur to remain stationary during testing. The heel was placed on a cushioned table which could be positioned such that the knee was at either 20° flexion or full extension. Patellar maltracking is more pronounced between full extension and 20° flexion when the patella is not in close articulation with the trochlear grove (Sheehan *et al.*, 2009). In addition, the patellar clamp used to track patellar motion has been previously validated to move together with the patella during knee flexion-extension between 0°–20° (Lin *et al.*, 2003).

Active infrared markers were placed on a custom-designed lightweight clamp mounted on the patella (Lin *et al.*, 2003) and on the femoral condyle clamp (fixed to the chair). Three-dimensional patellar movements relative to the femur were measured using an optoelectronic motion capture system (OPTOTRAK™ 3020, Northern Digital, Inc., Waterloo, Canada). To minimize the effect of skin movement of the thigh associated with stimulation-induced muscle contraction, patellar movement was calculated relative to the femoral condyle clamp.

Patellofemoral movements were described by joint coordinate systems (JCS) embedded in the femur and patella as previously described (Grood and Suntay, 1983; Lin *et al.*, 2003). The patellar flexion axis was the transepicondylar line of the femur. The Y-axis was the longitudinal axis of the patella and was the axis of patellar medial/lateral tilt. The third axis, perpendicular to the above axes, characterized medial/lateral rotation of the patella, with medial rotation defined as the distal pole of the patella moving medially in frontal plane. Linear shifts of the tibia and patella relative to the femur were described by translations along the three axes of the femoral coordinate system: medial-lateral translation along the femoral X-axis, proximal-distal translation along the femoral Y-axis, and anterior-posterior translation along the femoral Z-axis; where the positive directions of motion are listed first (for a right knee). The positive directions of patellar rotation for a right knee were flexion, medial tilt, and medial rotation. The zero position of the patella was defined as the position when the local patellar coordinate system and the local femoral coordinate system were parallel. Therefore, an initial patellar orientation with respect to the femur at full extension can be described. Translations and rotations of the patella and tibia relative to the femur were calculated from the measured infrared marker coordinates in six DOFs. For left knees appropriate sign changes were made to convert translations and rotations to the right-knee conventions.

To selectively activate an individual head of the quadriceps muscle, a pair of surface electrodes was placed on the skin above the targeted muscle component (VML, VMO, and VL) and constant-current electrical stimulation was delivered through the electrodes. The approximate location of the motor point (Warfel, 1993) for each muscle was located and marked with a skin marker. Starting with a low-intensity current, a pair of electrodes with conducting gel was placed on skin at the marked location and was moved around to locate the point where stimulation induced the strongest contraction with a constant current intensity. If no contraction was successfully induced, the intensity of the current was slightly increased and the procedure was repeated until an obvious muscle contraction was achieved.

Once the motor point was located, a train of constant current stimulation pulses was used to activate the muscle at 2-second intervals. The pulse width was 0.3 msec repeated at 25 pulses per second. The pulse train duration was 600 msec. The current amplitude was adjusted for each quadriceps component so that visually observable and palpable contraction of the target

muscle was elicited without significant current overflow to the surrounding muscles (monitored by palpation of the quadriceps tendon and visual inspection). To evaluate the stimulation-elicited contraction at the target muscle and current overflow to neighboring muscles, the M-wave (compound muscular action potential) was recorded for each muscle component during electrical stimulation in selected subjects. When no measurable M-wave was detected in the surrounding muscles while obvious contraction of the targeted muscle was induced, the stimulation was considered valid with negligible current overflow. Practically, a muscle had to produce visible/palpable contraction to contribute measurably to patellar tracking.

Each quadriceps component was stimulated at the motor point with the knee at full extension and at 20° of flexion, while 6 degree-of-freedom patellar tracking was simultaneously recorded. For each muscle at each knee angle, four trials were recorded and the mean value of the four trials used for further analysis. To minimize muscle fatigue, all sequential trials were separated by rest periods.

To compare patellar tracking across different subjects, the normalized direction of translation (DOT) and direction of rotation (DOR) were used to characterize the relative amplitude of the components of 3-D patellar movement as follows (Zhang *et al.*, 1998):

$$DOR_1=R_1/R, DOR_2=R_2/R, DOR_3=R_3/R, R=\sqrt{R_1^2+R_2^2+R_3^2} \quad (1)$$

$$DOTx=Tx/T, DOTy=Ty/T, DOTz=Tz/T, T=\sqrt{T_x^2+T_y^2+T_z^2} \quad (2)$$

where R_1, R_2 and R_3 were rotations about the three JCS axes of the patellofemoral coordinate systems. T_x, T_y and T_z were translations, which were normalized to the knee width at the level of the femoral epicondyles for each subject, along the X, Y and Z axes of the femur, respectively.

Differences in patellofemoral kinematics between groups were analyzed for each muscle using two-way ANOVA with repeated measures (group had two levels: healthy and PFP, and knee flexion angle had two levels: 0° and 20°). If an overall significant difference was detected, the Tukey's HSD post-hoc test was used to test for differences at specific knee flexion angles. Alpha and beta were preset to 0.05 and 0.20, respectively. The number of male and female subjects in each group was compared using the Fisher's exact test, and subject age, height, and weight were compared between groups using the Student's t-test.

RESULTS

There was no obvious resistance to manual medial or lateral patellar translation for the entire PFP group, except a single subject who exhibited noticeable tightness of the lateral parapatellar structures resulting in resistance to medial patellar glide. For each subject, consistent patellar movement was induced through electrical stimulation of an individual quadriceps component (Figure 2). Typical results from healthy subjects show that selective VMO activation caused patellar extension, medial tilt, lateral rotation, and medial, proximal, and anterior translation (Figure 2). VML activation induced patellar extension, medial tilt, and lateral rotation and medial, proximal, and posterior translation. VL activation induced patellar extension, lateral tilt, medial rotation, and lateral, proximal, and posterior translation.

The normalized DOR and DOT varied with knee angle and varied between healthy and subjects with PFP. In healthy subjects, the primary DOR during selective VMO activation was medial

tilt at full extension and patellar flexion at 20° of knee flexion (Table 2). In PFP subjects at both knee positions VMO activation caused medial patellar rotation compared to lateral rotation in healthy subjects ($p<0.01$, $p<0.001$ respectively; Figure 3). VMO activation also caused less patellar flexion in PFP subjects with the knee in 20° flexion ($p<0.01$). Finally, VMO activation caused proximal and anterior patellar translation in subjects with PFP compared to distal and posterior translation in healthy subjects at 20° flexion ($p<0.001$, $p<0.001$, respectively; Table 3).

There were no significant differences in patellar rotations between healthy and subjects with PFP during VML activation (Table 2). However in subjects with PFP at both knee positions, VML activation caused anterior patellar translation compared to posterior translation in healthy subjects ($p<0.05$, $p<0.001$, respectively; Table 3). At 20° flexion, VL activation caused lateral patellar translation compared to medial patellar translation in healthy subjects ($p<0.001$, Figure 4B).

In subjects with PFP, the initial static patellar position at full extension was extended ($p<0.001$), more laterally rotated ($p<0.05$), and shifted in the proximal direction (patella alta, $p<0.05$) compared to healthy subjects ($p<0.001$, Figure 5).

DISCUSSION

It has been shown that each quadriceps component is responsible for different portions of patellar movement during knee flexion-extension (Goh *et al.*, 1995; Heegaard *et al.*, 1994; Lin *et al.*, 2004; Sakai *et al.*, 2000). The action of the VMO component has been studied extensively (Goh *et al.*, 1995; Heegaard *et al.*, 1994; Koh *et al.*, 1992; Lin *et al.*, 2004; Sakai *et al.*, 2000; Zhang *et al.*, 2003) because the VMO is thought to be the most important quadriceps component for preventing abnormal patellar tracking (Fulkerson, 2002; MacIntyre *et al.*, 2006). In healthy subjects in the current study contraction of the VMO produced lateral patellar rotation. This counterintuitive finding is likely due to the specific anatomy of the obliquus portion of the vastus medialis muscle. Anatomical studies have shown that the muscle fibers of the VMO are more obliquely oriented than the fibers of the VML (Farahmand *et al.*, 1998), and it has been shown that the tendon associated with the VMO inserts on the medial side rather than the superior edge of the patella (Holt *et al.*, 2008). This oblique orientation and the medial location of the tendon insertion on the patella would produce medial patellar tilt and lateral rotation (inferior pole moving laterally in the frontal plane) with VMO contraction. In subjects with PFP, contraction of the VMO produced medial patellar rotation. This may be due to anatomical differences between healthy subjects and subjects with PFP (i.e. PFP subjects may have more superiorly-oriented VMO muscle fibers or a different tendon insertion site). Weakness in the oblique muscle fibers of the VMO in subjects with PFP could also contribute to this finding (Amis *et al.*, 2006; Thomee *et al.*, 1995; Young *et al.*, 1987).

In the healthy knee, the presence of obliquely oriented muscle fibers and a medial tendon insertion site also explains the modulation from extension to flexion and from proximal and anterior patellar translation to distal and posterior patellar translation as the knee moves from full extension to 20°. As the patella flexes, a medially oriented (as opposed to superiorly oriented) pull would cause the patella to flex further and translate in the medial, distal, and posterior directions. This modulation of action is consistent with the hypothesis that in the healthy knee the VMO stabilizes the patella at or near terminal extension (Goh *et al.*, 1995; Lieb and Perry, 1968).

In a recent study, Lin et al. (2004) found that VML activation produced patellar extension, medial tilt, and lateral rotation. Results from the current study show a similar patellar rotation patterns in both study populations. However, in subjects with PFP, VML activation produced

anterior patellar translation compared to posterior patellar translation in healthy subjects. Posterior patellar translation in healthy subjects suggests the VML also plays a role in stabilizing the patella during knee flexion by causing the patella to have a more firm articulation with the trochlear groove. The decrease in posterior stabilization force from the medial quadriceps components may suggest the presence of moment arm dysfunction (i.e. the line of action of the medial quadriceps muscle is altered in subjects with PFP syndrome) (Wilson and Sheehan, 2009).

In healthy subjects, VL activation produced lateral patellar translation at full extension and medial patellar translation at 20° flexion. At 20° of knee flexion the patella begins to articulate with the trochlear groove and VL contraction causes the patella to have a firm articulation with the trochlear groove. The proximal portion of the trochlear groove is oriented medially, and tends to guide the patella medially, so an increased articulation with the groove will cause a medial shift as the patella slides into the groove. These results are further supported by a recent *in vivo* study which found that in healthy subjects the patella translated laterally during the first 15° of knee flexion, but after 15° medial patellar translation occurred throughout the range of knee flexion during squatting (Wilson *et al.*, 2009). In the current study, VL activation produced lateral patellar translation at both knee positions in subjects with PFP. Similarly, the aforementioned study found consistent lateral patellar translation throughout knee flexion in subjects with PFP (Wilson *et al.*, 2009).

Excess lateral patellar translation in subjects with PFP syndrome has been shown in numerous previous studies (Brossmann *et al.*, 1993; MacIntyre *et al.*, 2006; Powers, 2000; Wilson *et al.*, 2009) and it is commonly thought to be related to medial quadriceps weakness. Weakness of the VMO quadriceps component may cause the extensor mechanism to be incapable of generating effective forces in opposition to the action of the VL. Ineffective opposition of the force from the VL may lead to an imbalance in the extensor mechanism and overloading of the lateral compartment of the patellofemoral joint, which may ultimately lead to pain (Fulkerson, 1997). Since patellar tracking is also affected by other factors such as the shape of the trochlear groove and the pattern of the joint contact, tracking patterns found in this study cannot be fully explained by the action of the quadriceps muscles.

A limitation of this study is the relatively small sample size as the sample was limited to unilateral limbs from only female subjects in our larger PFP patient population. VL activation caused trends toward increased lateral tilt and decreased medial rotation in subjects with PFP compared to healthy subjects, which did not achieve statistical significance, likely due to power considerations. However, inclusion of a homogeneous, non-paired population was deemed a more important consideration as the lack of significance for these two trends did not substantially impact the findings of this study. Due to the limited sample size caution must be taken when extrapolating findings from this study to a larger population.

In conclusion, this study provides an *in vivo* and noninvasive tool to characterize 3-D patellar tracking induced by *individual* quadriceps components in PFP. Results suggest that in healthy subjects the mechanical actions of some of the quadriceps are actively modulated with knee flexion angle. However, this modulation of action was not demonstrated in subjects with PFP. Lack of active modulation may result in decreased patellar stabilization during knee flexion leading to abnormal patellar tracking. In addition, the 3-D mechanical action of the VL is altered in subjects with PFP, which may lead to increased lateral patellar translation. Excess lateral patellar translation in PFP syndrome may be related to VMO weakness which may cause ineffective forces in opposition to the action of the VL.

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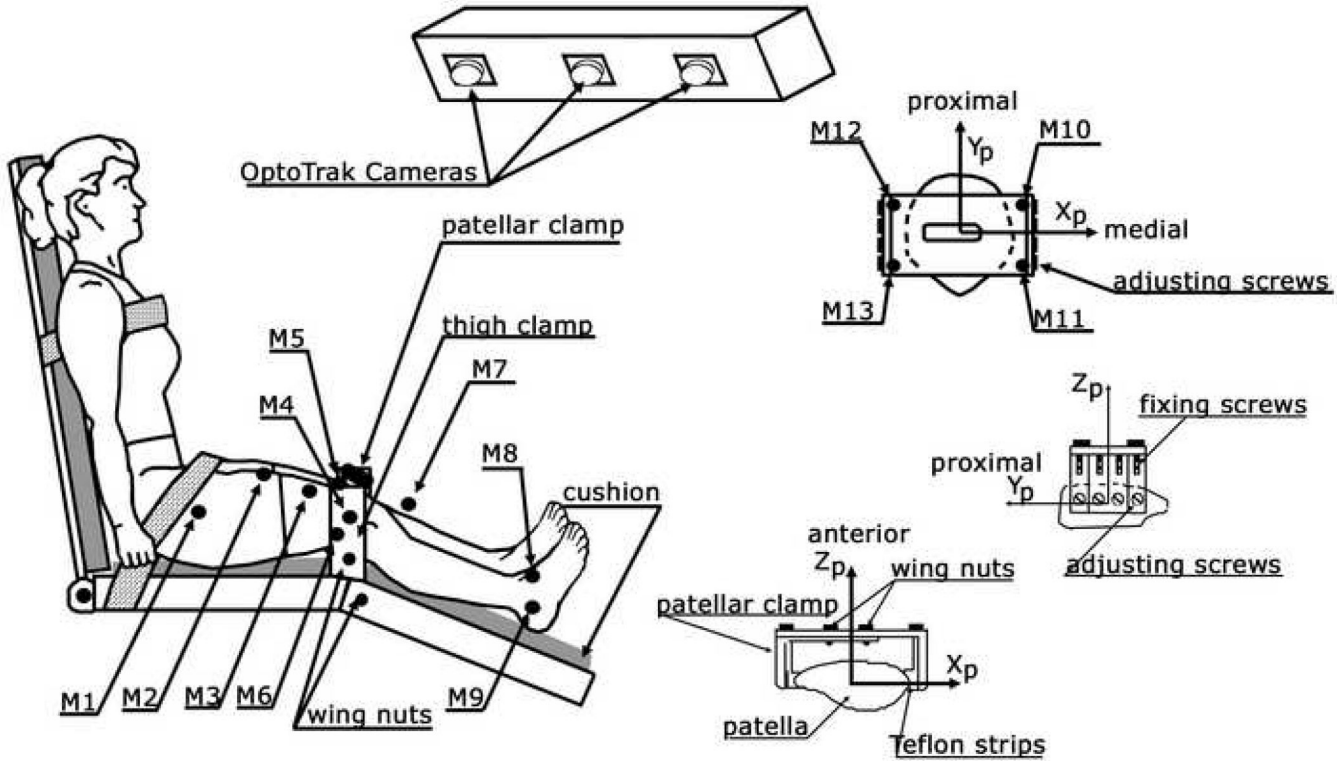

Figure 1.

Experimental setup for *in vivo* and noninvasive patellar tracking. The subject was seated with the thigh and trunk strapped to the seat and backrest. The femoral condyles were fixed from both the medial and lateral sides with a thigh clamp. Two pairs of wing nuts were tightened from the medial and lateral sides to fix the femoral condyles. Three markers were placed on the thigh (M1, M2, M3). Three markers were attached to the thigh clamp (M4–M6) with M4 placed on the knee flexion axis and lateral to the lateral epicondyle. Three markers were attached to the lower leg (M7–M9), and three were fixed to the four corners of the patellar clamp (M10–M13).

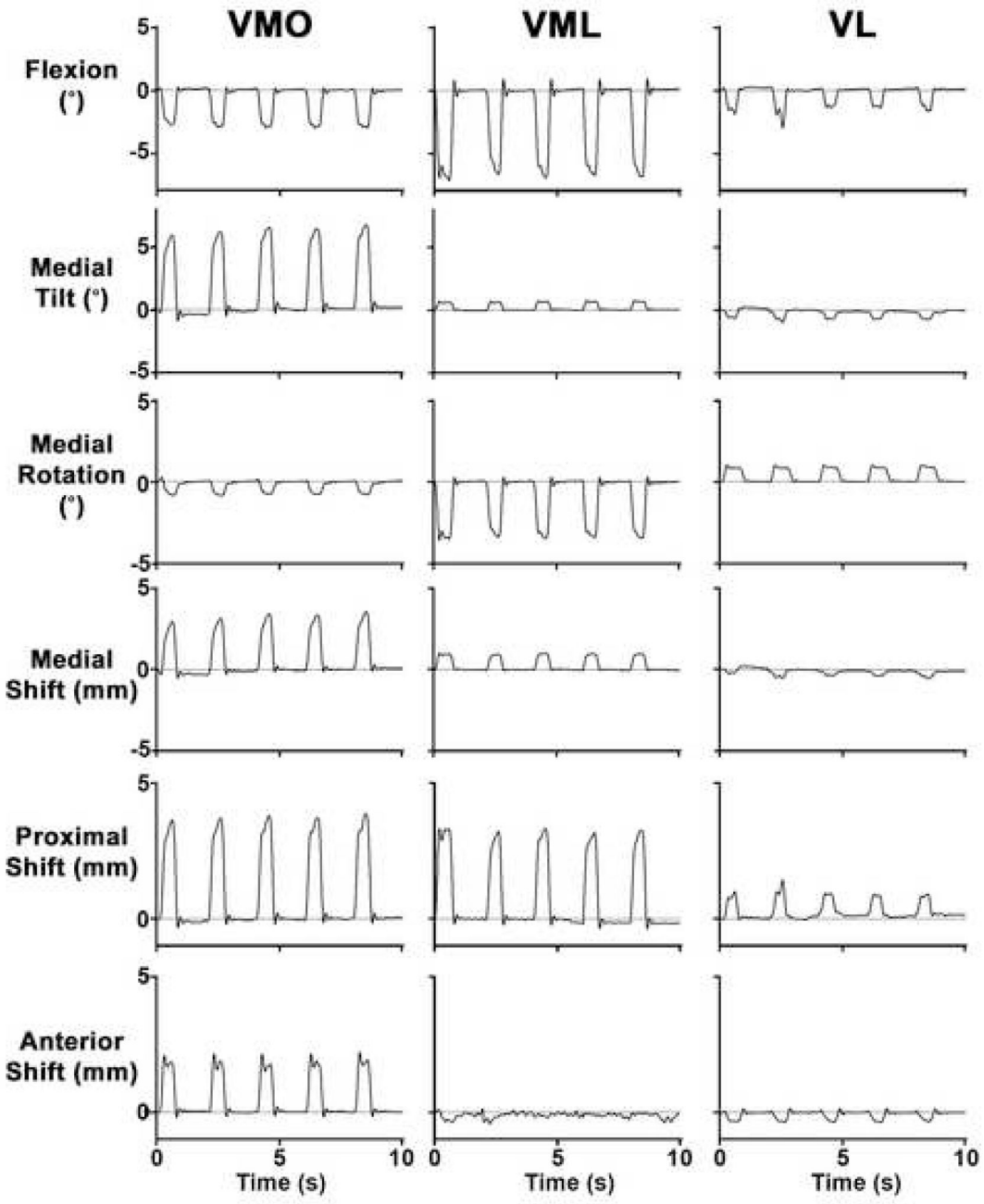

Figure 2.

Typical result for patellar tracking in healthy subjects induced through selective activation of individual quadriceps components with the knee at full extension, neutral tibial rotation, and neutral tibial abduction. The stimulation train (and thus the contraction) was repeated at 2-second intervals. From top to bottom, the six rows correspond to patellar flexion, medial tilt, medial rotation, medial shift, proximal shift, and anterior shift, respectively. The positive direction of each DOF is given for the ordinate. The zero position corresponded to the patellar position prior to stimulation.

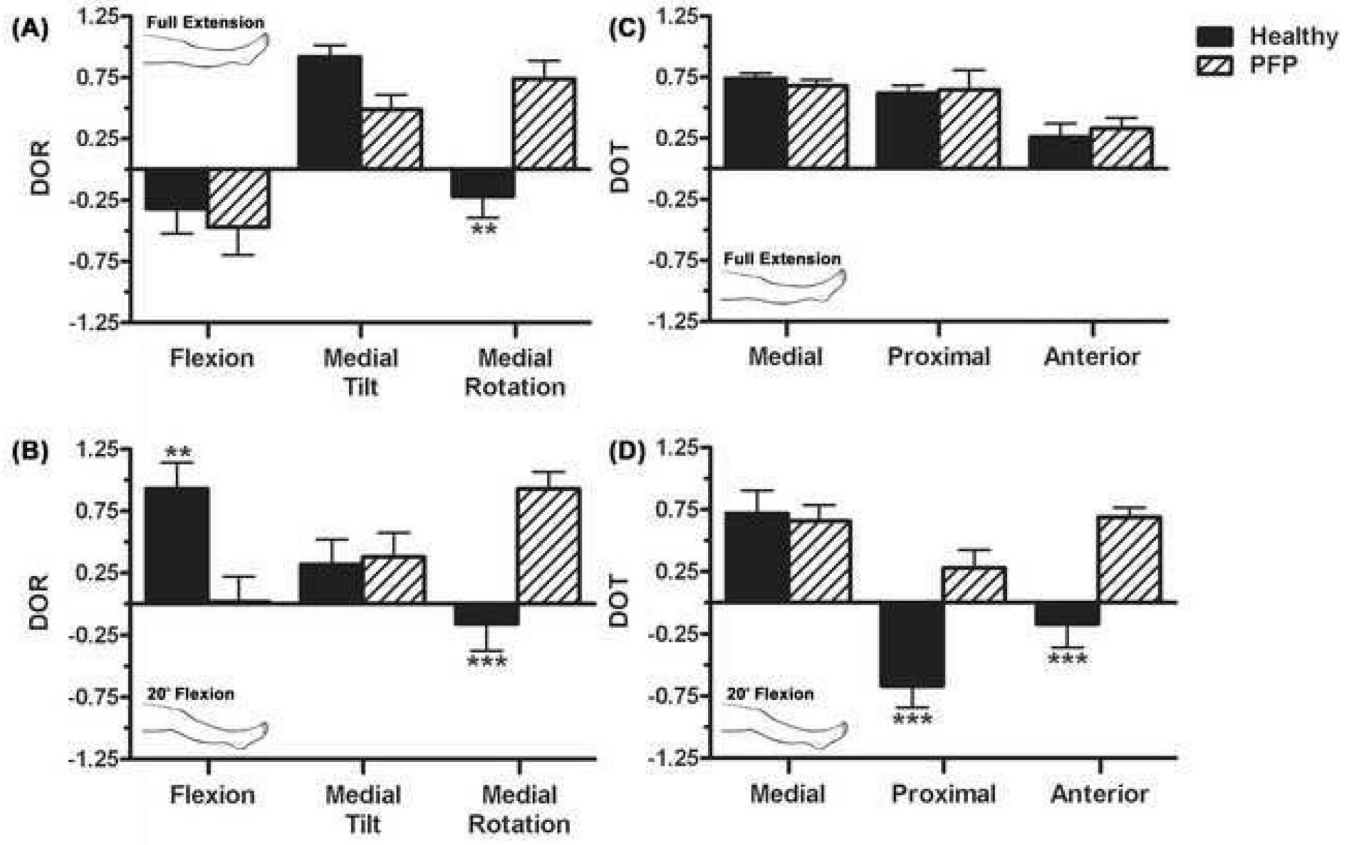

Figure 3.

Patellar tracking induced by selective activation of the VMO resulted in significantly more flexion, medial rotation and more anterior and proximal translation in subjects with PFP compared to healthy subjects. (A) Mean DOR for all subjects in each group with the knee in full extension. (B) Mean DOR for all subjects in each group with the knee at 20° flexion. (C) Mean DOT for all subjects in each group with the knee at full extension (D) Mean DOT for all subjects in each group with the knee at 20° flexion. Error bars represent standard error of the mean, ** $p<0.01$, *** $p<0.001$.

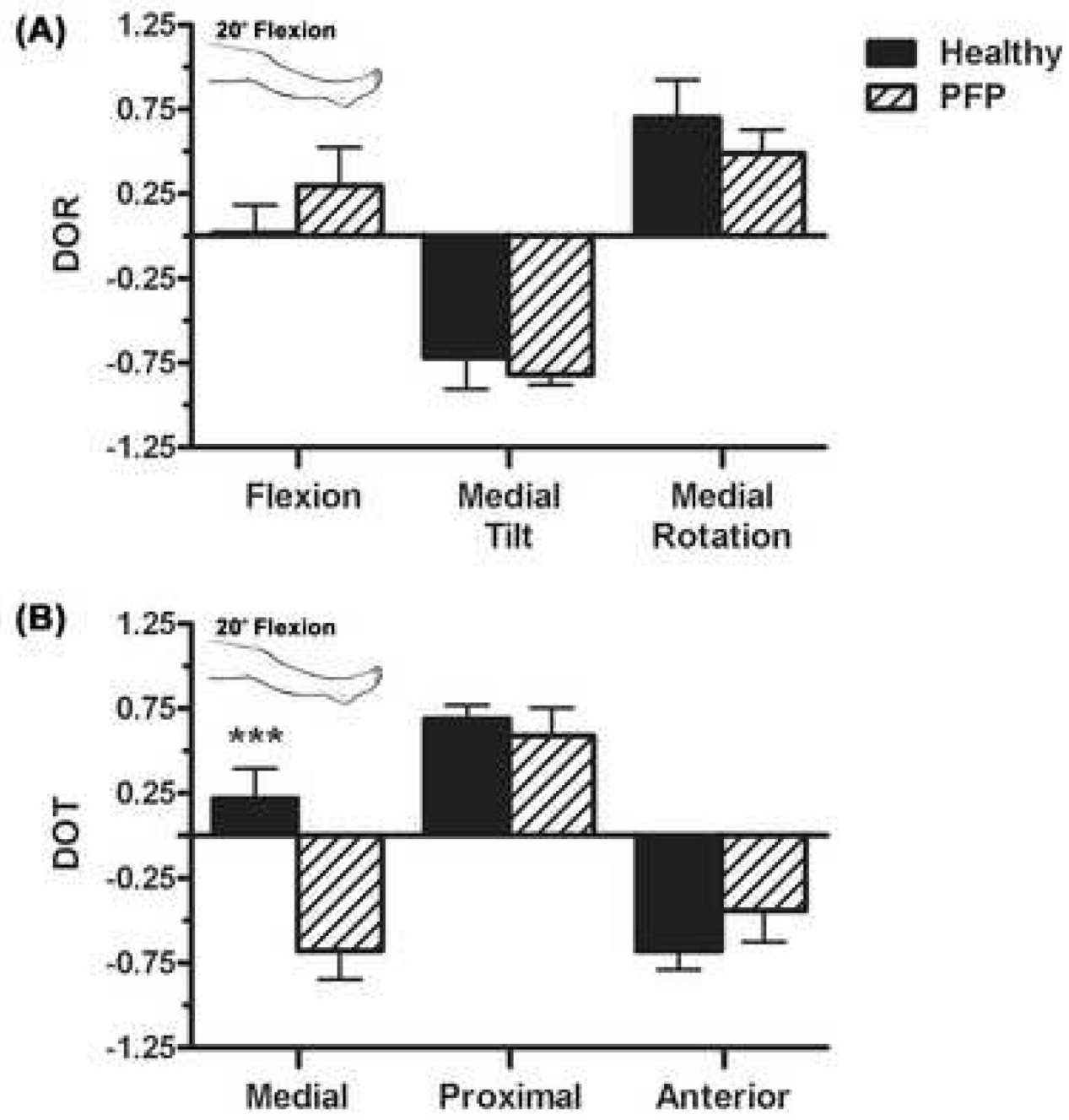

Figure 4.

(A) Selective activation of the VL generated less medial rotation and more lateral tilt in PFP subjects when the knee was at 20° flexion. (B) VL activation also generated lateral patellar translation in PFP subjects when the knee was at 20° flexion. Error bars represent standard error of the mean, *** $p<0.001$.

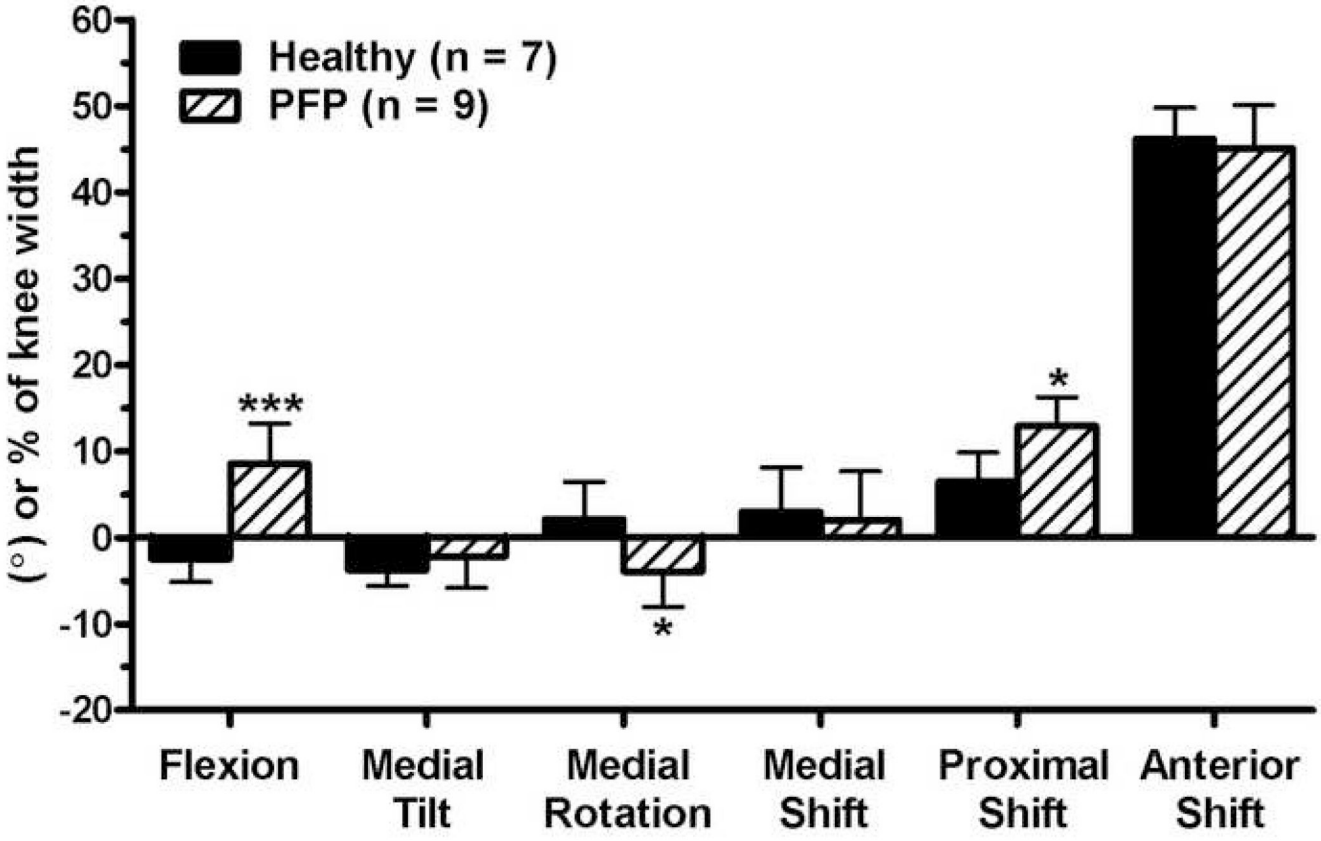

Figure 5.
The mean static position of the patella at full knee extension for healthy and subjects with PFP. Error bars represent one standard deviation, * $p<0.05$, *** $p<0.001$.

Table 1

Demographic characteristics of the subject population (Mean ±SD)

	Healthy Subjects	PFP Subjects	p-value
Number and Gender	7 female	9 female	0.99
Age (years)	27.8 (±8.5)	30.9 (±7.0)	0.44
Height (cm)	165.8(±7.0)	161.1 (±7.6)	0.23
Weight (kg)	66.1 (±22.8)	61.0 (±8.7)	0.54

Table 2

Patellar Tracking DORs for Selective Activation of the Individual Quadriceps Components (Mean ±SD).

		0°			20°		
		Flexion	**Medial Tilt**	**Medial Rotation**	**Flexion**	**Medial Tilt**	**Medial Rotation**
VMO	**PFP**	−0.47 (±0.69)	0.49 (±0.35)	**0.74 (±0.44)** **	**0.02 (±0.60)** **	0.38 (±0.58)	**0.93 (±0.41)** ***
	Healthy	−0.32 (±0.54)	0.92 (±0.24)	**−0.22 (±0.46)**	**0.93 (±0.56)**	0.32 (±0.53)	**−0.16 (±0.57)**
VML	**PFP**	−0.91 (±0.14)	0.06 (±0.44)	−0.40 (±0.34)	−0.84 (±0.49)	0.51 (±0.48)	−0.16 (±0.47)
	Healthy	−0.41 (±0.69)	0.36 (±0.40)	−0.84 (±0.30)	−0.49 (±0.47)	0.76 (±0.25)	−0.43 (±0.40)
VL	**PFP**	−0.71 (±0.72)	−0.51 (±0.36)	0.48 (±0.55)	0.30 (±0.67)	−0.82 (±0.19)	0.49 (±0.42)
	Healthy	−0.79 (±0.26)	−0.54 (±0.36)	0.29 (±0.50)	0.02 (±0.43)	−0.72 (±0.49)	0.70 (±0.59)

** $p<0.01$,

*** $p<0.001$

Table 3

Patellar Tracking DOTs for Selective Activation of the Individual Quadriceps Components (Mean ±SD).

		0°			20°		
		Medial	**Proximal**	**Anterior**	**Medial**	**Proximal**	**Anterior**
VMO	**PFP**	0.68 (±0.15)	0.65 (±0.48)	0.33 (±0.26)	0.66 (±0.38)	**0.28 (±0.43)** ***	**0.69 (±0.23)** ***
	Healthy	0.74 (±0.12)	0.62 (±0.17)	0.26 (±0.29)	0.72 (±0.49)	**−0.67 (±0.46)**	**−0.17 (±0.50)**
VML	**PFP**	0.37 (±0.30)	0.83 (±0.14)	**0.42 (±0.33)** *	0.63 (±0.22)	0.52 (±0.23)	**0.57 (±0.55)** ***
	Healthy	0.56 (±0.30)	0.83 (±0.21)	**−0.01 (±0.50)**	0.85 (±0.26)	0.39 (±0.36)	**−0.35 (±0.51)**
VL	**PFP**	−0.71 (±0.28)	0.69 (±0.55)	0.17 (±0.37)	**−0.68 (±0.50)** ***	0.59 (±0.49)	−0.44 (±0.56)
	Healthy	− 0.46 (±0.12)	0.88 (±0.09)	0.10 (±0.28)	**0.22 (±0.46)**	0.69 (±0.21)	− 0.68 (±0.29)

* $p<0.05$,

*** $p<0.001$