Research Report

Timing and Intensity of Vastus Muscle Activity During Functional Activities in Subjects With and Without Patellofemoral Pain

Background and Purpose. Differences in intensity and timing of muscle activity between the vastus medialis and vastus lateralis muscles have been hypothesized as contributing to lateral patellar tracking and patellofemoral pain (PFP). The purpose of this study was to ascertain whether there were differences in the activity of the vastus muscles that would be suggestive of patellar instability in subjects with PFP. Subjects. Twenty-six subjects with PFP and 19 subjects without PFP participated in the study. Methods. Fine-wire electromyography was used to record activity of the vastus medialis oblique, vastus medialis longus, vastus lateralis, and vastus intermedius muscles during level walking, stair climbing, and walking on ramps. Knee motion was assessed using a six-camera motion analysis system. Results. No differences in onset or cessation of muscle activity was found among the vastus muscles for either group, regardless of condition. Subjects with PFP demonstrated less activity of all vastus muscles for level walking and ramp walking than did subjects without PFP. Conclusions and Discussion. These results do not support the hypothesis that timing or intensity differences between the vastus medialis and vastus lateralis muscles are associated with PFP. [Powers CM, Landel R, Perry J. Timing and intensity of vastus muscle activity during functional activities in subjects with and without patellofemoral pain. *Phys Ther.* 1996;76:946–955.]

Key Words: *Electromyography, Gait, Patellofemoral pain.*

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Disturbance of the extensor mechanism is regarded as one the most common disorders of the knee.[1] Patellofemoral pain (PFP) is the prominent knee complaint among adolescents and young adults[2] and is a much more frequent finding in females than in males.[3] The incidence of PFP is greater in males than in females, however, when only athletes are considered.[4] Despite the high prevalence of PFP in the general population, its etiology and treatment remain controversial.

The mechanism most widely thought to cause PFP is abnormal patellar tracking, which can lead to excessive strain on both peripatellar retinacular supports and the patellar articular cartilage.[5] One hypothesis suggests that patients with PFP have an imbalance between the primary dynamic patellar stabilizers, which results in lateral tracking and malalignment.[1] In this model, the lateral pull of the vastus lateralis muscle (VL) is not adequately counteracted by the medial pull of the vastus medialis oblique muscle (VMO) and the vastus medialis longus muscle (VML). Several studies[6–9] have addressed this dynamic imbalance theory by examining the electromyographic (EMG) activity of the VMO and VL in patients with PFP. Although some investigators[7,9] have found differences in VMO and VL activity in this population, other investigators[6,8] have not found such differences. This conflicting evidence implies that the magnitude of motor unit activity may not be the sole contributor to dynamic patellar imbalance. Direct comparisons of these studies, however, are difficult because of differences in experimental technique and methods of assessing EMG data.

Asynchronous timing of vastus muscle contraction also has been postulated as contributing to patellar instability. In patients with PFP, the VL is hypothesized to contract earlier than the VMO rather than simultaneously.[10] This premise has been incorporated into the clinical treatment of patients with PFP with the use of biofeedback and muscle reeducation. The purpose of such treatment is to alter the timing of the VMO and VL, focusing on initiating VMO contraction before the VL to counteract any early laterally directed force on the patella (J McConnell, *The Advanced McConnell Patellofemoral Treatment Plan Course Notes*; The University of Sydney, Lidcombe, New South Wales, Australia; 1991).

Evidence in support of the asynchronous timing hypothesis was presented by Voight and Wieder,[10] who found that activation of the VMO in subjects with PFP was delayed compared with the VL during a monosynaptic reflex (patellar tendon tap). These findings, however, have recently been disputed by Karst and Willett,[11] who

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Table 1.
Comparison of Group Characteristics

	Comparison Group (n=19)			PFP[a] Group (n=26)			
	$\bar{X}$	SD	Range	$\bar{X}$	SD	Range	P
Age (y)	27.5	4.7	23–38	25.6	7.1	14–46	.32
Height (cm)	165.3	7.7	149.8–183.5	165.1	10.4	130.8–187.9	.92
Weight (kg)	59.2	7.5	46.8–74.1	63.9	9.8	42.0–85.4	.09

[a] PFP=patellofemoral pain.

reported no vastus muscle timing differences in their patient group. Although the conflicting data presented in these studies can be explained by methodological differences, as well as by the inherent variability of EMG data, continued research is necessary to establish whether timing differences actually exist in this population. In addition, investigation of vastus muscle timing during functional activities is essential, as it is during such tasks that PFP is typically reproduced. This information would contribute to the knowledge base regarding the etiology of PFP and would validate clinical assumptions on which current treatment protocols are based.

The purpose of this study was to test the hypothesis that subjects with PFP would demonstrate EMG patterns consistent with that proposed for compromised patellar stability (ie, delayed timing or reduced intensity of VMO activity relative to VL activity). To accomplish this task, vastus muscle activity was assessed during various functional activities (level walking, stair climbing, and walking on ramps). Knee joint motion also was recorded to document potential compensatory gait mechanisms in this population.

Method

Subjects

Subjects were 45 females ranging from 14 to 46 years of age (Tab. 1). Twenty-six subjects were diagnosed as having PFP (PFP group), whereas the remaining 19 subjects were free of any knee pain or pathology (comparison group). The subjects with PFP were recruited from orthopedic clinics in the Los Angeles (Calif) area and were screened to rule out ligamentous instability, internal derangement, or patellar tendinitis. Subjects were assigned to the PFP group if they had (1) pain (vague or localized) originating from the patellofemoral joint articulation (only patient histories relating to overuse or insidious onset were accepted) and (2) readily reproducible pain with at least two activities commonly associated with PFP (eg, squatting, stair climbing, kneeling, prolonged sitting, isometric quadriceps femoris muscle contraction).[12] Subjects with PFP were excluded from the study if they reported having (1) any previous knee surgery, (2) a history of traumatic patellar dislocation, or (3) any neurological involvement that would influence gait.

The comparison group was recruited from the student population at the University of Southern California and Rancho Los Amigos Medical Center (Downey, Calif). These subjects were selected based on the same criteria used for the PFP group except that the subjects had (1) no history or diagnosis of knee pathology or trauma, (2) no knee pain with any of the activities described, and (3) no limitations that would influence gait.

Procedure

All testing was done at the Pathokinesiology Laboratory of Rancho Los Amigos Medical Center. Prior to participation, informed consent was obtained from all subjects. Age, height, and weight were recorded to determine group homogeneity. For those subjects with bilateral symptoms, only the side with the worst symptoms was tested.

To record the timing and intensity of muscle activity, indwelling, bipolar wire electrodes (50-μm nickel-chromium alloy wire in a 25-gauge needle) were inserted into the muscle belly of the VMO, VML, VL, and vastus intermedius muscle (VI) using Basmajian's technique.[13] Confirmation of electrode placement was determined by mild electrical stimulation and voluntary muscle contraction. An FM/FM telemetry system* was used to transmit the EMG signal from the subject to a DEC 11/23 computer.† The system bandwidth was 150 to 1,000 Hz, with an overall gain of 1,000. The data acquisition rate for each channel was 2,500 Hz.

To allow for comparison of EMG intensity between subjects and muscles and to control for the variability of electrode placement, EMG data were normalized to the EMG values acquired during a maximal isometric knee extension effort. This was done on a Lido isokinetic dynamometer‡ with the subject seated and the knee flexed to 60 degrees. Sixty degrees of knee flexion was used because females without musculoskeletal impair-

* Biosentry Telemetry Inc, 20720 Earl St, Torrance, CA 90503.
† Digital Equipment Corp, 146 Main St, Maynard, MA 01754.
‡ Loredan Biomedical Corp, PO Box 1154, Davis, CA 95617.

ment can generate the greatest knee extensor torque in this position[14] and because this position provides greater patellar stabilization within the trochlear groove.[12] This position would potentially minimize quadriceps femoris muscle inhibition resulting from the pain associated with patellar instability.

Motion analysis was performed using a six-camera VICON motion system.[§] Each camera contained infrared light-emitting diodes (wavelength=940 nm) with a flash rate of 50 Hz. Reflective markers placed on the sacrum, anterior superior iliac spine (bilaterally), greater trochanter, anterior thigh, medial and lateral femoral condyles, medial and lateral malleoli, anterior tibia, dorsum of the foot, fifth metatarsal head, and posterior heel were used to determine sagittal-plane motion of the lower extremity.

Phasing of EMG activity during gait was determined by use of a Stride Analyzer.[‖] This system consisted of insoles containing compression-closing footswitches taped to the soles of the subjects' bare feet. Sensors at the heel, first and fifth metatarsal heads, and great toe responded to compression equal to or greater than 3 psi. Analog footswitch data were collected by the same DEC 11/23 computer used to acquire the EMG signals; therefore, synchronization of these data was automatic.

One practice trial of both free-speed and fast walking allowed the subject to become familiarized with the instrumentation. Level-walking trials were done on a 10-m walkway, with the middle 6 m designated for data collection. A four-step staircase with a step height of 15 cm and a tread depth of 27 cm was used for stair ambulation. Ramp walking was assessed on a 12-degree incline that was 6.1 m in length. Subjects performed two trials for each of the following conditions: free-speed and fast walking on a level surface, ascending and a descending ramp, and ascending and descending stairs. All subjects were given the opportunity to ambulate at their own self-selected speed (ie, either free-speed or fast walking). Electromyographic, footswitch, and motion data were collected simultaneously.

Following gait testing, the maximal isometric muscle test on the Lido dynamometer was repeated, with the maximal EMG activity being recorded. This was done to ensure that the intramuscular electrodes had not been displaced during the testing procedure.

Data Analysis

Digitally acquired EMG data for all gait conditions were full-wave rectified and integrated over 0.01-second intervals. Intensities were reported as a percentage of maximal isometric muscle test (% MIMT). To assess whether electrode displacement occurred during testing, all EMG data collected during the end isometric muscle test were screened for discernible drops or rises in intensity (ie, a 30%–40% change in intensity). If a noticeable change was evident for a particular insertion, then all acquired runs were examined to determine where the drop or rise occurred. All trials subsequent to that point were then normalized by the end muscle test EMG value. This procedure was necessary in less than 10% of all insertions (17/180).

Assessment of EMG timing (onset and cessation) was accomplished through the EMG Analyzer software.[‖] This software determined the onsets and cessations for all packets of EMG activity that exceeded an amplitude of 5% MIMT. Packets of EMG activity separated by an interval of less than 5% of the gait cycle were combined. Only the initial packet of EMG activity (which occurred from late swing through loading response) was assessed for onset, cessation, and mean intensity for the six gait conditions. Activity during this phasing is considered to be the normal timing of the vastus muscles during gait[15] and was shown to be the most consistent as well as the dominant EMG packet for the vastus muscles in this study. All EMG activity onsets and cessations were reported as a percentage of the gait cycle (% GC).

To allow averaging of data acquired from multiple strides and subjects, motion data were processed, digitized, and normalized to a stance phase that represented 62% of the gait cycle. This value is considered to be representative of normal walking[15] and was consistent with the average stance phase demonstrated by our subjects for all conditions. The knee joint motion was analyzed for minimum and maximum values at each phase of the gait cycle.

Statistical Analysis

Prior to analysis, descriptive statistics and the Wilk-Shapiro test for normality were calculated for all variables. Subject characteristics (age, height, and weight) were compared between groups using two-sample *t* tests.

To determine whether EMG timing varied between groups or muscles, a 2×4 (group×muscle) analysis of variance (ANOVA) for repeated measures on one variable (muscle) was performed. This analysis was repeated for each condition for EMG activity onset, cessation, and mean intensity. To determine whether knee motion differed between groups and conditions, a 2×6 (group×condition) ANOVA for repeated measures on one variable (condition) was performed. This analysis was repeated for the maximum degree of knee flexion for each phase of the gait cycle. Significant main effects

[§] Oxford Metrics Ltd, Unit 14, 7 West Way Botley, Oxford, England OX2 0JB.
[‖] B&L Engineering, 8807 Pioneer Blvd. Suite C, Santa Fe Springs, CA 90670.

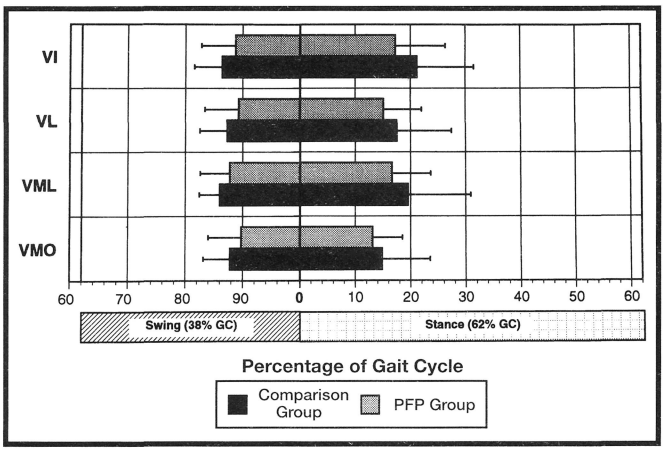

Figure 1.
Onset and cessation of electromyographic activity of the vastus muscles during free-speed walking (expressed as a percentage of the gait cycle) for subjects with patellofemoral pain (PFP group) and subjects without knee pain or pathology (comparison group). Onsets are indicated by the left edge of the horizontal bars. Cessations are indicated by the right edge of the horizontal bars. Error bars indicate one standard deviation from the mean. Zero percent of the gait cycle indicates initial contact. (VI=vastus intermedius muscle, VL=vastus lateralis muscle, VML=vastus medialis longus muscle, VMO=vastus medialis oblique muscle).

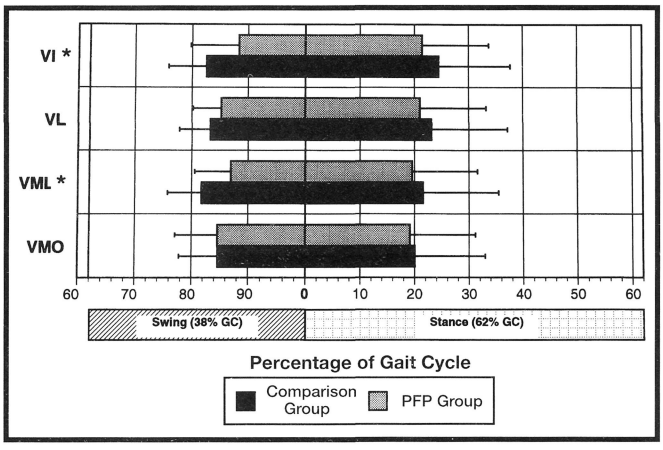

Figure 2.
Onset and cessation of electromyographic activity of the vastus muscles during fast walking (expressed as a percentage of the gait cycle) for subjects with patellofemoral pain (PFP group) and subjects without knee pain or pathology (comparison group). Onsets are indicated by the left edge of the horizontal bars. Cessations are indicated by the right edge of the horizontal bars. Error bars indicate one standard deviation from the mean. Zero percent of the gait cycle indicates initial contact. (VI=vastus intermedius muscle, VL=vastus lateralis muscle, VML=vastus medialis longus muscle, VMO=vastus medialis oblique muscle). Asterisk (*) indicates delayed onset of electromyographic activity in these muscles for the PFP group compared with the comparison group ($P<.05$).

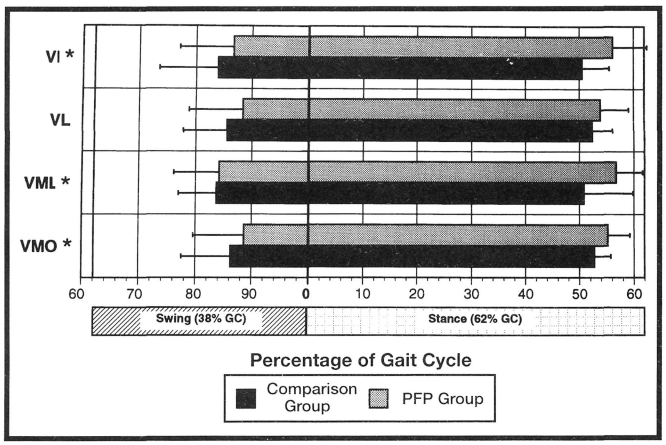

Figure 4.
Onset and cessation of electromyographic activity of the vastus muscles during stair descent (expressed as a percentage of the gait cycle) for subjects with patellofemoral pain (PFP group) and subjects without knee pain or pathology (comparison group). Onsets are indicated by the left edge of the horizontal bars. Cessations are indicated by the right edge of the horizontal bars. Error bars indicate one standard deviation from the mean. Zero percent of the gait cycle indicates initial contact. (VI=vastus intermedius muscle, VL=vastus lateralis muscle, VML=vastus medialis longus muscle, VMO=vastus medialis oblique muscle). Asterisk (*) indicates delayed cessation of electromyographic activity in these muscle for the PFP group compared with the comparison group ($P<.05$).

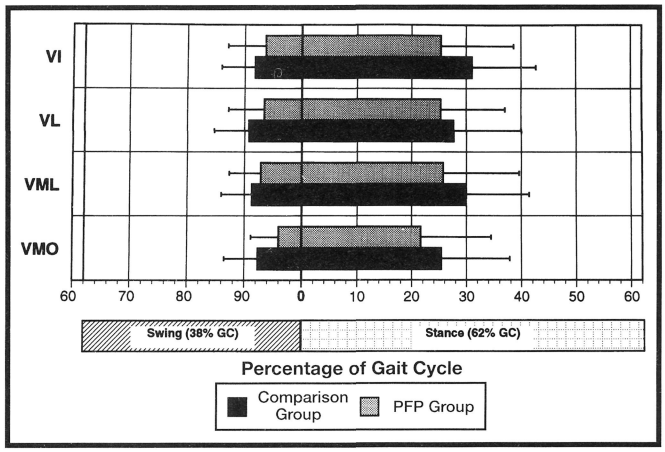

Figure 5.
Onset and cessation of electromyographic activity of the vastus muscles during ramp ascent (expressed as a percentage of the gait cycle) for subjects with patellofemoral pain (PFP group) and subjects without knee pain or pathology (comparison group). Onsets are indicated by the left edge of the horizontal bars. Cessations are indicated by the right edge of the horizontal bars. Error bars indicate one standard deviation from the mean. Zero percent of the gait cycle indicates initial contact. (VI=vastus intermedius muscle, VL=vastus lateralis muscle, VML=vastus medialis longus muscle, VMO=vastus medialis oblique muscle).

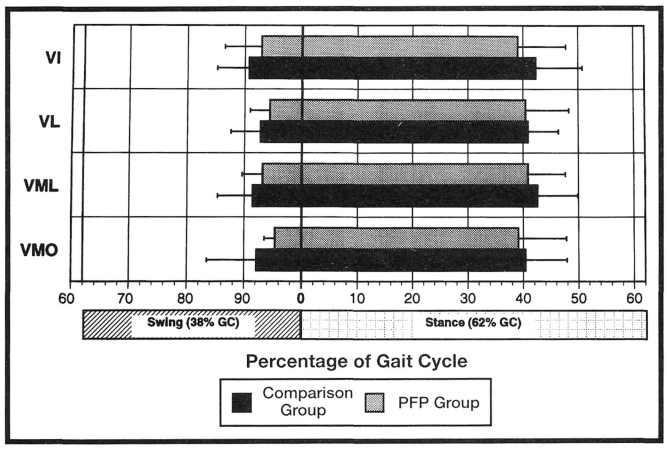

Figure 3.
Onset and cessation of electromyographic activity of the vastus muscles during stair ascent (expressed as a percentage of the gait cycle) for subjects with patellofemoral pain (PFP group) and subjects without knee pain or pathology (comparison group). Onsets are indicated by the left edge of the horizontal bars. Cessations are indicated by the right edge of the horizontal bars. Error bars indicate one standard deviation from the mean. Zero percent of the gait cycle indicates initial contact. (VI=vastus intermedius muscle, VL=vastus lateralis muscle, VML=vastus medialis longus muscle, VMO=vastus medialis oblique muscle).

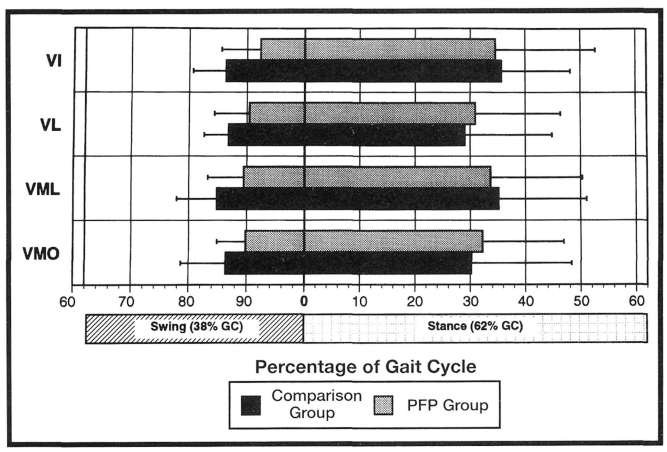

Figure 6.
Onset and cessation of electromyographic activity of the vastus muscles during ramp descent (expressed as a percentage of the gait cycle) for subjects with patellofemoral pain (PFP group) and subjects without knee pain or pathology (comparison group). Onsets are indicated by the left edge of the horizontal bars. Cessations are indicated by the right edge of the horizontal bars. Error bars indicate one standard deviation from the mean. Zero percent of the gait cycle indicates initial contact. (VI=vastus intermedius muscle, VL=vastus lateralis muscle, VML=vastus medialis longus muscle, VMO=vastus medialis oblique muscle). Onset of electromyographic activity was delayed for the PFP group compared with the comparison group when averaged across all muscles ($P<.05$).

were reported only if there were no significant interactions. If a significant interaction was found, the individual main effects were analyzed separately. A Tukey's *post hoc* test was used to identify significant comparisons. All significance levels were set at $P<.05$. Statistical calculations were performed with BMDP software.[*]

Results

Subject Characteristics

No differences were found between groups for mean age or height ($P>.05$; Tab. 1). Although the PFP group's mean weight was greater than that of the comparison group (63.9 kg versus 59.2 kg), this difference was not significant ($P=.09$; Tab. 1).

Electromyographic Analysis

Onset. No differences were evident in the onset of EMG activity among the vastus muscles within either group (no muscle effects). This finding was consistent for each gait condition tested (Figs. 1–6). When averaged across all conditions, the VMO and VL onset for the PFP group were very similar (VMO: 90.6% GC versus VL: 90.2% GC; Tab. 2). The same trend was seen in the comparison group (VMO: 88.3% GC versus VL: 87.7% GC; Tab. 2).

During ramp descent, a group effect (no interaction) was found. The onset of EMG activity in the vastus muscles of the PFP group was delayed compared with that of the comparison group when averaged across all muscles (86.0% GC versus 90.5% GC; $P<.01$) (Fig. 6). In addition, a group effect and a group×muscle interaction were found during fast walking. Breakdown of the muscles individually by group revealed a delayed onset in the PFP group compared with the comparison group for the VML (86.9% GC versus 81.7% GC; $P<.01$) and the VI (88.5% GC versus 82.6% GC; $P<.02$) (Fig. 2). No other group effects were found for onset of EMG activity.

Cessation. No differences were evident in the cessation of EMG activity among the vastus muscles within either group (no muscle effects) regardless of condition (Figs. 1–6). As with EMG activity onset, there was very little difference between the VMO and VL when averaged across all conditions for both the PFP group (VMO: 30.1% GC versus VL: 31.1% GC) and the comparison group (VMO: 30.6% GC versus VL: 31.7% GC) (Tab. 3).

A group effect and a group×muscle interaction were evident during stair descent. Breakdown of the muscles individually by group revealed a later cessation of EMG

[*] BMDP Statistical Software Inc, 1440 Sepulveda Blvd, Suite 316, Los Angeles, CA 90025.

Table 2.
Onset of Electromyographic Activity (Expressed as Percentage of Gait Cycle) of the Vastus Medialis Oblique Muscle (VMO) and the Vastus Lateralis Muscle (VL)[a]

	Comparison Group (n=19)				PFP[b] Group (n=26)			
	VMO		VL		VMO		VL	
Condition	$\bar{X}$	SD	$\bar{X}$	SD	$\bar{X}$	SD	$\bar{X}$	SD
FR	87.8	4.6	87.3	4.8	89.7	5.7	89.3	5.9
FT	84.6	6.8	83.3	5.5	84.6	7.5	85.2	5.0
AR	92.3	5.9	90.7	6.1	96.0	4.9	93.4	6.3
DR	86.4	7.7	86.8	4.2	89.8	4.9	90.4	6.0
AS	92.1	8.6	92.7	5.1	95.3	1.9	94.3	3.4
DS	86.2	8.7	85.5	7.7	88.7	9.0	88.4	9.6
$\bar{X}$	88.3	7.0	87.7	5.6	90.6	5.7	90.2	6.0

[a] No differences were found between the VMO and VL within either group for any of the conditions tested. (FR=free-speed walking, FT=fast walking, AR=ascending ramp, DR=descending ramp, AS=ascending stairs, DS=descending stairs.)
[b] PFP=patellofemoral pain.

Table 3.
Cessation of Electromyographic Activity (Expressed as Percentage of Gait Cycle) of the Vastus Medialis Oblique Muscle (VMO) and the Vastus Lateralis Muscle (VL)[a]

	Comparison Group (n=19)				PFP[b] Group (n=26)			
	VMO		VL		VMO		VL	
Condition	$\bar{X}$	SD	$\bar{X}$	SD	$\bar{X}$	SD	$\bar{X}$	SD
FR	14.8	8.7	17.6	9.7	13.1	5.7	15.1	6.9
FT	20.1	12.9	23.1	14.0	19.2	12.0	21.0	12.2
AR	25.4	12.5	27.7	12.3	21.6	12.9	25.3	11.7
DR	30.2	18.2	29.0	15.8	32.2	14.7	30.9	15.4
AS	40.4	7.5	40.7	5.5	39.1	8.7	40.5	7.8
DS	52.7	3.0	52.4	3.7	55.2	4.1	53.9	5.2
$\bar{X}$	30.6	10.5	31.7	10.2	30.1	9.6	31.1	9.9

[a] No differences were found between the VMO and VL within either group for any of the conditions tested. (FR=free-speed walking, FT=fast walking, AR=ascending ramp, DR=descending ramp, AS=ascending stairs, DS=descending stairs.)
[b] PFP=patellofemoral pain.

activity of the VMO, VML, and VI in the PFP group compared with the comparison group (VMO: 55.1% GC versus 52.7% GC, $P<.04$; VML: 56.1% GC versus 50.8% GC, $P<.02$; VI: 56.3% GC versus 50.5% GC, $P<.01$) (Fig. 4). No other group effects were found for cessation of EMG activity.

Mean intensity. There were no differences in mean intensity of EMG activity among the vastus muscles within either group (no muscle effects). As with EMG onset and cessation, the average intensities of the VMO and VL for each condition were very similar within both groups (Tab. 4).

Group effects (no interactions) were found, however, for the free-speed, fast-speed, ascend-ramp, and descend-ramp conditions. In general, the vastus muscle activity of the PFP group was less than that of the comparison group when averaged across all muscles (free speed: 12.5% MIMT versus 18.1% MIMT, $P<.02$; fast speed: 22.1% MIMT versus 30.2% MIMT, $P<.01$; ascend ramp: 13.9% MIMT versus 19.7% MIMT, $P<.01$; descend ramp: 12.2% MIMT versus 16.8% MIMT, $P<.01$) (Fig. 7).

Knee Motion

No differences in knee motion were found between groups for any of the conditions tested. This finding was consistent for all phases of the gait cycle. Although there was decreased loading-response knee flexion in the PFP group compared with the comparison group when averaged across all conditions (20.3° versus 23.6°), this difference was not significant ($P=.10$) (Fig. 8).

Discussion

Results of this study have demonstrated no differences in muscle activity within either group for the onset or cessation of EMG activity, regardless of the task. These findings have important clinical implications because, as Voight and Wieder[10] and McConnell (J McConnell, *The Advanced McConnell Patellofemoral Treatment Plan Notes*) have postulated, the VMO may contract later than the

Table 4.
Intensity of Electromyographic Activity (Expressed as Percentage of Maximal Muscle Test) of the Vastus Medialis Oblique Muscle (VMO) and the Vastus Lateralis Muscle (VL)[a]

	Comparison Group (n=19)				PFP[b] Group (n=26)			
	VMO		VL		VMO		VL	
Condition	$\bar{X}$	SD	$\bar{X}$	SD	$\bar{X}$	SD	$\bar{X}$	SD
FR	16.8	9.1	18.9	10.7	11.6	6.7	14.1	7.6
FT	30.0	9.1	30.7	16.7	23.3	10.1	23.7	7.8
AR	20.8	7.0	19.4	6.4	15.4	7.0	13.9	7.5
DR	16.7	10.6	16.8	7.5	11.8	5.0	11.8	4.0
AS	27.5	10.7	29.4	9.9	30.7	14.8	29.2	14.3
DS	20.1	13.0	19.6	10.2	18.8	8.3	12.4	5.5
$\bar{X}$	22.0	9.9	22.5	10.2	18.6	8.7	17.5	7.8

[a] No differences were found between the VMO and VL within either group for any of the conditions tested. (FR=free-speed walking, FT=fast walking, AR=ascending ramp, DR=descending ramp, AS=ascending stairs, DS=descending stairs.)
[b] PFP=patellofemoral pain.

VL in subjects with PFP, and thereby contribute to lateral patellar tracking.

Lieb and Perry[16] contended that the VMO is the primary medial patellar stabilizer, owing to its oblique fiber orientation (55° angle of insertion into the patella). In a mechanical study, Lieb and Perry[16] found that the function of the VMO was to counterbalance the lateral pull of the VL. The longer-fibered VML also provides medial patellar support; however, the angle of fiber orientation into the patella is only 15 to 18 degrees from the midline of the femur. This anatomical difference makes the VML a less effective medial stabilizer than the VMO. Theoretically, the function of either structure would be compromised if the neuromuscular activation were delayed or diminished.

The possibility of a temporal feedforward mechanism in which the VMO contracts before the VL to counteract the larger force capacity of the VL has been discussed.[17] This hypothesis is supported by the work of Voight and Wieder,[10] who noted VMO activity prior to that of the VL in individuals without knee impairment during a patellar tendon tap. Similarly, Grabiner et al[17] reported that VMO activity preceded VL activity during maximal isometric contractions in subjects without knee impairment. Despite the statistical significance of the difference, however, the temporal difference between these two muscles was only 5.6 milliseconds. These authors concluded that these results were not clinically meaningful and that without greater differences the feedforward-activation hypothesis should be contested.

In contrast to the results obtained from their subjects without knee impairment, Voight and Wieder[10] reported that reflex activity of the VL preceded that of the VMO in subjects with extensor mechanism disorders. Despite failure to report the magnitude of this timing difference, and the lack of evidence indicating that this phenomenon would be present in voluntary contractions, these authors hypothesized that this finding was indicative of a neurophysiologic motor control imbalance, and therefore contributory to patellofemoral joint dysfunction. In a subsequent study, Karst and Willett[11] found no evidence of timing differences between the VMO and the VL. Using techniques that improved on the procedures of Voight and Wieder[10] (such as increasing the temporal resolution of the reflex-latency measurement and controlling for subject height), these authors refuted the hypothesis of timing differences during reflex conditions. In addition, Karst and Willett reported that there were no onset timing differences during voluntary knee extension.

Our EMG results during functional gait activities support the conclusions of Karst and Willett[11] that timing differences between the VMO and the VL do not exist in patients with PFP, and therefore do not play a role in contributing to this disorder. Therefore, the clinical rationale for the use of biofeedback and muscle reeducation techniques in an attempt to alter the onset of VMO activity relative to that of the VL must be questioned.

In our subjects with PFP, all vastus muscles had decreased mean intensities compared with the vastus muscle of the subjects without PFP in four of the six conditions tested (free-speed and fast walking and ascending and descending a ramp). This decreased activity is suggestive of a quadriceps femoris muscle avoidance pattern, which is similar to the response seen in subjects with anterior cruciate ligament tears.[18] Perry[15] stated that subjects with weak quadriceps femoris muscles or painful knees avoid loading-response knee flexion, as it is this point in the gait cycle where the muscular demands and knee joint reaction forces are the greatest. Although this premise was supported by the work of Dillon et al,[19] who reported a reduction in

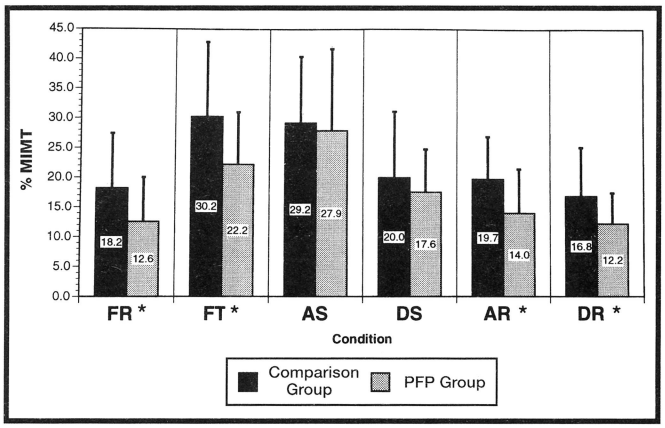

Figure 7.
Mean intensity of vastus muscle contraction (expressed as a percentage of maximal isometric muscle test [% MIMT]) between subjects with patellofemoral pain (PFP group) and subjects without knee pain or pathology (comparison group) for all conditions tested (FR=free-speed walking, FT=fast walking, AS=ascending stairs, DS=descending stairs, AR=ascending ramp, DR=descending ramp). Error bars indicate one standard deviation from the mean. Asterisk (*) indicates intensity of electromyographic activity was lower in the PFP group than in the comparison group ($P<.05$).

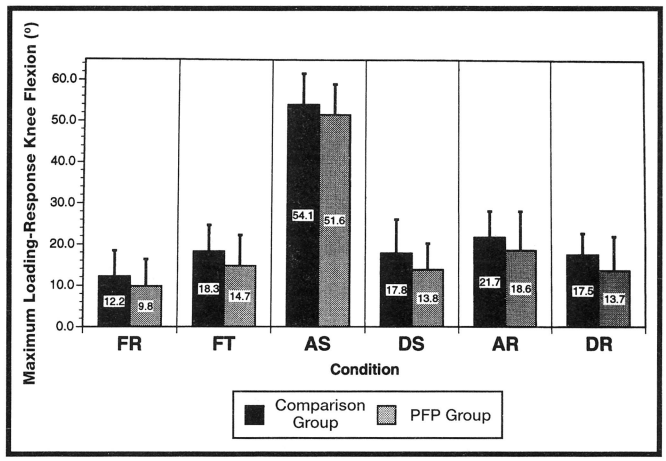

Figure 8.
Comparison of maximum knee flexion during the loading-response phase of the gait cycle between subjects with patellofemoral pain (PFP group) and subjects without knee pain or pathology (comparison group) for all conditions tested (FR=free-speed walking, FT=fast walking, AS=ascending stairs, DS=descending stairs, AR=ascending ramp, DR=descending ramp). Error bars indicate one standard deviation from the mean.

stance-phase knee flexion in eight subjects with PFP, the knee-motion results of our investigation do not adequately explain the differences in vastus muscle EMG activity between groups. Instead, the reduced EMG activity in the subjects with PFP was most likely the result of a decreased external knee flexion moment (not measured), which could have been accomplished through a subtle positioning of body weight over the knee-joint axis or a conscious effort to reduce walking speed.

Mean intensity of vastus muscle activity was not different between groups for both ascending and descending stairs, indicating that the higher muscular demand associated with these activities was unavoidable. This conclusion is logical because larger ranges of knee flexion are required to accomplish these tasks. Because patellofemoral joint reaction forces are directly related to amount of knee flexion and quadriceps femoris muscle force,[20] it is not surprising that ascending or descending stairs commonly reproduces PFP symptoms.

Within the PFP group, there were no differences in intensity of vastus muscle EMG activity for any of the conditions tested. Although EMG ratios were not calculated in this study, the VMO, VML, and VL had similar mean intensities, indicating that recruitment of the medial quadriceps femoris muscle was not compromised. This finding is in contrast to that reported by Mariani and Caruso[7] as well as Souza and Gross,[9] who found decreased VMO activity compared with that of the VL in subjects with PFP. These conflicting findings can be attributed to methodological differences, as these authors[7,9] based their conclusions of VMO insufficiency on nonnormalized EMG data obtained from small samples. We believe that the lack of normalized EMG data to control other variables not related to muscle function (eg, electrode placement) compromises the validity of these previous results.

The EMG timing for both groups during level walking is in agreement with the data of Adler et al,[21] who analyzed the timing patterns of the vastus muscles in individuals without knee impairment during free-speed and fast walking. According to Perry,[15] the onset of vastus muscle activity in terminal swing functions to reverse the swing-phase knee flexion and prepare the limb for initial contact. Continued activity to the beginning of mid-stance controls the external knee flexion moment in loading response and provides limb stability into single-limb support. In general, our results indicate that the onset of vastus muscle activity during free-speed walking occurred from 85% to 89% of the gait cycle (terminal swing), with termination of activity evident from 13% to 21% of the gait cycle (mid-stance). Overall, the timing of vastus muscle activity during fast walking for the comparison group demonstrated a slightly earlier onset of vastus muscle activity and later cessation compared with free-speed walking (Figs. 1, 2).

The PFP group demonstrated a delayed onset of activity for the VML and VI during fast walking compared with the comparison group, suggesting that the preparation phase for initial contact may have been compromised. The same finding was evident during descending ramps, as the onset of activity of all four vastus muscles was delayed. This delayed activity may have been the result of

the subjects with PFP anticipating a decreased muscular demand during the loading response, owing to the use of a quadriceps femoris muscle avoidance gait pattern. Conversely, individuals without PFP would anticipate the increased muscular demand and would be more likely to ensure adequate knee stability at initial contact.

The prolonged vastus muscle activity evident in both groups during stair and ramp ambulation reflects the increased muscular demand necessary to support the flexed knee posture associated with such activities. The reason why the PFP group had prolonged EMG activity of the VMO, VL, and VI during descending stairs is not entirely clear; however, it is possible that these subjects were more deliberate in controlling the rate of descent rather than "skipping" to the next step. This gait pattern was observed in a number of patients who were obviously cautious in descending the staircase.

Conclusion

This study demonstrated no differences in onset, cessation, or mean intensity of EMG activity of the vastus muscles within the PFP and comparison groups during functional activities. The PFP group, however, demonstrated a decrease in mean intensity of vastus muscle EMG activity during level-surface and ramp ambulation. This finding is indicative of a quadriceps femoris muscle avoidance gait pattern. The theory of an EMG timing or intensity difference among the different vastus muscles as it relates to PFP was not supported by this study.

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A Conference on this article follows on the next page.

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