

# 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 were found among the vastus muscles in 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.** The 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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**D**isturbance of the extensor mechanism is regarded as one of the most common disorders of the knee.<sup>1</sup> Patellofemoral pain (PFP) is the prominent knee complaint among adolescents and young adults<sup>2</sup> and is a much more frequent finding in females than in males.<sup>3</sup> The incidence of PFP is greater in males than in females, however, when only athletes are considered.<sup>4</sup> 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.<sup>5</sup> One hypothesis suggests that patients with PFP have an imbalance between the primary dynamic patellar stabilizers, which results in lateral tracking and malalignment.<sup>1</sup> 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<sup>6-9</sup> have addressed this dynamic imbalance theory by examining the electromyographic (EMG) activity of the VMO and VL in patients with PFP. Although some investigators<sup>7,9</sup> have found differences in VMO and VL activity in this population, other investigators<sup>6,8</sup> 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.<sup>10</sup> 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,<sup>10</sup> 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,<sup>11</sup> who

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**Table 1.**  
Comparison of Group Characteristics

	Comparison Group (n=19)			PFP <sup>a</sup> Group (n=26)			P
	$\bar{X}$	SD	Range	$\bar{X}$	SD	Range	
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

<sup>a</sup>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).<sup>12</sup> Subjects with PFP were excluded from the study if they reported having (1) any previous knee surgery, (2) a history of traumatic patellar disloca-

tion, 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- $\mu$ m 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.<sup>13</sup> 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.<sup>†</sup> 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<sup>‡</sup> 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<sup>14</sup> and because this position provides greater patellar stabilization within the trochlear groove.<sup>12</sup> 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.<sup>8</sup> 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.<sup>11</sup> 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 inter-

vals. 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.<sup>11</sup> 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<sup>15</sup> 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<sup>15</sup> 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.

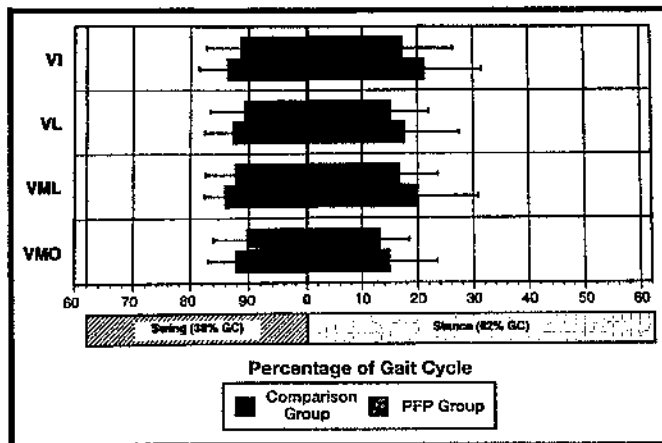
#### Statistical Analysis

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.

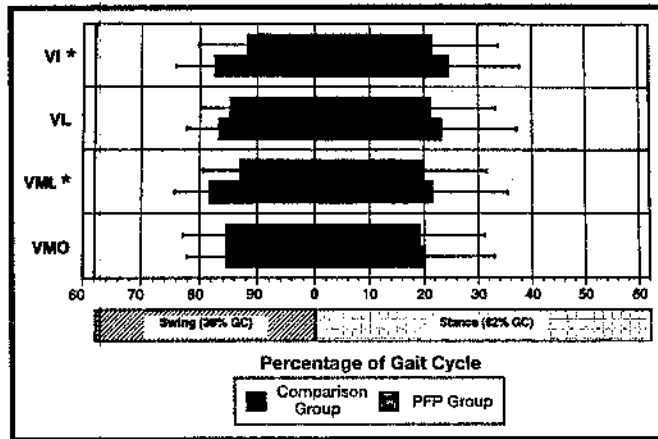
To determine whether EMG timing varied between groups or muscles, a 2×4 (group×muscle) analysis of variance (ANOVA) for repeated measures on one variable (muscle) was performed. This analysis was repeated for each condition for EMG activity onset, cessation, and mean intensity. To determine whether knee 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

<sup>8</sup> Oxford Metrics Ltd, Unit 14, 7 West Way Botley, Oxford, England OX2 0JB.

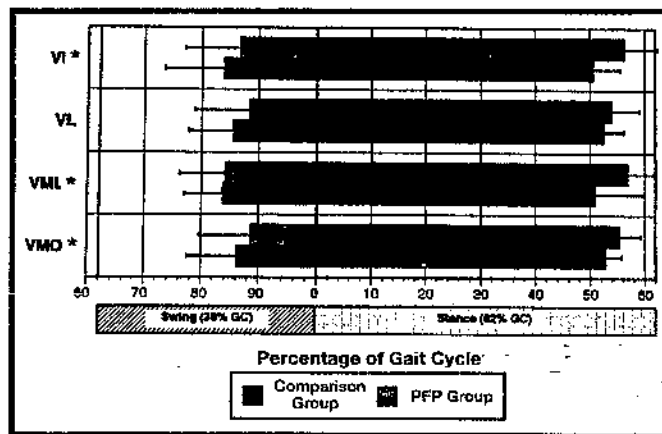
<sup>11</sup> B&L Engineering, 8807 Pioneer Blvd, Suite C, Santa Fe Springs, CA 90670.



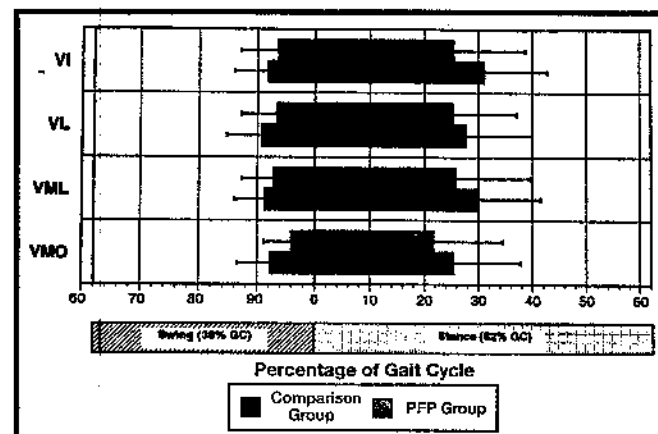
**Figure 1.** Onset and cessation of electromyographic activity of the vastus muscles during free-speed walking (expressed as a percentage of the gait cycle) for subjects with patellofemoral pain (PFP group) and subjects without knee pain or pathology (comparison group). Onsets are indicated by the left 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).



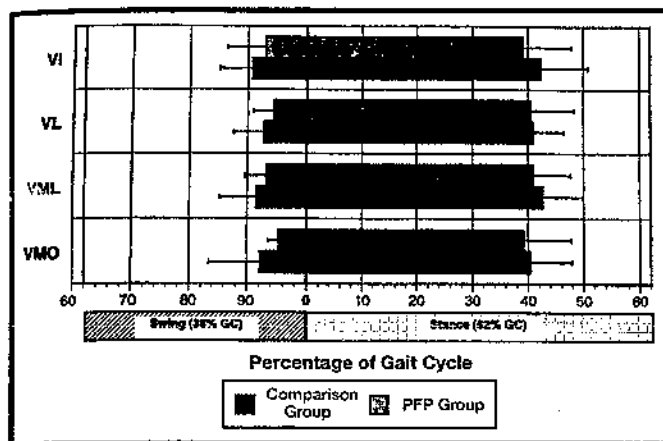
**Figure 2.** Onset and cessation of electromyographic activity of the vastus muscles during fast walking (expressed as a percentage of the gait cycle) for subjects with patellofemoral pain (PFP group) and subjects without knee pain or pathology (comparison group). Onsets are indicated by the left edge 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 ( $P < .05$ ).



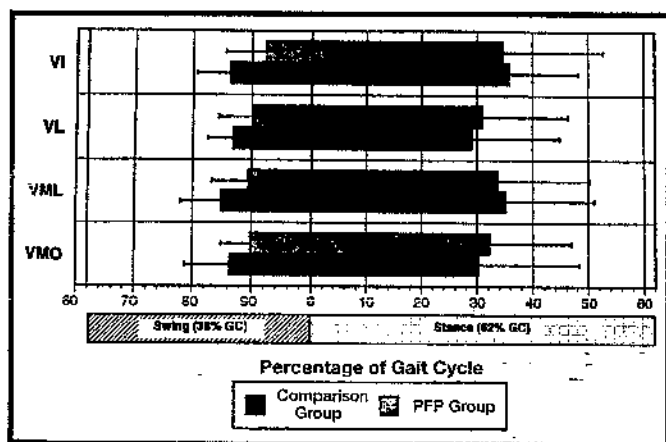
**Figure 4.** Onset and cessation of electromyographic activity of the vastus muscles during stair descent (expressed as a percentage of the gait cycle) for subjects with patellofemoral pain (PFP group) and subjects without knee pain or pathology (comparison group). Onsets are indicated by the left edge 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  $\times$  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  $\times$  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)<sup>a</sup>

Condition	Comparison Group (n=19)				PFP <sup>b</sup> Group (n=26)			
	VMO		VL		VMO		VL	
	$\bar{X}$	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

<sup>a</sup>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.)

<sup>b</sup>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)<sup>a</sup>

Condition	Comparison Group (n=19)				PFP <sup>b</sup> Group (n=26)			
	VMO		VL		VMO		VL	
	$\bar{X}$	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

<sup>a</sup>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.)

<sup>b</sup>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<sup>10</sup> 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)<sup>a</sup>

Condition	Comparison Group (n=19)				PPF <sup>b</sup> Group (n=26)			
	VMO		VL		VMO		VL	
	$\bar{X}$	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

<sup>a</sup>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.)

<sup>b</sup>PPF=patellofemoral pain.

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

Lieb and Perry<sup>16</sup> 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<sup>16</sup> 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.

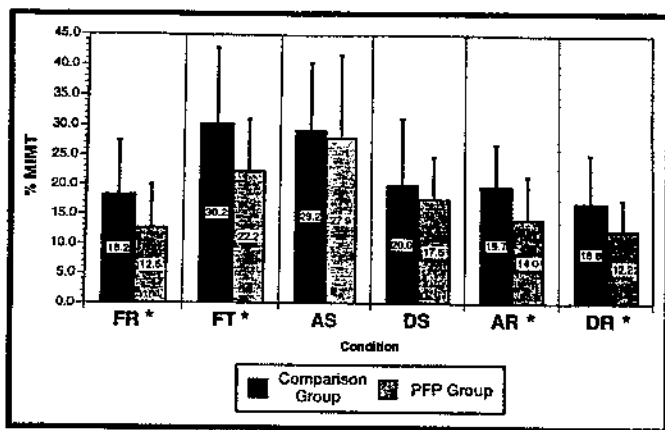
The possibility of a temporal feedforward mechanism in which the VMO contracts before the VL to counteract the larger force capacity of the VL has been discussed.<sup>17</sup> This hypothesis is supported by the work of Voight and Wieder,<sup>10</sup> who noted VMO activity prior to that of the VL in individuals without knee impairment during a patellar tendon tap. Similarly, Grabiner et al<sup>17</sup> 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<sup>10</sup> 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 phenome-

non 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<sup>11</sup> found no evidence of timing differences between the VMO and the VL. Using techniques that improved on the procedures of Voight and Wieder<sup>10</sup> (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<sup>11</sup> 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.<sup>18</sup> Perry<sup>15</sup> 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,<sup>19</sup> who reported a reduction in



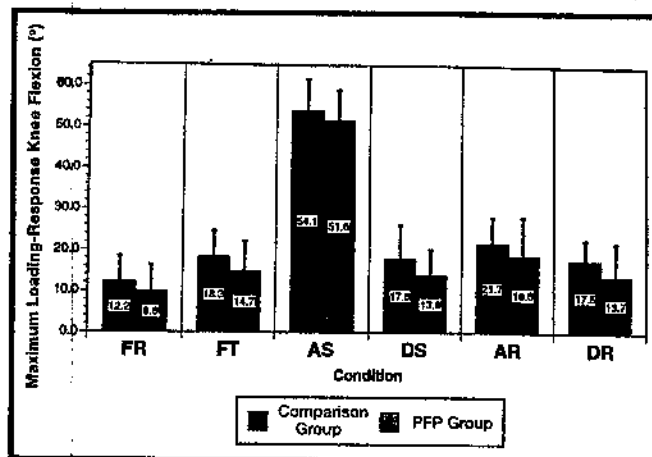
**Figure 7.**

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 ( $P < .05$ ).

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,<sup>20</sup> 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<sup>7</sup> as well as Souza and Gross,<sup>9</sup> 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



**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.

authors<sup>7,9</sup> 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,<sup>21</sup> who analyzed the timing patterns of the vastus muscles in individuals without knee impairment during free-speed and fast walking. According to Perry,<sup>15</sup> 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 VL 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.**

### References

- 1 Fox TA. Dysplasia of the quadriceps mechanism: hypoplasia of the vastus medialis muscle as related to the hypermobile patella syndrome. *Surg Clin North Am.* 1975;55:199-226.
- 2 Reider B, Marshall JL, Ring B. Patellar tracking. *Clin Orthop.* 1981;157:143-148.
- 3 Outerbridge RE. Further studies on the etiology of chondromalacia patellae. *J Bone Joint Surg [Br].* 1964;46:179-190.

- 4 Levine J. Chondromalacia patellae. *The Physician and Sportsmedicine.* 1979;7:41-49.
- 5 Insall J. Patellar malalignment syndrome. *Orthop Clin North Am.* 1979;10:117-122.
- 6 MacIntyre DL, Robertson GE. Quadriceps muscle activity in women runners with and without patellofemoral pain syndrome. *Arch Phys Med Rehabil.* 1992;73:10-14.
- 7 Mariani PP, Caruso I. An electromyographic investigation of subluxation of the patella. *J Bone Joint Surg [Br].* 1979;61:169-171.
- 8 Moller BN, Jurik AG, Tidemand-Dal C, et al. The quadriceps function in patellofemoral disorders: a radiographic and electromyographic study. *Arch Orthop Trauma Surg.* 1987;106:195-198.
- 9 Souza DR, Gross MT. Comparison of vastus medialis obliquus:vastus lateralis muscle integrated electromyographic ratios between healthy subjects and patients with patellofemoral pain. *Phys Ther.* 1991;71:310-316.
- 10 Voight ML, Wieder DL. Comparative reflex response times of vastus medialis obliquus and vastus lateralis in normal subjects and subjects with extensor mechanism dysfunction: an electromyographic study. *Am J Sports Med.* 1991;19:131-137.
- 11 Karst GM, Willett GM. Onset timing of electromyographic activity in the vastus medialis oblique and vastus lateralis muscles in subjects with and without patellofemoral pain. *Phys Ther.* 1995;75:813-823.
- 12 Fulkerson JP, Hungerford DS. *Disorders of the Patellofemoral Joint.* Baltimore, Md: Williams & Wilkins; 1990.
- 13 Basmajian JV, De Luca CJ. *Muscles Alive: Their Functions Revealed by Electromyography.* 5th ed. Baltimore, Md: Williams & Wilkins; 1985.
- 14 Lieb FJ, Perry J. Quadriceps function: an electromyographic study under isometric conditions. *J Bone Joint Surg [Am].* 1971;53:749-758.
- 15 Perry J. *Gait Analysis: Normal and Pathological Function.* Thorofare, NJ: Slack Inc; 1992.
- 16 Lieb FJ, Perry J. Quadriceps function: an anatomical and mechanical study using amputated limbs. *J Bone Joint Surg [Am].* 1968;50:1535-1548.
- 17 Grabiner MD, Koh TJ, Draganich LF. Neuromechanics of the patellofemoral joint. *Med Sci Sports Exerc.* 1994;26:10-21.
- 18 Berchuck M, Andriacchi TP, Bach BR, Reider B. Gait adaptations by patients who have a deficient anterior cruciate ligament. *J Bone Joint Surg [Am].* 1990;72:871-877.
- 19 Dillon FZ, Updyke WF, Allen WC. Gait analysis with reference to chondromalacia patellae. *J Orthop Sports Phys Ther.* 1983;15:127-131.
- 20 Huberti HH, Hayes WC. Patellofemoral contact pressures. *J Bone Joint Surg [Am].* 1984;66:715-724.
- 21 Adler N, Perry J, Kent B, Robertson K. Electromyography of the vastus medialis oblique and vasti in normal subjects during gait. *Electromyogr Clin Neurophysiol.* 1983;23:643-649.

*Invited clinicians and researchers discuss a research report's implications for practice, research, education, and administration.*

In "Timing and Intensity of Vastus Muscle Activity During Functional Activities in Subjects With and Without Patellofemoral Pain," Powers, Landel, and Perry test a commonly held notion underlying current treatment of patients with patellofemoral pain. With Editor Jules Rothstein, G Kelley Fitzgerald, Greg Karst, Terry Malone, and Kevin Wilk consider the implications when the mechanisms used to explain treatments are not viable—and when there are no data on the effectiveness of those treatments.

**Rothstein:** As early as the first two sentences of their introduction, Powers and colleagues make the assumption that a disturbance of the extensor mechanism causes patellofemoral pain. Do you find this in your own practice?

**Malone:** First, what is meant by "disturbance"? It's a difficult term to define. "Alteration of function" might better reflect the phenomenon.

**Rothstein:** Alteration of what specific function?

**Malone:** Lower-extremity function related to quadriceps femoris muscle activity.

**Fitzgerald:** In my experience, on initial examination, patients with patellofemoral pain show a

decreased ability to produce torque. Quadriceps deficit is associated with patellofemoral pain. Whether the pain causes the deficit or whether the deficit causes the pain is unknown.

**Rothstein:** What is the likelihood of encountering patients who have quadriceps weakness but no patellofemoral pain?

**Fitzgerald:** It's very unlikely that we would see individuals with quadriceps weakness unless they had pain.

**Rothstein:** The weakness itself is not profound, then.

**Fitzgerald:** Right.

**Wilk:** This illustrates one of the problems of combining all patients with anterior knee pain together or all patients with patellofemoral pain together. We aren't accurately describing what's going on; we're just grossly classifying them as "patients with pain." The authors of this paper, for instance, reported that they had 26 patients with patellofemoral pain. Would we say that we had 26 patients with ligamentous insufficiency of the knee? No. We'd say that we had 26 patients with anterior cruciate ligament insufficiency or posterior cruciate ligament insufficiency.... The presentation of patients' symptoms may alter the EMG [electromyographic] activ-

ity. If patients have an underconstrained patella—one that moves too much because of hyperelasticity and instability—they have specific characteristics, as do patients who have been immobilized for a long time and have a patella that is hypomobile. Or there may be biomechanical factors, that is, malalignment factors, such as an excessively pronated foot or a tremendous amount of femoral anteversion or tibial torsion. Or a patient may have been hit on the kneecap and therefore may have trauma-induced pain. But the authors classify all the patients in their study as having patellofemoral pain, and the reader is to assume that patellofemoral pain and quadriceps weakness go hand in hand. Which isn't always the case.

**Rothstein:** You're suggesting four categories of patients. Do you use these subclassifications in your practice?

**Wilk:** Yes.

**Karst:** I think of "disturbance" in broader terms. There could be structural problems, or there could be motor control problems—which has been proposed in the literature and which obviously is what these authors are talking about. The reason we lump together all patients with patellofemoral pain is that we really don't know the etiology of patellofemoral pain, and not everyone will agree on categories.

**Rothstein:** The classification that Kevin [Wilk] suggests is based on examination findings more than on etiology, however.

**Wilk:** Thus, the treatment plan is based on those findings. I don't believe that everyone with patellofemoral pain has muscle imbalance or motor control problems.

## Testing the Notions Underlying Practice

**Rothstein:** We're questioning some ubiquitous notions. Here's another: All people with patellofemoral pain have a tracking problem. Or do they?

**Wilk:** No. But then again, I'm not sure we can agree on what "normal" tracking is. We know that there is some lateral excursion of the patella as the lower extremity goes into extension and flexion. In the normal subject, the patella tends to track in a lateral "C" pattern, as has been described in the literature.<sup>1,2</sup> One notion is that the VMO [vastus medialis oblique muscle] is the primary structure that stabilizes the patella in the trochlea. As noted in this paper, studies by Lieb and Perry<sup>3,4</sup> have shown that the VMO attaches to the patella at an approximately 55-degree angle. But not everyone's VMO attaches at that angle. In fact, most patients I see have VMO dysplasia, in which the VMO is absent, has atrophied, or attaches at a very low angle, similar to the longest fibers of the VM [vastus medialis muscle]. From a tracking standpoint, there are a number of structures—osseous and ligamentous—that provide stability and that are just as important as the VMO.

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**Malone:** I use a classification scheme similar to the one that Kevin uses, only I break it down a little differently. Patellar instability can be either active—an inability to control the patella due to motor problems or pain—or passive, due to ligamentous incompetence.

**Rothstein:** Tell us more about the examination procedure.

**Malone:** To determine whether a patient has passive instability, I look at gross instability, typically near extension. When the patient is relaxed, I move the patella medially and laterally and inferiorly and superiorly, noting the gross movement pattern facilitated through my tactile stimulation. Of course, it's important not to dislocate the patient's patella! But you do need to determine the movement pattern and assess the inherent ligamentous restraint. In the literature, there are some good descriptions of the tethering bands—meniscal-patellar and tibial-patellar, sometimes described as patellofemoral ligaments—particularly in a text produced by the Hughston Orthopedic Clinic,<sup>5</sup> in which Emily Craig, the medical illustrator, created models that further delineate those structures. If there is a ligamentous incompetency, that is, if there is insufficient passive restraint, the patient should not be exercised in extension. The patella

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normally is laterally biased in extension; in these patients, it's dramatically laterally biased.

**Rothstein:** We should note that this is based on clinical observation. It hasn't been measured, and published data supporting the concept have not appeared.

**Malone:** That's correct... Some patients have an active inability to control the patella. When they go through a range of motion in a loaded state—both closed kinetic chain and open kinetic chain—they have a great deal of difficulty moving smoothly through the range and report that it "feels like I can't control my kneecap." These patients have a lack of motor control because of a deficient firing pattern or the inhibitory influence of pain. I use the following analogy with my patients: "It's like a bobsled run. If you don't keep your bobsled in the middle of the run and you bang against the sides, you have problems. The same thing happens when you don't keep your patella in a well-controlled tracking pattern."

**Rothstein:** This is a *hypothesized* mechanism.

**Malone:** True. We should never put bad science to good clinical observation.

**Rothstein:** The definition of "good clinical observation" usually is in the eye of the beholder, however. None of the patient classification systems we've discussed so far have been tested in outcomes studies, let alone in reliability or classification studies. We shouldn't assume that a given belief system is better than what's already been published in the literature.

**Malone:** There are three other areas to observe in patients with patellofemoral pain—tension, friction, and compression. Patients may have difficulty in jumping; landing

hurts them. That may be due to eccentric activation; however, in normal jumping, many people use the stretch-shortening cycle, as described by Komi and Bosco.<sup>6</sup> Many of these patients also report that they have pain when they move into a flexed position to potentiate the jump. This may be patellar tendinosis, that is, a muscle tendon unit problem. Some patients—frequently they are runners—have trouble with repetitive flexion-extension activities. There may be fat-pad irritation or problems with the iliotibial band, the plica, or the tethering band that attaches to the tibial-meniscal area or to the femur itself. Other patients may have compression problems, described as "excessive lateral compression syndrome" by Ficat and Hungerford.<sup>7</sup> I believe these problems aren't solely lateral—they can involve any area of the vertical ridge. I like to divide patients into these groupings because then I can have a specific program—a protocol that follows a strong evaluative sequence—for each.

**Fitzgerald:** When a patient has recurrent patellofemoral dislocation, there's no question that malalignment is involved; however, I agree that patellofemoral pain is not related to malalignment as often as we may think it is. This study doesn't support muscle imbalance that would relate to malalignment. From my clinical observations, the Q angle, which has been used as a measure of malalignment, doesn't seem to be highly correlated with pain problems in the patellofemoral region, and surgeries to correct alignment don't seem to be highly successful. All of these factors indicate that alignment isn't the real culprit. Both Terry [Malone] and Kevin have suggested other factors, such as flexibility of structures associated with the knee and the patellofemoral joint. Adaptive shortening of those tissues might expose the knee to added stresses. Anything

that would increase patellofemoral compression might create a repetitive trauma syndrome, which might be the cause of pain in many cases. The real culprit, then, may be the way structures around the knee deal with stresses applied during functional activities. If there's a quadriceps deficit, perhaps the quadriceps isn't able to dissipate loads across the knee during activities. If tissues aren't flexible or if there is adaptive shortening of muscles, compression forces may increase to levels beyond what is normal during functional activities.

### Tied Up in Anatomical Structure

**Rothstein:** We've heard some intriguing hypotheses about a variety of patellofemoral problems. Let's try to forget for a moment what we learned as physical therapists. From a motor control standpoint, does the timing theory described by the authors seem reasonable?

**Karst:** When you consider the structures involved, the theory makes *a priori* sense. Physical therapists tend to look at this problem anatomically, focusing on the medial force vector from the VMO and the lateral force vector from the VL [vastus lateralis muscle]. The problem is that anatomy isn't the whole answer. Compare the vastus muscles with the deltoid muscles, for example. We may think of anterior and posterior deltoids as being similar muscles, but we also know that they can be activated differently. They can act as antagonists or as synergists, just as, according to recent research,<sup>8,9</sup> the vastus muscles and the rectus femoris muscle do very different things. The vastus muscles, however, seem to function together. Looking at the anatomy, it makes sense to say that there could be vastus muscle imbalances in magnitude or timing; however, there is a

great deal of evidence to suggest that those imbalances don't occur, that those muscles are "hardwired" together.

**Wilk:** The knee is the most biomechanically complex joint in the human body, excluding the spine. It's complex not just because of the biomechanics but because of the significant anatomical variations, which are similar to those of the shoulder joint, with the static stability restraints. Terry brought up the tethering bands, the ligamentous bands that stabilize the patella. In a study on these static restraints, Conlan et al<sup>10</sup> found that the medial patellofemoral ligament provided more than 50% of the restraining force for preventing lateral dislocation of the patella. But the most interesting finding was that among 19 dissections of cadaveric specimens, one third did not have an eloquent medial patellofemoral ligament. The most important stabilizing structure in the knee from a static standpoint often is not even present!

**Rothstein:** Do we know if these individuals had had symptoms?

**Wilk:** Unfortunately, no clear association has been substantiated. In clinical practice, we can't see beneath the skin, of course. But we can palpate, and sometimes when we set out to palpate the medial patellofemoral ligament, we don't find it. We then can provide resistance to the VMO and palpate its border to roughly calculate the angle of insertion. In my experience, the patients who have the highest incidence of symptoms, such as slender individuals, usually are deficient in the ligament or have a high angle of VMO insertion into the patella.

**Karst:** To follow up on active and passive instability: It's a good point that there are *passive* anatomical structures, including muscles when

they're not activated, and *active* anatomical structures. I'd be cautious, however, in moving the patella while the muscle is relaxed. It's difficult to know the level of the forces that the therapist exerts on the patella as compared with forces exerted during real-life movement. Think of the anterior cruciate ligament and what we really test when we do a manual ligamentous muscle test. We barely stress it beyond the toe region of the stress-strain curve when we do the clinical test, whereas physiological loading results in much greater stress to the ligament.<sup>11</sup> That may be the case with patellofemoral articulation as well. We know from the motor control literature that when there are a number of synergistic muscles across a joint, it's difficult to "parcel out" the relative forces. Likewise, even though the knee is relaxed, it's difficult to determine the relative importance of the forces.

**Malone:** It's vital to recognize that, as mentioned previously regarding the Q angle, measuring isn't appropriate. Patients usually complain of pain on one side, but most of the time they have a fairly equal Q angle from right to left. If a patient has malalignment or an abnormal Q angle on the right, the same is probably true on the left—but the patient may complain of right anterior knee pain. That should point to a multifactorial problem. There is a lack of reliability in every patellofemoral study in terms of the measurements. It's a very inaccurate process, fraught with the danger of saying too much about too little.

**Rothstein:** I can't help but reflect on the interesting nature of all of these hypotheses and marvel at how wonderful physical therapists are at using anatomy. It's our great tool. But I'm also reminded of the neurophysiologist who, in considering a mechanism, said, "All we need now is a trained anatomist who can

provide a pathway to prove that this concept is viable." Sometimes we need a physiologist to eliminate the nonviable hypotheses, because we are extraordinarily tied up in anatomical structure, and we relate that structure to function. But testing our ideas becomes very difficult in the intact human being, and that underscores the necessity of going beyond the mechanistic study to the testing of classification schemes. In addition to asking, "Are the mechanisms correct?" we need to ask, "Do the mechanisms lead to a useful scheme of treatment?" So far, our discussion has been a fascinating expression of anatomical and physiological concepts. But how could we ever test them?

**Malone:** Some of these concepts have been tested. In the journal *Isokinetics and Exercise Science*, Conway et al<sup>12</sup> reported force output and perceived pain with the use of taping or a Palumbo brace. The subjects were cadets at the US Air Force Academy; they all had anterior knee pain. They were tested in a five-step sequence: initially as their own control group; next, either braced or taped, after which they were control subjects; then conversely braced or taped, after which they were control subjects again. This sequence was conducted in an open chain with a KIN-COM<sup>®</sup>,\* looking at concentric and eccentric movement and using a pain scale. There was a significant increase in torque output in both braced and taped conditions—concentrically and eccentrically with the tape, and very close to concentrically and eccentrically with the brace. Perceived pain decreased dramatically under both conditions, but the increase in torque output was not correlated well at all with the decrease in perceived pain.

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**Rothstein:** Was this pain perceived during exercise?

**Malone:** Yes.

**Rothstein:** I submit, then, that the question addressed by that study was not really whether the condition was improving. Examination of pain during exercise is viewed as less and less justifiable in terms of reimbursement. We need to look for studies in mainstream, widespread publications that deal with improvement in function—and that give definitions. With the KIN-COM®, for example, the leg is fixed, which does not meet the definition of open kinetic chain given by Steindler,<sup>13</sup> who stated that the kinetic elements in the chain must be defined. If the leg is attached to the machine, the chain is closed through the machine. This is an example of problems in terminology, and it illustrates the need for publication in robustly reviewed journals. The purpose of the Powers et al study was to test the hypothesis that subjects with patellofemoral pain would demonstrate EMG patterns consistent with the theory that timing and intensity differences are associated with patellofemoral pain. We have to remember that this study is looking at the viability of a theory, not the viability of a treatment. The treatment might in fact work, but we can't tell from this study.

**Fitzgerald:** But the results of this study *do* cause me to rethink the cause of the problem. As I discussed earlier, the study results don't support the theory of an imbalance between the VL and the VMO in terms of a timing problem, which has been a prevalent idea. But the authors did find an overall reduction in quadriceps activity in the various functions that were measured. That suggests that we might want to consider discarding the notion of an imbalance between the heads of the vastus muscles, but that a quadriceps deficit in general may

be part of the problem or can be used as a marker to determine when someone is getting better. If patients with training are able to increase quadriceps activity to a level similar to that of nonpatients, that would indicate that quadriceps activity was the problem.

**Karst:** I agree that the study doesn't support the timing hypothesis. The magnitude issue is interesting. The authors found that the magnitude—expressed as a percentage of maximal voluntary isokinetic contraction at 60 degrees—did not differ between groups for stair climbing, but that it did differ between groups for level walking and ramp walking. The authors' proposed explanation wasn't that the subjects were *unable* to adequately activate the quadriceps. The authors hypothesized that the subjects made a subtle change in kinematics during level walking and ramp walking and thus decreased the need for activation, but that they apparently were unable to make such a change in kinematics during stair climbing. It's difficult to interpret this. The patients used a smaller percentage of their maximal isometric contraction to do most tasks than did the people who had no pain.

**Rothstein:** This was what the authors called "quadriceps muscle avoidance pattern."

**Wilk:** I've never entirely accepted the concept of "reversal pattern," which is described in this manuscript and by Voight and Wieder<sup>14</sup> and which holds that there is a reversal of normal firing patterns. In my opinion, Voight and Wieder didn't prove that hypothesis; they never tested the subjects once they were pain-free. Testing the onset timing deficit is difficult in the clinic, whether you use a biofeedback unit or something else. I'm not sure that the timing difference would have any relevance to clinical

practice. For the most part, in our clinic, we look not at onset but at ratios and duration. What is the magnitude of the VMO contraction, and to what degree? We hypothesize that if there is a somewhat acceptable VMO-to-VL ratio, it should induce appropriate tracking of the patella in the superior direction without excessive lateral or medial excursion. There occasionally are patients who actively subluxate their patella medially, usually after surgery. It was interesting to me that in this study, the experimental group exhibited a lower magnitude of EMG activity [Tab. 4] for both the VMO and the VL in five out of six test conditions. The only test condition that did not illustrate this difference was that of ascending stairs, when the experimental group exhibited a slightly higher intensity of EMG activity. Although there is no significant statistical difference, there may be real differences that could be clinically relevant.

**Rothstein:** When we use EMG biofeedback units in practice, we rarely normalize, and we therefore have to be careful when we anecdotally describe what we do clinically. What does this paper tell us about normalization?

**Malone:** This was a very well-controlled normalized EMG study. Using fine wire gave the authors the opportunity to get good information, and they expressed that information well. Because they used such a well-controlled normalization process, it's difficult to make direct comparisons with other papers. In fact, the authors pointed out that their results differ from those of other papers because of this. It was interesting to see there was "quadriceps avoidance" across all conditions. It may be an inhibitory influence, it may be a learned response to minimize activation lower in the range of motion, it may be related to eccentric activity—but somehow, there is an altered response. Ben-

nett and Stauber<sup>15</sup> established that the primary difference is eccentric. It would have been interesting to have the subjects in the Powers et al study do some eccentric activity with the fine wires in place to see whether the activity was altered more dramatically than concentric activity was altered in some of these patterns. Bennett and Stauber said that a certain percentage of patients with patellofemoral pain have eccentric weakness; other researchers have found that the same percentage of the healthy population also have that weakness.<sup>16</sup> In the Conway et al<sup>12</sup> study, none of the cadets exhibited that eccentric weakness, yet they had anterior knee pain.... EMG activity is extremely difficult to quantify, especially when dealing with smaller percentages. Our clinic did an unpublished study on consistency of EMG activity in raising and lowering a known amount of weight from day to day, and we found that in the healthy population, there may be as much as a 12% to 15% variation in EMG activity. And that's while maintaining the same amount of weight and keeping the electrodes as consistently placed as possible. Again, we have to be careful about saying too much about too little.

### **Biofeedback: Time to Hang Up the Machines?**

**Rothstein:** Based on variability of findings of previous studies, on the attention that the authors of this paper paid to methodological issues, and on the fact that their findings agreed with the Karst and Willett study<sup>17</sup> as a result of those methodological consistencies—should we hang up the EMG machines?

**Fitzgerald:** It's not time to give up on biofeedback treatment. We can't tell from this study whether the quadriceps deficit came first, and the study doesn't support using a biofeedback device to restore bal-

ance among the various heads of the quadriceps. In some activities, there was a delay in onset in some of the muscles during fast walking and stair descending, so there still may be a timing problem, but it may refer to the whole quadriceps group rather than to specific heads. Biofeedback may be useful in improving magnitude of activity or timing of the entire muscle group.

**Rothstein:** If you were a managed care organization [MCO], would you pay for that?

**Fitzgerald:** Probably not. Many of these patients get better through a thorough training program for quadriceps strengthening, through flexibility exercises, and through functional activity modifications—without ever going near a biofeedback device. If I were an MCO, I wouldn't pay for biofeedback unless someone showed me that using such a device would make the patient better faster at a lower cost.

**Wilk:** Biofeedback is useful under specific conditions. Using the classification scheme I proposed earlier, if a patient has patellar instability or hypermobility, we'd want to enhance the dynamic stabilization, and biofeedback would be very useful for that. However, it wouldn't be the best treatment for a patient who's been immobilized and who has a tight knee joint, even though there may be some quadriceps atrophy. We tend to get enamored of taping, bracing, isokinetics—whatever the mood of the month is—but the fact is that those treatment methods may be indicated not for every pathology related to the patella, but rather for very specific pathologies.

**Rothstein:** From a payment standpoint, then, would you say that when a case can be made to justify application of biofeedback, it is worth reimbursing?

**Wilk:** Yes, under specific circumstances and clinical conditions.

**Malone:** I agree. There are times when biofeedback can be used to help an individual regain some level of conscious awareness. When patients can feel something, see it, and integrate it, they can increase their ability to use it more functionally.

**Wilk:** Haven't some studies shown that biofeedback can selectively recruit the VMO independent of the VL?

**Malone:** Since the LeVeau and Rogers<sup>18</sup> article in 1980, there have been a couple of other studies. But I haven't seen anything that was convincing to me as a critical reader.

**Karst:** I don't believe there has been any good evidence. The studies by LeVeau and Rogers and Wise et al<sup>19</sup> both had some problems. Wise et al did not normalize the EMG data from session to session, and there were saturation problems. LeVeau and Rogers did normalize—to 80% of MVIC [maximal voluntary isometric contraction]—but the training and testing were done at levels less than 25% of MVIC, well below the threshold for strength training.

**Malone:** Wise et al also used a fairly small patient group.

**Karst:** The clinical use of biofeedback is an important issue. I don't know how many therapists are using it to address timing, but I do know that there is widespread use in addressing relative magnitudes. Here's how I break down the theoretical usefulness of a dual-channel EMG biofeedback device. If you don't move the electrodes and the patient doesn't perspire under them within a given session, changes in overall magnitude within that session will be meaningful. But you have to be careful between sessions.

Even if the patient lifts the same weight from day to day, the absolute magnitude of the EMG activity will be different, no matter how carefully you place the electrodes, which is why you need to have the patient do a known submaximal contraction day to day to normalize. There's a great deal of misconception, from what I've heard in continuing education courses particularly, that there should be a one-to-one ratio between the medial and lateral components, and that attaching electrodes alone should tell us something about that. It doesn't, however, because of the differences in placement and subcutaneous tissue. The VMO has less subcutaneous tissue over it than the VL does....

**Rothstein:** What does the literature tell us?

**Karst:** If you compare studies that have looked at nonnormalized EMG findings, the VMO-to-VL ratio is almost always greater than 1, whether it's in subjects with or without patellofemoral pain, whereas in normalized EMG studies, the ratio tends to be around 1, which means that the medial and lateral aspects of the quadriceps don't change much from the normalizing contraction. The vastus muscles are used in the same way, regardless of the type of contraction and regardless of the location in the range. In the study by Souza and Gross,<sup>20</sup> the nonnormalized VMO-to-VL ratios were very low—around 0.5—because they oriented their VMO electrodes perpendicular to the fibers instead of longitudinal to the fibers. That's an example of how small differences in placement can make a big difference in raw magnitudes. It shows why those raw magnitudes don't mean much.

**Fitzgerald:** Another issue is that the EMG activity seen in the VMO may not represent the same amount of force or tension being generated by the VL, even if there is the same

level of electrical activity. When there is an imbalance related to malalignment, you're talking about balancing the forces being generated; and even when the amount of EMG activity is the same, you can't assume that the level of forces is the same.

**Rothstein:** How would you use an EMG biofeedback unit?

**Karst:** Each day, have the patient do a submaximal contraction in a way that you can reproduce—for instance, while holding a 5-lb weight at a 45-degree knee angle. That reading—an average of 5 seconds would be typical—would be the patient's 100% for that day, and you would quantify the patient's maximal contraction during a particular exercise in terms of that normalizing contraction. If you do the same thing every day, and express the data in terms of the percent of the activity for that day, you can compare data across days, similar to the way that Powers and colleagues compared data across subjects. There is some evidence from Yang and Winter<sup>21</sup> that using submaximal contractions is more reliable because people tend to be unreliable in generating maximal contractions.

**Rothstein:** The consensus of this group seems to be that there is a great use for the clinical biofeedback unit—when there is an appropriate theoretical model for using it. There also is a need for normalization, very careful consideration of electrode placement, and proper methodology, because without them, you'd be creating artifacts.

**Karst:** I also agree with the authors' conclusion that we should question the use of temporal analysis with EMG biofeedback, primarily because those devices don't have the temporal resolution to do the kind of analysis done in this study—which is similar to the kind of analysis that my colleagues and I

reported for both reflex and voluntary quadriceps activity.<sup>17</sup>

## The Patients: Typical or Atypical?

**Rothstein:** Are the patients in this study typical or atypical?

**Wilk:** Every time I read a paper about patients with anterior knee pain or patellofemoral pain, I never really know who the patients are. The subjects in this study are no exception. The authors tell me who the patients *aren't*—they aren't people who have had surgery, they aren't people who have dislocations, they aren't people with neurologic involvement. But I don't know what kinds of problems these individuals have, other than vague or localized pain or pain with squatting or other activities. I don't know whether these are the types of patients that I see in my clinic, for instance.

**Malone:** I agree.

**Fitzgerald:** In the clinic, therapists collect additional information, but for the purposes of this study—assessment of movement and EMG patterns in patients with anterior knee pain—the authors chose "classic patients": those with pain on squatting, stair climbing, kneeling, prolonged sitting. Other knee problems were ruled out. Many of the patients I see would fit that category.

**Wilk:** But are there articular cartilage problems? Is there passive or active instability? Do the patients subluxate the knee? Are the knees too tight? Do the patients have fat-pad syndromes or saphenous neuritis? All of those symptoms could be present with a variety of diagnoses. I make a plea to authors writing about patellofemoral pain: Please be more specific about what kinds of patients are involved! We traditionally have clumped all of our treatments together—biofeedback, mus-

cle stimulation, aquatic therapy, taping—but is every type of treatment effective for everyone under every condition?

**Rothstein:** According to this discussion, the authors' conclusion did not support the concept that they tested. You've provided an explanation that may make the concept viable. You've suggested that the heterogeneity of the patient group may have made for unfair testing of the theory. That is, the concepts of muscle imbalance and tracking problems may apply to subcategories of patients, but we'd have no way of knowing that through this study.

**Fitzgerald:** But you *can* start an investigation with a broad picture, on a large scale, choosing general characteristics that most people with patellofemoral pain have. I don't disagree with the notion of dividing patients into subcategories and finding out how that classification would affect findings. But there is something else to consider: Maybe the testing procedures weren't intense enough. In my experience, many patients become symptomatic only after a long period of activity—10 or 15 minutes of running, for example. In this study, muscle activity was measured after the subjects performed the activity for only a few trials. I suggest testing to see whether there is an imbalance after someone has run on a treadmill for 10, 15, or 20 minutes. That would be closer to what might happen in a real-life functional situation.

**Karst:** It would be nice if there were clear-cut categories to test. But the problem is that there isn't any agreement on what those categories should be. And remember, we're a panel of experts. What about new graduates? They don't have our perspectives, and many of them are treating patients with patellofemoral pain. They can go to any number of weekend continuing education

courses where they will be told that people with these general symptoms should be treated on the basis of the timing theory.

**Wilk:** Do you think patients could have an alteration in onset timing if they continued to perform these activities or performed very specific movements? And would there be a change from week to week?

**Karst:** A subgroup of six patients who participated in our timing study<sup>17</sup> were followed. Results showed that pain as measured using a visual analog scale decreased after 4 weeks of conservative treatment, but nothing changed in terms of EMG timing or magnitude.<sup>22</sup> We didn't follow a large enough sample to have statistical power, however.

**Malone:** LeVeau and Rogers<sup>18</sup> studied 10 healthy student volunteers aged 22 to 29 years who had 3 weeks of training. Percentages in the Powers et al study naturally look very different from those percentages.

### The Implications of Being Barefoot

**Rothstein:** I was curious about one methodological aspect of this study. Because of the device used—the Stride Analyzer—the patients were barefoot. How might barefootedness affect the findings? What do we know about the kinetics and kinematics? What do patients tell us about their symptoms while barefoot versus their symptoms while wearing shoes?

**Wilk:** I asked biomechanists at our facility to read this paper because I wasn't familiar with the motion analysis system used. From a gait standpoint, according to the biomechanists, this system is accurate, but it may not be for higher-speed activities. In the study, the authors referred to "fast gait." They

didn't supply a lot of biomechanical data—obviously they were looking for other kinds of information—but data related to knee flexion angle and onset, for instance, might have supported or refuted the concept of quadriceps avoidance related to the flexion moment. The authors alluded to flexion moment, but they did not supply sufficient data for us to examine whether it was the flexion moment.

**Wilk:** I apologize because I have to keep referring to the classification scheme! Under certain conditions—such as an articular cartilage lesion due to an overconstrained joint or trauma due to a blow to the knee—the patient's gait pattern is affected.

**Fitzgerald:** Earlier I questioned whether the testing procedures were intense enough. Perhaps they *were* intense enough, given that the subjects were barefoot.

**Rothstein:** Would general quadriceps avoidance be more likely to occur when the patient is barefoot?

**Fitzgerald:** If barefootedness had played a major role in this study, I would have expected more variability in the results. Otherwise, we would have to assume that every patient has a similar type of foot, which I find hard to believe.

**Rothstein:** Where would you expect to see the variability?

**Fitzgerald:** If there had been variability in the amount of pronation or supination during stance phase, and if that variability was important, I would have expected to see differences in the EMG activity. But clearly most of the people in this study responded in the same way.

**Wilk:** Do patients with patellofemoral pain tell you that they hurt more in or out of shoes?

**Malone:** Some feel better in shoes, some feel better out of shoes, depending on the type of problem. If there are vague medial problems, plica irritations, or friction-related conditions, they might feel better in shoes because they wouldn't pronate as much and the medial drag would be minimized.

**Wilk:** Therapists therefore can make a strong case for the use of orthoses by people with patellofemoral pain even when orthoses aren't indicated by the attending physician.

### Effects of Speed and Weight

**Rothstein:** Is there a difference in complaints among patients with patellofemoral pain when it comes to different walking speeds?

**Wilk:** I never hear a patient say, "I can't walk fast" or "I can't walk slow." Some say that they can't go up or down stairs, slowly or quickly.

**Fitzgerald:** I agree. I also hear complaints about prolonged sitting or jogging.

**Rothstein:** The authors pointed out that weights and heights were similar between the groups. What are the implications of that?

**Malone:** For many of these patients, weight is an important variable. We used to think that it was the young women who didn't have "adequate control," particularly when they were overweight—that they had an "odd-shaped patella." It's important to note that we see patellofemoral pain in men as well as women.

**Wilk:** I haven't seen anything that would correlate with height. But with kneeling and squatting, weight is an issue. If it's a question of quadriceps insufficiency, however, we should be hearing more patient

complaints about not being able to walk fast or run. When people run, they assume a position of greater flexion than they do when they walk; the flexion moment therefore is greater, and more demands are made on the quadriceps. Instead, I hear, "I can't go up and down stairs," "I can't get up once I'm sitting down," or "I often have to straighten my knee."

**Rothstein:** But has anyone systematically documented a relationship between weight and patellofemoral pain?

**Wilk:** I am aware of only one published study that documents the relationship between the patient's body weight and total knee replacement surgery and postoperative rehabilitation.<sup>23</sup>

**Malone:** I've never read a well-controlled study that has documented it. Anecdotally, in a study published in a non-peer-reviewed journal in the 1960s, subjects wore a weight belt only in the morning. Fewer pain complaints were recorded in the afternoon, when they weren't wearing the belt.

**Rothstein:** Historically, in osteoarthritis, there is the notion that weight is a factor, but in fact it's only a factor in medial compartment disease and has more to do with use than with weight, such as when people abduct when they walk. This would be a topic for an interesting descriptive or epidemiological study. The common wisdom—that weight plays a role—has never really been tested through the collection of data.

**Malone:** We also should mention that the articular cartilage on the patella is the thickest in the human body. It's not that way by accident. The forces are enormous.

**Wilk:** If you asked clinicians who treat a lot of patients with patel-

lofemoral problems, they'd probably say that it's the slender patients who have a higher incidence of patellofemoral dysfunction.

**Fitzgerald:** I can't say one way or the other. I've had an assortment of patients.

**Malone:** Again, it all goes back to classification systems. Unfortunately, some people have more than one problem. Many of the slender individuals who have trouble controlling the patella fall into the first classification scheme, which is based on active or passive instability. Conversely, those who have arthritic problems in the medial-tibiofemoral area may have severe patellofemoral pain because they have both types of conditions.

### Will This Study Change Your Practice?

**Rothstein:** As an editor, this conversation makes me salivate for descriptive case studies. The number of phenomena that need to be described is extraordinary—as is the number of clinicians who are treating these patients and who could write these descriptions. We can only hope that this Conference will stimulate them to do so.... Will this study change what you do in practice or research?

**Karst:** We all like to have our views confirmed, and this paper did that for me. It has some direct implications for the use of biofeedback. I don't think it's necessary to give up on biofeedback, but it is necessary to think critically about how we use it. The paper underscores the need for more descriptive information, for agreement on categorization of patients, and for theory underpinning blanket protocols. Are there specific VMO exercises that we should use? A number have been proposed. Cerny<sup>24</sup> showed that you can do many different quadriceps

exercises and VMO/VL ratios don't change much! We do know that people feel better when they're taped or braced; we don't know the mechanism. There is conflicting evidence about whether taping or bracing changes the position of the patella, and, if so, about how long that lasts if the patient moves when the tape is in place. Not for long, I'd say.... We need to tease out which parts of the protocol are helping patients and which ones are just wasting our time and our patients' time.

**Malone:** Protocols are appropriate; but you need four to six protocols, depending on how you classify the patients, to adequately address the specific needs of a given patient group. Mangine<sup>25</sup> used the term "evaluation-based protocol," and that fits here. You evaluate, fit the patient into a proper grouping, and apply a protocol. But you have to be willing to adjust according to further evaluation.

**Karst:** That's a good point. Taping, for instance, helps some people. But can you determine how to tape people by palpating the patella? As Fitzgerald and McClure<sup>26</sup> found when they studied the reliability of tests for patellofemoral alignment, apparently not!

**Fitzgerald:** I don't think this study would immediately change anything I'm doing with my patients. I never did buy into the VMO-VL imbalance or the need to do specific exercises to restore that balance. My approach has been to provide a well-rounded quadriceps strengthening program. The study raises interesting questions, however. Why is there reduced activity in the quadriceps of patients with patellofemoral pain? Was it the reduced activity that caused the problem, or vice versa? The answers have implications for evaluation and treatment. If we had information about ground reaction forces, we could determine

whether a compensatory pattern occurred. Would the kinematics and kinetics explain the reduction in quadriceps activity? If so, that information could be used both as a marker to determine whether a patient is compensating for the problem and as a guide to developing training programs. If the pain is under control but the compensatory pattern still exists, there may be strategies that therapists can use to restore normal patterns and prevent future problems. If the reduction in activity remains even when the pain is under control or eliminated, that may be why there are patients with chronic knee problems.

**Wilk:** But what about recruitment of the VMO? I have a feeling that Greg [Karst] believes that we have a certain firing or recruitment pattern, regardless of the type of exercise or movement, and that we cannot "bias" the VMO. Is that correct?

**Karst:** I wouldn't go that far! There's no blanket prescription. I'm not sure that individuals *couldn't* learn to preferentially activate the VMO with an individualized exercise program based on EMG analysis, but we have no data to support that yet. When you look across a group of patients, as Cerny did, using a variety of exercises, the results just wash out. But you might be able to tailor the exercises for an individual patient. As far as specific strengthening, there are two criteria to meet: The patient has to reach an overload range, and there has to be a major difference in the relative levels of activation of the VMO and VL if you want to change the muscle itself. You also have to consider whether you are trying to make the muscles hypertrophy so that the same motor commands will give more force, which would make sense, or whether you are trying to carry gains over to functional activities, which no one has studied yet.

**Wilk:** There are specific movements and specific ranges in the motion that bring about certain muscle activity within the quadriceps. One of the classic examples and a good contrast is the knee extension versus the squat. During knee extension, there is a specific muscle activity pattern at 90 degrees and a different one at 30 degrees. The same is true with the vertical squat.

**Karst:** There has been some evidence of changes within range of motion, but I'm concerned that when testing is done—as most of it is done—with surface electrodes, there is a large change in the relationship between the electrode and the fibers of the VMO as the angle changes. I sincerely doubt that we are recording from the same part of the muscle when we're at different ranges. That is, I doubt we have the same fiber-to-electrode orientation. The question is still open.

**Wilk:** I appreciate that point of view, but we have a paper in press that states the opposite. We did a biomechanical study looking at three different movements—the knee extension, the squat, and the leg press—using surface EMG. We used motion analysis to analyze the joint shear and compressive forces. We used surface EMG because the subjects did not like to be stuck with a needle. There were dramatic and consistent differences, especially between the knee extension and the squat, within the muscles.<sup>27</sup>

**Karst:** Was there a big difference at the same angle for the two exercises?

**Wilk:** Yes.

**Karst:** If the knee angle is the same, that would be a valid comparison.

**Wilk:** We've also taken it one step further, comparing our results with

those of other studies. The findings have been very similar.

**Rothstein:** We could probably talk for hours! This is one of those areas of practice in which there are good points of view both ways. One of the points that's been brought out in this discussion is that we have a series of treatments that many people believe in—whether it's training, tracking, or taping—but when we study them, we find that the mechanisms may not be viable. But we also have to remember that we don't know whether the treatments themselves work. We need further data. There is room for mechanistic and theoretical studies in addition to general outcomes studies. This Conference characterizes the research questions and highlights the concerns that every therapist in this area of practice should have.

## References

- 1 Hungerford DS, Barry M. Biomechanics of the patellofemoral joint. *Clin Orthop*. 1979; 144:9-15.
- 2 Warren RF, Arnoczky SP, Wickiewicz TL. Anatomy of the knee. In: Nicholas JA, Hershman EB. *The Lower Extremity and Spine in Sports Medicine*. St Louis, Mo: CV Mosby Co; 1986:676-677.
- 3 Leib FJ, Perry J. Quadriceps function: an electromyographic study under isometric conditions. *J Bone Joint Surg [Am]*. 1971;53: 749-758.
- 4 Leib FJ, Perry J. Quadriceps function: an anatomical and mechanical study using amputated limbs. *J Bone Joint Surg [Am]*. 1968; 50:1535-1548.
- 5 Hughston JC. *Knee Ligaments: Injury and Repair*. St Louis, Mo: Mosby-Year Book Inc; 1993.
- 6 Komi TV, Bosco C. Utilization of stored elastic energy in leg extensor muscles by men and women. *Med Sci Sports Exerc*. 1978;10:261-265.
- 7 Ficat P, Hungerford D. *Disorders of the Patellofemoral Joint*. Baltimore, Md: Williams & Wilkins Co; 1977.
- 8 Soderberg GL, Minor SD, Arnold K, et al. Electromyographic analysis of knee exercises in healthy subjects and in patients with knee pathologies. *Phys Ther*. 1987;67:1691-1696.
- 9 Karst GM, Jewett PD. Electromyographic analysis of exercises proposed for differential activation of medial and lateral quadriceps femoris components. *Phys Ther*. 1993;73:286-299.
- 10 Conlan T, Garth WP, Lemons JE. Evaluation of the medial soft tissue restraints of the extensor mechanism of the knee. *J Bone Joint Surg*. 1993;75:682-693.
- 11 Noyes FR. Functional properties of knee ligaments and alterations induced by immobilization: a correlative and histological study in primates. *Clin Orthop*. 1977;123:210-242.
- 12 Conway A, Malone TR, Conway R. Patellar alignment/tracking alteration: effect on force output and perceived pain. *Isokinetics and Exercise Science*. 1992;2(1):9-17.
- 13 Steindler A. *Kinesiology of the Human Body Under Normal and Pathological Conditions*. Springfield, Ill: Charles C Thomas Publishers Inc; 1973.
- 14 Voight ML, Wieder DL. Comparative reflex responses in normal subjects with extensor mechanism dysfunction: an electromyographic study. *Am J Sports Med*. 1991;19: 131-137.
- 15 Bennett J, Stauber W. Evaluation and treatment of anterior knee pain using eccentric exercise. *Med Sci Sports Exerc*. 1986;18: 526-530.
- 16 Trudell-Jackson E, Meske N, Highenboten C, Jackson A. Eccentric/concentric torque deficits in the quadriceps muscle. *J Orthop Sports Phys Ther*. 1989;11:142-145.
- 17 Karst GM, Willett GM. Onset timing of electromyographic activity in the vastus medialis oblique and vastus lateralis in subjects with and without patellofemoral pain syndrome. *Phys Ther*. 1995;75:813-823.
- 18 LeVeau B, Rogers C. Selective training of the vastus medialis muscle using EMG biofeedback. *Phys Ther*. 1980;60:1410-1415.
- 19 Wise HH, Fiebert IM, Kates JL. EMG biofeedback for patellofemoral pain syndrome. *J Orthop Sports Phys Ther*. 1984;6:95-103.
- 20 Souza DR, Gross MT. Comparison of vastus medialis obliquus:vastus lateralis muscle integrated electromyographic ratios between healthy subjects and patients with patellofemoral pain. *Phys Ther*. 1991;71:310-320.
- 21 Yang JF, Winter DA. Electromyography reliability in maximal and submaximal isometric contractions. *Arch Phys Med Rehabil*. 1983;64:417-420.
- 22 Willett GM. *Patellofemoral Disorders: Is Timing of Vastus Medialis Obliquus and Vastus Lateralis Muscle Activation a Factor?* Omaha, Neb: University of Nebraska Medical Center; 1994. Master's thesis.
- 23 Stern SH, Insall JN. Total knee arthroplasty in obese patients. *J Bone Joint Surg [Am]*. 1990;72:1400-1404.
- 24 Cerny K. Vastus medialis oblique/vastus lateralis muscle activity ratios for selected exercises in persons with and without patellofemoral pain syndrome. *Phys Ther*. 1995;75: 672-683.
- 25 Mangine RE, ed. *Physical Therapy of the Knee*. 2nd ed. New York, NY: Churchill Livingstone Inc; 1995.
- 26 Fitzgerald GK, McClure PW. Reliability of measurements obtained with four tests for patellofemoral alignment. *Phys Ther*. 1995;75: 84-92.
- 27 Wilk KE, Escamilla RF, Fleising GS, et al. The comparison of tibio-femoral joint forces and electromyography during open and closed kinetic chain exercises. *Am J Sports Med*. In press.

## ● Author Comment

We would like to thank the conference participants for their discussion regarding the clinical and theoretical implications of our research. The etiology of patellofemoral pain (PFP) continues to be poorly understood, making effective treatment a difficult task for the physical therapist. Although there have been many hypotheses proposed as to the cause of PFP, objective data supporting many of these theories are lacking. We agree with the conference members that the ability to subclassify patients with PFP based on objective findings would assist in guiding rehabilitation efforts. At the present time, however, there is no such classification system that has been shown to be based on valid and reliable measurements. Until the mechanism(s) of PFP can be better defined and identified clinically, the rationale behind the various treatment protocols used to treat patients who have this disorder will remain debatable and guided mostly by clinical experience and observation.

The dynamic theory of patellar instability (vastus medialis oblique muscle [VMO] versus vastus lateralis muscle [VL]) has been commonly accepted by clinicians and readily incorporated into clinical practice. The primary problem with this hypothesis is that clinical assessment of "muscular imbalance" is typically based on electromyographic (EMG) findings obtained from dual-channel biofeedback units. As was emphasized in the conference, without proper normalization techniques (ie, to control for potential EMG variability as a result of electrode placement, subcutaneous fat displacement, and so on), comparisons between muscles cannot be made. Thus, these EMG data must be viewed with caution, and care must be taken not to overinterpret their meaning. Furthermore, it appears that using EMG biofeedback to

assist in "selectively recruiting" the VMO or to alter the neuromuscular timing or "control" of this structure may not be justified, as there is no objective evidence in the literature to support this practice. This is not to suggest, however, that biofeedback has no place in the treatment of patients who have PFP. For instance, biofeedback appears to be effective in the promotion of general quadriceps femoris muscle recruitment, which seems to be a common problem in this population. It would be interesting to assess whether this tool could be used to reverse the deficits identified in the current study.

Regardless of the various rationales used to treat patients with PFP, the common link among these methods is that all encourage strengthening of the extensor mechanism. Because the vastus muscles appear to work synergistically, emphasizing a general quadriceps femoris muscle strengthening program may be more cost-effective and less time-consuming than targeting the VMO specifically (keeping in mind patellofemoral joint biomechanics and sensible exercise progression). We submit that emphasis placed on VMO strengthening is most likely inducing a generalized quadriceps femoris muscle strengthening effect and that the focus on this structure may not be necessary. Clinical trials are obviously needed to determine which treatment or exercise protocols will provide the most effective results.

We appreciate the opportunity to express our views on this topic and hope that this work stimulates much-needed clinical research in this area.

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