Onset Timing of Electromyographic Activity in the Vastus Medialis Oblique and Vastus Lateralis Muscles in Subjects With and Without Patellofemoral Pain Syndrome

Background and Purpose. Inappropriate neural control of the quadriceps femoris muscle group has been implicated in patellofemoral pain syndrome (PFPS). This study investigated the timing of initial electromyographic (EMG) activity of the vastus medialis oblique muscle (VMO) and the vastus lateralis muscle (VL) in asymptomatic subjects and subjects with PFPS during reflex and voluntary muscle activity. Subjects. Fifteen symptomatic subjects (SYMP group) (9 with bilateral symptoms) and 12 asymptomatic subjects (ASYMP group) participated. Both knees were tested in the ASYMP group and only the symptomatic knees were tested in the SYMP group, resulting in a total of 24 data sets from each group. Methods. Electromyographic data were recorded from the VMO and VL under three conditions: reflex knee extension (RFLX) elicited by a patellar tendon tap, and active knee extension in non-weight-bearing (NWB) and weight-bearing (WB) situations. For each condition, EMG activity onset times for the VMO and VL were determined from ensemble averages of four trials. Results. There were no differences between the SYMP and ASYMP groups with respect to the relative timing of initial VMO and VL activity under any of the three conditions tested. Mean timing differences for both groups were less than 0.25 milliseconds under reflex conditions and less than 4 milliseconds for active knee extension under both WB and NWB conditions. Conclusion and Discussion. These findings contradict a previous report of differences in reflex timing related to PFPS. Differences in the relative timing of onset of EMG activity of the VMO and VL during voluntary knee extension were not significant between SYMP and ASYMP groups, and were not related to the relative timing differences observed during reflex testing. [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 syndrome. Phys Ther. 1995:75:813-823.]

Key Words: Electromyographic biofeedback, Motor control, Reflex latency.
medialis oblique muscle (VMO) insufficiency. Despite the diversity of terms associated with PFPS, there is general agreement that improper tracking of the patella within the trochlear groove is a major factor in most cases of PFPS.2–6 During flexion and extension of the knee, tracking of the patella is affected by a variety of forces that tend to displace the patella either medially or laterally. These forces may be categorized as passive forces such as those exerted by bony structures, connective tissue structures, and inactive muscles, or as active forces, which come into play only when the nervous system activates the contractile machinery of muscles attached to the patella. Some authorities believe that PFPS is associated with an imbalance of these forces in which the laterally directed forces are excessive in relation to the medially directed forces acting on the patella.

As a result, the goal of conservative treatment for PFPS has often been to reduce laterally directed passive forces by stretching structures such as the lateral retinaculum and the iliotibial band. This goal has been coupled with attempts to increase the medially directed active force exerted by the VMO relative to the laterally directed active force exerted by the vastus lateralis muscle (VL).6,7 If an imbalance in the active medial and lateral forces exerted by the VMO and VL is responsible for initiating or perpetuating PFPS, the underlying mechanism for this active force imbalance could be morphological (eg, hypertrophy of the VMO relative to the VL) or functional (eg, inadequate neural drive to the VMO relative to the VL). In the former case, excessive lateral force on the patella could result because VMO force production could be inadequate in some situations despite maximal neural drive to the VMO. In the latter case, even an adequately hypertrophied VMO could produce less than adequate forces if neural drive to the VMO is either of insufficient magnitude or inappropriately timed, such that the VL is recruited far enough in advance of the VMO to cause a temporary medial-lateral force imbalance during the initial phase of knee extensor activity.

The idea that PFPS might result from inappropriate patterns of neural activity seems reasonable, and has been suggested frequently in the literature.8–16 This concept of viewing PFPS as a motor control problem appears to be gaining in popularity among physical therapists, and treatment of PFPS by using electromyographic (EMG) biofeedback to alter the relative magnitude or timing of VMO and VL activity is being advocated by equipment manufacturers and in continuing education courses. Despite this growing popularity, a literature review revealed very little data supporting possible differences between asymptomatic subjects and patients with PFPS as regard to VMO and VL activity patterns.

With respect to evidence suggesting alterations in the relative magnitude of VMO and VL activity being associated with PFPS, there are conflicting reports. The studies8,17 most commonly cited in support of PFPS-related changes in relative magnitude, however, offer limited evidence for such changes. Mariani and Caruso reported that seven of eight patients diagnosed with subluxation of the patella demonstrated less EMG activity in the VMO than in the VL during active knee extension in the seated position, but their conclusions were based only on qualitative, visual analysis of raw EMG records from five asymptomatic subjects (three male, two female) and eight patients (one male, seven female). A more recent study by Souza and Gross is frequently cited as having demonstrated differences in VMO:VL activity ratios between asymptomatic subjects and subjects with PFPS, but their normalized EMG data revealed no differences between groups. Though they also reported that the nonnormalized EMG data did indicate decreased VMO:VL ratios in subjects with PFPS, there is good reason to believe that the results based on the normalized data have greater validity.10,18,19 Furthermore, other studies examining the relative magnitude of VMO and VL during isometric or isotonic20,21 knee extension exercises or during running22 have failed to show changes in the relative magnitude of VMO and VL activity to be associated with PFPS. Thus, although the issue of PFPS-related changes in relative magnitude of VMO and VL EMG activity is not fully resolved, current evidence suggests that such imbalances are not a critical feature in PFPS.

Recently, numerous authors11,13–16 have suggested that PFPS is associated with alterations in the relative timing of muscle activity in the VMO and VL, but to date only one published study has addressed this issue. Voight and Wieder examined reflex EMG activity elicited by a patellar tendon tap in 41 asymptomatic subjects and 16 patients with PFPS. They reported that the reflex response of the VMO occurred earlier than that of the VL in asymptomatic subjects, whereas the reverse was true in patients with PFPS. They attributed this difference to a faster VL response time in the patient group as compared with the asymptomatic group. They did not examine VMO and VL EMG activity during voluntary knee extension.

Voight and Wieder's findings have potential clinical significance in terms of (1) the etiology of PFPS, because the findings imply that a motor control problem may underlie the condition; (2) the treatment of PFPS, because the findings suggest that emphasis should be placed on biofeedback aimed at normalizing the relative timing of onset of activity of the VMO and VL; and (3) the diagnosis and ongoing evaluation of PFPS, because the findings imply that relative reflex latencies of the two muscles could provide a measure related to the severity of the dysfunction.

The clinical significance of the reflex timing differences reported by Voight and Wieder, however, depends on two factors not addressed in their study. First, the functional relevance of their findings rests on the assumption that changes in reflex latencies are associated with similar changes in
relative timing of onset of activity of the VMO and VL during voluntary activation of the knee extensors. Second, the magnitude of any PFPS-associated changes in timing must be sufficient to result in consequential muscle force imbalances. The actual magnitude of the mean reflex latency difference between VMO and VL onsets of activity was not reported in the Voight and Wieder study, making it difficult to assess the importance of the reported change. In addition, interpretation of the reflex data presented by Voight and Wieder is problematic because the range of reflex latencies they reported differs markedly from that reported in previous studies quantifying patellar reflex latency\(^{21-29}\) and because the effect of subject height, which is strongly correlated with reflex latency,\(^{28-30}\) was not accounted for when comparing absolute reflex latencies between groups.

Based on the considerations discussed, we carried out this study with the following purposes in mind:

1. To quantify more accurately any differences in reflex latencies of the VMO and VL in asymptomatic subjects and patients with PFPS and to test for differences between those groups. In order to do so, we have refined the techniques for eliciting the reflex and triggering data collection, improved the temporal resolution of the latency measurement, and eliminated subject height as a confounding factor by comparing relative, rather than absolute, VL-VMO latencies between groups.

2. To quantify VL-VMO onset differences during voluntary muscle activation under both weight-bearing (WB) and non-weight-bearing (NWB) conditions in order to test for possible timing changes associated with PFPS and to test the assumption that reflex latency differences are indicative of similar latency differences during voluntary knee extensor activity.

3. To determine whether the magnitudes of any latency differences are clinically meaningful in terms of using EMG biofeedback in an attempt to alter the temporal pattern of muscle force production by the VMO and VL.

**Method**

**Subjects**

Two groups of subjects were recruited for the study. All subjects were volunteers and were required to be at least 12 years of age and to provide informed consent prior to participation. Additional inclusion criteria for the asymptomatic (ASYMP) group were (1) no history of knee injury requiring diagnosis or treatment by a medical practitioner and (2) no present complaint of knee pain during activity or at rest. Additional inclusion criteria for the symptomatic (SYMP) group were (1) a diagnosis of patellofemoral joint dysfunction (Tab. 1), (2) a report of anterior knee pain associated with activity or prolonged sitting (eg, "movie-goer’s sign"), and (3) no history of knee surgery within 1 year prior to participation in this study.

All of the subjects in the SYMP group reported activity-related anterior knee pain of at least 1 year’s duration, and five reported pain while sitting. Three subjects in the SYMP group reported an onset of PFPS following surgery for meniscal or ligamentous injury. Two of the conditions were the result of direct trauma, and the remainder of the subjects in this group reported an onset of PFPS related to sporting or physical recreation activities.

Both knees were tested on all subjects, but for subjects with only one symptomatic knee, data for the contralateral asymptomatic knee were not included in the analyses. Thus, the 24 data sets for the SYMP group were obtained from 9 subjects with bilateral PFPS and 6 subjects with unilateral PFPS, and the 24 data sets for the ASYMP group were from 12 subjects with no history of knee problems. Complete descriptive statistics for both groups are provided in Table 2.

**Data Collection**

After arrival at the laboratory, subjects provided a verbal history of any medical problems affecting the lower extremities, and anthropometric data were recorded. After preparing the skin by shaving (when necessary) and cleansing with isopropyl alcohol, pairs of bipolar, silver/silver chloride EMG electrodes (8 mm diameter; 12 mm interelectrode distance) with on-site preamplifiers (gain=55±10%) were applied to the distal VL and VMO using double-stick adhesive tape. Electrode placement was standardized by having each subject actively extend the knee maximally and placing the inferior edge of the preamplifier casing at the most distal palpable portion of each muscle belly. Electrode pairs were aligned along the longitudinal axis of the muscle fibers by orienting the electrodes oblique to the shaft of the femur at angles of 50 and 15 degrees for the VMO and VL, respectively.\(^{3,31}\) In addition to the EMG electrodes, a small piece of flexible, plastic-backed copper foil was taped

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**Table 1. Frequency Distribution of Diagnoses for the Symptomatic Subject Group**

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>No. of Subjects</th>
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<tbody>
<tr>
<td>Extensor mechanism malalignment</td>
<td>7</td>
</tr>
<tr>
<td>Subluxating or dislocating patellae</td>
<td>7</td>
</tr>
<tr>
<td>Chondromalacia patellae</td>
<td>5</td>
</tr>
<tr>
<td>VMO(^*) insufficiency</td>
<td>4</td>
</tr>
<tr>
<td>Medial synovial fringe entrapment</td>
<td>1</td>
</tr>
</tbody>
</table>

\(^*VMO =\) vastus medialis oblique muscle.
over the patellar tendon to serve as part of a contact switch for triggering data collection during reflex testing.

Data were collected under three different conditions: (1) reflex activity of the knee extensors elicited by a patellar tendon tap (RFLX), (2) active knee extension while NWB, and (3) active knee extension while WB. For the RFLX and NWB conditions, subjects were seated on a table with the knee flexed to 90 degrees, and the test conditions were presented alternately until four repetitions of each were completed. During RFLX tests, subjects were instructed to close their eyes and remain relaxed.

During NWB tests, subjects were told to remain relaxed until a verbal command was given by one of the investigators, at which time they were to actively perform a brisk, small-amplitude knee extension movement similar to that elicited by the patellar tendon tap. To avoid displacing the EMG electrodes or the tendon tapping device by changing subject position, the four repetitions of the WB test were performed following the seated RFLX and NWB tests. For the WB tests, each subject performed a lateral step-up onto a 8-cm step. Prior to each WB trial, the subject was instructed to place the leg to be tested on the step and to relax that leg as much as possible by bearing weight on the opposite lower extremity. The 8-cm step height resulted in an initial position of approximately 40 degrees of hip and knee flexion for most subjects. On verbal command from one of the investigators, the subject would then step up by actively extending the knee of the test leg. The subject was allowed to practice the voluntary tasks as needed prior to the initiation of data collection, and the investigators monitored EMG activity on-line using a dual-channel oscilloscope to ensure that the subject was relaxed prior to initiation of either reflex or voluntary activity.

A custom-designed pendulum device was used to elicit the patellar reflex and to trigger data collection during the reflex tests. Once adjusted to the subject, the metal crossbar of the pendulum could be used to deliver a tap that was repeatable in location and magnitude by dropping the pendulum from a standard height (typically 5 cm from the patellar tendon). In addition, this device provided a digital trigger that initiated sampling by the data-collection computer at the instant when the metal crossbar initially contacted the copper foil overlaying the patellar tendon. During the voluntary extension conditions (NWB and WB), the data-collection computer was manually triggered by the investigator coincident with the verbal cue given to the subject. A 1-second sampling window was used for all three conditions, and the subject was informed that the voluntary tasks were not reaction time tasks, but that the subject merely needed to initiate the movement within the 1-second window following the verbal starting cue.

Electromyographic signals from the preamplifiers were fed to a GCS 67 multichannel EMG amplifier,* where they were high-pass filtered (cutoff=40 Hz) and amplified (gain=500–2,000, as appropriate) before being sampled at a rate of 4,000 Hz per channel by a WATSCOPE® 12-bit analog-to-digital converter and stored for off-line analysis.

### Data Analysis

Onsets of initial EMG activity in the VMO and VL were determined using an interactive computer graphics program developed by the principal investigator and based on the criteria suggested by Walter.32 For each subject, an ensemble average of the digitally rectified EMG data from the four trials under a given condition was created, and a computer algorithm was used to estimate the time of initial EMG activity in each muscle based on the point in the record at which the EMG amplitude exceeded a threshold value based on the standard deviation of the mean resting baseline amplitude as measured during the first 15 milliseconds of the 1-second sampling window. Based on the computer estimate of the first EMG onset, the EMG data were displayed along with vertical cursors indicating the computer-estimated EMG onsets. The time frame of the data displayed was limited to a 160-millisecond window beginning 50 milliseconds prior to first EMG activity, thus allowing the screen resolution to match the actual sampling resolution of 0.25 milliseconds. The program also allowed for manual movement of the cursors in cases where the computer-estimated onset was clearly inappropriate due to low-level activity well in advance of the EMG bursts associated with movement onset.32 Such manual correction was necessary in approximately 20% of the cases under WB and NWB conditions, but was virtually never required for reflex latency determination due to the consistency and clarity of the EMG data under the reflex condition (Fig. 1).

Because data collection for the RFLX trials was triggered by the tendon tap itself, the ensemble averaging of the

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A reflex condition, none of the 16 data sets required manual correction of the computer-estimated onsets, so the mean test-retest difference was zero and the ICC(3,1) was 1.00. For the NWB and WB conditions, the ICC(3,1) was .99 and .87, respectively, and the mean absolute difference in test-retest OTD values was 0.266 milliseconds for the NWB condition and 1.063 milliseconds for the WB condition.

Comparisons of OTD values between SYMP and ASYMP groups were carried out using independent-sample $t$ tests and, in cases where the assumption of a normal distribution was violated ($P<.05$ for the Komogorov-Smirnov normality test), Mann-Whitney rank-sum tests. Pearson product-moment correlation coefficients were calculated to test for the expected correlation between subject height and absolute reflex latency. The relationship between relative onset timing of the VMO and VL under the three test conditions (RFLX, NWB, and WB) was examined using a repeated-measures ANOVA to test for differences across conditions and by calculating correlation coefficients to determine whether OTD values under reflex conditions were associated with similar differences during active extension under WB and NWB conditions. All statistical analyses were carried out using SigmaStat, version 1.01.

**Results**

For both SYMP and ASYMP groups, absolute reflex latencies for the VMO and VL ranged from 21 to 30 milliseconds and were strongly correlated with subject height ($r=.86$ for VMO and $r=.81$ for Vl; $P<.001$ for both).

Table 3 provides complete descriptive statistics regarding absolute reflex latencies for both groups. The scatter-plot in Figure 2 illustrates the correlation between absolute VMO reflex latency and subject height for both groups. In marked contrast to the correlation between height and absolute reflex latency, correlations between OTD and height were not statistically significant under all three conditions ($r=-.01$, $r=-.1$, and $r=.1$ respectively).
Table 3. Absolute Reflex Latencies for the Vastus Medialis Oblique (VMO) and Vastus Lateralis (VL) Muscles in Response to Patellar Tendon Tap

<table>
<thead>
<tr>
<th>Group</th>
<th>Muscle</th>
<th>Reflex Latency (ms)</th>
<th>X</th>
<th>SD</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Asymptomatic</td>
<td>VMO</td>
<td>25.95</td>
<td>1.57</td>
<td>22.25-29.5</td>
<td></td>
</tr>
<tr>
<td></td>
<td>VL</td>
<td>25.76</td>
<td>1.75</td>
<td>21.25-28.75</td>
<td></td>
</tr>
<tr>
<td>Symptomatic</td>
<td>VMO</td>
<td>25.95</td>
<td>1.94</td>
<td>21.75-29.75</td>
<td></td>
</tr>
<tr>
<td></td>
<td>VL</td>
<td>25.89</td>
<td>1.97</td>
<td>22.0-29.5</td>
<td></td>
</tr>
</tbody>
</table>

The OTD of initial EMG activity in the VL and VMO was quantified under each of the three conditions (RFLX, NWB, and WB). For the RFLX condition, the OTD (X±SD) was 0.01±0.44 milliseconds for the SYMP group and -0.19±0.52 milliseconds for the ASYMP group. A two-tailed, independent-samples t test revealed no significant difference between groups (P>.16) and yielded a high power value (power=.86 for α=.05). Under the NWB condition, SYMP and ASYMP group OTD values were -0.29±3.61 and 1.34±3.48 milliseconds, respectively, whereas the corresponding OTD values for the WB condition were 0.72±2.78 and 2.41±7.05 milliseconds. Because the data for these two conditions violated the normality assumption (P<.05, Komogorov-Smirnov normality test), Mann-Whitney rank-sum tests were applied as a nonparametric alternative to the t test. Again, there were no statistically significant differences between SYMP and ASYMP groups under either voluntary condition (P=.44 and P=.28 for NWB and WB, respectively). A graphical summary of OTD results for both groups under all three conditions is presented in Figure 3.

A histogram is presented in Figure 4 to better illustrate the distribution of voluntary OTD values for the SYMP group during WB and NWB active knee extension. This figure illustrates that the onset of EMG activity in the VMO and VL differed by less than 5 milliseconds for almost all subjects in the SYMP group, and that the distribution of OTD values was quite similar under WB and NWB conditions.

In order to examine the possibility that OTD values obtained under RFLX conditions might be related to OTD values under other conditions, we computed Pearson product-moment correlation coefficients using data from all subjects. As shown in Table 4, there were no significant correlations between reflex OTD values and those obtained under either of the voluntary knee extension conditions. Comparison of OTDs across test conditions (Friedman repeated-measures ANOVA on ranks) revealed a significant difference (P<.001), with post hoc multiple comparisons (Student-Newman-Keuls method) indicating that significant differences (P<.05) in OTD existed between the RFLX condition and both voluntary conditions (WB and NWB), but not when comparing WB and NWB conditions.

Discussion

Comparison of Reflex Findings With Previous Studies

Absolute reflex latencies. We have provided data regarding the absolute reflex latencies of the VMO and VL in order to make two points. First, the fact that the means and ranges of our absolute reflex latency measurements are very comparable to those reported...
in a number of previous publications\textsuperscript{24–29} supports the credibility of our technique. Second, the demonstration that subject height is strongly correlated with absolute reflex latencies, although not correlated with the OTD, supports our contention that the OTD (rather than absolute reflex latency) is the appropriate variable to be used to test for PFPS-related timing differences.

**Relative reflex latencies.** The primary purposes of our study were to quantify any differences in the relative onset timing of reflex-elicited VMO and VL EMG activity and to test for differences in timing related to the presence of PFPS. Two major conclusions emerge from our analysis of the results regarding reflex latencies of the VMO and VL. First, no differences existed between the two groups (ASYMP versus SYMP) with respect to the relative reflex latencies of the VMO and VL. Second, regardless of the presence or absence of PFPS, any differences in VMO and VL reflex latencies were extremely small, with the mean difference being less than 0.1 millisecond and the absolute maximum difference observed being only 1.25 milliseconds. Our findings are in disagreement with those of Voight and Wieder.\textsuperscript{11} The following discussion provides a detailed comparison of these two studies and addresses possible causes for the contradictory findings.

Voight and Wieder\textsuperscript{11} describe three types of statistical comparisons to support their conclusion that differences in reflex latency exist between groups of asymptomatic subjects and patients with PFPS. The first type of comparison reported was a within-group, paired t-test comparison of absolute VMO and VL latencies, not a comparison between groups. Of the two measures that provide a comparison between groups, the first measure reported by Voight and Wieder was a chi-square analysis of that demonstrated a dependency between the muscle firing order and the type of

### Table 4. Pearson Product-Moment Correlation Coefficients for Onset Timing Differences Between Reflex Extension (RFLX) and Active Extension Under Non-weight-bearing (NWB) and Weight-bearing (WB) Extension Conditions

<table>
<thead>
<tr>
<th>Condition</th>
<th>NWB</th>
<th>WB</th>
</tr>
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<tbody>
<tr>
<td>RFLX</td>
<td></td>
<td></td>
</tr>
<tr>
<td>n</td>
<td>48</td>
<td>48</td>
</tr>
<tr>
<td>(r)</td>
<td>.50 (NS)</td>
<td>.55 (NS)</td>
</tr>
</tbody>
</table>

*NS = not significant.
subject.119123 We do not believe this approach to be optimal because it takes ratio data (time) and reduces it to nominal data (category), thus resulting in a loss of information contained in the original data.35

The other between-group type of comparison Voight and Wieder reported was a comparison of absolute latency values for each muscle using independent-sample t tests. Based on those tests, they reported that "the patients demonstrated a significantly faster vastus lateralis response time than the normal subjects,"119131 with no statistically significant difference between groups for the VMO latencies. Although this is an appropriate statistical procedure for between-group comparisons, the variable chosen for comparison (absolute reflex latency) is problematic. Because most of the reflex latency is due to time required for nerve conduction in the afferent and efferent components of the reflex loop, absolute reflex response times are strongly related to subject height.21-23 Thus, if the groups differed in mean height, as seems likely given the markedly unequal distribution of male and female subjects in the two groups (male:female ratios of 17:24 in the asymptomatic subject group versus 10:6 in the patient group), we would expect the group with shorter subjects to exhibit faster responses in both VMO and VL. Finally, it is difficult to accurately assess the magnitude of any latency differences in the Voight and Wieder study as no means or standard deviations were reported. Based on the frequency distributions depicted in Figures 3 and 4 of their article, however, it would appear that despite reaching statistical significance, the mean differences must have been quite small, similar to our results.

In our study, we not only attempted to match the groups in terms of gender and anthropometric characteristics, but we completely avoided the possibility of such confounding factors by making our between-groups comparison based on the difference between VL and VMO onset latencies for each subject, rather than on absolute reflex latency measurements. The independent-samples t test comparing reflex OTD values between the ASYM and SYMP groups demonstrated no significant difference between groups and good statistical power, indicating a low possibility that this negative finding was the result of a Type II statistical error. Although we believe that our analysis approach was the appropriate one, we reanalyzed our data using the methods described by Voight and Wieder11 in order to determine whether the disparate conclusions of the two studies were simply due to different statistical testing procedures. This reanalysis of our data using the methods described by Voight and Wieder did not result in any statistically significant results, such as those they reported. Thus, it does not appear that the differing conclusions regarding reflex latencies in these two studies are merely due to differences in data analysis.

A more likely explanation for the contradictory findings lies in the differences in the data-collection procedures used in the reflex testing portion of the two studies. In our study, we used a pendulum device to provide a standardized tendon tap that triggered computerized data collection at a rate of 4,000 Hz, thus providing temporal resolution of 0.25 milliseconds for computer-aided, off-line determination of EMG onsets. This procedure resulted in highly reproducible reflex EMG activity (Fig. 1) with clearly defined onsets. Furthermore, the absolute reflex latencies recorded using these methods compare well with previous studies of patellar tendon reflexes24-29 and are highly correlated with subject height, as would be expected because reflex latency is primarily due to nerve conduction time and is thus proportional to the length of the afferent and efferent neural paths.

In the study by Voight and Wieder,11 the reflex was elicited with a manual reflex hammer, which triggered a single sweep of a dual-channel oscilloscope from which the latencies of VMO and VL were determined visually. The authors stated that the latencies were "recorded in milliseconds,"119131 but they did not give any indication of what degree of temporal resolution was actually possible. Even if the actual resolution was 1.0 millisecond, our data suggest it would likely be inadequate, because only 4 of the 96 tests of voluntary knee extension conducted in our study resulted in VL-VMO reflex latency differences of 1 millisecond or more. Furthermore, the data presented in Figures 3 and 4 of the Voight and Wieder's article indicate that they recorded reflex latencies well below 10 milliseconds in a number of subjects in both of their groups. Based on the results of previous patellar reflex studies24-29 and on the upper limits of peripheral nerve conduction velocity, such short latencies are clearly unphysiological, suggesting that mechanical artifacts in the EMG record or problems related to the data-collection trigger may have introduced error in those data. Thus, we believe that the contradictory conclusions reached in these two studies were related to data-collection methodology, and furthermore that the more refined methodology used in our study provided reliable data on VMO and VL reflex latencies. The remaining questions, regarding VL-VMO latencies during voluntary activity and the relationship between relative latencies measured under reflex and voluntary conditions, were not addressed in the study by Voight and Wieder, but were addressed in our study, as discussed in the next section.

Relative Timing of VMO and VL EMG Onsets During Voluntary Activity

Comparison of relative timing under reflex and voluntary conditions. Another issue addressed by our study concerns whether VL-VMO latency differences under reflex conditions are associated with similar latency differences during voluntary activation of these muscle groups. This issue is an important one, because the interpretation of Voight and Wieder's results11 as being indicative of a motor control problem11,16 relies on the implicit assumption that a reflex timing
difference between the VMO and VL is associated with a similar timing disparity during voluntary activation of the knee extensors. Furthermore, the possible use of reflex latency testing as an assessment tool for patients with PFPS depends on the existence of a strong relationship between OTD values measured under reflex and voluntary conditions. If such a relationship exists, reflex latency testing might provide a reliable and relatively simple means of assessing the efficacy of treatment regimens for PFPS.

Our results indicate that for both groups, there was a tendency for a greater OTD value (ie, a relatively earlier VMO onset) during voluntary knee extensor activity than during reflex activity, with no difference in relative onset timing between the WB and NWB conditions. We considered the possibility that the small differences in OTD observed under reflex conditions might somehow be magnified during voluntary activity, in which case it is possible that reflex OTD values might still be related to voluntary timing differences despite the differences in OTD magnitude between the reflex and voluntary conditions. To rule out that possibility, we assessed the correlation coefficients between VL-VMO latency differences under reflex conditions and both WB and NWB active knee extension conditions. As indicated in Table 4, the Pearson product-moment correlation coefficients for all subjects in both voluntary and reflex conditions were extremely small and not statistically significant for these comparisons. This lack of association between the relative timing of VMO and VL onsets during reflex and voluntary activities suggests that such reflex testing is not a good indicator of the relative timing of muscle activity during voluntary activity, and thus is not likely to be useful for assessment of PFPS.

**Comparison of voluntary onset timing differences in subjects with and without PFPS.** Although numerous authors have speculated that timing differences between VMO and VL onsets during voluntary activity might be a cause of PFPS, we were unable to find any published data directly addressing this possibility. We examined this issue using much the same approach used in the reflex latency testing discussed earlier. The primary conclusion reached was that no difference in OTD existed between the ASYMP and SYMP groups during voluntary knee extension under either the WB or NWB condition. The variability in these data, however, was considerably greater than in the reflex data, and because the data failed to meet normal distribution criteria, we were compelled to report nonparametric statistical comparisons for the voluntary OTD data (though the conclusions based on t tests were identical to those based on the Mann-Whitney rank-sum tests). In comparison with the reflex OTD results, the voluntary OTD tests had relatively low statistical power, suggesting that the lack of statistical significance in between the SYMP and ASYMP groups during voluntary activity should be viewed with caution.

Given the greater variability of the OTD under voluntary conditions and the possibility of a Type II statistical error, a primary question regarding the voluntary OTD in patients with PFPS is whether the magnitude of any timing difference would be large enough to be detectable using clinical EMG biofeedback devices. This question is important because of the widespread acceptance among physical therapists of the use of clinical EMG biofeedback devices in attempts to teach patients to voluntarily alter the relative timing of the VMO and VL. To our knowledge, the data presented here are the first to allow objective evaluation of the theoretical basis for this use of EMG biofeedback. Grabiner and associates, in a recent review of literature, cited unpublished observations regarding the relative onset timing of the VMO and VL during isokinetic knee extension in asymptomatic subjects. Their report of a mean difference of 5.6 milliseconds (VMO first) is similar to the differences reported here for WB and NWB extension, and they concluded that “although the difference was statistically significant, the functional significance of this earlier activation may be challenged.”

In order for a patient with PFPS to use EMG biofeedback information to modify the relative timing of VMO and VL onsets, both the biofeedback device and the patient’s sensory system must have sufficient temporal resolution to detect the OTD. As shown in Figure 4, our data demonstrate that for most SYMP group subjects the onset of activity in the VMO and VL during voluntary activity differed by less than 5 milliseconds, with the maximum difference observed being only 13 milliseconds. Though we have not done an exhaustive review of the technical specifications of all available clinical biofeedback devices, we are unaware of any clinical EMG biofeedback devices that are capable of the temporal resolution necessary to detect such small OTDs. More importantly, data regarding the ability to discriminate the relative onsets of two closely timed sensory cues indicate that even if the feedback provided to a patient has infinite resolution, the human sensory system is unable to resolve such small time differences.

In a recent study by Artieda et al., which compared subjects with Parkinson’s disease with asymptomatic control subjects, the visual discrimination threshold (the minimal detectable difference in time of presentation of two visual cues) was determined by having the subjects determine whether two light-emitting diodes came on simultaneously or separately. They found that for the control subjects, the minimum detectable difference in the onset timing of the two lights averaged 68.7 milliseconds, an interval that is much greater than any of the OTD values that we observed in either group under any of the three test conditions. Thus, it seems unlikely that visual feedback in the form of light bars, analog meters, or even raw EMG signals on an oscilloscope screen would allow the observer to detect in real time the small differences in VMO and VL onset timing that we measured in our study.
Limitations of Our Study

Although the inclusion criteria for the SYMP group were designed to recruit a typical sample of patients with PFPS who might be seen by physical therapists, the relatively small sample size (15 symptomatic subjects with a total of 24 symptomatic knees) might limit the generalizability of the findings presented here. We saw no indication in our data that there were subgroups within the SYMP group (eg, subjects with diagnoses of “VMO insufficiency” versus subjects with diagnoses of “extensor mechanism malalignment”) that demonstrated differing EMG responses. To completely rule out that possibility, however, would require a study with a larger sample and inclusion of diagnostic subgroups as a covariant in the analyses.

Additional possible limitations of this study relate to the identification of EMG onsets and possible variability in the subjects’ responses under the voluntary conditions. The difficulties inherent in determinations of EMG onsets have been discussed by other authors,32,35 and the suggestion has been made that computer algorithms, used either alone35 or in an interactive mode,32 enhance the reliability of onset detection. Because the interactive computer algorithm used in our study for detecting EMG onsets was not 100% successful in determining onsets under the WB and NWB conditions, the possibility exists that experimenter bias or unreliability might affect the OTD determination for those conditions. We attempted to minimize these possibilities by having all computer-assisted OTD determinations made by one investigator (GMK) who has had considerable prior experience in such analyses.36,37 Finally, because knee extension velocity was not tightly controlled under the WB and NWB conditions, we cannot exclude the possibility that this factor could influence the findings, though we know of no published evidence suggesting that the relative timing of VMO and VL onset varies with knee angular velocity (or, for that matter, with any other kinematic or kinetic variable).

Clinical Implications

Our data regarding relative reflex latencies of the VMO and VL indicate that it is unlikely that such reflex testing is useful for screening for PFPS or as a means of ongoing assessment of treatment efficacy for this problem. With respect to the OTD during voluntary knee extension, these results do not completely rule out the possibility that there might be small differences (eg, a few milliseconds) in onset timing between symptomatic and asymptomatic subjects, but the magnitude of such differences was, in all cases studied here, too small to be detected with typical EMG biofeedback methods.

Clinical EMG biofeedback devices typically use linear envelope detectors with long time constants and analog-to-digital converters with slow sampling rates, thus severely limiting the temporal resolution of the EMG feedback and making them inappropriate for attempting to detect onset timing differences in the range of those observed in our study.38,39 Furthermore, because of these characteristics, differences in the absolute amplitude of the EMG activity from two muscles activated at precisely the same time could be misinterpreted as a timing difference simply because the larger-amplitude signal reaches the threshold for initiation of the feedback signal (ie, moving the meter or turning on the light-emitting diode) sooner. This confounding effect of signal amplitude is exacerbated by the fact that the absolute amplitude of surface EMG signals is affected not only by the degree of motor unit activity but by many other factors, including electrode orientation and spacing, skin-electrode interface, and amount of subcutaneous fat underlying the electrodes. We believe that EMG biofeedback is an important clinical tool with a number of legitimate uses, but therapists should be aware of the capabilities and limitations of EMG biofeedback, particularly when applying it to problems such as the one addressed in our study.

Conclusions

The data presented here provide strong evidence that there is no significant difference in the relative timing of reflex-elicited VMO and VL EMG activity when comparing asymptomatic subjects and patients with PFPS. Similarly, there were no timing differences between groups during voluntary knee extension under WB and NWB conditions, and the magnitude of VL-VMO timing differences was in all cases very small in relation to the temporal resolution of clinical EMG biofeedback devices and in relation to the ability of the human sensory system to discriminate the onset of two closely timed feedback signals. Relative VL-VMO timing under reflex conditions was not associated with the relative timing during voluntary conditions. For both SYMP and ASYMP groups, VMO and VL onsets were essentially simultaneous during reflex testing, although the VMO tended to be activated earlier during voluntary knee extension as compared with reflex extension. These results do not support the conjecture that altered timing of VMO and VL activity plays a role in initiating or perpetuating PFPS. In addition, these findings emphasize the need for greater awareness of the capabilities and limitations of EMG biofeedback devices with respect to their use in assessing or treating temporal aspects of muscle activity patterns.

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References


