In vivo and Noninvasive Load Sharing among the Vasti in Patellar Malalignment

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ABSTRACT

MAKHSOUS, M., F. LIN, J. L. KOH, G. W. NUBER, and L. ZHANG. In vivo and Noninvasive Load Sharing among the Vasti in Patellar Malalignment. Med. Sci. Sports Exerc., Vol. 36, No. 10, pp. 1768–1775, 2004. Purpose: It is not clear how the knee extension torque is distributed quantitatively among the lateral and medial vasti in patellofemoral pain (PFP) patients with patellar malalignment, which was investigated in vivo and noninvasively in ten PFP patients and eleven controls. We hypothesized that the vastus medialis oblique (VMO) and vastus medialis longus (VML) of PFP patients contribute less to knee extension than that in controls. Methods: Electrical stimulation was used to activate each vastus component selectively. The relationship between the knee extension torque generated by each individual vastus component and the corresponding compound muscle action potential (M-wave) was established over different contraction levels, which was used to calibrate the corresponding voluntary EMG signal and determine torque ratios of VMO/VL (vastus lateralis), VMO/VML, VML/VL and (VMO+VML)/VL during voluntary isometric knee extension. Results: The VMO and VML of PFP patients contributed significantly less to knee extension than their counterparts in controls. The combination of VMO and VML generated comparable amount of extension torque as the VL in the controls, while it produced significantly lower extension torque than that of the VL in the PFP patients. In addition, the VMO/VL was lower than VMO/VML and VML/VL in both PFP and control groups. Conclusions: Compared to controls, the VMO and VML in the PFP patients contributed significantly less to the knee extension torque. The approach can be used to investigate load sharing among quadriceps muscles in vivo and noninvasively, in both healthy subjects and patients with patellofemoral disorder and patellar malalignment. Key Words: QUADRICEPS, KNEE, STIMULATION, TORQUE RATIO, M-WAVE

Patellofemoral pain (PFP) is a common, painful musculoskeletal condition that affects both adults and adolescents, especially the physically active (43). Biomechanical analyses suggest that normal alignment and function of the patellofemoral joint depend on appropriate balance of forces exerted on the patella by the medial and lateral vasti and passive structures. Imbalanced torques generated by the lateral and medial vasti (VMO, VML and VL) are thought to be an important etiological factor in PFP with patellar malalignment and maltracking in the femoral groove (26,15,29). The net force on the patella in PFP patients may be directed too laterally, resulting in predisposition to lateral subluxation or dislocation of the patella (29). Imbalanced torque distribution (load sharing) among the VMO, VML and VL in PFP patients is considered related to the tightness of the lateral structures (13), changes in the Q-angle (31), and muscle fiber orientation and attachment (23). However, there is a lack of quantitative information on load sharing among medial and lateral vasti in the PFP patients.

The relative contributions of the quadriceps components have been evaluated using several methods including EMG-based approaches (1,2,29,33,35,40), cadaver-based experiments (15,26), and computer modeling (17). Goh et al. (15) used the cross-sectional areas of muscles to estimate the force ratios, which could vary several folds even within normal anatomical variations (4) and only gave potential force generating capacity (38). By implanting force transducers into the muscle tendons of elbow flexors of small monkeys (24) and triceps surae muscles in cats (14), load sharing has been evaluated in vivo. However, the invasive procedure made it impractical to implant a force transducer into each of the quadriceps components of human subjects. Recently, we investigated in vivo load sharing among the VL, vastus intermedius (VI), rectus femoris, and vastus medialis (VM) components in healthy subjects and found that the different quadriceps components contributed differently to total knee extension torque depending on the total knee extension torque.

The purpose of this study was to determine, in vivo and noninvasively, torque distribution (torque ratios) among the VMO, VML and VL during isometric voluntary knee extension in PFP patients and normal controls. It was hypothesized that the VMO and VML of the PFP patients contribute less to knee extension than their counterparts in normal controls. It was also hypothesized that the medial portion of the quadriceps, that is, the combination of the VMO and
FIGURE 1—Experimental setup: The subject was seated and a short-leg (ankle and distal leg) cast was fixed to the aluminum beam through two half-rings that could be adjusted and locked in multiple degrees of freedom for appropriate alignment. The seat was adjusted to align the knee flexion axis with the z-axis of the six-axis force sensor at 60° knee flexion. The knee was clamped at the femoral condyles. The thigh, hip and trunk were strapped to the seat using seat-belts to obtain a stable position during the experiment. The target and actual torques were displayed on the motor.

VML (VMO+VML), generates roughly the same amount of knee extension torque as the VL in normal subjects but contributes much less to knee extension than the VL in PFP patients. Quantitative evaluation of torque distribution among the medial and lateral vasti in the PFP and normal groups will help us better understand the mechanisms underlying PFP and evaluate treatment outcome more accurately.

METHODS

Eleven healthy subjects (age: 33.2 ± 7.5 yr; mass: 74.9 ± 12.9 kg; height: 172.9 ± 8.2 cm; Q-angle: 15.0 ± 2.7°; 7 males and 4 females) and 10 PFP patients (age: 29.1 ± 5.3 yr; mass: 64.5 ± 15.2 kg; height: 167.3 ± 11.0 cm; Q-angle: 18.7 ± 3.9°; all females) participated in the study. All the PFP subjects were diagnosed as having PFP by physicians involved in this study. They experienced patellofemoral pain during functional activities and had lateral patellar malalignment. It was measured by sunrise or Merchant view radiographs. One of the subjects had prior history of patellar lateral subluxation with subsequent therapeutic treatment, while none of participating PFP patients had received treatment within 6 months before the study. The study was approved by the Institutional Review Board of Northwestern University and informed consent was given by each subject in writing before the experiment.

Experimental setup

After the Q-angle measurement (bilateral weight bearing in standing) was taken, the subject was seated upright with the knee at 60° flexion (Fig. 1). The ankle and distal leg were fixed to the distal end of an aluminum beam located on the lateral side of the leg using a fiberglass-tape cast. The proximal end of the beam was mounted onto a six-axis force sensor (JR3 Inc., Woodland, CA, U.S.) (Fig. 1). The chair was adjusted in multiple degrees of freedom (proximal-distal, medial-lateral, anterior-posterior of the leg and rotation about the tibial long axis) to achieve appropriate alignment of the knee and force sensor, with the Z-axis of the six-axis force sensor aligned with the knee flexion axis. The knee was clamped at the femoral condyles and the thigh, hip and trunk were strapped to the seat using seat-belts to ensure a stable position during experiment.

Selective activation of individual muscle

The skin was cleaned with alcohol pads before applications of the electrodes. As suggested by a number of investigators (36,6,33), the VMO, VML and VL can be differentiated by their respective anatomic angle relative to the long axis of the femur (VMO: 45°–55°, VML: 10°–20°, and VL: 35°–45°) and through palpation during successive cycles of isometric contractions and relaxations. Each muscle was marked with a centerline passing through the tendon and along the belly of the muscles. The VMO, VML or VL was activated selectively (one component at a time) using a four-channel custom-made electrical stimulator controlled by a PC (44). Bipolar stimulation was used due to its localized current flow and selective activation. A pair of surface electrodes with 1-cm-diameter conducting disks and about 3-cm (adjustable) interval between two disks was used to deliver constant-current stimulation. The electrodes were initially placed on the marked line on the muscle and moved around on the muscle belly to search for the motor point where the strongest contraction was induced by the stimulation, as determined by visual inspection, palpation of the corresponding quadriceps tendon, subject’s feeling, and/or the induced torque (44). If needed, the interval between the two electrodes and the stimulation current amplitude was adjusted in order to find the motor point with the strongest contraction. Conducting gel (Parker Lab, Inc., New Jersey) was used to provide a good contact between the electrodes and skin. Each quadriceps component was activated using a train of stimulation pulses. The stimulation pulse (width 0.3 ms) was repeated at 25 pulses per second. The duration of the stimulation pulse train was 600 ms (Fig. 2), which resulted in 15 stimulation pulses. The evoked M-wave from each of the muscles was recorded through a pair of SOFT-E™ monitoring surface electrodes (Kendall-LTP, Chicopee, MA, U.S.) together with the isometric knee extension torque.

An electronic device was custom developed to record both the compound muscular action potential (M-wave) and voluntary EMG signals (44). Since the stimulation artifact during the constant current stimulation was much higher in amplitude than the M-wave, a special circuit was designed to minimize the artifact during the delivery of stimulation pulses. A computer was used to control the stimulator and the custom-made M-wave recording device. M-waves were recorded at both the targeted muscle being stimulated and surrounding muscles to monitor potential current overflow. If significant M-wave was observed only from the targeted
muscle but not the others, it would indicate that the targeted muscle was activated selectively and the knee extension torque was generated primarily by the targeted head of muscle (44).

Protocol

The M-waves and knee extension torque were recorded at 2 kHz after low-pass filtering (900-Hz cutoff). Moments and forces at the knee were recorded using the six-axis force sensor. The stimulation and recording procedure was randomly repeated for VMO, VML and VL muscles. The amplitude of the constant-current stimulation was adjusted systematically across trials to activate each muscle over a range of contraction (44). It was started at very low current systematically across trials to activate each muscle over a range of contraction (44). It was started at very low current

A linear relationship between the peak-to-peak M-wave amplitude and peak knee extension torque was characterized by the slope K_i (i = VMO, VML, VL) (Fig. 3). This relationship was used to calibrate the corresponding voluntary EMG signals for the individual heads of the quadriceps muscle. Since the same electrodes were used to record both the M-wave and the voluntary EMG signals for each muscle, electrode location, size and material properties, etc., were matched between the M-wave and EMG signals for each muscle. The individual muscle’s torque–M-wave relationship established in the stimulation experiment could thus be used to calibrate the corresponding voluntary EMG signal. Similar to the linear relationship between the joint torque and M-wave amplitude of each muscle established through electrical stimulation, it was assumed that the relationship between the mean EMG linear envelope of a muscle and the mean joint torque generated by the muscle within a window of steady contraction during the isometric and submaximal voluntary contraction was in a linear pattern and characterized by the slope G_i (i = VMO, VML, VL) (8,10,18,34). Furthermore, since the same electrodes were used to record the M-wave and EMG signals during the stimulation-induced and voluntary contractions during knee extension, it was assumed that (44):

$$\frac{G_{\text{VMO}}}{K_{\text{VMO}}} = \frac{G_{\text{VML}}}{K_{\text{VML}}} = \frac{G_{\text{VL}}}{K_{\text{VL}}} = C$$

The corresponding EMG signals during voluntary contractions were calibrated with these M-wave–torque relationships (Eq. (1)). The torque ratios of VMO/VL, VMO/VML, VML/VL and (VMO+VML)/VL were then determined as follows:

![FIGURE 2—Representative M-waves recording during stimulation of the VMO. Top: A train of stimulation pulses elicited a series of M-waves in the VMO. The stimulation pulse (0.3-ms width) was repeated at 25 pulses per second. Duration of the stimulation pulse train was 600 ms. Bottom: Multiple M-waves corresponding to the train of stimulation pulses were aligned and averaged to calculate the mean M-wave signals and the peak-to-peak amplitude.](image-url)
EMG was the mean amplitude of EMG linear envelope generated by the ith muscle (i = VMO, VML, VL) during the steady voluntary target matching period.

The torque ratio and knee extension torque were fit with a second-order polynomial. This relationship was used to fit the data across different torque levels within the generated torque levels for each subject. Mean and standard deviation (mean ± SD) of the torque ratio among the muscles were then calculated across multiple subjects at the increment of 1.0 N-m.

Two-way analysis of variance (ANOVA) with repeated measures (over the different torque levels) was used to test whether each of the torque ratios (VMO/VL, VMO/VML, and VML/VL) were equal to 1 or less than 1, and the test was repeated for the PFP and normal groups. In other words, whether contributions of the different muscles involved (the numerator and denominator of a torque ratio) to the knee extension torque were equal to each other or the muscle in the numerator contributed less than that in the denominator. All statistical analyses were performed with the SAS statistical software package (SAS Institute, Cary, NC). The significance level was set at 0.05.

\[
\frac{VMO}{VL} = \frac{G_{VMO}EMG_{VKO}}{G_{VL}EMG_{VL}} = \frac{C K_{VKO}EMG_{VKO}}{C K_{VL}EMG_{VL}} = \frac{K_{VKO}EMG_{VKO}}{K_{VL}EMG_{VL}}
\]

\[
\frac{VMO}{VML} = \frac{G_{VMO}EMG_{VM}]}{G_{VML}EMG_{VML}} = \frac{C K_{VM]}EMG_{VM}]}{C K_{VML}EMG_{VML}} = \frac{K_{VM]}EMG_{VM]}}{K_{VML}EMG_{VML}}
\]

\[
\frac{VML}{VL} = \frac{G_{VML}EMG_{VM]}}{G_{VL}EMG_{VL}} = \frac{C K_{VM]}EMG_{VM]}}{C K_{VL}EMG_{VL}} = \frac{K_{VM]}EMG_{VM]}}{K_{VL}EMG_{VL}}
\]

\[
\frac{VMO + VML}{VL} = \frac{G_{VMO}EMG_{VM]}}{G_{VL}EMG_{VL}} = \frac{C K_{VM]}EMG_{VM]}}{C K_{VL}EMG_{VL}} = \frac{K_{VM]}EMG_{VM]}}{K_{VL}EMG_{VL}}
\]

\[\text{FIGURE 4}—\text{The torque ratios among the muscles (VMO/VL, VMO/VML, and VML/VL) measured at each voluntary knee extension torque level for both the normal (N = 11) and PFP (N = 10) groups. Mean value and standard error of the mean (mean ± SE) are indicated by the symbols and vertical bars, respectively. A second-order polynomial was used to fit the data for each of the torque ratio. The bars are only given in one direction for visual clarity.}\]
RESULTS

The $R^2$ value for the linear regression fitted between the peak-to-peak M-wave amplitude and peak knee extension torque across various levels of contractions (Fig. 3) was found to be high, indicating a linear relationship between the peak-to-peak M-wave amplitude and peak knee extension torque.

The torque ratios of VMO/VL, VMO/VML and VML/VL measured at each voluntary knee extension torque level in the normal and PFP subjects are shown in Figure 4. The contributions of the VMO and VML to the knee extension torque were less than the VL in both normal and PFP subjects and across all levels of knee extension torque tested since the ratios of VMO/VL and VML/VL were found significantly smaller than one ($P < 0.0001$) for both ratios in normal and PFP groups. Similarly, the contribution of the VMO to knee extension was less than that of the VML since VMO/VML was significantly smaller than one ($P < 0.0001$) in both normal and PFP subjects and at all levels of knee extension torque tested.

The torque ratios of VMO/VL, VMO/VML and VML/VL in the normal controls varied from 0.36 ± 0.07 to 0.47 ± 0.09, from 0.65 ± 0.01 to 0.68 ± 0.06, and from 0.55 ± 0.08 to 0.64 ± 0.07, respectively, in the knee extension torque range of 5 to 45 N·m. The VMO/VL was significantly smaller than the VMO/VML and VML/VL across all levels of the knee extension torque (both with $P < 0.0001$). The torque level was found to have no significant effect on any of the torque ratios.

For the PFP patients, the VMO/VL was significantly smaller than the VMO/VML and VML/VL across all the levels of knee extension torques (both with $P < 0.0001$). The torque ratios of VMO/VL, VMO/VML and VML/VL in the PFP patients varied from 0.11 ± 0.08 to 0.16 ± 0.07, from 0.33 ± 0.12 to 0.36 ± 0.16, and from 0.29 ± 0.14 to 0.43 ± 0.07, respectively, in the knee extension torque range of 5 to 30 N·m. The torque level was found to have no significant effect on any of the torque ratios.

The VMO/VL, VMO/VML and VML/VL torque ratios in the PFP patients were significantly smaller than their counterparts in normal controls, across all levels of knee extension torque tested ($P = 0.0002$, $P < 0.001$, $P = 0.0006$, respectively).

In normal controls, the VMO+VML generated roughly the same amount of knee extension torque as the VL (Fig. 5). The (VMO+VML)/VL varied from 0.91 ± 0.12 to 1.14 ± 0.17 over the knee extension torque range in the normal controls, and it was not significantly different from 1 ($P = 0.825$). In contrast, the (VMO+VML)/VL varied from 0.40 ± 0.11 to 0.59 ± 0.12 in the knee extension torque range in the PFP subjects, and it was significantly smaller than 1 ($P = 0.022$). The torque level was also found to have no significant effect on the (VMO+VML)/VL in both groups. Compared with that of normal controls, the (VMO+VML)/VL in the PFP patients was significantly smaller over all levels of knee extension torque tested ($P < 0.001$).

**DISCUSSION**

An in vivo and noninvasive approach was used to investigate the knee extension torque distributions among the VMO, VML and VL muscles in PFP and control subjects. Compared with their counterparts in control group, the VMO and VML in PFP patients contributed significantly less to the knee extension torque. In the controls, the VMO and VML in combination generated a comparable amount of knee extension torque as the VL did, while in the PFP patients the VMO+VML combination contributed significantly less to the knee extension torque than the VL. The in vivo and noninvasive approach helps us gain insight into the mechanisms underlying PFP and patellar malalignment, and potentially evaluate treatment outcomes more accurately.

It is well-documented that the VMO affects patellofemoral mechanics considerably by stabilizing the patella medially during knee extension, as well as by improving the efficiency of the quadriceps mechanism (26). Our results showing lower VMO/VL and (VMO+VML)/VL torque ratios in the PFP patients suggest altered activation and reduced contribution of the VMO and VML to the knee extension torque. In the PFP patients, the medial quadriceps muscle (VMO) may not be as effective as it is in normal subjects, as indicated by the lower VMO/VL ratio shown in Figure 4. This may interact with other biomechanical factors, such as the tightness of the lateral soft tissues (13) and the malalignment of the patella, and contribute to patellofemoral dysfunctions.

Quadriceps components in normal controls are synergistically activated to extend the knee and to maintain the patella in balance in the femoral trochlea, as indicated by the torque ratio of (VMO+VML)/VL, which was about 1 (Fig.
In contrast, in the PFP patients, the VMO and VML contributed significantly less in generating the knee extension torque as was indicated by the much lower (VMO + VML)/VL torque ratio in the PFP patients. In the same group of PFP patients, we found significantly larger lateral tilt (the patellar rotation about its proximal-distal axis and the lateral edge moves posteriorly) during knee voluntary extension, as compared to their counterparts in normal controls (28,27). The lower contributions of the VMO+VML to knee extension and the excessive lateral patellar tracking in the PFP patients may be related to each other.

The patellofemoral pain made it more difficult for the PFP patients to track a time-varying torque superimposed on a steady level of extension torque for a period of time (43), especially at higher levels of knee extension torque. Therefore, the majority of the PFP patients did not generate knee extension torque as high as that of the normal controls. As a result, the data reported for the PFP group are over the knee extension torque range of 0–30 N-m, which was the common torque range reached by all the PFP patients. In contrast, the torque range for normal controls was 0–45 N-m. Of note is that, for the task of isometric knee extension used in this study, which is a single-joint, single muscle group, and nonweight-bearing task, it was difficult for a subject to track a time-varying torque and maintain a steady level of extension torque as high as the level in squatting and stair climbing, which had a peak value about 75 ~ 250 N-m (11,12,41,39,9). Similar to our finding, Steinkamp, et al. has reported that patients with patellofemoral joint arthritis tolerated leg extension less than that for leg press (41). Furthermore, 30 N-m was the torque level that every PFP patient could perform the target tracking and matching. Some patients actually generated higher than 30 N-m torque. However, for consistent analysis, the commonly reached range of 0~30 N-m was used for the PFP population.

As direct measurement of the vastus muscle force is very difficult, especially in human subjects, usually EMG studies have been used to elucidate mechanisms underlying patellar pain and malalignment (32,16,3,40,29,36,33,35). Previous studies on potentially unbalanced activations between the quadriceps components in PFP patients have controversial results. Moller et al. (32), Grabiner et al. (16) and Boucher et al. (3) found no significant difference in the amplitude of VMO and VL EMG between the normal and PFP groups. Powers et al., (35) found less EMG activity of all vastus muscles in PFP patients compared to normal controls. Mariani (29) found less EMG (nonnormalized) activity of the VM compared with that of the VL in the preoperative patients. Activation of the VMO in PFP patients was found to be less and force rise was slower than in the normal controls (33). Souza and Gross (40) found lower VMO/VL EMG ratio in PFP patients than normal controls using non-normalized EMG.

Normalization of the EMG signals is important in evaluating the amplitude of quadriceps contraction. In prior studies (22,1), EMG normalization was usually done with respect to its corresponding amplitude under maximal voluntary contraction. This method of normalization might be affected by the pathological conditions and was dependent on the motivation and skill of the subjects. In addition, it is difficult for the PFP patients to generate the maximal voluntary contraction, as was observed in our study. The new EMG normalization method used in this study related the EMG of a quadriceps head directly to the corresponding joint torque generated by that head, which may provide potentially more accurate normalization of EMG signals and thus torque determination. Moreover, using the same electrodes to record the M-wave and EMG signals during the stimulation-induced contraction and voluntary knee extension, the substantial variations of the EMG signals associated with factors like the electrode size, shape, location, inter-electrode distance, skin preparation etc. were minimized. The electronic device we developed to reject the stimulation artifact used a blanking period with adjustable duration (44). Even if there were some artifacts left, they only affected the initial phase of the M-wave. For establishing the torque–M-wave relationship, only the peak-to-peak amplitude of the M-wave was used, which occurred several milliseconds after the stimulation. Therefore, the residual artifacts would not affect the validity of the data analysis.

Repeatability of the M-wave recording (Fig. 2) was good during the trains of stimulation pulses in each muscle and subject. The peak knee extension torque increased with the M-wave amplitude, and the relationship between them over the range of contraction levels was approximately linear for each quadriceps component, as corroborated by the high $R^2$ values for each of the muscles (Fig. 3).

In this study, the muscle activity of the patellar medial and lateral stabilizers (VMO, VML and VL) was investigated. The limitation of this study is that the remaining quadriceps components were not evaluated due to the difficulty involved in evaluating the deep muscles. The experiment itself was time consuming, which was especially difficult for the PFP patients.

The 60° knee flexion used in this study was the position with the highest forces in the quadriceps tendons and patellar ligament (21) due to increased tightness of the musculotendon structures of the patella. It was also reported that maximal torque and integrated EMG under isometric conditions occurred at 50° and 70° for males and females, respectively (5). However, further study needs to be done at different joint positions to investigate dependence of torque ratios among VMO, VML and VL on joint position in PFP patients and normal controls.

Although the Q-angle was found significantly larger in PFP patients than that of normal controls, this significance should be interpreted with caution because of the dominance of female PFP patients in this study, considering that female individuals tend to have larger Q-angles (19,20).

It can be concluded that the VMO and VML in the PFP patients contributed significantly less to the knee extension torque as compared to their counterparts in normal
controls. Our study provides an in vivo and noninvasive quantitative method to assess muscular imbalance and to evaluate progress in rehabilitation. It can help develop and evaluate various rehabilitation protocols for PFP patients to achieve balanced vastus contractions, including both conservative techniques like biofeedback (42), electrical stimulation (2), taping (30), active exercise (30,7,6), and surgical interventions (37) such as lateral release or plication of the VMO.

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