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Muscle Force Cannot Be Directly Inferred From Muscle Activation: Illustrated by the Proposed Imbalance of Force Between the Vastus Medialis and Vastus Lateralis in People With Patellofemoral Pain

Electromyography (EMG) is widely used to measure muscle activation during both isometric and dynamic actions. Although it provides valuable information about the neural control of movement, it does not provide direct information about muscle force. For example, at the same activation level (percent of maximal voluntary contraction), a muscle with a bigger physiological cross-sectional area will produce more force than a smaller muscle. In addition, because of the well-known force-length and force-velocity relationships, higher muscle force can be produced at the same activation level if the muscle operates at a more optimal length and/or velocity. Consequently, inferring muscle force from EMG signal amplitude is even more problematic during dynamic actions compared to isometric actions. Further, between muscle activation and force production there is an electromechanical delay. Considered together, it is clear that a comprehensive understanding of the sharing of force between muscles cannot be based on EMG alone, but requires a more direct estimation of the force produced by individual muscles or muscle region. Although the limitations associated with inference of force from EMG are well known, they have received little attention in the clinical literature.

This clinical commentary discusses these limitations within the context of the imbalance of force production between the vastus medialis (VM) and vastus lateralis (VL) muscles related to the etiology of patellofemoral pain (PFP). We aim to demonstrate that only a biomechanical

SYNOPSIS: Muscle force cannot be directly inferred from neural drive assessed using electromyography (EMG). Although the limitations associated with inferring force from EMG are well known, this has received little attention in the clinical literature. This commentary discusses these limitations within the context of the imbalance of force production between the vastus medialis (VM) and vastus lateralis (VL) muscles, which has been speculated to contribute to the development and/or persistence of patellofemoral pain. The balance of neural drive between vasti muscles is most frequently measured with 2 approaches: (1) the onset of VM EMG relative to that of the VL, and (2) the ratio of the EMG signal amplitude of the VM and VL. Here, we demonstrate that this classical approach cannot determine whether an imbalance of force exists between the VM and VL. Considerations such as altered electromechanical delay (time between the onsets of muscle activation and patellar motion) in people with patellofemoral pain may lead to a reconsideration of the classical interpretation of the onset of VM EMG signal relative to that of the VL. Also, beyond the amplitude of the neural drive, muscle force depends on several biomechanical factors (eg, specific tension and physiological cross-sectional area). Therefore, the VL/VM activation ratio does not provide information about the VL/VM force ratio, which is ultimately the most important information from a clinical perspective. Although the literature includes defenses for both the existence and absence of this force imbalance in people with patellofemoral pain, a reconsideration of the methods used to assess this imbalance is needed. *J Orthop Sports Phys Ther 2015;45(5):360-365. Epub 26 Mar 2015. doi:10.2519/jospt.2015.5905*

KEY WORDS: *anterior knee pain, biomechanics, electromechanical delay, electromyography, force*

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approach can resolve the potential issue of force imbalance related to PFP. The arguments presented are not only relevant for PFP but can also be extended to other musculoskeletal conditions in which a force imbalance between agonist muscles or regions within muscles is suspected.

Imbalance of Force Production Between the VM and VL

It is a long-held belief that altered force sharing between the heads of the quadriceps muscle plays an important role in the pathophysiology of PFP. The VM and VL share the functional role of knee extension with the rectus femoris and vastus intermedius. Because of its anatomy (ie, orientation and attachment of muscle fibers to the patella), the distal part of the VM (vastus medialis obliquus) is considered important for medial control of the patellofemoral joint.[23] Based on this proposal, an imbalance of force generation between the VM and VL has been speculated to contribute to the development and/or persistence of PFP.[15] One approach to the rehabilitation of PFP has therefore aimed to restore force balance between the VM and VL by specifically enhancing the activation (amplitude and/or timing) of the VM.[1,7,10,27] Although the approach has been shown to be efficacious in randomized clinical trials,[7] the potential to disassociate activation of the VM and VL is controversial, and the biomechanical importance of a putative disassociation in the neural drive is not clear. Although the literature includes defenses for both the existence and absence of a contribution of disassociation to the development and/or persistence of PFP, we argue that the current literature lacks the methodological approaches to resolve this issue.

Delayed Activation Is Not Evidence of Delayed Force Production

The balance of neural drive between vasti muscles is most frequently assessed using EMG, and is measured using 2 approaches: (1) the onset of VM EMG relative to that of the VL, and (2) the ratio of the EMG signal amplitude of the VM and VL. Delayed onset of VM EMG relative to VL EMG (of approximately 20 milliseconds) has been reported in individuals with PFP during stepping[7,8,10] and postural tasks.[9] This delayed VM activity has long been thought to contribute to patellar maltracking and development (and/or persistence) of PFP.[15,36] In support of this hypothesis, a simulation study demonstrated that a delay of as little as 5 milliseconds of VM EMG onset relative to that of the VL during running increases the compression forces on the lateral patellofemoral joint.[28] However, this difference in activation latency is not always observed in people with PFP.[14,26] In addition, a systematic review[5] that considered the relative onset of the VM and VL found large variations between studies (eg, onset of VM EMG ranged from 17.5 milliseconds before to 50 milliseconds after VL onset during stair ascent/descent) and between individuals within studies. The authors[5] concluded that there may be a trend in delayed onset of the VM relative to the VL in individuals with PFP, but not all individuals may exhibit this altered control. This is consistent with the results of a study by Pal et al,[31] who demonstrated a positive correlation between delayed onset of VM EMG during gait and patellar tilt in a subgroup of 15 out of 40 people with PFP who were classified as "maltrackers" (ie, having abnormal patellar tilt). Further, it has been argued that PFP is a multifactorial condition related to modified biomechanics at the foot[6] or hip[35] in addition to the knee, and thus suboptimal vasti activation may only be relevant for a subgroup of individuals with PFP. Therefore, it is possible that at least part of the discrepancy between studies and individuals within a study might be explained by the selection of the participants.

However, it is important to note that a delayed onset of EMG signal does not provide direct evidence of delayed force production. This is because (1) the onset of EMG signal may not accurately represent the onset of neural drive to the muscle but, rather, the latency of myoelectric activity under the recording surface electrodes, and thus depends on the electrodes' location relative to the motor point (spatial variations can produce a timing difference of up to 20 milliseconds[19]); and (2) there is a time lag (referred to as electromechanical delay) between onset of neural drive and force production (**FIGURE**). This electromechanical delay reflects both electrochemical processes (synaptic transmission and excitation-contraction coupling) and mechanical processes (force transmission).[29] These mechanical processes depend on muscle architecture and mechanical properties that may be modified in people with PFP. A recent study[4] that used high–frame-rate ultrasound (200 Hz) showed that individuals with PFP had a longer VM (about 12 milliseconds) and a shorter VL (about 7 milliseconds) electromechanical delay (defined in their study as the time between the onset of muscle electrical stimulation and onset of patellar motion) compared to healthy controls. This relatively longer electromechanical delay observed in the VM than in the VL, in combination with the delayed onset of VM EMG activity reported in some (but not all) studies, provides some evidence that a possible delay in onset of VM force production (ie, the potential for a mechanical imbalance between the VM and VL) may be underestimated when considering EMG alone. In addition, even in the absence of delayed VM EMG signal, a change in electromechanical delay may still result in an imbalance of force between these muscles (**FIGURE**). To our knowledge, electromechanical delay in the VM and VL has only been measured in the study discussed above.[4] Additional work using a higher temporal resolution (5 milliseconds),[4] that is, a higher frame rate, would strengthen this result.

With these issues in mind, greater consideration of delayed force (rather than delayed EMG signal) in the VM is required, and this may be achieved using ultrafast ultrasound imaging (sampling

frequency up to 5 kHz).[18,29] This relatively new technique could be used during various tasks to (1) confirm whether the delayed onset of VM EMG signal is associated with a delayed onset of muscle and/or patellar motion, and (2) precisely quantify the time lag between the onset of VM and VL motion.

Imbalance of EMG Signal Amplitude Is Not Evidence of Force Imbalance

There is no consensus regarding an altered VL/VM activation ratio in people with PFP compared to pain-free controls. Although no difference in activation ratio has been reported in people with PFP by some studies,[34] others have reported a linear relationship between patellar tilt and VL/VM activation ratio in people with PFP who exhibit patellar maltracking.[30] It is important to note that regardless of the population (people with PFP or pain-free controls), the mean ratio is very often greater than 1 (eg, 1.4 in the study by Pal et al[30] and approximately 1.1 in the study by Wong et al[39]), indicating greater activation of the VL than the VM. Large variability in activation ratio has been demonstrated between participants (eg, ranging from approximately 0.8 to 3 in the study by Pal et al[30]). This variation may be partly explained by methodological considerations when estimating neural drive using EMG (eg, cross-talk, signal cancellation, amplitude normalization).[13,17] However, beyond limitations inherent to the EMG technique, even if accurate quantification of VM and VL neural drive were possible, it would not provide information on the balance in muscle force, which is ultimately the most important information from a clinical perspective. Relative force cannot be inferred from neural drive alone, because muscle force also depends on the combination of several biomechanical factors. Among them, the most important to consider for the comparison of VL and VM force are specific tension (T_{spe}, defined as maximal force per unit area) and physiological cross-sectional area (PCSA, defined as the area of a muscle perpendicular to its fibers). Note that other factors, such as the force-length and force-velocity relationships, contribute[3] but are not expected to differ considerably between the VL and VM, although this remains to be confirmed. During a maximal isometric action, the maximal force produced by an individual muscle (F_m^{max}) can be calculated as follows (equation 1): $F_m^{max} = PCSA \times T_{spe}$.

Consequently, the force produced by an individual muscle during a submaximal isometric action (F_m^{sub}) can be calculated as (equation 2) $F_m^{sub} = PCSA \times T_{spe} \times \%_{activation}$, where $\%_{activation}$ is the neural drive normalized to that recorded during maximal voluntary contraction. It is evident from equation 2 that accurate information on balance in muscle force between the VM and VL should (at least) consider both PCSA and T_{spe} in addition to neural drive. This is challenging for several reasons. First, in vivo estimation of T_{spe} is not a trivial task, and this underpins the large range of reported values (median, approximately 25 N/cm^2 [250 kPa]; range, 9.7-62.8 N/cm^2 for ankle plantar flexor muscles[24]). Animal[33] and human[11] experiments suggested that T_{spe} depends on muscle typology, with a higher T_{spe} associated with type II muscle fibers. A lower percentage of type II fibers in the VM than in the VL in healthy controls[20] is likely associated with a lower T_{spe} for the VM. Whether fiber-type composition of the VM is modified in people with PFP is unknown. Second, individual muscle force strongly depends on PCSA and, therefore, on both muscle volume and pennation angle. Using dif-

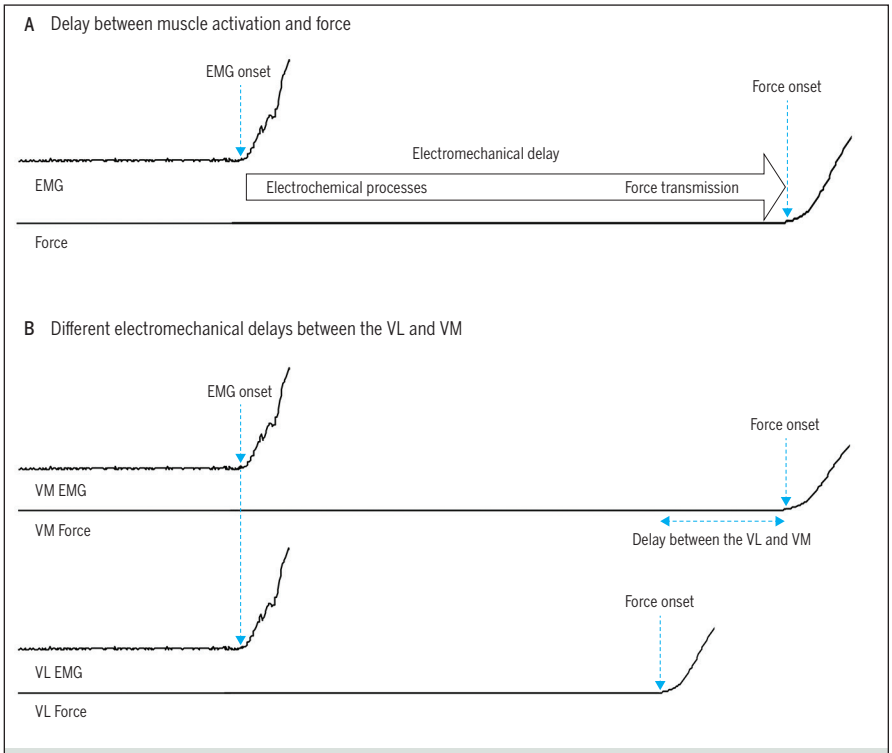

FIGURE. Schematic representation of the influence of electromechanical delay on the onset of force production. (A) The time lag between onset of myoelectrical activity and force production (electromechanical delay) reflects both electrochemical processes (ie, synaptic transmission, excitation-contraction coupling) and mechanical processes (ie, force transmission along the muscle and tendon). (B) Although the electromechanical delay has been shown to be similar between the VM and VL in pain-free participants (about 25 milliseconds), longer delay has been reported in the VM (about 38 milliseconds) than in the VL (about 18 milliseconds) in people with patellofemoral pain.[4] With consideration of this difference, this example illustrates that an absence of difference of EMG onset times might still lead to difference in onset of force production. Abbreviations: EMG, electromyography; VL, vastus lateralis; VM, vastus medialis.

fusion tensor magnetic resonance imaging, Kan et al[21] reported that the PCSA of the VL (distal oblique fibers) is twice that of the VM (distal oblique fibers) in healthy participants. Similarly, a higher volume (approximately 1.5 times) was found for the VL compared to the VM in healthy controls.[16] In addition to the reported higher activation and the possibly higher T_{spe} of the VL than of the VM, these data strongly suggest that VL force is systematically higher than VM force in pain-free controls. Therefore, we cannot consider that the force production is balanced between the VL and VM in pain-free controls. Surprisingly, despite the importance of muscle PCSA to infer muscle force, there is a paucity of information regarding potentially altered muscle architecture and volume in people with PFP. Based on measurements of muscle volume alone, Pattyn et al[32] reported a selective atrophy of the VM (distal oblique region) relative to the VL in people with PFP. If confirmed by PCSA measurements, this suggests that a similar activation ratio between the VM and VL would lead to a lower force produced by the VM relative to the VL in people with PFP.

Finally, 2 other mechanical factors are important to consider. First, the force produced by a muscle is the combination of active (discussed above) and passive components. In this manner, decreased quadriceps flexibility has been suggested as an intrinsic risk factor for the development of PFP.[38] This is likely related to an increased tightness/stiffness of these muscles, which may affect joint moments/load at rest and during movement. This passive component cannot be measured by EMG but could be estimated on each individual muscle from elastography measurements.[25] Second, muscle geometry (line of action and moment arm) should be considered to determine the mechanical effect of force on the patella. However, there is preliminary evidence that quadriceps geometry is not different between individuals with and without PFP.[37]

Estimating Individual Muscle Force Is Challenging

From the limitations highlighted above, it is clear that an accurate quantification of the balance of force between muscles belonging to the same muscle group requires an accurate quantification of individual muscle force. However, there is no noninvasive experimental technique to measure individual muscle force. Although musculoskeletal models can be used, they are based on many assumptions that may ultimately prove to limit their usefulness in clinical practice. More importantly, in the absence of experimental measures of muscle force, these models cannot be validated.[12] Consequently, estimation of individual muscle force remains one of the main challenges in biomechanics.

As largely discussed in the literature, physiological and nonphysiological factors can limit the ability of EMG to accurately quantify neural drive.[13,17] Further, EMG cannot quantify passive force. In contrast, muscle shear modulus (ie, stiffness) measured using shear-wave elastography is strongly linearly related to both passive[22,25] and active force.[2] Of note, during isometric actions, the linear relationship between muscle stiffness and muscle force is stronger (mean R^2 = 0.977) than the linear relationship between EMG signal amplitude and force (mean R^2 = 0.936).[2] Consequently, measurement of muscle stiffness normalized to that recorded during maximal voluntary contraction might give reliable information about relative muscle force (ie, sum of the active and passive components). This information, in combination with muscle PCSA, might give a more accurate estimation of individual muscle force and, therefore, a more direct assessment of the balance of muscle force. However, this remains to be validated. In addition, because shear-wave elastography measurements are currently provided at up to 1 sample per second (eg, supersonic shear imaging technique; SuperSonic Imagine, Aix-en-Provence, France), only isometric actions and slow dynamic passive stretching (less than 10°/s) can be studied.

SUMMARY

Because estimation of individual muscle force remains one of the main challenges in biomechanics, the issue of imbalance of timing and/or amplitude of force between the VM and VL in people with PFP has been mainly addressed using a neurophysiological approach (relative onset and ratio of muscle activation). This has led to contradictory findings, that is, studies showing both the existence and the absence of an activation imbalance between the VL and VM in people with PFP. We contend that this debate cannot be addressed without the use of a biomechanical approach. For example, to determine if a relative delay of force production is likely to underpin PFP, the possible difference in electromechanical delay between the VL and VM needs to be considered. Further, if the ratio of force between the VL and VM is to be considered, concurrent consideration of muscle activation (or, ideally, muscle stiffness), muscle PCSA, and passive force is required (T_{spe} being more difficult to estimate), as they are susceptible to being different between healthy controls and people with PFP.

This clinical commentary presents 4 main considerations: (1) based on published EMG and PCSA data, it is likely that VL force is systematically higher than VM force in healthy, asymptomatic individuals; (2) current literature cannot determine whether the balance of force between the VM and VL is altered in patients with PFP compared to healthy, asymptomatic individuals; (3) a concurrent measurement of muscle activation (using EMG) or, more ideally, stiffness (using elastography) and muscle PCSA could provide the first indirect way to assess the balance of force between these muscles, and determine if this long-held belief is supported; and (4) the development of a method to accurately estimate individual muscle force remains a crucial

step in being able to determine if balance of force production between muscles belonging to the same muscle group is altered. Ultimately, this method could assist in the identification/subgrouping of individuals with PFP, and other musculoskeletal conditions, who demonstrate a dysfunction in muscle coordination. If so, this will provide the information needed to allow clinicians to more efficiently target appropriate, individualized rehabilitation strategies. ◉

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