INTRODUCTION:

Patellofemoral (PF) pain syndrome (PFPS) is one of the most common problems of the knee. A potential source of this pain is a force imbalance around the knee leading pathological PF kinematic (maltracking). In turn, this causes elevated joint contact stresses, which ultimately results in PF pain. The source of this force imbalance is still open to debate with some postulating the cause to be delayed timing or loss of strength in the vasti medialis (VM) [1]. Yet, numerous studies refute these claims as well [2]. Since muscle force cannot be measured directly, without highly invasive techniques, previous studies have relied on electromyography (EMG). Yet in patients with PFPS the assumptions required to estimate muscle force from EMG may not be valid [3]. Thus, the purpose of this study was to determine how a loss of VM force alters 3D in vivo PF and tibiofemoral (TF) kinematics during a volitional extension task.

METHODS:

To date, 21 asymptomatic females with no history of knee pain, trauma, and surgery have been enrolled in this IRB approved study. During the first visit, subjects provided signed consent, had a history and physical, and then were placed supine in a magnetic resonance (MR) imager (3.0 T, Philips Medical Systems, Best, NL). For dynamic scanning, the knee was bent and supported on a cushioned block. A customized coil holder held a pair of flex coils medial and lateral to the knee. Subjects cyclically flexed and extended their knee while a cine-phase contrast (CPC) MR image set (x,y,z velocity and anatomic images frames) was acquired [4]. Dynamic cine images were also acquired to establish anatomical coordinate systems. With the knee in full extension, 3D static images were acquired. The scanning protocol was saved so that identical dynamic scanning parameters could be used for the second visit. If the data acquired revealed a valid exclusion criteria, the subject was removed from the study (n=5), if not, the subject was asked to return within a week (n=16, 26.7±8.6years, 164.5±6.4cm, 57.3±7.4kg).

For the second visit, scanning began immediately after administering a motor branch block to the VMO. A single physician performed all nerve blocks. Using ultrasound (US) guidance and electrical stimulation, the femoral nerve motor branch to the VMO was localized and then 3 cc of 1% lidocaine was injected. The absence of a twitch response (visual surface inspection and B mode US) upon percutaneous electrical stimulation of the motor nerve indicated a complete or near complete block of the VMO. If the twitch response was not ablated (n=6), the procedure was repeated at a second site with up to 2 cc of 1% lidocaine. Immediately following the muscle block, the volunteer was transported to the MR scanner with the coil holder returned to the same location. Using reference marks on both the coil holder and the skin over the subject’s knee, the subject was placed in as similar a position as possible to the first exam. All scanning occurred within 20 minutes of completing the muscle block. Lidoacaine has a minimum effective period of 1 hour.

The PF and TF kinematics, both pre-and post-injection (pre-I and post-I) were quantified through integration of the CPC data and were interpolated to single knee angle (KA) increments. The pre-I and post-I kinematics were compared using paired Students’ t-test. Correlations between the change in kinematics post-I and the pre-I kinematics were quantified at the 5° KA increments.

RESULTS:

Post-I, the patella shifted laterally (Figure 1, max = 1.9±1.9mm), whereas the tibia rotated externally (max=3.2°±3.8°) and shifted laterally (max=1.6±2.0mm). These changes were 4.1-4.7 times greater than the average subject repeatability [4]. An insignificant trend in PF lateral tilt was seen post-I. Post-I PF lateral shift was correlated with pre-I PF superior shift (r=0.52, KA = 15°) and valgus rotation (0.59-0.70, KA = 20°-35°). TF external rotation and lateral shift were not correlated with any pre-I kinematics.

DISCUSSION:

This study provides the first in vivo data pertaining to the contributions of the isolated VMO to PF and TF kinematics. A VMO nerve block produced kinematics changes that mirrored the difference in axial plane kinematics seen between patients diagnosed with PFPS and controls (previously acquired identical imaging protocol: maximal voluntary isometric contraction injection paradigm: medial shift = -1.6mm, medial tilt = 1.0° (NS), alta = 2.7mm, and flexion 2.3mm). Yet, the muscle block likely produced a greater loss in VM strength than that experienced by individuals with PFPS and the functional loss of the VMO did not produce changes in the other planes of motion.

To relate the kinematic post-I changes to those seen in PFPS, it is important to understand that there are likely subgroups within the PFPS population [5], each with unique kinematic alterations of varying etiologies. Dividing the PFPS cohort into medial and lateral maltrackers resulted in a lateral maltracking group that demonstrated increased PF lateral shift (3.5mm), superior shift (4.1mm), lateral tilt (3.9°), and flexion (2.4°) and a medial maltracking group that demonstrated increased PF medial shift (1.5mm), medial tilt (5.0°) and flexion (2.0°). The VMO block accounts for a portion of the lateral shift and tilt seen in the lateral maltrackers. Ligament laxity would likely increase this shift and tilt, as well as increase the patellar ligament length, resulting in the observed PF superior shift (patella alta). This alta reduces the femoral groove’s influence on the PF kinematics, increasing lateral shift (as supported by the correlations within this study) and lateral tilt. Therefore, a combination of VM weakness and ligament laxity could account for the kinematic variations in the lateral maltracking group, with these changes likely leading to PF pain. As demonstrated previously [5], a higher lateral femoral sulcus combined with a normative PF superior location in the medial maltrackers limited lateral PF shift. Thus, in this subgroup a loss of VM strength likely result in increased contact force between the lateral femoral sulcus and the patella, resulting in PF pain. The primary limitation was a lack of power for the correlations. Work is ongoing to increase the sample size.

SIGNIFICANCE:

It is clear when comparing the post-I changes to the kinematics associated with PFPS that VMO weakness is most likely a major factor in, but not the sole source of, PF maltracking. Thus, isolated VM strengthening will likely not correct PF maltracking in most individuals. These results will help foster the next generation of PFPS treatments focused on first elucidating subject-specific factors leading to PF pain in order to design interventions specifically targeting these factors.

REFERENCES:


gavellif@cc.nih.gov