INTRODUCTION
Patellofemoral (PF) pain syndrome (PFPS) is one of the most common problems of the knee, characterized by idiopathic anterior knee pain aggravated by deep knee flexion, prolonged sitting, and repetitive flexion/extension. The most widely accepted theory in regards to the source of PF pain is that a force imbalance around the knee leads to PF static malalignment and dynamic 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) or an imbalance in the passive structures [1]. Yet, numerous studies refute these claims as well [2]. Unfortunately, muscle force cannot be measured directly, without highly invasive techniques. Thus, the force imbalance between the VM and vasti lateralis (VL), reported previously, is based on electromyographic recordings (EMG), from which muscle force can be approximated based on a series of assumptions. An earlier study clearly demonstrated that, in patients with PFPS, a muscle may not contribute to useful work even if it is active [3]. In such an instance the assumptions required to estimate muscle force from EMG are not valid. Therefore, the purpose of this study was to determine how a loss of force in the VM alters the dynamic control of 3D in vivo PF kinematics during a volitional extension task.

METHODS
To date, 18 asymptomatic females with no prior history of knee pain, trauma, leg surgery, or contraindications to having an MRI have been enrolled in this IRB approved study, involving two visits. Recruitment targeted females, as this is the population in which PFPS is most prevalent. During the first visit, subjects provided signed consent and had a history and physical along with a clinical evaluation of the knee joint (Q-angle, laxity, etc). Subjects were excluded from the study if they had any contra-indications of having an MRI, any history of anterior knee pain, lower leg surgery, joint pathology (ligament/meniscus tears, arthritis, etc), or abnormal knee joint kinematics (defined as any single PF or tibiofemoral (TF) kinematic parameter being more than 2 SD from a previously defined control average). If no exclusion criteria were discovered, the subject proceeded to the MR unit for both dynamic and static scanning. The dynamic scanning all participants were placed supine in an MR unit for both dynamic and static scanning. For the dynamic criteria were discovered, the subject proceeded to the MR scanner so that the identical dynamic scanning parameters could be used for the second visit.

For the second visit, scanning began immediately after administering a motor branch block to the VM. Using ultrasound (US) guidance and electrical stimulation, the femoral nerve motor branch to the VM was localized. After near nerve or motor point location was confirmed, 3 cc of 1% lidocaine was injected. A single physician (Dr. Alter, board certified by ABPM&R and ABEM) performed all nerve blocks. Evaluation of the effectiveness of the motor blockade was assessed by absence of visible twitch (visual surface inspection and B mode US) with percutaneous electrical stimulation of the motor nerve. In addition to the twitch response, the absence of VM contraction was confirmed in a similar manner while the subject performed a maximal isometric contraction of the quadriceps. If the twitch response was not ablated, the procedure was repeated at a second site with up to 2 cc 1% lidocaine. Confirmation of the effectiveness of the block was performed as noted above. An absence of a twitch response upon stimulation indicated a complete or near complete block of the VM. Although force measurements were taken pre- and post-block, they were not used to define the block of the muscle, as there is no in vivo data, to date, in regards to the isolated force capability of the VM. Immediately following the
The PFPS and controls cohorts. Further, VM loss did not produce changes in the other planes of motion. Specifically, the PFPS cohort demonstrated increased PF superior displacement, flexion, valgus, and TF external rotation, as compared to the control cohort. Thus, the loss in VM function cannot explain all the kinematics changes in PFPS cohort and it is most likely that VM weakness is a major factor in, but not the sole source of, PF maltracking.

To relate the kinematic changes seen following a VM block to those seen in PFPS, it is important to understand that there are likely subgroups within the PFPS cohort, each with unique kinematic alterations of likely varying etiologies. In a previous study the PFPS cohort was divided into two groups (lateral and non-lateral maltrackers [6]). The lateral maltrackers, demonstrated increased PF lateral and superior shift, lateral tilt, flexion, valgus rotation and TF external rotation. Based on the current results, the loss of VM function could account for a portion of the lateral shift and tibial external rotation. An increase in ligament laxity would likely increase this shift and rotation, as well as increase the patellar ligament length, resulting in a PF superior shift (patella alta). This alta reduces the influence of the femoral groove on PF kinematics, increasing PF lateral shift (as supported by the correlations within this study), lateral tilt and valgus rotation. Therefore, a combination of VM weakness and generalized ligament laxity would account for the kinematics variations in the lateral maltracking group, with these changes likely leading to PF pain.

The non-lateral maltrackers demonstrated increased PF flexion and increased TF external rotation only. From the current results, it can be stated that VM weakness accounts for a majority of the TF external rotation, yet the expected increase in lateral PF displacement is not observed. As demonstrated previously [7], the higher lateral femoral sulcus combined with a normative PF superior shift in the non-lateral maltrackers prevents the lateral PF shift. Thus, in this subgroup, a loss of VM strength would be compensated for by increased contact force between the lateral femoral sulcus and the patella, resulting in PF pain.

RESULTS AND DISCUSSION

Post-injection, the patella shifted lateral (max = 1.7±1.7mm, p=0.004, KA=15°), whereas the tibia rotated externally (max=3.7°±3.5°, p=0.003 KA=15°), and the tibial origin shifted laterally (max=2.6±2.5mm, p=0.04, KA=12°) (Figure 2). These changes were 4.1-4.7 times greater than the subject repeatability for knee joint kinematics [4]. An insignificant trend of PF lateral tilt was seen post-injection. The remaining degrees of freedom for the PF and TF joints, demonstrated post-injection changes that were less than the subject repeatability. PF post-injection lateral shift was correlated with pre-injection PF superior displacement (r=0.48) and valgus rotation (r=0.59). TF external rotation and lateral shift were not correlated with any pre-injection kinematics, but were correlated with each other (r=0.81), indicating that the change in the TF origin was due to external TF rotation.

The loss in VM function produced kinematics changes that mirrored the difference in axial plane kinematics seen between patients diagnosed with PFPS and controls, measured using the identical pre-injection paradigm. Even though the muscle block likely produced a greater loss in VM strength than that experienced by patients with PFPS, the post-injection changes in PF lateral shift and TF external rotation were only 59% and 92% of the differences between the PFPS and controls cohorts[5]. Further, VM loss did not produce changes in the other planes of motion. Specifically, the PFPS cohort demonstrated increased PF superior displacement, flexion, valgus, and TF external rotation, as compared to the control cohort. Thus, the loss in VM function cannot explain all the kinematics changes in PFPS cohort and it is most likely that VM weakness is a major factor in, but not the sole source of, PF maltracking.

CONCLUSIONS

In the current study, a loss of VM function lead to increased lateral PF shift and external TF rotation. This supports the fact that the VM exerts a medially directed force on the patella [8] and an internal rotation moment on the tibia via the patellar tendon. Combining these results with past results pertaining to kinematics and bone shape alterations in patients with PFPS supports two paths to PFPS in two kinematically unique subgroups. Future work is required to provide further evidence to the validity of these paths.

REFERENCES