DOES KNEE EXTENSOR MUSCLE IMBALANCE CAUSE CHANGES IN PATELLAR TRACKING?

Andrew Sawatsky, Tim Leonard, Walter Herzog

University Of Calgary
email: asawatsky@kin.ucalgary.ca

INTRODUCTION

Patellofemoral pain syndrome (PFPS) is one of the most common knee disorder seen by clinicians. The main region of pain is the lateral aspect of the patellofemoral joint. Force imbalance in the knee extensor muscles has been associated with changes in patellofemoral tracking [1] and is suspected to cause PFPS. Weakness of the vastus medialis (VM) relative to the vastus lateralis (VL) is the usual imbalance associated with PFPS. The fibers of VM are aligned about 50° from the femoral axis and this structural arrangement has been thought to pull the patella medially while VL has been assumed to provide the corresponding lateral balance [2].

A commonly used treatment for PFPS is exercises directed at increasing VM strength to balance the medial-lateral tensions. Although the idea that muscle imbalance between VM and VL causes maltracking of the patella, has intuitive appeal and anecdotal support, it has not been tested in an intact knee loaded by muscular contraction.

The purpose of this study was to create an animal model of quadriceps muscle imbalance and to test the effect of VM weakness, and thus VM/VL imbalance, on patellar tracking. Muscle imbalance was produced by eliminating the action of VM by cutting it in mid-belly. We hypothesised that transection of VM causes a medial-lateral knee extensor imbalance causing the patella to shift laterally in the femoral groove.

METHODS

All experiments were performed on knees (n=9) of skeletally mature New Zealand White rabbits (mass 4.8-5.7kg). Patellar tracking was recorded with a high speed camera at 200Hz and a spatial resolution of 80µm.

Two markers were placed on the patella via a bone screw and two further markers on the femur via bone pins, to record frontal plane movements of the patella relative to the femur. The hip and knee of the rabbits were fixed in a stereotaxic frame, while the tibia was attached to a lever arm connected to a motor allowing for knee extensions and flexions at controlled speeds. Movements of the patella relative to the femur were recorded for passive and active concentric and eccentric knee extensor contractions, before and after VM transection.

Knee extensor activation was produced through femoral nerve stimulation using a nerve cuff-type electrode. All contractions were performed at maximal levels of stimulation and a frequency of 40Hz. All (passive and active, concentric and eccentric) tests were executed at an angular speed of 40°/s moving from 30° to 90° of knee flexion. Patellar tracking was recorded with the knee joint and VM intact and then again following VM transection.

RESULTS AND DISCUSSION

The primary result of this study was that patellar tracking was not affected by VM ablation (Figure 1). This result suggests that even the biggest possible muscle imbalance of the knee extensor complex (zero force in the VM) did not affect the medial-lateral tracking of the patella. This result is in stark contrast to findings by Goh et al. (1995) who suggested, in human cadaveric knees, using wires and pulleys to load the patellofemoral joint, that simulated weakness of the VM caused immediate and pronounced shifts of the patella to the lateral edge of the femoral groove. There are several reasons why our results may differ from those obtained in the literature, but the most compelling appears to be that we measured patellar
tracking using the actual, intact muscles to produce force through nerve stimulation, rather than an artificial loading apparatus which, by design, makes assumptions on how muscles act on the patella. However, our results are consistent with previous observations in the rabbit knee that demonstrated that “isometric” contractions of the knee extensors also did not produce the “expected” lateral shift of the patella in the femoral groove following VM ablation [4].

Figure 1 Exemplar result of patellar tracking before and after VM transection for concentric conditions. The distances shown are measured relative to the origin of an inertial reference system with origin at the medial femoral condyle. The movement starts at the top-left (arrow) with the muscles relaxed. Upon activation, there is a distinct, medial shift of the patella, which is reversed upon deactivation. The patellar tracking before and after VM ablation is virtually identical with differences that are within the resolution of our system.

Patellar tracking, however, was greatly affected by activation of the muscles. The patellae in all experiments tracked more laterally along the femoral groove for passive concentric and eccentric contractions, compared to the corresponding active contractions (Figure 2). Activation, always caused a medial shift of the patella, and when activation was absent in the passive trials, the patella always moved along a lateral path during knee extension and flexion.

Concentric and eccentric active contractions also produced different patellar tracking patterns, but these were less systematic than those observed between the passive and active trials and need further quantification (Figure 2). However, it appears that in general, eccentric contractions might cause a greater medial shift of the patella than concentric contractions. Therefore, if lateral tracking of the patella is implicated in PFPS, as has been suggested in the literature, and has been thought to occur with VM weakness, then eccentric contractions should be the least likely to cause PFPS syndrome.

Figure 2 Exemplar results of patellar tracking for a passive movement, an active eccentric and an active concentric movement with the VM intact. The distances shown are measured relative to an inertial reference system with origin at the medial femoral condyle. Note the medial shift of the patella for the active compared to the passive movements and for the eccentric compared to the concentric movement.

CONCLUSIONS

VM weakness does not produce changes in patellar tracking in the rabbit knee. This result may have important clinical implications for PFPS in humans, and challenges a long-held clinical paradigm.

Reference List


ACKNOWLEDGEMENTS

CIHR, the Canada Research Chair Programme in Molecular and Cellular Biomechanics and the AHFMR Team Grant on OA.