

Changes in Patellofemoral Contact Pressure due to Imbalance of the Knee Extensors

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INTRODUCTION

Muscle imbalance in the knee extensor muscles has been associated with problems in patellofemoral tracking (Powers, 2000), joint pain, and osteoarthritis. Changes in patellar tracking and pressure distribution are suspected to cause patellofemoral pain (PFP). The main region of pain is the lateral aspect of the patellofemoral joint. Weakness of the vastus medialis (VM) relative to the vastus lateralis (VL) is the usual imbalance associated with PFP (Cowan et al., 2002; Lefebvre et al., 2006). The fibers of VM are aligned about 50° to 55° medially, and this structural arrangement has been thought to pull the patella medially while the vastus lateralis has been assumed to provide the corresponding lateral balance.

A commonly used treatment for PFP is exercises directed at increasing the strength of the VM in the hope to balance the lateral tensions. Although the idea that muscle imbalance causes maltracking of the patella and so causes pain makes intuitive sense and has clinical and anecdotal support, it has not been tested scientifically.

The purpose of this study was to create an animal model of quadriceps imbalance and to test the effect of extreme imbalances on patellofemoral contact pressures. Muscle imbalance was produced by eliminating the action of VM by cutting it in mid-belly. We hypothesized that the elimination of VM causes a lateral shift of the patellofemoral contact pressures, and might cause lateral patellar subluxation.

METHODS

All experiments were performed on knees (n=9) of six months old New Zealand white rabbits (mass 4.9-5.9kg). Measurements of patellofemoral contact pressures were performed before and after VM transection at a variety of force levels. Experiments were approved by the Animal Ethics Review Committee of The University of Calgary.

In order to assess changes in patellofemoral contact pressures, low grade and medium grade pressure sensitive film (Fuji) was used. Prior to measurement, rabbits were placed into a stereotaxic frame with only the tibia and foot of test limb being mobile. The film was inserted into the joint through bilateral, retinacular, 20-25mm incisions while the joint was fully extended. The tibia was then placed behind a tibial restraining bar at a knee angle of approximately 90°, as determined with a hand held goniometer. The tibial restraining bar measured knee extensor forces. Patellofemoral contact pressures were measured while stimulating the knee extensor muscles through the femoral nerve at supra-maximal currents and frequencies of 200, 60, 50, 40, and 30Hz. Contact pressures were always measured with the low grade film (range = 1-16Mpa). When the low grade film was saturated, measurements were also made using the medium grade film (range = 18 - 52MPa). Contact area measurements were all determined from the low grade film, while peak and average pressures were determined from the low and medium grade films.

Contact area was calculated by integrating across the low grade Fuji film. Peak pressure was obtained as the maximal pressure in an area of at least 0.25mm². Average pressure

was calculated as the total contact force divided by the total contact area.

RESULTS AND DISCUSSION

Peak pressures before and after VM transection were similar in each animal and remained similar when averaged across all animals (figure 1). Average pressure increased with increasing knee extensor forces and remained the same before and after VM transection (figures 2). Contact area size and shape also remained virtually identical before and after VM transection, and so did the pressure distribution (results not shown).

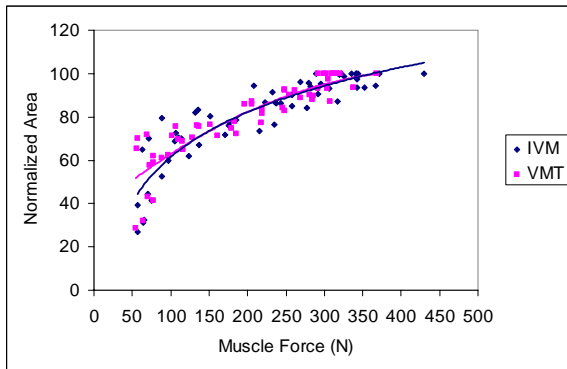


Figure 1 Change in normalized area as a function of muscle force. Across all Rabbits in both hind limbs for IVM and VMT

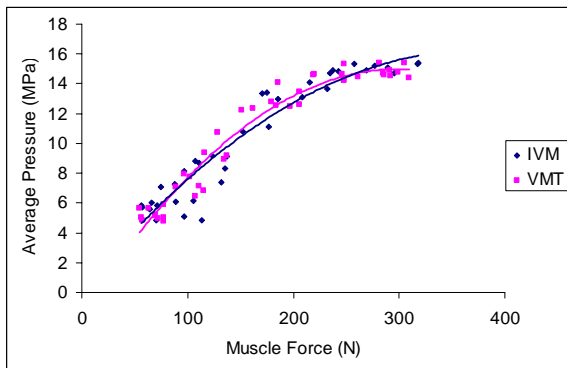


Figure 2 Average pressures as a function of knee extensor forces across all animals with the VM intact (VM) and transected (VMT)

Clinical and anecdotal evidence has led to the common notion that knee extensor imbalance is a cause for PFP associated with changes in patellofemoral contact pressure distribution. One common treatment for PFP is the strengthening of the VM because it has been assumed implicitly that weakness of VM relative to VL is the primary cause of the joint pain. Specific strengthening of the VM is supposed to correct this muscle imbalance, thereby equalizing the medial and lateral forces acting on the patella, allowing for proper patellar tracking in the femoral groove (Cowan et al., 2002). However, here we found that extreme changes in knee extensor imbalance produced by complete elimination of the VM by transection had no visible and consistent effect on patellofemoral contact pressure distributions and contact areas in the New Zealand White rabbit. Although these results may not translate directly to the human condition, it questions the clinical assumption that muscle imbalance causes changes in patellofemoral pressure distributions and joint pain.

References

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