The effects of articular, retinacular, or muscular deficiencies on patellofemoral joint stability

A BIOMECHANICAL STUDY IN VITRO

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Normal function of the patellofemoral joint is maintained by a complex interaction between soft tissues and articular surfaces. No quantitative data have been found on the relative contributions of these structures to patellar stability. Eight knees were studied using a materials testing machine to displace the patella 10 mm laterally and medially and measure the force required. Patellar stability was tested from 0˚ to 90˚ knee flexion with the quadriceps tensed to 175 N. Four conditions were examined: intact, vastus medialis obliquus relaxed, flat lateral condyle, and ruptured medial retinaculae. Abnormal trochlear geometry reduced the lateral stability by 70% at 30˚ flexion, while relaxation of vastus medialis obliquus caused a 30% reduction. Ruptured medial retinaculae had the largest effect at 0˚ flexion with 49% reduction. There was no effect on medial stability. There is a complex interaction between these structures, with their contributions to loss of lateral patellar stability varying with knee flexion.

The function of the patellofemoral joint is normally maintained by a complex interaction between soft tissues and bony structures. The structures responsible for its stability can be divided into three groups: the active stabilisers represented by the quadriceps muscles, the passive stabilisers particularly the retinacula and the static stabilisers represented by the articular surfaces. In normal knees these structures act in harmony to maintain stability of the joint. However, this complex interaction can be disrupted by pathology or trauma, which may result in patellofemoral instability.

Instability such as patellar subluxation or dislocation is common and may require treatment by conservative or surgical methods. More than 100 surgical procedures have been described for treating patellofemoral instability, including a combination of lateral retinacular release, medial capsular reefing or patellar tendon transfer. Despite these procedures the rate of recurrence remains high. The underlying problem is that patellofemoral instability can be subtle and multi-factorial. There is a lack of useful information about the relative importance of different pathologies, or of the mechanical effectiveness of the different surgical techniques. In order to make the correct diagnosis and choose an appropriate treatment, it would be helpful to understand the relative contributions of the different patellar stabilising mechanisms. Although there have been reports of the effects of particular stabilising mechanisms, none has studied the relative contributions of the different active, passive and static patellar stabilisers.

This study examined patellar stability in the objective mechanical sense and was not concerned with subjective instability. Its aim was to measure the forces required to cause the patella to sublux medially and laterally, allowing the effects of various abnormalities associated with patellar instability to be quantified.

Materials and Methods

Specimens and specimen preparation. Eight knees were obtained at post-mortem with ethical approval and the informed consent of relatives. They were sealed in polyethylene bags and frozen at -20˚C prior to use. The mean age of the donors was 69 years (57 to 87). The specimens included 20 cm each of femur and tibia. The specimens were prepared and mounted using methods which have been described previously. The skin, underlying fat and muscles, other than the distal quadriceps were removed. Care was taken to preserve the retinaculae and the fascia of the quadriceps muscles intact. The quadriceps were then separated as accurately as possible into six components: rectus femoris (RF), vastus intermedius (VI), vastus lateralis longus (VLL), vastus lateralis obliquus (VLO), vastus medialis longus (VML), and vastus medialis obliquus (VMO).
The distal tendinous fibres of the muscles merged together and were left intact to ensure that the actions of the muscles were as physiological as possible. Vastus intermedius was separated from the femur.

Cloth strips were wrapped and looped over the proximal end of each muscle component and attached by stitching through the whole muscle bulk. Rectus femoris and vastus intermedius were looped together to form a central muscle group. The cloth strips provided a firm attachment for a muscle loading cable for each of the five muscle groups (RF+VI, VLL, VLO, VML, and VMO).

**Experiment set-up.** A stability testing rig was mounted in an Instron 1122 materials testing machine (Instron Ltd., Buckinghamshire, UK). The stability rig was in two parts, one fixed to the base of the Instron, the other suspended from the loadcell in the moving crosshead (Fig. 1). The fixed part consisted of a femoral mounting on a steel baseplate with pulleys fixed onto the base of the Instron. The moving part was a three-degree-of-freedom mounting allowing for patellar translation and rotation in the sagittal plane. The moving crosshead of the Instron was used to displace the patella laterally and medially in a controlled manner while measuring the force required. The knee was mounted via an intramedullary femoral rod in the mounting device on the base of the Instron machine with the line joining the posterior femoral condyles describing the medial-lateral axis, parallel to the displacement axis. The centre of rotation of the knee was assumed to be at the femoral epicondyle. Subsequently, the quadriceps muscles were loaded. The knee was orientated with the lateral aspect uppermost, and with the tibia flexing in a horizontal plane (Fig. 1). The initial femoral rotation and its alignment were ensured by the cementing technique of the intramedullary rod. The quadriceps components were each loaded by cables routed via pulleys to hanging weights and a total load of 175 N was applied. The muscle groups were tensioned in their anatomical directions and in proportion to their physiological cross-sectional areas.

The patella was connected via the three-degree-of-freedom linkage to the load cell on the moving crosshead of the Instron. At the patella, a ball-bearing inside the patella, centred 10 mm deep to the anterior surface, allowed natural tilt and other rotations of the patella (three-degrees-of-freedom) when it was displaced laterally and medially (Fig. 1). This arrangement allowed five-degrees-of-freedom while controlling the medial-lateral translation.

Patellar force-displacement behaviour was tested at 0, 10, 20, 30, 45, 60, and 90° knee flexion. Extension of the knee was blocked at each angle by a vertical rod, anterior to the tibial rod, at a radius of 120 mm from the epicondyle. The patella was cyclically displaced 10 mm laterally and 10 mm medially from its stable position at 100 mm/min. The fourth load vs displacement cycle was recorded at each knee flexion angle. The relative stiffness of the Instron load frame and specimen mounting was very high (1250 Nmm⁻¹) when compared with the tissues resisting patellar displacement. Therefore, crosshead motion was a sufficiently accurate representation of patellar motion.

**Patellar stability measurement protocols.** Measurements of patellar stability were performed in four testing conditions: intact knee, VMO malfunction, lateral trochlear dysplasia, and with the medial patellofemoral ligaments ruptured. The tests were performed in that particular order in order to assess the effects of each condition on its own. The pathological abnormalities were simulated as follows: VMO malfunction was simulated simply by relaxing the VMO muscle (taking the weights off). The VMO tension was shared out among the other muscle parts maintaining a total tension of 175 N. This was done immediately after the force-displacement behaviour was recorded at each angle of flexion in the ‘intact’ knee.

Lateral trochlear dysplasia was simulated by cutting a subchondral wedge out of the lateral condyle to flatten the lateral facet (Fig. 2). The patellar force-displacement behaviour was recorded using the same protocol mentioned above.

Medial retinacular structures ruptured: a double hook was used to pull the patella laterally until the medial reti-
naculae ruptured. The quadriceps muscles were relaxed (except rectus femoris which had 20 N tension). This was done to protect the patella from excessive force because it fractured under both 175 N quadriceps tension and the lateral displacing force. The double hooks were positioned proximally and distally under the medial border of the patella and were pulled laterally with a crosshead speed of 10 mm/min until the medial structures ruptured. The lateral trochlea was reconstructed by repositioning the original bone wedge and adding wooden fillers which had the same thickness as the saw cuts. This assembly was fixed by a bone screw. The testing protocol was then resumed.

Data analysis. The reduction of the restraining forces from the ‘intact’ level for each simulated pathology at different angles of knee flexion was examined by analysis of variance with a Tukey post test and an alpha level of 0.05.

Results
The mediolateral stability of the patella for the intact knee has been described previously. The mean patellar restraining force vs knee flexion angle curves at 10 mm lateral displacement are shown in Figure 3. For the intact knee, 10 mm lateral displacement needed a mean force of 126 N at 0˚ flexion reducing to a minimum of 75 N at 20˚ flexion and rising to 125 N at 90˚ flexion. VMO release had a significant effect on the lateral restraining force throughout flexion (Fig. 3). Flattening the lateral facet of the femoral trochlea reduced the lateral restraining force...
Results
The mean patellar restraining force versus knee flexion angle curves at 10 mm medial displacement are shown in Figure 7. For the intact knee, 10 mm medial displacement needed between 144 N and 239 N from 0° to 90° knee flexion. It is clear that these simulated pathologies had no practical effect on medial patellar stability.

Discussion
This paper has presented data which we believe to be the first attempt to demonstrate the relative effects of various abnormalities on patellar stability. Although a loss of tension from the VMO had an effect on lateral stability throughout knee flexion, this was overshadowed by the greater effect caused by flattening the lateral facet of the trochlea from 0° to 60° knee flexion. In the extended knee a rupture of the medial retinacular structures had the largest effect.

In addition to studying lateral displacement of the patella, which causes most clinical problems, this study also displaced the patella medially and this is the first time that this has been studied. Medial patellar subluxation may occur as a result of surgical overcorrection of lateral malalignment. The retinacular, muscular and articular abnormalities studied have all been related to lateral patellar instability. It was not surprising to find that they had no significant effect on patellar medial stability. There is published data showing that division of the lateral retinaculæ reduces lateral patellar stability.

If the VMO was relaxed, the force required to displace the patella 10 mm laterally was reduced approximately 30% between 20° and 90° knee flexion. In the extended knee, this loss of stability reduced to 14% which was an unexpected finding. The VMO tension acts both medially and posteriorly and is known to resist lateral patellar displacement. It has been noted that the VMO is the first part of the quadriceps to weaken, and the last to be rehabilitated. Clinical observations linking loss of active full knee extension to VMO atrophy have led to the belief that VMO has its main role in the last 15° of active knee extension. Many conservative treatments are based on this hypothesis. In contrast, the present study found the smallest effect of VMO relaxation was in terminal knee extension and a number of electromyographic studies have failed to find selective vastus medialis activity in this arc. In summary, though, this experiment showed that a quadriceps imbalance caused by loss of VMO tension caused a significant reduction in lateral patellar stability throughout knee flexion. However, without knowing how this effect compared with those of the retinacular or articular geometry, we cannot conclude that VMO is the most important patellar stabiliser.

If the lateral facet of the trochlea had its slope reduced to zero, the force required to displace the patella laterally fell 70% at 20° knee flexion. This reduction was the largest single effect in these experiments in the arc of flexion where dislocations occur most often in life. In the normal knee,
the combined effect of the patellar tendon and quadriceps tensions is to pull the patella posteriorly and laterally, and this is resisted by the slope of the lateral trochlear facet.\textsuperscript{26,27} Mechanically, therefore, it is clear that a loss of that slope will lead to lateral patellar displacement occurring more readily. In a lateral radiograph of a normal knee the line of the base of the trochlear groove remains within the outline of the anterior femoral lateral condyle, which is the edge of the trochlea. However, if the trochlea is dysplastic and is flat mediolaterally, the base of the groove meets the outline of the condyle on the radiograph. This is the ‘crossing sign’ described by Dejour et al\textsuperscript{28} who saw it in 96\% of their cases with objective patellar instability and in only 2\% of their controls. As the trochlear groove becomes flatter, so the sulcus angle seen on a ‘skyline’ radiograph increases. This angle has been correlated with the severity of other features of extensor mechanism dysplasia.\textsuperscript{29} In this study, the slope of the lateral trochlear facet was 22° ± 5° (mean ± SD) at the contact zone for 20° knee flexion.\textsuperscript{30} This was reduced to 0° in all of the knees when testing the effect of trochlear geometry on lateral patellar stability.

If the medial passive restraints were ruptured by displacing the patella laterally, the force later required to displace the patella 10 mm laterally was reduced 49\% in the extended knee, and to a decreasing extent as the knee flexed. Figure 5 shows how the effect of the medial retinaculum increased rapidly in the last 20° of knee extension. Previous cutting studies have found that the medial patellofemoral ligament was the single most important passive restraint to lateral patellar displacement at or close to full knee extension.\textsuperscript{13,15} The role of these structures has not been studied previously at other angles of knee flexion. As the knee reaches full extension, the patella moves proximally out of engagement with the trochlear groove, and becomes mobile mediolaterally when the muscles are relaxed. Tensing the quadriceps normally pulls the patella proximally and slightly laterally, and patellar mobility is lost. The loss of this stability after rupturing the medial retinaculum suggests that the retinaculae are tensed by the proximal movement of the patella, with a mechanism akin to pulling up the ridge of a tent until the guy ropes become tight on either side.

Although these experiments have provided information about the relative contributions of several distinct simulated pathologies to loss of patellar stability, the limitations of working \textit{in vitro} should be kept in mind. The most serious of these relates to the use of isolated joints, rather than studying the knee as an integral part of the functioning, loaded, lower limb. In life, the symptoms of patellar instability often appear as a sudden dynamic event which cannot be simulated. This work measured the underlying inherent stability of the patella onto which sudden loading or moving events may be superimposed \textit{in vivo}. It is this inherent stability that controls whether the patella subluxes easily. The majority of joint force \textit{in vivo} is a reaction to the muscle tensions. These were limited in this work because we knew from prior experience\textsuperscript{16} that higher tension caused tearing of the loading cables from the muscle heads.

Previous work\textsuperscript{31} has shown that the results will not be affected greatly by changes in overall muscle force. In addition, the muscle tension was constant at all angles of knee flexion, whereas in life the tension increases as we squat down. This may have affected the relative VMO contribution in the flexed knee in this work. The complete relaxation of VMO in this study probably caused its maximum effect, compared to partial weakness in vivo. The modification of the trochlear geometry was a reverse of the Albee procedure\textsuperscript{32} that has normally been used to treat trochlear dysplasia. However, there may also be abnormalities of the soft tissues and of limb alignment in such cases \textit{in vivo} which could not be simulated in this work. The advantages of working \textit{in vitro} include the ability to control the mechanics accurately, and then to make paired, within-specimen, comparisons of the effects of the simulated pathologies. This study provides information that will help to plan objective studies \textit{in vivo}.

The quantitative results of these experiments have provided objective evidence relating to the relative effects of simulated abnormalities for the muscles, joint geometry or retinaculae on mediolateral patellar stability. Figure 6 shows clearly that there was a complex interaction between them, with their contributions to loss of lateral patellar stability varying with knee flexion. Although non-surgical treatment is always preferable, the results suggest that the role of VMO may be less important than abnormal trochlear articular geometry, and that the medial retinaculum become more important for patellar stability as the knee extends. In addition, Figures 3 to 5 show that the patella was least stable at 20° knee flexion in the normal knee and that continued to be the case with each of the three simulated pathologies.

The Instron material testing machine was donated by the Arthritis Research Campaign (ARC). Dr Senavongse was supported by the Thai government and a project grant from the ARC.

No benefits in any form have been received or will be received from a commercial party related directly or indirectly to the subject of this article.

References


