(i) Biomechanics of the knee joint

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Abstract
The knee joint has biomechanical roles in allowing gait, flexing and rotating yet remaining stable during the activities of daily life, and transmitting forces across it. Geometrical, anatomical and structural considerations allow the knee joint to accomplish these biomechanical roles. These are addressed and discussed in this article.

Keywords biomechanics; joint; knee; kinematics; ligaments; tissue mechanics

Introduction — basic function of the knee joint

The knee joint comprises two distinctly separate joints/articulations; the tibiofemoral (TF) joint — the articulation of the femur over the tibia — and the patellofemoral (PF) joint — the articulation of the patella over the femur. The main roles of the knee joint complex are (1) to allow locomotion with (a) minimum energy requirements from the muscles and (b) stability, accommodating for different terrains, and (2) to transmit, absorb and redistribute forces caused during the activities of daily life.

Clinical interest in the knee joint stems from the frequency of injury and pain resulting from degenerative changes, and there are mechanical factors that are associated with this. The aim of this article is to review the knee joint from a clinically driven mechanics viewpoint.

The knee joint acts as a pivot between the two longest bones in the human body whilst the strongest muscles in the body (the quadriceps muscles) act across it. The TF joint has a wide range of motion, reaching up to 160° of flexion (rotation in the sagittal plane), with coupled rotations in the other two planes; this leads to incongruency between articulating surfaces across part of the range of motion. Similarly, the PF joint has a complex, three-dimensional range of motion across TF joint flexion in order to allow for minimal quadriceps contraction to extend the knee. This complex mechanism of knee joint motion means that the geometry itself is not adequate to maintain stability, requiring input from passive soft tissue (e.g. ligaments) and muscle tensions. It also means that large forces acting on small articulating areas generate high articular stresses, commonly called joint contact pressure. The complexity of knee joint behaviour is a result of the individual behaviour of and interaction between three different factors:

- static stability — geometry and anatomy of the joint surfaces.
- active stability — muscle contraction.
- passive stability — ligaments, menisci and retinacula.

Knee joint kinematics

The primary motion of the TF joint is rotation in the sagittal plane (flexion—extension); therefore a simplified description is that it acts as a hinge joint in the sagittal plane. The complex three-dimensional motion of the TF joint can be described through six degrees of freedom (three rotations and three translations) in a clinical joint coordinate system (Figure 1). Full extension (i.e. 0° flexion) is usually defined when the long axes of the tibia and femur are aligned in the sagittal plane. Active knee flexion is possible primarily through hamstring contraction and usually reaches 130°, whereas passive flexion can reach 160°. Some individuals can hyperextend (i.e. negative flexion) to up to −5° flexion. When at full extension, the knee allows for optimized weight support and stability. If the body was at rest in some other angle of flexion, then the vertical line of action of the ground reaction force would pass posterior to the knee joint, which would mean that the quadriceps muscles would need to do work to maintain posture; this would be energetically...
inaccurate, hence tiring, and the muscle force would increase the load on the TF joint. Thus, flexion contracture is disabling. Similarly, hyperextension becomes stable only because of large tensions in the posterior capsular structures when the line of action of the body weight passes anterior to the flexion–extension axis of the knee.

The most frequent knee movement occurs during gait. Figure 2 shows TF joint (knee) flexion in gait. Knee motion is dictated by energy considerations that require the centre of gravity of the body to move forward with minimal other upper body movement (up–down or medial–lateral), and by optimal capacity to absorb the impact at heel strike. In order for the toes of the swinging leg not to be dragged on the ground, the knee flexes up to approximately 70° during the swing phase of gait. When the leg has swung past the other and just before heel strike the quadriceps muscles contract to bring the knee to full extension and the foot forwards. After heel strike the knee does not remain extended as this would mean upward movement of the body and the leg acting as a rigid strut unable to absorb the impact load; therefore, the knee joint flexes up to 15° in mid stance phase by stretching the quadriceps muscles, thus absorbing energy.

Flexion is not the only rotation that takes place during gait. Prior to heel strike, as the knee extends from approximately 30 to 0° flexion, the tibia externally rotates by up to 30°. This is known as the ‘screw-home’ mechanism and is thought to occur in order to tighten the soft tissue structures and lock the knee geometry prior to accommodating the impact load of weight bearing. This rotation is linked to corresponding rotations at the hip and ankle. For example, immediately after heel strike the knee flexes hence the tibia rotates internally, a motion that is coupled with foot eversion; this leads to deformation (hence “softening”) of the foot structures that allows for energy absorption.

Articular kinematics

The geometry of the articulation and the actions of the passive and active stabilizers define the path of articulation. The outline of the femoral condyles in the sagittal plane is longer than the anterior–posterior dimension of the tibial plateau (Figure 3). This means that if flexion was occurring purely by a rolling motion, then the femur would roll off the tibial plateau well before the knee reached full flexion. At full extension the femur has a large contact area with the tibial plateaus and presses anteriorly on the meniscal horns. As the knee flexes, contact moves posteriorly towards the posterior meniscal horns and the contact area with the tibial plateaus is reduced as lesser radii of curvature of the femoral condyles are sequentially coming into contact. Tibial rotation occurs during flexion (the reverse of the screw-home); the medial tibial plateau is slightly concave whereas the lateral tibial plateau is flat or slightly convex in the sagittal plane; this means that the centre of contact in the medial side remains relatively constant in terms of anterior–posterior position, but the lateral condyle rolls posteriorly towards the posterior horn of the mobile lateral meniscus until the meniscus starts resisting further translation by stretching circumferentially. Thus, tibial rotation is essentially occurring about a medial axis. From then on the femoral condyles have almost circular sagittal sections and the anterior cruciate ligament (ACL) is in tension, resisting further posterior translation of the femur relative to the tibia; this results in the femur sliding anteriorly and rolling posteriorly at the same time. In deep flexion contact occurs almost solely between the femoral condyles and the posterior meniscal horns with very little cartilage-to-cartilage contact, especially in the medial side where the meniscus is constrained from displacing further posteriorly off the tibial posterior rim; further tibial internal rotation occurs as well (up to approximately 15°), which causes the posterior horn of the lateral meniscus to displace off the posterior rim of the tibial plateau.

The PF joint articulation occurs between the patella and the femoral trochlear groove. The complex articular surface of the patella can be roughly split into lateral (larger) and medial slightly concave facets of congruent shape to that of the femoral trochlea when the knee is flexed; these facets are separated by a convex ridge. At full TF joint extension the PF joint contact occurs at the distal end of the patella (Figure 4a). As flexion increases the patella engages into the femoral trochlear groove and the contact area spreads across the width of the patella and moves proximally (Figure 4b). In deep flexion contact occurs only laterally and medially on the patella; the lateral facet articulates on the distal aspect of the lateral femoral condyle, and the ‘odd’ medial facet contacts against the medial femoral condyle at the edge of the intercondylar notch. The increase of PF contact area with knee flexion is a clever mechanism that controls the magnitude of stresses by spreading the ever increasing PF joint load with knee flexion over a larger area. Analysis of the patellar kinematics requires all six degrees of freedom to be accounted for as the three-dimensional movement is complex. The reader is referred to textbooks for more detail, but the most frequent clinical problems relate to abnormal medial–lateral translations and rotations (patellar tilting) during knee flexion–extension. These abnormalities can result from factors including a shallow trochlear groove, inadequate passive soft tissue restraints, or abnormal balance of tensions between the components of the quadriceps—especially deficient vastus medialis obliquus tension.

Loading and articular mechanics

The loads that the joint surfaces experience are a result of external forces (for example ground reaction forces) and the muscle forces that are required to maintain posture and facilitate body movement. Ligament forces are passive internal forces...
Figure 3 Knee joint kinematics in the sagittal plane during gait. a Extension: contact is located centrally. b Early flexion: posterior rolling; contact continuously moves posteriorly. c Deep flexion: femoral sliding; contact is located posteriorly; the unlocking of the ACL prevents further femoral roll back.

PF joint force at a extension and b 90° flexion, showing geometrically the increase of PF joint force with flexion. (PT: patellar tendon, Q: quadriceps muscles, PF: patellofemoral, TF: tibiofemoral).

MINI-SYMPOSIUM: SOFT TISSUE SURGERY IN THE KNEE

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which are developed in response to joint motion or external loading and provide alternative pathways for load transmission through the joint. The anatomical and structural complexity of the knee joint means that forces within the different structures cannot be measured with accuracy during daily activities. Attempts to measure such forces require both experimental data and numerical modelling that utilize optimization algorithms which minimize energy expenditure. A seminal study in the late 1960s estimated TF joint forces of 3.4 body weight (BW) when walking, 4.3 BW when ascending stairs and 4.0 BW when descending a ramp,\(^4\,^5\) whilst a later study calculated up to 8.5 BW TF joint forces when walking downhill.\(^6\)

In the PF joint, when the knee is near extension, the lines of action of the patellar tendon (PT) and quadriceps muscle (Q) are almost co-linear in the sagittal plane, resulting in a small joint force (Figure 4a). However, as the knee flexes, the angle between the lines of action of the PT and Q reduces, resulting in an ever increasing PF joint force up to approximately 70° knee flexion, where the PT tension is approximately 70% that of Q (Figure 4b). This is partially accommodated for by the geometry of contact; as aforementioned, the contact area between the femur and the patella increases with knee flexion, therefore maintaining physiological contact stresses (since stress = force/area). Beyond 70° knee flexion, the quadriceps wraps around the trochlea, so this effect does not increase.

A daily activity that generates large knee joint forces and could be injurious to a sensitive population like the elderly is rising up from a chair (Figure 5). This requires up to 100° of knee flexion to bring the body to the upright position and, hence, entails maximal quadriceps tension. The line of action of the BW is approximately 200 mm behind the knee. In order to maintain equilibrium between the flexing and extending moments, and assuming that half of BW is transferred through each leg, the PT tension needs to reach approximately 3 BW, resulting in a TF joint force of approximately 3.5 BW and a PF joint force of approximately 5.5 BW. Thus, although the PF joint is not interposed in the weight-bearing structure of the leg, it cannot be taken to be less heavily-loaded than the TF joint.

The discussion on joint loading up to now has been limited to the sagittal plane. It has been shown that there is an adduction...
moment in the TF joint in the frontal plane when walking. The normal leg has approximately 6° valgus angle between the anatomical axes of the femur and tibia in the frontal plane in the extended knee, whereas the mechanical axis — which joins the centres of the hip, knee and ankle — is approximately straight. Hip adduction is needed to position the foot of the load-bearing leg under the BW in the stance phase of gait. Thus, the line of action of the BW passes medial to the centre of the medial femoral condyle, which means that all compressive joint force should be transferred through the medial condyle. However, the quadriceps muscles act also to extend the knee during stance, and their line of action passes through the middle of the joint, therefore balancing the load between the two compartments to some extent. It has been shown that the line of action of the TF joint force is located at the medial compartment and moves by up to 40 mm in the medial–lateral sense during gait, with knee stability maintained by tension in the ilio-tibial tract. Thus, the medial condyle force is larger than the lateral, and this is reflected by the relative areas and congruence of the two articulations.

Looking at the extended PF joint in the frontal plane, there is a resultant lateral force acting on the patella stemming from the lines of action of the PT and Q; this is often referred to as the Q-angle effect (Figure 6). The Q-angle is defined as the angle between the line of action of the PT and the resultant line of action of the quadriceps muscles; it is approximately 12–15° in males and 15–18° in females in the extended knee and reduces with flexion due to reversal of the screw-home mechanism, which causes medialization of the tibial PT attachment (the tibial tubercle). The Q-angle effect tends to sublux the patella laterally. This is resisted geometrically by the depth of the trochlear groove, with the sulcus angle being the single best predictor of symptoms of instability. Thus, trochleoplasty to deepen the groove in a dysplastic knee is mechanically logical and increases objective patellar stability significantly. The patella is most unstable at approximately 10–30° of knee flexion, with a Q-angle effect, albeit smaller than in full extension, is resisted solely by the soft tissues (the retinacular structures).

Articular mechanics
Looking closer into the mechanics of the articulation, the forces discussed above are transferred to the articulating surfaces and can be split into compressive and frictional/shear components. The compressive force is distributed over the contact area of the articulation to result in contact stresses within the structures involved. The magnitude of these stresses depends on both the magnitude of the force and the contact area. The articulating geometry does not allow for full conformity between the compartments in contact in TF or PF joints, and therefore is not ideally designed to minimize contact stresses. In the PF joint this is accommodated for by the thick articular cartilage (the thickest in the body) and an ever increasing contact (conforming) area with knee flexion up to approximately 90° flexion (as aforementioned, the joint force increases with knee flexion). In the TF joint this is accommodated for by the presence of the medial and lateral menisci. These are crescent-shaped fibrocartilages, wedge-shaped in cross section. Their tissue includes strong collagen fibres running primarily in a circular manner around their periphery. They sit on the rim of the tibial plateau and are

![The knee joint in the frontal plane.](image)

**Figure 6**

The knee joint in the frontal plane. **a** The Q-angle is defined as the angle between the line of action of the patellar tendon, PT and the line of action of the resultant quadriceps muscles, Q. The Q-angle effect results in a lateral force on the patella, L. **b** The quadriceps muscle force results from summing direction and magnitude of the quadriceps components acting on the patella. The lines of action of each component lie on the frontal plane, except for the oblique muscles, which describe an angle with the sagittal plane as well (RF: rectus femoris, VI: vastus intermedius, VML: vastus medialis longus, VMO: vastus medialis obliquus, VLL: vastus lateralis longus, VLO: vastus lateralis obliquus).
attached to the tibia primarily through insertional ligaments at their horns, therefore allowing for increased mobility within the articulation.

As the femur compresses the menisci onto the tibia, the menisci conform to the femoral condyles and are squeezed out radially; this causes them to stretch circumferentially. The tissue is held by the insertional ligaments at the bone attachments and hoop (circumferential) tension develops in the menisci to resist further deformation (Figure 7). The TF contact area is increased by the menisci, thereby decreasing the contact stress on the articular cartilage and minimizing the cartilage-to-cartilage contact. The insertional ligaments are key in this function, as they act as anchors to the tibial plateau; loss of an insertional ligament would mean complete loss of the load-bearing function of the menisci.9

The mobility of the menisci allows them to maximize the degree of conformity of the articulation across knee flexion. In addition to the insertional ligaments, meniscal motion is defined by the geometry of the articulation; as discussed previously, as the knee flexes the contact moves posteriorly towards and onto the posterior meniscal horns. Furthermore, the tibial plateau is concave on the medial side but flat or slightly convex on the lateral side; this means that the lateral meniscus goes ‘downhill’ with knee flexion, whereas the medial meniscus is squeezed onto the tibial rim. This in turn results in larger posterior translations of the lateral meniscus compared to the medial meniscus.

The importance of the menisci in increasing the contact area, hence reducing the contact stresses on the articular cartilage, is supported by the prevalence of osteoarthrosis after meniscectomy.10 The load-bearing role of the menisci is a result of their geometry, microstructure and attachments to the tibia; if any of those three elements is disrupted, then meniscal function will be hampered. For example, a circumferential tear, which is a split in-between the circumferential fibres, will not affect their load-bearing role; however, a radial tear that cuts across the load-bearing fibres or an insertional ligament rupture will de-function the tissue.

As in any motion that involves loaded surfaces sliding over one another, frictional forces develop during knee joint motion. Human joints are similar to mechanical bearings that transfer forces between body segments and it is remarkable that they undergo millions of cycles of loading without showing symptoms of wear. This is due to the joint lubrication that the synovial fluid provides. The presence of synovial fluid means that contact between the bearing surfaces is reduced. When knee motion entails high velocities and light loads, a film of fluid is present between the surfaces, and that ensures very low friction. When short-term impact loads are involved (as seen in heel strike), the fluid is trapped between the surfaces as there is not enough time or space for it to escape, thus generating a “squeeze film” effect which protects the surfaces from direct contact and assists in distributing and absorbing the shock load. In the case whereby the squeeze film effect is insufficient the surfaces are protected at a molecular level by the large protein molecules contained in the synovial fluid; this molecular layer acts as a boundary lubricant.11

**Soft tissue mechanics**

The soft tissues provide dynamic stability to the knee joint. The muscles load the joint according to the required movement and provide active stability. Ligaments, menisci and retinacular structures provide passive stability by resisting excessive...
displacements between the bony segments and control the path of motion of the joint. When external loads are applied to the joint the ligaments stretch and develop tensile force that resists further elongation, thus tethering the joint and maintaining stability. The joint is more stable when loaded under compression, whereby the articular surfaces are pushing against each other, therefore directly transmitting the load from one bony segment to the other (as opposed to transmission via insertions of passive stabilizers). Disruption of a passive restraint may lead to symptomatic “instability” of the knee, when the bones move uncontrollably in relation to each other. When the joint is displaced in a controlled manner, in an anterior drawer test, for example, there will be a tendency for abnormally large displacement due to the applied force, also termed as excessive joint laxity. Laxity may be defined as either the displacement due to the applied force, also termed as excessive 'coupled' motions (such as the tibial internal rotation which accompanies anterior drawer) are usually ignored.

During a laxity test on the knee, more than one passive stabilizer is utilized to maintain stability. For example, the primary restraint to anterior drawer is the ACL, but secondary restraints are employed such as the menisci and the medial collateral ligament (MCL), especially at large displacements. The primary restraint is the tissue that is better aligned to the applied displacement (or force line of action), hence is stretched directly and develops a greater tensile force than the secondary restraints to resist the displacement. However, if a primary restraint is injured, the secondary restraints will have to resist the same displacement as before without being as well aligned as the primary restraint had been; this means that they need to stretch a lot more than before in order to generate the tensile force required to maintain the same level of laxity. Therefore, although knee joint laxity might remain largely unaffected after a primary restraint rupture due to the presence of secondary restraints, the latter are naturally overloaded; this might lead to further soft tissue damage, or ‘stretching-out’.

Our present understanding of the function of the main knee joint ligaments in restraining applied joint displacements is summarized in Table 1 and in the following paragraphs (Table 1).

### Cruciate ligaments: the ACL is the primary restraint to anterior tibial translation. It also prevents hyperextension, acts as a secondary restraint to internal and valgus tibial rotations at full extension and controls the screw-hone motion of the knee. The posterior cruciate ligament (PCL) is the primary restraint to posterior tibial translation, being most effective in mid to deep flexion; near extension the PCL slackens and the posterolateral structures take over the role of primary restraints. Acting together, the ACL and PCL control the anterior—posterior rolling and sliding kinematics of the TF joint during flexion—extension.12

### Medial collateral ligament and posteromedial corner: the medial collateral ligament (MCL) is the primary restraint to valgus angulation and internal tibial rotation and a secondary restraint to external tibial rotation. It is also a secondary restraint to anterior tibial translations when those are large or when the tibia is externally rotated. The posteromedial corner (PMC) acts as a stabilizer to the extended knee, especially in the presence of concurrent internal tibial rotation and posterior drawer.13

### Lateral collateral ligament and posterolateral corner: the lateral collateral ligament (LCL) is the primary restraint to varus angulation, but slackens with knee joint flexion, which reduces its restraining capability. It is a secondary restraint to posterior translations when those are large and, combined with the posterolateral corner (PLC), is a primary restraint to external tibial rotation.14

### The meniscus–meniscal ligaments construct: the meniscus—meniscal ligament complex (meniscus, meniscal insertionligaments, meniscofemoral ligaments (MFLs) and deep MCL (dMCL)) acts as a primary restraint to tibial rotation and as a secondary restraint to anterior—posterior translation. Specifically, the MFLs are significant restraints to posterior drawer and the dMCL is a secondary restraint to valgus at 60–90° flexion.9

### Patellar retinacula and the medial patellofemoral ligament: lateral and medial retinacular structures are the passive stabilizers of the PF joint. The lateral retinacular tissue consists of several functional units that are on the one hand implicated in causing patellar lateral tilt if they are pathologically tight and on the other restraining the patella from lateral dislocation in normal use. The strong transverse attachment from the patella to the ilio-tibial tract is tightened as the knee flexes, while the capsular lateral patellofemoral ligament is tightest in extension.15

The main functional unit of the medial retinaculum is the medial patellofemoral ligament (MFL). It acts as the primary passive restraint to lateral patellar displacements and assists in controlling patellar motion, especially guiding the patella into the trochlear groove in early knee flexion. If the patella dislocates

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**Table 1**

<table>
<thead>
<tr>
<th>Applied displacement</th>
<th>Flexion angles (°)</th>
<th>Primary restraint</th>
<th>Secondary restraint</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior drawer</td>
<td>0</td>
<td>ACL</td>
<td>ITB, MCL, LCL, menisci</td>
</tr>
<tr>
<td></td>
<td>30</td>
<td>ACL</td>
<td>MCL</td>
</tr>
<tr>
<td></td>
<td>60—120</td>
<td>ACL</td>
<td>Menisci, MCL</td>
</tr>
<tr>
<td>Posterior drawer</td>
<td>0—30</td>
<td>PLC, PMC</td>
<td>aPCL, LCL, MFLs</td>
</tr>
<tr>
<td></td>
<td>40—120</td>
<td>aPCL</td>
<td>pPCL, MFLs</td>
</tr>
<tr>
<td>Varus rotation</td>
<td>0—60</td>
<td>LCL</td>
<td>ITB, PCL</td>
</tr>
<tr>
<td>Valgus rotation</td>
<td>0</td>
<td>MCL, PMC</td>
<td>ACL</td>
</tr>
<tr>
<td></td>
<td>30</td>
<td>MCL</td>
<td>ACL</td>
</tr>
<tr>
<td></td>
<td>60</td>
<td>MCL</td>
<td>Menisci, MCL</td>
</tr>
<tr>
<td>External tibial rotation</td>
<td>0—90</td>
<td>LCL, PLC</td>
<td>Menisci, MCL</td>
</tr>
<tr>
<td>Internal tibial rotation</td>
<td>0—30</td>
<td>PMc</td>
<td>ACL, MCL, menisci</td>
</tr>
<tr>
<td>Hyperextension</td>
<td>60</td>
<td>MCL</td>
<td>ACL, menisci</td>
</tr>
</tbody>
</table>

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latterly, or can be subluxed abnormally far laterally, then the MPFL must have been damaged.\textsuperscript{15}

**Conclusion**

This exposition has shown that both the TF and PF joints are subjected to large compressive forces during normal activities, principally as a result of the muscles having their lines of action passing close to the axis of TF joint flexion—extension. This mechanical disadvantage means that large muscle tensions are needed to resist the externally-applied loads. At the same time, the exact paths of motion of the bones while the knee is flexing—extending depend critically on the interactions of the ligaments, and this article has shown that there will be a hierarchy of importance in resisting specific bone—bone displacing loads. By imposing the loads which act on each of the main ligaments in turn, damage to these primary restraints can be diagnosed. Finally, the role of the menisci in act on each of the main ligaments in turn, damage to these primary restraints can be diagnosed. Finally, the role of the menisci in spreading the load-bearing areas of the TF joint is clear, and so the hugely increased prevalence of TF joint degenerative changes that follows loss of meniscal function is unsurprising.

Taken together, these observations draw a picture of many structures acting in harmony to maintain normal knee function, while knowledge of their specific roles should aid in diagnosis and evaluation of treatment.

**REFERENCES**


**Nomenclature**

*BW:* body weight;  
*TF:* tibiofemoral;  
*PF:* patellofemoral;  
*PT:* patellar tendon;  
*Q:* quadriceps muscle;  
*ACL:* anterior cruciate ligament;  
*PCL:* posterior cruciate ligament;  
*aPCL:* anterior bundle of the PCL;  
*pPCL:* posterior bundle of the PCL;  
*MCL:* medial collateral ligament;  
*dMCL:* deep medial collateral ligament;  
*LCL:* lateral collateral ligament;  
*ITB:* ilio-tibial band;  
*PMC:* posteromedial corner;  
*PLC:* posterolateral corner;  
*MFLs:* meniscofemoral ligaments;  
*MPFL:* medial patellofemoral ligament.