

Pelvic stability

influences lower-extremity kinematics

The functional interactions between the intrinsic biomechanics of the knee and the ankle and foot have been well researched and studied.^{1,2} In comparison, little research and few in-depth studies explore the biomechanical impact an unstable pelvis can have on lower-extremity kinematics and pathomechanics.

Movement, stability, and dynamic control of the hip and knee are results of joint-to-joint configuration, forces created by muscles crossing these joints, forces affected by weight-bearing and dynamic-resistance status, and the position of the hip. The most important contributing influence on the knee and patellofemoral joint is pelvic position.

Rotary alignment of the long axis of the femur to the hip is dictated by femoral version.³ Anteversion of the femoral head, associated with inclination of the pelvis, makes it possible for flexion movements of the hip joint to be transposed into rotary movements of the head of the femur.⁴ Thus these rotary movements of the femur depend largely on acetabulum position, compression of the femur in the acetabulum from muscle activity during open kinetic chain (OKC) activities, and from weight-bearing during closed kinetic chain (CKC) activities. Kinetic rotational direction of the femur is guided and limited by the rotation and tilt of the pelvis, and by pelvic femoral soft tissue (Figure 1). An anterior tilt or

rotation of the pelvis promotes overuse of the hip flexors during beginning stance and swing phases of gait, as well as forward pelvic rotation, femoral internal rotation, medial displacement of the femoral range of rotation, genu valgus, genu recurvatum, subtalar eversion, and forefoot or rearfoot pronation (Figure 2).

An anteriorly positioned pelvis, increased lordosis of the lumbar sacral spine, increased extension of the thoracic lumbar spine, and increased hip flexion lead to pathokinesiologic effects on the knee. These usually begin with an imbalance of strength between the abdominal obliques and the rectus femoris

Pelvic position is the foremost contributing influence on patellofemoral joint biomechanics.

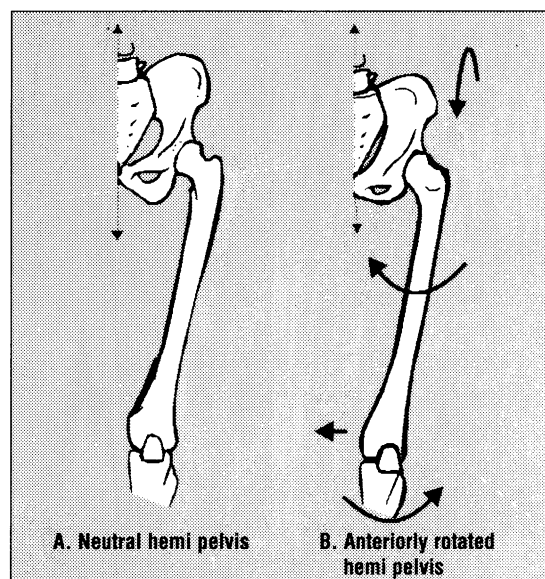


FIGURE 1A and 1B. An anterior tilt of the pelvis promotes overuse of the hip flexors and displacement or rotation of all subsequent elements in the kinetic chain.

and psoas. Dysynchronism of antagonistic muscles contracting synchronously, when the antagonistic muscle should not be contracting, also contributes to lumbopelvic instability. For example, if the external obliques contract with the paravertebrals upon back extension, pelvic floor stability during hip flexion activities is compromised. Hypertonic, lengthened hamstring musculature that contracts during knee extension, providing dynamic eccentric stability for the hip, can be torn.

Dysynchronism

Dysynchronism versus asynchronism is associated with both an imbalance of agonistic-to-antagonistic strength and a mismatch of gravitational, compressive, or rotational forces. Dysynchronism may also lead to passive biomechanical malalignment and active movement dysfunction. Configuration of movement, accuracy of movement (coordination), repeatability of movement (proprioception), alteration of movement (balance), and maintenance of end position achieved by movements (posture) are all influenced by dysynchronism and strength imbalance.

The posterior rotators of the hip play a role in the most influential muscle weakness pattern associated with anterior knee pathology and lateral femoral patellar compression. The hamstrings, as a group, are the only dynamic stabilizers of the knee and, along with the phasic abdominal obliques, transverse abdominals and gluteals, stabilize the pelvis on dynamic demand. This weak and lengthened musculature, along with joint instability at the pubis and sacroiliac joints, can create trunk, pelvic, hip, knee, and ankle-and-foot instability. Instability of the pelvis promotes ligamentous laxity of the knee, exaggerated Q angles, genu capsular strain, overcompensation by the paravertebrals, iliopsoas, and hip abductors for postural support, pubic shear, and gait overstride.

The most important movement of the hip is internal and external rotation, which activates receptors of the inferior and superior portions of the capsule. In cases of postural spondylolisthesis, exaggerated lumbar lordosis, or an anterior pelvic tilt, external rotation of the femur is limited and neuromuscular control of iliofemoral rotation is dampened. Since postural influences are processed through brain-stem motor integration, dampened afferent proprioceptive orientation of the hip and knee may produce abnormally high reflex muscular splinting, and the resultant destabilization may increase propensity for injury.

Joint afferent fiber activity also assists the muscle spindle afferent fibers in inhibiting antagonistic muscle activity under conditions of rapid lengthening and associated periarticular tissue distortion, both of which accompany unexpected postural perturbations. When input arises simultaneously from cutaneous tissue, joint muscle, and vestibular receptors, a unique pattern of afference is generated. Elicited long-latency, involuntary postural adjustments then occur. This acts to stabilize the body and bring its center of gravity into a state of equilibrium. If a state of equilibrium or balance is abnormal secondary to abdominal oblique, gluteal, and hamstring muscle weakness, afferent input may activate antagonistic muscle and periarticu-

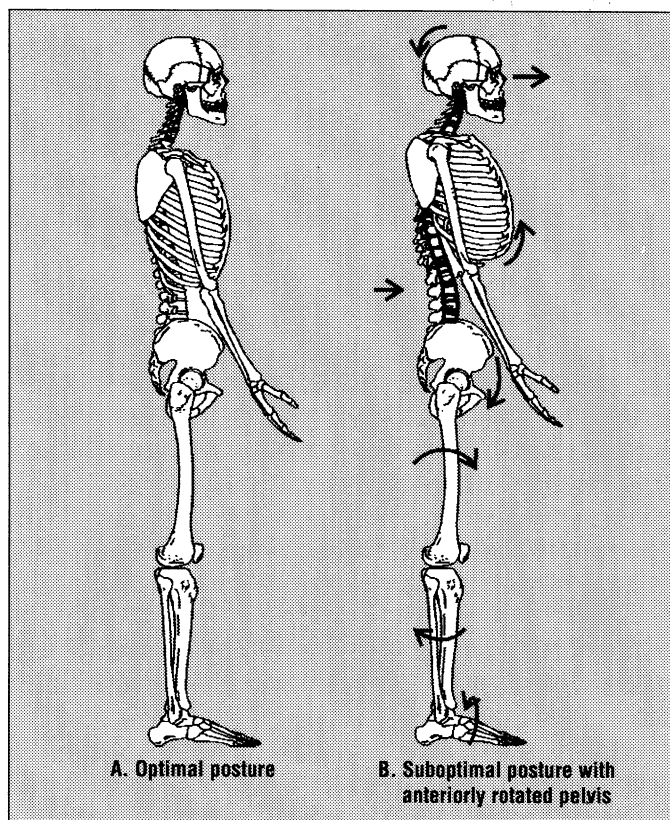


FIGURE 2A and 2B. The sequence of displacements from an anterior pelvic tilt includes forward pelvic rotation, femoral internal rotation, medial displacement of the femoral range of rotation, genu valgus, genu recurvatum, subtalar eversion, and forefoot or rearfoot pronation.

lar tissue on rapid lengthening. This may increase the chances for anterior cruciate ligament (ACL) rupture, meniscal perforation, or a collateral tear. Full range of motion at the hip and knee is therefore limited because of decreased iliofemoral and femorotibial ligament kinesthetic awareness secondary to guarded, restricted, and compensatory activity.

From midstance to toe-off, an anteriorly positioned pelvis and an internally rotated femur also increase the stress on the popliteus and ACL, since this muscle and ligament restrict excessive external rotation of the tibia on the femur. In this situation, forefoot push-off is enhanced by ACL and medial collateral ligament laxity, since more tibial external rotation is needed under a femur or hip that will not rotate externally. This is often referred to as excessive version of the knee and is defined as the static rotation of the femur with respect to the tibia, in full knee extension.^{5,6} If the ligaments, capsule, and derotational musculature of and around the knee do not allow compensatory movement for rotation lost at the iliofemoral joint (secondary to anterior pelvic inclination and pelvic instability), torsion through the tibia will result in tibial stress fractures, provided there is pronation control offered by a shoe with good subtalar support.²

Anterior orientation of the pelvis on the femur and the accompanying compensatory external rotation of the femur at rest or during gait decreases the length/tension ratios and mechanical action of the iliopsoas, gluteus maximus, piriformis,

quadratus femoris, sartorius, pectineus, adductor longus, and adductor magnus, since these muscles are aligned in a shortened position.

The psoas has the greatest difficulty in externally rotating the femur as it originates at an osseous site that has come forward and down with respect to its neutral alignment. External rotation of the femur and tibia at midstance, terminal stance, and preswing is limited in extreme hip flexion, since muscle function of the hip is dependent on its position in the range (of motion) and the availability of motion of the proximal and distal segments. Lateral rotators (iliopsoas, pectineus, and adductor magnus) of the hip joint become medial rotators from a position of extreme medial rotation of the femur or on extreme hip flexion. Adductors that are flexors in the neutral hip joint may become extensors in the flexed hip joint.

Rethinking knee pain

Traditionally, clinical attribution of patellofemoral pain is to patellar or knee extensor mechanism malalignment.⁷ Angulation in the coronal plane (genu valgus) or rotation in the transverse plane (femoral anteversion) has been studied extensively.⁸ The intuitive assumption that the patella is tracking abnormally is supported by Q angles, patellar tilt, and subluxation. However, the pathologic entity responsible for the increase in lateral patellofemoral contact force may not be the patella, but the underlying torsional direction of the femur's lumbopelvic position (Figure 3). The patella is a passive component of the extensor mechanism. It is totally dependent on the soft tissue surrounding it and the morphological conditions of the underlying femur.⁹ Biomechanical testing has confirmed that an increase in patellofemoral contact pressure occurs with "internal rotational deformity" of the femur.¹⁰ Therefore, a biomechanical basis for rehabilitation programs involving the patellofemoral joint should include strengthening and neuromuscular training programs that reposition the anteriorly rotated pelvis into a neutral static and dynamic state of control.

Although physical therapists provide stabilization training for individuals with low back problems, few take the opportunity to alter pelvifemoral pathomechanics or correct lumbopelvic instability where they exist relative to femoral patellar dysfunction. The intricate relationship between the lumbar spine, pelvis, and femur can be restored through a well defined, specific, and individualized abdominal oblique strengthening program and a hip-to-femur repositioning program.¹² A typical patellofemoral exercise program that develops quadriceps muscles can be pursued only after normal biomechanics of the hip to the femur have been restored and maintained.

Repositioning of the hip, and ultimately restoration of the alignment relationship of the femur to the patella during CKC activity, requires hamstring and abdominal oblique control of the lumbopelvic joints. Programmable resistance, independent of speed and gravity, applied to the hamstring muscle permits iso-

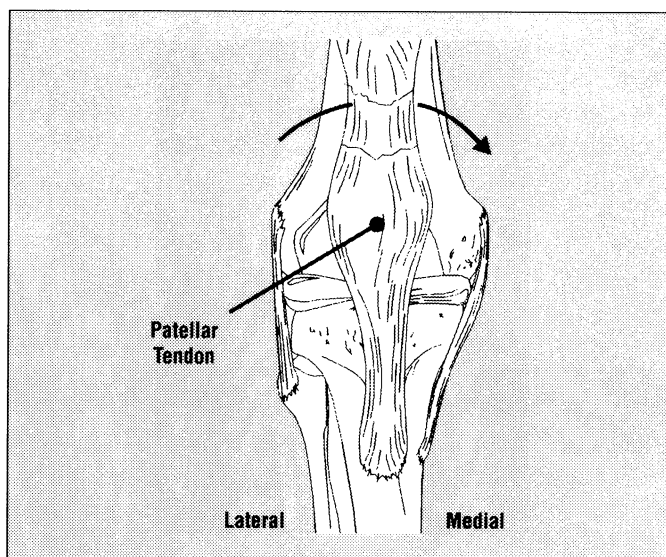



FIGURE 3. Biomechanical testing has confirmed that an increase in patellofemoral contact pressure occurs with "internal rotational deformity" of the femur.

lated hamstring contractions independent of overused, neuronal patterned hip flexors. Femoral patellar dysfunction can be reduced in individuals who show positive outcomes when their hip flexors and abductors are tested, using the Thomas and Ober testing procedures, and their passive femoral rotation is limited externally. By properly applying "feed forward" resistance to the hamstrings, normal dynamic alignment and stability of the hip can be improved, and activities that demand quadriceps development can occur without interference from knee instability or dysfunctional pelvifemoral tone.

Traditional assessment and treatment of the knee with anterior symptomology focuses on the coronal position of the patella. However, this sesamoid bone is a passive element of the extensor mechanism whose position is predetermined by the rotational orientation of the femur to the hip. By addressing limitations in hip extension, lumbar flexion, and femoral external rotation, lumbopelvic influences on pelvifemoral and femoral patellar biomechanics can be altered or in many instances alleviated. Ultimately, lumbopelvic control is needed for normal femoral patellar relationships to occur. 

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