

The Influence of Altered Lower-Extremity Kinematics on Patellofemoral Joint Dysfunction: A Theoretical Perspective

Christopher M. Powers, PT, PhD¹

Although patellofemoral pain (PFP) is recognized as being one of the most common disorders of the lower extremity, treatment guidelines and underlying rationales remain vague and controversial. The premise behind most treatment approaches is that PFP is the result of abnormal patellar tracking and/or patellar malalignment. Given as such, interventions typically focus on the joint itself and have traditionally included strengthening the vastus medialis oblique, taping, bracing, soft tissue mobilization, and patellar mobilization. More recently, it has been recognized that the patellofemoral joint and, therefore, PFP may be influenced by the interaction of the segments and joints of the lower extremity. In particular, abnormal motion of the tibia and femur in the transverse and frontal planes may have an effect on patellofemoral joint mechanics. With this in mind, interventions aimed at controlling hip and pelvic motion (proximal stability) and ankle/foot motion (distal stability) may be warranted and should be considered when treating persons with patellofemoral joint dysfunction. The purpose of this paper is to provide a biomechanical overview of how altered lower-extremity mechanics may influence the patellofemoral joint. By addressing these factors, better long-term treatment success and prevention may be achieved. *J Orthop Sports Phys Ther* 2003;33:639-646.

Key Words: knee, patella, patellofemoral, pain

Although patellofemoral pain (PFP) is a common clinical finding in a wide range of individuals,^{1,12,39} the incidence is greater in physically active populations.^{3,5,19,38} Despite its high prevalence, however, treatment guidelines and underlying rationales remain vague and controversial.³⁵ This is supported by the fact that there is no agreement on how PFP should be treated. For example, a myriad of conservative procedures have been advocated,^{7,26,35,42,46,48,49} and numerous surgical techniques described.^{2,8,15,24,47}

A commonly accepted hypothesis concerning the etiology of PFP is related to increased patellofemoral joint stress and subsequent articular cartilage wear.^{10,16,17,28,40} Patellar malalignment and/or abnormal patellar tracking is thought to be one of the primary precursors of patellofemoral joint pathology.^{11,13,28,40} Acceptance of this theory is evident in clinical practice, as most interventions are focused on the patellofemoral joint itself, with the intention of influencing patellar motion (ie, strengthening the vastus medialis oblique, stretching, patellar taping, patellar bracing, soft tissue mobilization, and patellar mobilization).^{7,26,35,42,46,48,49}

It has been recognized by several authors that the patellofemoral joint may be influenced by the segmental interactions of the lower extremity.^{6,7,21,27,34,45} Abnormal motion(s) of the tibia and femur in the transverse and frontal planes are believed to have an effect on patellofemoral joint mechanics and therefore PFP. An understanding of how lower-extremity kinematics may influence the patellofemoral joint is important, as interventions to control abnormal lower-extremity mechanics are not focused on the area of pain, but, instead, on the segments and joints proximal and distal to the patellofemoral joint.

The purpose of this paper is to provide a theoretical overview of how altered lower-extremity kinematics may influence the patellofemoral joint. This will be accomplished by reviewing normal and abnormal lower limb kinematics in relation to patellofemoral joint function. In addition, current literature in this area will be examined.

THE INFLUENCE OF LOWER-EXTREMITY KINEMATICS ON THE PATELLOFEMORAL JOINT

The normal alignment of the lower extremity predisposes the patella to laterally directed forces. This phenomenon has been de-

¹ Associate Professor and Director, Musculoskeletal Biomechanics Research Laboratory, Department of Biokinesiology and Physical Therapy, University of Southern California, Los Angeles, CA. Send correspondence to Christopher M. Powers, Department of Biokinesiology and Physical Therapy, University of Southern California, 1540 E Alcazar St, CHP-155, Los Angeles, CA 90089-9006. E-mail: powers@usc.edu

scribed by Fulkerson and Hungerford⁹ as the “law of valgus” and occurs because the 2 primary forces acting on the patella, the resultant quadriceps force vector and the patellar tendon force vector, are not collinear. As a result, contraction of the quadriceps creates a lateral force vector acting on the patella (Figure 1A).³⁰

Clinically, this offset in force vectors is defined by the quadriceps angle (Q angle), which is measured as the angle formed by the intersection of the line drawn from the anterior superior iliac spine (ASIS) to the midpoint of the patella and a proximal extension of the line drawn from the tibial tubercle to the midpoint of the patella (Figure 1A).³⁰ Although the Q angle has been shown to accurately reflect the angle of the resultant quadriceps muscle force vector in the frontal plane, the magnitude of this force vector and resulting lateral force acting on the patella has been reported to be underestimated by this clinical measurement.⁴¹ Nonetheless, a larger Q angle would tend to create a larger lateral vector and potentially a greater predisposition to lateral patellar tracking when compared to a smaller Q angle.⁴¹

It should be noted that the relationship between the Q angle and clinical signs and symptoms has not always been consistent.²³ This suggests that the Q angle may be problematic in a subpopulation of those with PFP, and that etiologic factors unrelated to the Q angle may be more dominant in certain individuals. However, other possible reasons for the lack of association between the Q angle and PFP may be related to the fact that there has been no consensus with respect to how this measurement should be taken (ie, standing vs sitting vs supine; quadriceps contracted vs relaxed).²³ Perhaps more

importantly is the fact that this measurement typically is taken statically, therefore, the contribution of abnormal segmental motions and muscle activation to the Q angle during dynamic activities may not be appreciated.

When the patella is seated within the trochlear groove (ie, beyond 20° of knee flexion), an increase in the Q angle can result in increased lateral facet pressure as the patella is being forced against the lateral femoral condyle.¹⁸ Huberti and Hayes¹⁸ documented the effects of an increased Q angle by measuring patellofemoral contact pressures in 12 fresh cadaver specimens and found that a 10° increase in the Q angle resulted in a 45% increase in peak contact pressure at 20° of knee flexion. It should be noted that when the Q angle was decreased 10° from the normal physiological position, increases in contact pressures also were observed. With respect to an increase in the Q angle, when the patella is not firmly seated within the trochlear groove (ie, between 0° to 20° of knee flexion) or in the presence of inadequate lateral bony support (ie, patella alta or trochlear dysplasia), quadriceps contraction may cause lateral patellar subluxation.³³

Although structural deformities (ie, femoral anteversion, coxa vara, laterally displaced tibial tuberosity) can lead to an increase in the Q angle, abnormal motions of the lower extremity also may contribute. With this in mind, the 3 principal lower-limb motions that may influence the Q angle (ie, tibial rotation, femoral rotation, and knee valgus) will now be discussed in detail.

Tibial Rotation

The Q angle can be influenced distally through motion of the tibia relative to the femur. External

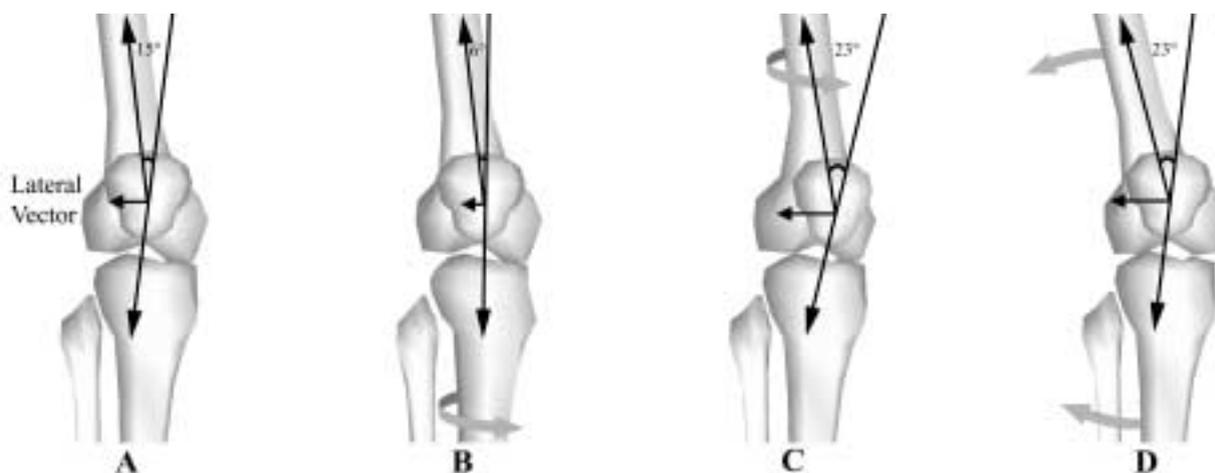


FIGURE 1. (A) The Q angle is measured as the angle formed by the intersection of the line drawn from the anterior superior iliac spine to the midpoint of the patella and a proximal extension of the line drawn from the tibial tubercle to the midpoint of the patella. Normal alignment of the tibia and femur results in an offset in the resultant quadriceps force vector (proximal) and the patellar tendon force vector (distal), creating a lateral vector acting on the patella; (B) tibia internal rotation decreases the Q angle and the magnitude of the lateral vector acting on the patella; (C) femoral internal rotation increases the Q angle and the lateral force acting on the patella; (D) knee valgus increases the Q angle and the lateral force acting on the patella.

rotation of the tibia moves the tibial tuberosity laterally, thereby increasing the Q angle, while tibial internal rotation decreases the Q angle by moving the tibial tuberosity medially (Figure 1B). In turn, tibial rotation can be influenced by subtalar joint motion.^{29,31} Internal rotation of the tibia is coupled with subtalar joint pronation, while external rotation of the tibia is coupled with subtalar joint supination.^{29,31} Normal subtalar joint pronation occurs during the first 30% of the gait cycle, during which the tibia internally rotates 6° to 10°. ^{4,14,37} This motion is in response to the inward rotation of the talus as it falls into the space created by the inferior and lateral movement of the anterior portion of the calcaneus.³¹

As a result of this close biomechanical relationship between the rearfoot and the tibia, abnormal pronation has been hypothesized to contribute to patellofemoral joint dysfunction.^{6,20,27,34} Typically, pronation is considered abnormal if the amount of motion is excessive or occurs at the wrong time (ie, when the foot should be supinating). When relating excessive pronation to various clinical entities, an assumption is made that abnormal pronation results in excessive tibial internal rotation and that this motion results in a rotatory strain on soft tissues of the lower extremity. While this may be the case with respect to the tibiofemoral joint, the same assumption does not hold true for the vertically aligned patellofemoral joint. In fact, excessive tibia internal rotation caused by abnormal subtalar joint pronation would actually *decrease* the Q angle and the lateral forces acting on the patella (Figure 1B).

Tiberio⁴⁴ described a scenario by which excessive pronation could affect normal patellofemoral joint function. This author postulated that to achieve knee extension in midstance, the tibia must externally rotate relative to the femur to ensure adequate motion for the screw-home mechanism. To compensate for the lack of tibial external rotation caused by the failure of the foot to resupinate, the femur would have to internally rotate on the tibia such that the tibia was in relative external rotation. Theoretically, compensatory internal rotation of the femur would permit the screw-home mechanics to allow for knee extension.⁴⁵ In turn, excessive internal rotation of the femur would move the patella medially with respect to the ASIS and the tibial tuberosity, thereby increasing the Q angle and the lateral component of the quadriceps muscle vector (Figure 1C).

It is conceivable that femoral rotation relative to the pelvis may have a greater influence on the line of action of the rectus femoris, while femoral rotation relative to the tibia may have more influence on the line of action of the vasti. Nonetheless, there appears to be a plausible biomechanical explanation by which pronation could influence the patellofemoral joint;

however, in order to do so, such motion would have to ultimately influence the femur.

An assumption made in the above scenario is that if excessive pronation is evident in midstance, then excessive internal rotation of the tibia also would be evident. However, a study by Reischl and colleagues³⁷ reported that the magnitude of foot pronation was not predictive of the magnitude of tibial or femoral rotation. In addition, the magnitude of tibial rotation was not predictive of the magnitude of femoral rotation, indicating that excessive rotation of the tibia did not translate into excessive femoral rotation. This is not surprising, considering that the knee is designed to absorb rotatory forces through its transverse-plane motion. It should be noted that all individuals in this study demonstrated pronation and tibial internal rotation during early stance, however, this motion was not a 1:1 ratio. This finding is consistent with other authors who have reported that the ratio of rearfoot eversion to tibial rotation is quite variable, ranging from 2.5:1 during walking³⁴ and from 1.5 to 1.8:1 during running.^{25,29} In addition, the data of Reischl and colleagues³⁷ support the findings of Kernozek et al,²⁰ who reported no association between rearfoot motion and the dynamic Q angle during the stance phase of gait. Individual factors, such as the orientation of the subtalar joint axis and the amount of transverse-plane motion between the rearfoot and the lower leg, likely influence the degree to which pronation can influence the magnitude of tibial rotation.

To date, only 2 studies have compared foot pronation in persons with PFP to a control group. Messier et al²⁷ found no significant differences in maximum pronation, maximum pronation velocity, and total rearfoot movement in 36 runners evaluated (16 with PFP and 20 controls). This led these authors to state that rearfoot movement variables were not significant etiologic factors in the development of PFP. Powers and colleagues³⁴ performed 3-dimensional motion analysis during self-selected free- and fast-walking velocities on 24 females with PFP and 17 controls, and found no group differences with respect to the magnitude and timing of peak foot pronation and tibia rotation. While certain individuals in this study did demonstrate the patterns of motion described by Tiberio,⁴⁵ by no means was this pattern consistent across all persons studied.

Taken together, the results of the above-noted studies suggest that one cannot assume a cause-and-effect relationship between abnormal pronation and PFP. The fact that not all persons with PFP demonstrate abnormal pronation, and that the magnitude of lower-extremity rotation cannot be predicted by the magnitude or timing of foot pronation, lends credence to this statement. However, it is entirely possible that certain individuals with PFP may

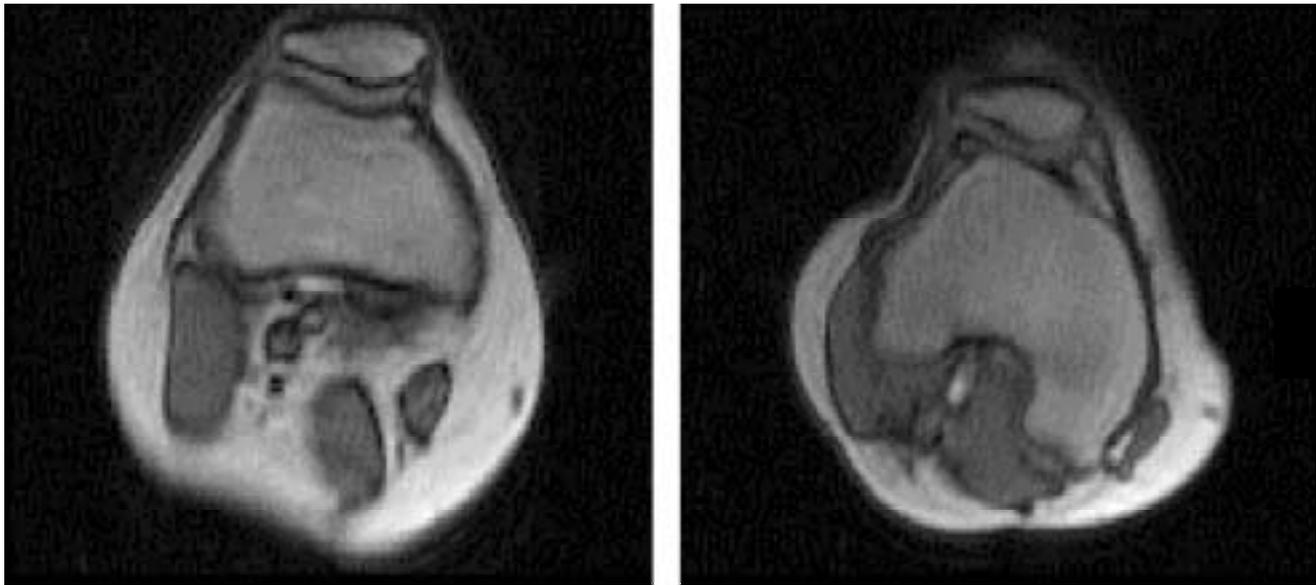


FIGURE 2. Kinematic magnetic resonance imaging of the patellofemoral joint during a single-limb stance step-down maneuver. The image on the left was taken with the knee flexed while the image on the right was obtained with the knee extended. It is apparent in the image on the left that the femur is internally rotating underneath the patella, giving the impression of lateral patellar tracking and tilt. (Reproduced with permission from Powers CM. *Patellofemoral Disorders: Home Study Course*. La Crosse, WI: American Physical Therapy Association; 2000. ©2000 Orthopaedic Section, APTA.)

demonstrate abnormal foot pronation (and subsequent lower-extremity rotations) that could be contributory to PFP. This concept is supported by the work of Eng and Pierrynowski,⁶ who reported that the use of soft orthotics combined with exercise was more effective in reducing PFP symptoms than exercise alone. It is likely that orthotics are beneficial for a subpopulation of persons with PFP and future studies should be conducted to determine the characteristics of such individuals so that optimal treatment can be administered.

Femoral Rotation

The Q angle can be influenced proximally through rotation of the femur. As described above, increased femoral internal rotation may result in a larger Q angle, as the patella would be moved medially with respect to the ASIS (femoral rotation relative to the pelvis) and/or the tibial tuberosity (femoral rotation relative to the tibia) (Figure 1C). Consequently, femoral external rotation could decrease the Q angle, as the resultant line of action of the extensor mechanism would be more in line with the ASIS and the tibial tuberosity.

Apart from increasing the Q angle and the laterally directed forces on the patella, femoral internal rotation can influence patellar alignment and kinematics.^{36,44} Because the patella is tethered within the quadriceps tendon, it is not obligated to follow the motions of the femur (ie, trochlear groove), especially when the quadriceps muscles are contracted. In fact, during weight-bearing activities, internal rotation of the femur can occur independent of patellar

motion.³⁶ Using dynamic MRI methods during a single-leg partial squat, Powers et al³⁶ demonstrated that the primary contributor to lateral patellar tilt and displacement in a group of individuals with patellar instability was femoral internal rotation and not patellar motion (Figure 2). It should be noted, however, that the influence of femoral internal rotation on patellar malalignment was more pronounced in terminal knee extension (less than 10° of knee flexion) as opposed to greater knee flexion angles. Nonetheless, this finding calls into question the long-held assumption that subluxation is the result of the patella moving on the femur. While this may be the case during non-weight-bearing activities, in which the femur is fixed (ie, during knee extension in sitting), this study provides evidence that lateral subluxation during weight-bearing activities may be the result of the femur rotating underneath the patella.

Additional evidence in support of the concept that excessive femoral rotation may be problematic in individuals with PFP has been provided by Lee et al,²¹ who examined the influence of fixed rotational deformities of the femur (ie, anteversion) on patellofemoral joint mechanics in cadaveric knees. Using pressure-sensitive film, these authors reported that 30° of femoral internal rotation significantly increased patellofemoral stress (force per unit area) when the knee was flexed beyond 30°. Increases in patellofemoral stress also were observed after simulating 20° of femoral anteversion; however, this difference was not statistically different from baseline (neutral alignment) measurements.

During normal walking, the femur has been reported to internally rotate approximately 7° during the first half of stance.²² However, wide variability in normative values has been reported.^{34,37} As described above, excessive internal rotation of the femur during midstance may be a compensatory mechanism to ensure normal knee screw-home mechanics in the presence of the combination of abnormal pronation and excessive tibia internal rotation.⁴⁵ However, motion of the femur also can be influenced proximally. The hip joint offers a great deal of mobility and is dependent on adequate muscular control for stability. Clinically, weakness of the hip external rotators (ie, gluteus maximus and deep rotators) can result in a “rolling in” of the femur during early stance,⁴³ and therefore, may have an adverse effect on the patellofemoral joint. In addition, excessive femoral anteversion can bias the lower extremity into internal rotation and may result in the clinical appearance of “squinting patellae” and/or a toed-in gait.³⁰

To date, only 1 study has studied femoral rotation in a group of individuals with PFP. Powers et al³⁴ reported that during self-selected free-walking speeds, persons with PFP had *less* femoral internal rotation than control subjects during early stance. On the average, the group with PFP demonstrated 2.1° of femoral *external* rotation compared to 1.6° of femoral internal rotation in the control group. These authors speculated that this motion was a compensatory strategy to reduce the Q angle. However, it should be noted that wide variability existed in both groups and that the average value reported was not reflective of all subjects. Assessment of femoral rotation during higher-demand activities, such as running and stair climbing, would be indicated, as deviations may become more apparent during tasks in which compensatory strategies are harder to maintain.

Knee Valgus

Apart from abnormal motions in the transverse plane, excessive frontal-plane motions can influence the patellofemoral joint. Most notably, valgus at the knee may increase the Q angle, as the patella would be displaced medially with respect to the ASIS (Figure 1D). In comparison, a varus position of the knee could decrease the Q angle, as the patella would be brought more in line with the ASIS.

Knee valgus may be the result of femoral adduction (relative to the pelvis), tibial abduction (relative to the femur), or the combination of both. As noted in the subject shown in Figure 3, the apparent knee valgus when landing from a jump appears to be coming from the combination of femoral adduction and tibial abduction. However, the apparent knee valgus of the subject shown in Figure 4, when performing a step-down maneuver, appears to be

more the result of femoral adduction, as the tibia is relatively vertical. Although both of these examples illustrate cases of excessive knee valgus, the contributions to this motion will likely vary from patient to patient as well as in the activity being assessed.

Excessive femoral adduction during dynamic tasks can be the result of weakness of the hip abductors, in particular, the gluteus medius. The upper fibers of the gluteus maximus and the tensor fascia latae also assist in abduction at the hip and may, if weak, contribute to excessive thigh adduction.³² Tibial abduction may be the result of excessive pronation or frontal plane motion at the ankle. However, it should be noted that tibial abduction also can be an accommodation to femoral adduction.

Structural abnormalities at the hip may predispose an individual to knee valgus. An example is coxa vara, which is defined as a femoral neck-shaft angle less than 125° .³⁰ In addition, a wider-than-normal pelvis has the potential to increase valgus at the knee, as the angulation of the femur in the frontal plane would have to be greater to maintain a normal stance width. Furthermore, a wider pelvis also would move the center of mass of the body more medial to the hip joint center, thereby increasing the adduction moment created by gravity during stance. In the presence of hip abductor weakness, such an increase



FIGURE 3. Example of excessive knee valgus when landing from a 30.5-cm drop. It is apparent from this picture that femoral adduction (relative to pelvis) and tibial abduction (relative to the femur) are contributing to this knee position.



FIGURE 4. Example of excessive knee valgus while performing a step-down maneuver. It is apparent from this picture that femoral adduction (relative to the pelvis) is the primary contributor to this knee position, as the tibia is relatively vertical.

in the external moment could exceed the strength capacity of the hip abductors, resulting in a contralateral pelvic drop and excessive hip adduction.

CLINICAL IMPLICATIONS

Several authors have stressed the importance of a classification system for PFP, based on potential etiological factors, including suspected lower-quarter biomechanical faults.⁷ Development of a sound treatment program for the individual with PFP should take into consideration all potential factors contributing to this syndrome. The decision to treat the lower extremity needs to be based on a systematic biomechanical evaluation, in particular, a thorough analysis of gait and functional movements. This is important, as not all patients with PFP will demonstrate lower-limb abnormalities and/or lack of dynamic control.

Once a lower-extremity abnormality is identified, a decision should be made as to the cause of the observed deviation. Structural factors are not necessarily amenable to conservative interventions, however, atypical motions resulting from muscle weakness

and/or poor neuromuscular control can be addressed. When considering potential contributors to abnormal motions, it should be realized that segments can be influenced from the ground up and/or from the hip and pelvis down. For example, “medial collapse” of the lower limb during stair descent may be the result of abnormal motion originating from the foot and ankle, hip, or a combination of both (Figure 5). Obviously, the decision to address a specific segment or joint will vary from patient to patient as will the course of treatment.

SUMMARY

This paper describes a biomechanical rationale by which segmental motions of the lower extremity may affect the patellofemoral joint. Although excessive motions of the tibia and femur in the frontal and

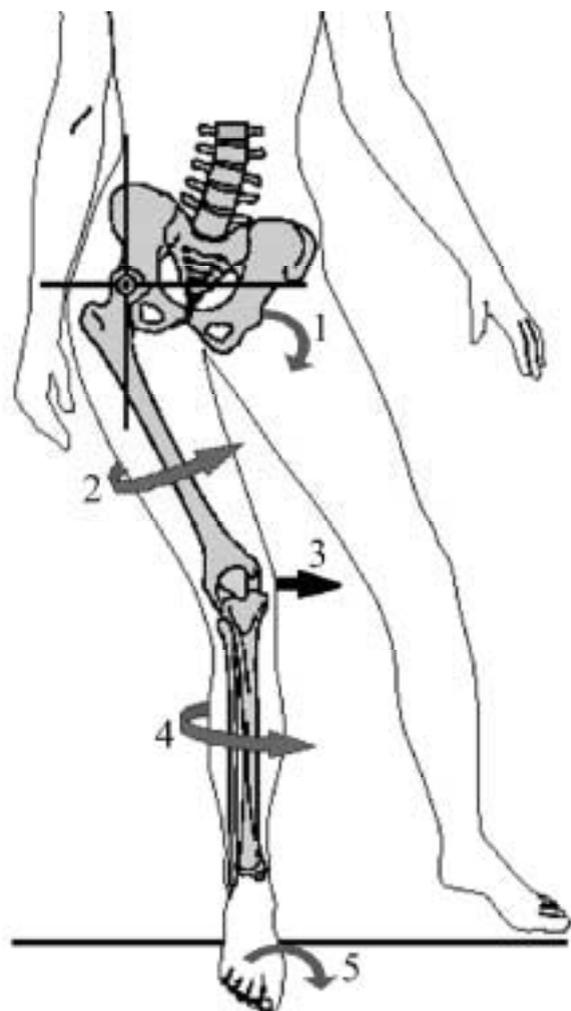


FIGURE 5. Schematic showing the potential contributions of the various lower-extremity segments to abnormal alignment: (1) contralateral pelvic drop, (2) femoral internal rotation, (3) knee valgus, (4) tibia internal rotation, and (5) foot pronation. (Adapted with permission from Götz-Neumann K. *Gehen Verstehen: Ganganalyse in der Physiotherapie*. Düsseldorf, Germany: Thieme-Verlag, Inc.; 2002. ©2002 Thieme-Verlag, Inc.)

transverse planes can influence patellofemoral joint mechanics and PFP, existing research suggests that such abnormalities are not a universal finding in this population. Therefore, interventions aimed at controlling hip and pelvic motion (proximal stability) and ankle and foot motion (distal stability) may be warranted in a subpopulation of persons with PFP.

Through an understanding of the potential contribution of the lower-extremity kinematics to patellofemoral joint dysfunction, it is hoped that clinicians will incorporate such information to better guide the examination process and treatment decisions for individuals who present with this complicated disorder. By identifying persons who could benefit from such interventions, better treatment outcomes may be achieved. Additional research is needed to (1) further evaluate the role of lower-extremity kinematics and kinetics in contributing to patellofemoral joint disorders, (2) develop valid and reliable classification systems to better identify those who may benefit from an intervention addressing lower-extremity function, and (3) evaluate clinical outcomes associated with interventions to address lower-extremity impairments.

ACKNOWLEDGMENTS

The author would like to thank Sam Ward, PT, PhD, Yu-Jen Chen, PT, MS, Li-der Chan, PT, MS, and Kornelia Kulig, PT, PhD, for their critical review and assistance with this manuscript.

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