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Stabilizing Anterior Cruciate Ligament Injuries: Biomechanical Requirements of Orthotic Design

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THE ACL IN ITS NORMAL AND INJURED STATES

The anterior cruciate ligament (ACL) is located in the intra-articular area of the tibio-femoral joint. It is attached on one end to a fossa on the posterior aspect of the medial surface of the lateral femoral condyle. This attachment is in the form of a segment of a circle, with its anterior border straight and its posterior border convex. Its long axis is angled somewhat forward from the vertical, and the posterior convexity is parallel to the posterior articular margin of the lateral femoral condyle. The other end of the ACL is attached to a fossa anterior, and lateral to the anterior tibial spine[2] (Figure 1).

The ACL itself consists of collagen and elastic fibers conformed in a band-like fashion. The band can be divided into two sections: an Anterior Medial Band (AMB) which is taut during flexion and lax near full extension, and a Posterior Lateral Band (PLB) which is taut during extension and lax in higher degrees of flexion.[2, 6, 39] The ACL's loosest position occurs between 40° and 50° of flexion, where neither the PLB nor the AMB have significant tension.[18]

The stability of a knee at any single moment is a function of four factors: ligament tightness, congruency of the articular joint surfaces, the effect of internal forces from the menisci, and the effect of muscle action.[22] For example, joint loading has been shown to significantly reduce anterior/posterior laxity—probably due to increased joint congruency and meniscal compression.[32] Cabaud describes the ACL as an essential structure on which mammalian knee joint stability depends, and as the "Keystone" of control in the complex fluid flexion and rotation of the normal knee.[6] The ACL has been shown extensively to be the primary structure controlling anterior drawer of the tibia in relation to the femur.[4, 6, 7, 11, 18, 19] In fact, values as high as 88.8% of the total resisting force to anterior tibial motion have been attributed to the ACL.[16] Internal rotation of the tibia increases tension on the ACL in all degrees of flexion.[6, 11, 18] The ACL serves to transfer force from the anterior superior tibia to the posterior distal femur. And, finally, it assists in limiting internal rotation and contributes to the rotation centers of the normal knee.

ACL Injuries

ACL injuries are quite prevalent and, as society becomes more oriented to fitness and sports activity, are becoming increasingly more common. Torg presents statistics showing that 69% of all knee injuries presented for arthroscopy for "internal derangement" of the knee involved damage

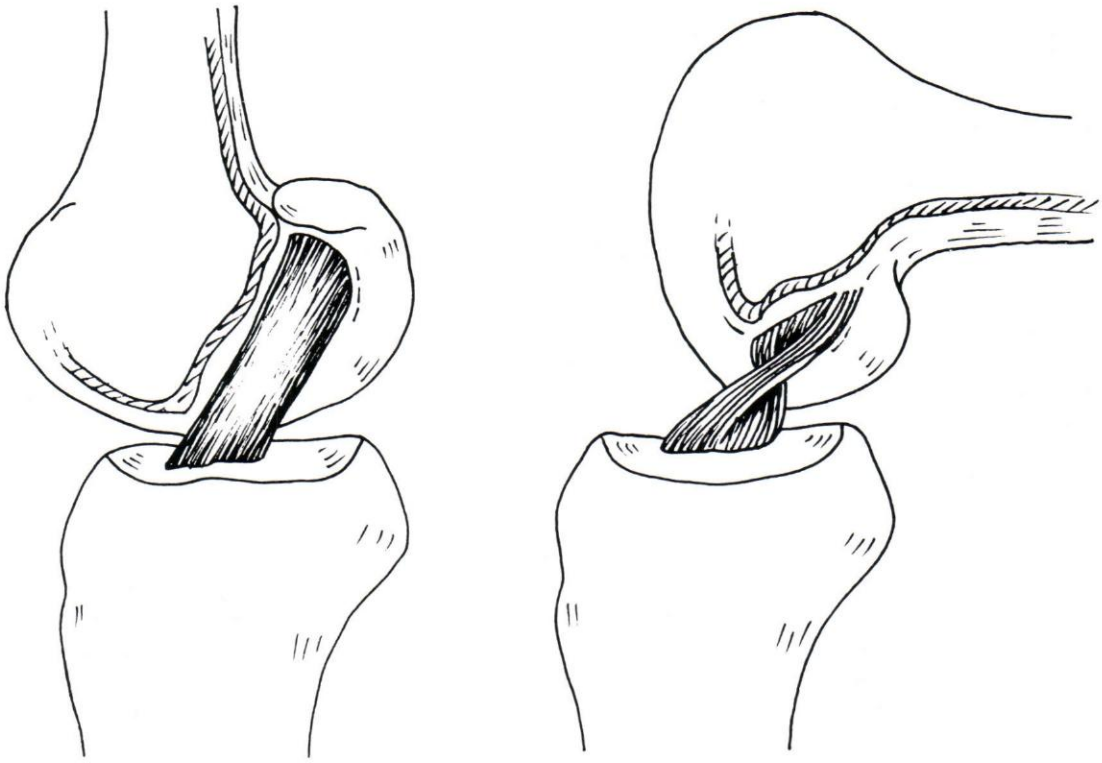

Figure 1. The anterior cruciate ligament.

to the ACL.[36] The literature describes four mechanisms capable of producing forces on the ACL larger than it can transfer, thereby severing it. The most common of the four is external rotation and abduction, the force mechanism present in most football and skiing injuries. An external rotatory moment is created by a cleated shoe in combination with an internal hip rotation. The same type of force is applied by a ski acting as a lever arm. When a large abductive force is applied to the tibia by another player or the skier continuing downhill and then firing his femoral adductors, the ACL tears.[8,18] A second common method of injury is extreme internal rotation of the tibia, such as crossing of ski tips.[6,11,22] The third injury mechanism is hyperextension to the degree where the posterior capsule and posterior cruciate have already been compromised. The last injury mechanism is a large anteriorly-directed posterior force, such as clipping in football.[18]

Anterior Drawer

Many studies have documented the difference in anterior drawer, both in vivo and in vitro, when the ACL is torn. Kochan, et al. found that at 20° of flexion with 200 N (Newtons) or about 45 lbs of anterior tibial force applied, the anterior drawer increased from 5.9 ± 2.1 mm for a normal knee to 12.2 ± 4.2 mm for an ACL-deficient knee.[19] These numbers may be affected by the limitation of internal tibial rotation upon anterior drawer caused by a locked foot plate used in these studies. Daniels, et al., using a less accurate KT-2000 and allowing for tibial rotation, obtained values for cadaver and in vivo knees at 89 N (20 lbs). Cadaver studies showed an increase from 5.8 ± 2.3 mm for the intact knee to 12.1 ± 2.9 mm when the ACL is severed (mean increase was 6.3 ± 2.0 mm, with a range of 2 to 10 mm). In vivo, this same study obtained values of

7.4 ± 1.7 mm for the normal knee and 13.0 ± 3.5 mm with the ACL absent.[7]

In a study by Fukubayashi, et al. anterior drawer increased 30% when free tibial rotation was allowed. The study explained that this rotation was internal and that the largest anterior drawer was obtained at 30° of flexion (7.0 mm at 100 N—22.5 lbs). They also demonstrated that anterior drawer of an ACL-deficient knee increased 2.5 times that of the intact knee betwen 15° and 45° of flexion at this same force level.[11] While actual anterior drawer values need to be determined using larger sample sizes and consistent force levels, the fact remains that anterior drawer of the tibia is primarily controlled by the ACL, and that these motions are extremely small in magnitude. To control anterior drawer to within normal values means to restrict movement to less than 1/5". At this point, the stiffness of the force versus displacement curve is high (83 ± 17 N/mm for normal knees and 52 ± 18 N/mm for ACL-deficient knees at 200 N of force), and increased force yields a less proportional increase in anterior drawer.[25] Allowing even 1/2" of anterior tibial motion is severely detrimental to secondary structures and normal knee motion.

Rotatory Instabilities and Pivot Shift

The most problematic and destructive occurrence to the ACL-deficient patient is not necessarily pure anterior drawer, but the resulting rotatory instabilities that are allowed to occur. These instabilities have been classified by Hughston, et al. and have received considerable recent attention in the literature.[14, 15] The reasons for this attention is the realization that rotatory instabilities cause severe degenerative changes and jeopardize secondary structures, which can be further disabling to the patient. The long term degenerative effects of rotatory instabilities are not yet known, but early evidence suggests the acceleration of degenerative changes.

Rotatory instabilities are classified as four major types. Here, we will focus on the two types that are related to anterior tibial movement: Anterior Lateral Rotatory Instability (ALRI), in which the lateral aspect of the tibia subluxes anteriorly and Anterior Medial Rotatory Instability (AMRI), where the medial aspect of the tibia subluxes abnormally in the anterior direction.[14, 15]

ALRI is the more debilitating of the two anterior rotational instabilities, primarily because the lateral compartment experiences greater stress during extension than does the medial compartment. The greatest stress occurs on the posterior lateral horn of the lateral meniscus.[15] Hughston, et al. continues, stating that this can be demonstrated by the fact that the posterolateral ligaments are bigger and stronger than the medial structures, possibly due to evolutionary reaction to the increased stress.[15] ALRI is brought about by two events: increased anterior drawer of the tibia and a shift of the transverse axis from its normal position to a point further medial. The literature identifies this latter event as the Pivot Shift Mechanism.

The normal location of the transverse axis (the center of tibial rotation) between 0° and 30° of flexion is slightly posterior and slightly medial to the bi-section of the tibial plateau.[37, 38, 39] As higher degrees of flexion occur, its location migrates slightly medially and posteriorly.[3] (Figure 2).

When the ACL is absent, the instant center of the knee moves from this position.[10] The center of rotation moves medially and posteriorly from its normal point to a point well inside the medial compartment, as shown in Figure 3.[9, 22] The effect of this pivot shift is an increase in the length of the lever arm to the lateral tibial condyle. This allows production of a larger angular radius.[35] The combination of the pivot shift (increased lever arm) and increased anterior drawer allows the lateral tibial condyle to significantly sublux anteriorly. A number of studies have noted the action of the femur sliding off the posterior lateral condyle of the tibia.[12, 23] This sliding causes impingement of the posterior horn of the lateral meniscus and will most likely cause damage to this structure over time.[5] It is important to understand that ALRI is a two-event situation and that

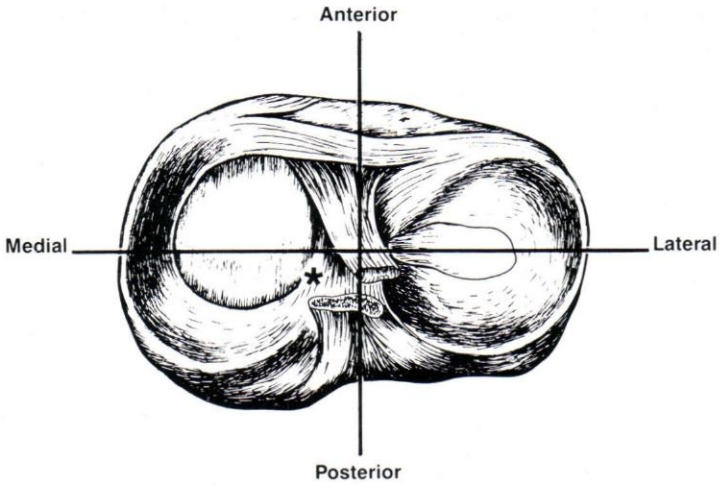

Figure 2. The tibial plateau in a normal knee, showing location of normal rotation center at 20° flexion.

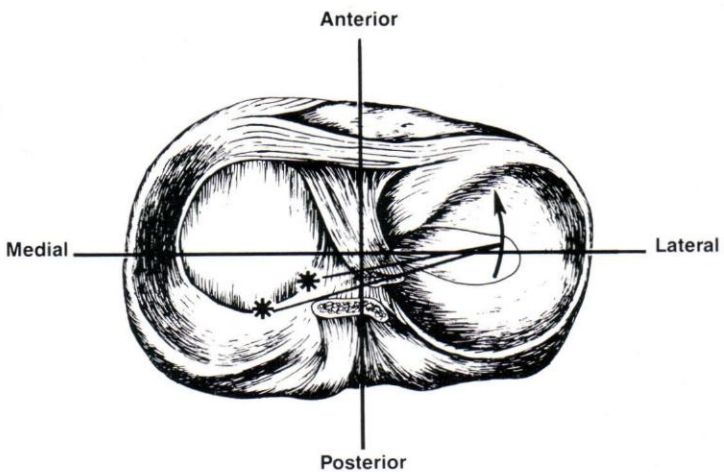

Figure 3. ALRI. Tibial plateau in an ACL injured knee, showing pivot shift. Lever arm to lateral tibial plateau increases.

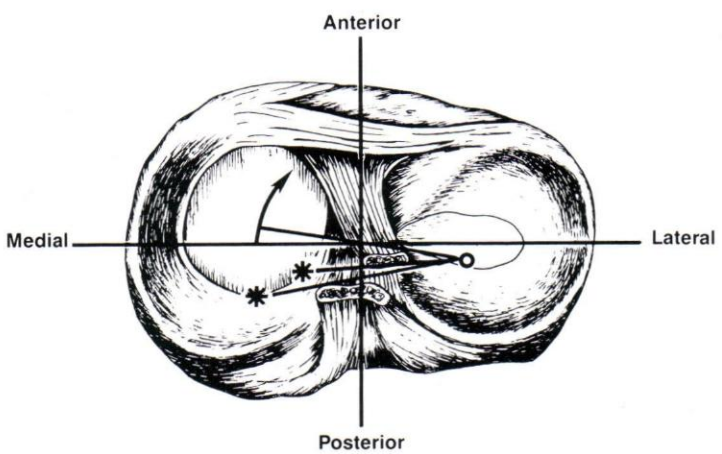

Figure 4. AMRI. The tibial plateau, showing both ACL and MCL pivot shift. Lever arm to lateral tibial plateau becomes significant.

rotatory instability is an anterior/posterior motion, and is not controlled primarily by medial/lateral stabilizers. It is reasonable to expect, then, that by limiting anterior drawer, ALRI will be reduced.

In injuries tested within two weeks of arthroscopy, the pivot shift was more sensitive in accurate diagnosis (88.8%) when compared to the Lachman and Anterior Drawer Sign Tests. In cases presented after two weeks, the Lachman and Pivot Shift tests were not found to be significantly different (84.6%).[16] The primary influencing factor here was the change in the center of rotation.

AMRI occurs primarily in the patient who has a combination of injuries which compromise the medial collateral ligament (MCL) as well as the ACL. Isolated sectioning of the MCL does not produce significant increases in anterior drawer, or in AMRI while the ACL is intact.[32,34] When the ACL and MCL have both been fully incapacitated, the pivot center of the knee shifts far laterally to the lateral compartment. The long lever arm to the medial aspect of the tibia, in combination with increased anterior drawer, allows anterior medial tibial subluxation.[33] (Figure 4).

This discussion of rotatory instabilities indicates that these harmful motions are anterior/posterior in direction and, therefore, are primarily controlled by the ACL. The ACL is a large contributor to the location of the transverse axis of rotation. Not until it is absent do the medial/lateral structures largely influence the location of this rotation center significantly.

ACL Tears

The area of the ACL that tears has been correlated in the literature to the strain rate at which the force is applied. The strength and function of the ACL under loading is determined by the structural arrangement of the fibers and the proportion of elastic to collagen fibers.[13,28] When the strain rate is slow, the ligament/bone complex is compromised first, and complete separation usually occurs in this area. The elastic modulus of canine preparation under slow strain rate was shown to be 200 megapascals.[13] At a fast strain rate, tears occur in the mid-ligament section.[26,27] Even though an ACL may appear intact arthroscopically, electron microscopy shows the micro fibrils of the collagen fibers to have been disrupted, decreasing the effectiveness of the ACL in controlling anterior tibial forces.[17,26,27]

The force required to disrupt the ACL varies widely due to differences in the size and shape of individuals. Older individuals have less ACL strength than younger adults.[27] Noyes, et al. demonstrated this, presenting maximum ACL forces of 1730 ± 660 N (388 ± 148 lbs) in a group of 16 to 26 year olds and 734± 266 N (165 ± 60 lbs) for a 48 to 86 year old group. Further, their study concluded that the ratio between ligament separation and body mass was 33 N/kg for the 16 to 26 year old group and 10 N/kg for 48 to 86 year olds.[27] Trent, et al. discussed the force per unit strain on the ACL (resting length times average stiffness of force versus deflection curves) as being 310 kg (141 lbs).[37] Resultant forces in knees range from 480 ± 35 N (107 ± 7.8 lbs) during walking, to 1020 ± 48 N (229 ± 10.7 lbs) during running, and 3280 ± 103 N (737 ± 23 lbs) in world-class long-jump athletes.[24] Forces generated during running have been presented as being 250% of the body weight.[1]

Since ligament tears do occur, it follows that the forces to which knees are sometimes subjected exceed these normal values. Complex ligamental tears imply even higher force levels. It is important to recognize that the forces to which knees are subjected are in the range of hundreds of pounds. The energy that the knee's ligaments attempt to transfer is extremely large, occasionally more than the ligaments can absorb. Even more important is the realization that the ligaments must contain motion of less than 7mm ($^3/_{10}$″) while sustaining these forces. It is this problem that today's surgeons, physical therapists, and orthotists face when attempting to rehabilitate the knees that have sustained ACL tears.

Secondary Ligamental Structures

The secondary structures that assist in anterior tibial resistance should also be considered. Once the ACL has been torn, there is minimal secondary ligamental back up. When an ACL-deficient knee is required to stabilize against anterior tibial subluxation, the force must be absorbed and transferred by the secondary structures. Secondary restraints may often block clinical laxity tests, but commonly stretch out in reaction to increased stress, and do not stabilize the higher forces experienced during activity. In the ACL-deficient knee, the anterior restraining structures absorb force in the following sequence: iliotibial tract (24.8%), mid-section of the medial area of the capsule (23.3%), mid-section of the lateral area of the capsule (20.8%), medial collateral ligament (16.3%), and the lateral collateral ligament (12.4%).

The hamstrings are also positioned to help resist anteriorly-directed forces on the tibia. They are seldom flexed during the weight-bearing phase and, therefore, are unable to restrict abnormal motion.[30] External rotation of the knee increases tension in the medial structures of the knee, whereas internal rotation tightens the iliotibial tract, lateral structures, and possibly the posterior cruciate.[20] Shoemaker and Markolf showed that normal knees could produce a maximum torque of 20 ± 6.7 Newton-meters (N-m) externally and 30.9 ± 9.6 N-m internally against a fixed force plate (20° of flexion, hips extended, neutral foot position). In the same study, they showed that in vitro ligament failure occurred at 41.3 ± 10.6 N-m in external rotation.[31] This implies that even when fully flexed, the internal rotators of the knee cannot resist the forces that are applied to the knee. In summary, clearly there is no structure in the normal knee evolutionarily designed for or capable of transferring force with the efficiency of the ACL.

Secondary anterior force producing muscle activity is a very important point, since the quadriceps are very powerful and must be contracted during any bent-knee activity. Isometric quadriceps activity significantly increases the strain within the ACL relative to normal passive strain at flexion angles of 10° to 45°. The hamstrings can reduce some strain during simultaneous quadriceps activity between 0° and 30° of flexion, but not a significant amount.[30] Perry, et al. studied quadriceps stabilizing forces for flexed knees in relation to femoral head force. They reported that the quadriceps had to produce 75% femoral head force at 15° of flexion, 210% at 30°, and 410% at 60°. They explained that a 225 N (50 lb) force on the femoral head, in combination with the quadriceps stabilizing activity, produced over 600 N of tibio-femoral joint force.[29] It is important to realize that although only a portion of quadriceps force is translated into anterior tibial force, it must be considered in all treatment plans of ACL deficiencies.

The ACL in Summary

The ACL is the primary knee structure that provides significant stabilization of anterior and internal rotation movements of the tibia. The small magnitude of motion and the large force levels that must be controlled in an ACL-deficient knee present a difficult treatment problem to the rehabilitation team. ALRI results from a two-part composite event: a medial and posterior shift of the transverse axis of rotation combined with an increase in anterior drawer. Such events allow a significant subluxation of the lateral tibia and impingement of the posterior lateral menisci. ALRI is the most detrimental abnormal motion that can occur when the integrity of the ACL has been compromised. And, finally, secondary ligamental structures or muscle activity are of little benefit in resisting abnormal motion.

BIOMECHANICAL CONSIDERATIONS IN ORTHOTIC DESIGN

The ultimate orthotic design for treatment of the ACL-deficient knee would be one in which normal knee motion occurred when the orthosis was in use. It would be

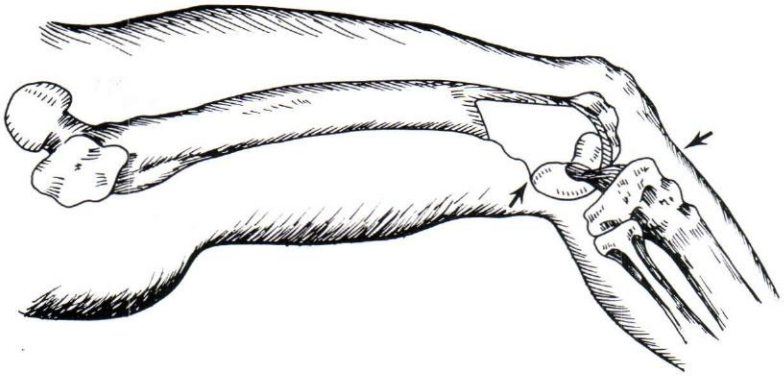

Figure 5. Ideal stabilizing forces.

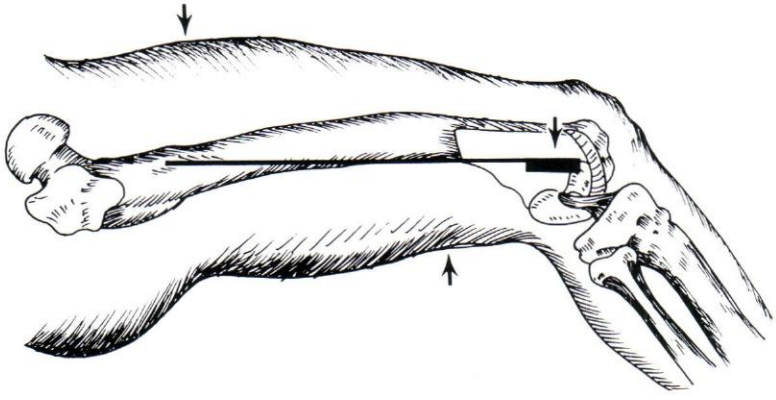

Figure 6. Two-point pressure system creating stable lever arm. Creates posteriorly directed force at joint center.

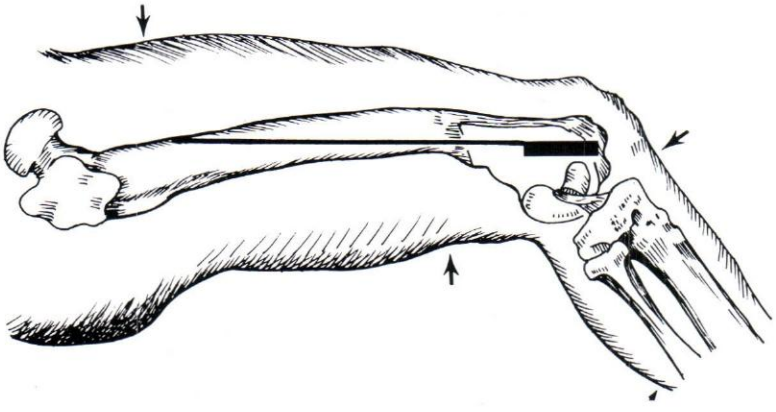

Figure 7. Four-point stabilization system required to orthoticly stabilize ACL deficient knees.

comfortable, light weight, and remain in perfect alignment at all times.

Perhaps the paramount test of an orthosis is how effectively it prevents re-injury when the patient experiences a force and situation similar to the one that originally tore the ACL. The orthosis must transfer enough force to prevent injury to the secondary structures, which become stressed in the absence of the ACL. The orthosis should generate relative motion between the tibia and femur that closely resembles normal motion. To accomplish this, the orthosis must transfer force from the anterior tibia to the distal posterior femur, replicating the primary function of the ACL. The orthosis should also resist internal rotation of the tibia on the femur, a secondary function of the ACL.

An Anatomical Joint

The ideal orthosis should incorporate in its design a joint that replicates the exact anatomical motion of the knee, that is, it rotates internally upon flexion. The foremost and most critical function of the joint is to transfer force in the anterior posterior plane, without allowing anterior motion of the tibia. If the joints are anatomical and are held in rigid relationship to the femur, the tibia will trace anatomically.

At this time, a joint of pure anatomical design and force transference does not exist. There are a number of joint designs on the market that are advertised as being "anatomical." However, most of these incorporate a slide pivot feature, which allows almost no resistance to forward motion. These designs do not restrict anterior drawer of the tibia.

An Effective Force Transfer Mechanism

Next, an ideal orthosis must stabilize the joints in relation to the femur so that their relationship does not change during activity. The main objective is to transfer force from the anterior superior tibia to the posterior distal femur. If equal and opposite forces are directed posteriorly on the anterior superior tibia, and anteriorly on the posterior distal femur, the relationship of the tibia and femur remains constant (Figure 5).

The application of pressure directly to the anterior superior tibia presents no problem, since it lies directly under the skin. However, applying force to the posterior distal femur is another matter. Pressure may not be placed directly on this area due to the large concentration of circulatory and nervous system anatomy beneath. Therefore, it becomes necessary to employ an alternate method of force transfer.

To create this stabilizing situation, a two-point pressure system must be implemented. The first pressure point is located on the femur, as far distal as comfortable knee flexion will allow. The second point is on the anterior superior thigh. These two points create a lever-arm system of stabilization with a mechanical advantage, as shown in Figure 6.

Poor anterior/posterior test results have characterized many of the orthoses presently available on the market. When the tibia moves anteriorly, the force is transferred to the joints. If the joints transfer this force effectively, they must be stabilized by the orthosis at the posterior femur. In conventional orthoses, this stabilization is effected by either an elastic or non-elastic strap. Elastic straps cannot limit motion to a few millimeters under hundreds of pounds of force. Non-elastic strapping, on the other hand, does not stretch, but instead imbeds itself into the soft tissue with a compression force equal to the original anterior tibial force. The distance the strap imbeds into the soft tissue under hundreds of pounds of force is more than the few millimeters of normal anterior motion that can be allowed. Static tests have shown that both elastic strap models, such as Lenox Hill, and non-elastic strap designs, such as C.Ti., control anterior drawer effectively only at force levels of 15 and 20 pounds, respectively. Neither, then, can effectively control anterior subluxation under dynamic situations.[3]

Other Considerations

The ideal orthosis must incorporate three other features in its design. First, it must produce a resistant force equivalent to the anterior force generated by quadriceps contraction. Second, the tibial section of the orthosis must not be allowed to leave the tibia when the leg reaches terminal swing. The orthosis has anterior inertia and does not have hamstrings to decelerate it as the human leg does.

Third, if the orthosis cannot restrict abnormal anterior tibial motion as precisely as the ACL does, it must limit extension to –10° of the full position. Limiting extension is necessary due to the increase in ligament stress and articular pressure, classically termed the "screw-home" mechanism. If the knee is abnormally rotated when approaching full extension, secondary structures such as the menisci can be pinched and damaged. Since an ideal joint design does not exist, an extension-limiting feature must be incorporated in the design of the orthosis. Once again, compression of the soft tissue on the femur using elastic and non-elastic straps creates problems. They cannot exert proper force on the posterior femur and gastrocnemius to control extension and hyperextension forces.

Knee Orthoses In Summary

The primary concern in the design of an ideal orthosis must be to limit anterior drawer to less than 10 mm of motion under hundreds of pounds of force. To accomplish this, the orthosis must incorporate joints that can effectively transfer force in all degrees of flexion and at least four pressure points (Figure 7): a two-point stabilization system to resist anterior joint motion, a point on the anterior superior tibia to transfer force to the joints, and a fourth point in the distal posterior gastrocnemius are to help decrease the orthosis' own inertial energy at terminal swing. It must also counteract the anterior tibial force generated by quadriceps contraction. Finally, the ideal orthosis must limit full extension to avoid the strong pivot shift which occurs during the "screw-home" mechanism.

IN REVIEW

The anterior cruciate ligament is the main stabilizing structure to control anterior drawer of the tibia. It also resists internal rotation of the tibia at any single degree of flexion. Its primary function by anatomical attachment is to transfer force from the anterior superior tibia to the posterior distal femur.

The difference in the amount of motion that occurs in normal and ACL-deficient knees is very small (approximately 7 mm, or $^{3}/_{10}$"). The knee can potentially experience forces of several hundred pounds.

When the ACL is compromised, rotational instabilities occur. The severity of these rotational instabilities is determined by the total amount of damage sustained by the secondary structures. The combination of the medial and posterior shift of the transverse center of rotation and increased anterior drawer produces a large angular moment of the lateral tibial condyle. This subluxation allows the femur to slide off the posterior lateral corner of the tibia and often causes impingement of the posterior horn of the lateral menisci.

The primary function of an orthosis designed for the ACL deficient patient is to replicate a normal ACL, reducing abnormal anterior motion of the tibia by transferring the force to the femur.

A minimum of four pressure points are required to stabilize the large forces involved in the ACL-deficient knee. The femoral section must be contoured to apply forces accurately in minimizing soft tissue compression and internal tibial rotation at single degrees of flexion. The femoral section must utilize a two-pressure-point design to resist anterior joint motion.

Further research must be conducted to produce the ideal orthosis—one which prevents degenerative changes and injury to secondary structures, and allows patient activity with comfort and security.

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