Frank R. Noyes
Sue Barber-Westin
Editors

ACL Injuries in the Female Athlete

Causes, Impacts, and Conditioning Programs

With DVD-ROM

Springer

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Preface

The dilemma of the gender disparity in anterior cruciate ligament (ACL) injuries began nearly 20 years ago when unsettling data began to appear in the *American Journal of Sports Medicine* from the editors' orthopedic and sports medicine center and the National Collegiate Athletic Association:

> The injury rate of serious knee ligament injuries among female athletes was 5.75 times that of male athletes, a difference that proved to be statistically significant (1994) [6].
> Specifically, female soccer players had an ACL injury rate at least twice as high as male players in any given year. Female basketball players had an ACL injury rate at least three times that of male players in 4 of the 5 years sampled (1995) [1].

Although no scientific data existed to determine why female athletes in certain sports were suffering higher rates of ACL injuries than their male counterparts, there were enough theories of causative risk factors to support the initiation of special training programs designed to decrease this problem;

> One starts an action simply because one must do something.
>
> – T.S. Eliot

Although it may have seemed premature to promote training of female athletes involved in soccer, basketball, and other high-risk activities, a few pioneers set forth to improve or change aspects of these athletes' movement patterns that seemed to inherently predispose them for ACL injury:

> This training may have a significant effect on knee stabilization and prevention of serious knee injury among female athletes (1996) [3].

> This prospective study demonstrated a decreased incidence of knee injury in female athletes after a specific plyometric training program (1999) [4].

Following the publication in 1999 of a neuromuscular training program that reduced the incidence of ACL injuries in female high school athletes, this topic generated tremendous interest almost instantly. Researchers from around the world were soon involved in studying risk factors hypothesized to cause the gender disparity and developing other training programs designed to reduce the incidence of noncontact ACL injuries. At the time of writing, over 300 original research investigations had been published that focused on ACL injuries in the female athlete.

Having been at the forefront of this research topic, the editors find it refreshing to see the amount of intellectual energy and dollars that have been devoted to this area. There is little doubt that the findings first described in 1994–1995 did not represent a trend or fad but a truly important problem worthy of bringing the interest of the best minds involved in sports medicine to try to solve or reduce this problem. In fact, multiple "ACL research retreats" (occurring in 1999, 2001, 2003, 2005, 2006, 2008, 2010, 2012) and consensus statements from organizations such as the International Olympic Committee [7] demonstrate the attention and emphasis the female athlete ACL injury dilemma has received internationally.

As shown in this textbook, many more investigators are studying the causative factors producing the higher incidence of ACL injuries in female athletes than are involved in prevention training. Debate exists regarding the continuing problem of deciphering the true risk factors, and in fact, there remain questions on the exact mechanisms of this injury. Public health experts stress the critical need to understand the etiology of why athletic injuries occur because

> Prevention cannot be instigated until this information is available because the specific focus and targeting of prevention programs is unclear [2].

So, at least for ACL injuries, a paradox exists in that we are still in the process of understanding the mechanisms and risk factors for the injury, yet prevention programs have reduced the incidence in some female athletic populations. This is true both for a few individual training programs and for meta-analyzed data:

> The findings from this review lend support to ACL injury prevention programs designed to prevent unopposed excessive quadriceps force and frontal-plane or transverse plane (or both) moments to the knee and to encourage increased knee flexion angle during sudden deceleration and acceleration tasks (2008) [9].

> Our study indicated strong evidence in support of a significant effect of ACL injury prevention programs. Our pooled estimates suggest a substantial beneficial effect of ACL injury prevention programs, with a risk reduction of 52% in the female athletes and 85% in the male athletes (2012) [8].

If it seems we are getting ahead of ourselves, that may be true. However, the ever-growing interest in ACL injury prevention training is indicative that many health professionals, athletes, coaches, and parents believe that some type of preventative effort is better than nothing. In fact, the editors' nonprofit research foundation has certified over 1,360 individuals to conduct neuromuscular ACL injury prevention programs in their communities and medical practices. A recent Bing search of "ACL Injury Prevention Training" revealed 515,000 hits, highlighting the popularity of this topic.

Unfortunately, not everyone has jumped on the bandwagon regarding ACL injury prevention training. Many authors have noted problems convincing coaches to agree to add this type of training to their practice schedules or to train their athletes before the season begins. In a recent study [5], 258 high school coaches in the Chicago area were invited to participate in a coach-led ACL injury prevention training program. Only 95 (37 %) enrolled. There

remains a tremendous need and responsibility of medical health professionals to educate those involved with female athletes of the devastating consequences of ACL injuries and the need to prevent them.

One potential solution to the "coach-not-interested" problem is to provide training programs that both enhance athletic performance and reduce the incidence of ACL injuries. This textbook describes programs designed for high-risk sports such as soccer and basketball that have accomplished both of these goals.

Another solution is to study and identify simple field tests to detect athletes with neuromuscular problems and imbalances that require correction. While laboratory work must continue using the most advanced three-dimensional motion, force plate, electromyographic, and other equipment available, realistic and cost-effective tests are required. These could be incorporated into preseason physicals done by physicians or conducted by coaches as part of their athlete testing regimen. Several such field tests are detailed in this book.

This textbook was designed to compile the many different approaches taken by clinicians and scientists regarding the female ACL injury problem. Our goal is to highlight the findings and current viewpoints of some of the individuals actively involved in this area of research. We are grateful to the guest authors, many of whom have published extensively on this topic, for their contributions to this effort.

It is our hope that someday, ACL injury prevention training will truly be widespread and perhaps even a part of routine physical education classes at schools. We agree with a recent consensus statement that we need to consider

> …increasing our focus on the youth athlete and taking more of a public health approach in our injury-risk screening and injury-prevention strategies in this population [10].

Only through widespread use of prevention training will the female ACL injury problem be solved or at least significantly reduced. Until then, it remains the responsibility of those clinicians and scientists involved to continue their efforts to educate the general public and conduct research in the areas of risk factors, risk screening, and prevention programs.

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Part VI Future Directions

Acronyms

ACL	Anterior cruciate ligament
AE	Athlete-exposure
AM	Anteromedial
AMI	Arthrogenic muscle inhibition
ANOVA	Analysis of variance
AP	Anteroposterior
BMD	Bone mineral density
BMI	Body mass index
B-PT-B	Bone-patellar tendon-bone
BSSTM	Behavioral and social science theories and models
CD	Compact disc
CNS	Central nervous system
cm	Centimeters
COF	Coefficient of friction
COM	Center of mass
COP	Center of pressure
COMP	Cartilage oligomeric matrix protein
DEXA	Dual energy x-ray absorptiometry
DPSI	Dynamic postural stability index
EMG	Electromyographic or electromyography
ER	External rotation
FAST-FP	Functional Agility Short-Term Fatigue Protocol
FIFA	Federation Internationale de Football Association
FPPA	Frontal plane projection angle
GAG	Glycosaminoglycans
GMAX	Gluteus maximus
GMED	Gluteus medius
GRF	Ground reaction force
GRFV	Ground reaction force vector
rGRFV	Resultant ground reaction force vector
GTO	Golgi-tendon organs
HBM	Health Belief Model
IC	Initial contact
ICC	Intraclass correlation coefficients
IEMG	Integrated electromyography
IKDC	International Knee Documentations Committee
IR	Internal rotation
IR/ER	Internal rotation / external rotation

H:Q	Hamstrings-to-quadriceps
JPS	Joint position sense
KIPP	Knee Injury Prevention Program
kg	Kilograms
KLIP	Knee Ligament Injury Prevention
KT and KT-2000	Knee arthrometer
LESS	Landing Error Scoring System
M	Meters
min	Minutes
MMPs	Metalloproteinases
mo	Month
MRI	Magnetic resonance imaging
MSFT	Multi-stage fitness test
ms	Milliseconds
MVC	Maximal voluntary contraction
MVE	Maximal voluntary excursions
NCAA	National Collegiate Athletic Association
NFL	National Football League
N	Newtons
Nm	Newton meters
OA	Osteoarthritis
PA	Posteroanterior
pEKAbM	Peak external knee abduction moment
PEP	Prevent Injury and Enhance Performance
PL	Posterolateral
PCL	Posterior cruciate ligament
pTIRM	Peak tibial internal rotation moment
QH	Quadriceps-hamstrings
RM	Repetition max
s	Seconds
SEBT	Star Excursion Balance Test
SEPs	Somatosensory evoked potentials
SLO-FP	Slow Linear Oxidative Fatigue Protocol
SPECT	Single-photon emission computed tomography
STG	Semitendinosus-gracilis
TIMP	Tissue inhibitors of metalloproteinases
TDPM and TTDPM	Threshold for detection of passive motion
TRIPP	Translating Research into Injury Prevention Practice
TTDPM	Threshold to detect passive motion
U.S.	United States
vGRF	Vertical ground reaction force
VMO	Vastus medialis oblique
VO_2max	Maximal oxygen uptake
WIPP	Warm-up for Injury Prevention and Performance
wk	Week
×	Times
yr	Year
3-D	3 dimensional
2-D	2 dimensional

Part I

Introduction

The ACL: Anatomy, Biomechanics, Mechanisms of Injury, and the Gender Disparity

1

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Contents

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Introduction

Anterior cruciate ligament (ACL) tears are the most common, complete ligamentous injuries that occur in the knee joint. In the United States (US), ACL tears occur in an estimated 1 in 3,500 individuals each year [6], and although exact data are not currently available, it is believed that approximately 125,000–200,000 ACL reconstructions are performed annually. Few national registries exist to document ACL injuries and reconstructions. The National Survey of Ambulatory Surgery from the US reported that 127,446 ACL reconstructions were performed in 2006 [41]. The Swedish National Registry was used to calculate the incidence of ACL injury in 2002, which was 0.81/1,000 inhabitants per year in individuals aged 10–64 years [29], with over 3,000 reconstructions performed annually [43]. In Norway, the annual incidence of cruciate reconstructions from 2004 to 2006 was reported to be 85 per 100,000 in citizens 16–39 years old [31]. The annual rate of cruciate reconstructions in this age group was 1,168. In the United Kingdom, a trauma institution that treated all adults from a population of 535,000 reported an ACL injury rate of 8.1 per 100,000 citizens per year [20]. The population in that investigation was 13–89 years old. It is reasonable to estimate that one million ACL injuries occur yearly worldwide.

Regardless of nationality, the majority of patients who sustain ACL injuries and undergo reconstruction are athletes <25 years old who are frequently involved in high school, collegiate, or

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league sports [93]. At least two-thirds of ACL tears occur during noncontact situations while an athlete is cutting, pivoting, accelerating, decelerating, or landing from a jump [13, 15, 84]. The costs of treatment of ACL tears are substantial, and athletes who suffer concomitant meniscus tears that require resection or other ligament tears are at increased risk for premature osteoarthritis [2, 34]. As well, athletes may suffer from deterioration of emotional health regardless of the treatment of the ACL injury [17, 46, 63, 73, 91].

The initial reports of a higher incidence of noncontact ACL injuries in female athletes compared to male athletes participating in soccer and basketball appeared in the medical literature in 1994 [50] and 1995 [4]. Since then, researchers worldwide have spent considerable time and effort in attempting to understand why this disparity exists in these and other sports and if interventions such as neuromuscular retraining can lessen the difference in injury rates between genders.

Critical Points

- United States: ACL injuries ~1 in 3,500 individuals; 125,000–200,000 ACL reconstructions annually.
- Swedish National Registry: >3,000 ACL reconstructions annually.
- Norway: patients 16–39 years old: 1,168 ACL reconstructions annually.
- Worldwide: one million ACL injuries annually.
- Majority are athletes <25 years old involved in high school, collegiate, or league sports.

Anatomy

Overview

Many authors have described the anatomy of the ACL [89]. The ACL is an average of 38 mm in length and 11 mm in width. This ligament originates on the medial aspect of the lateral femoral condyle (Fig. 1.1). The origin, which may be oval or semicircular in appearance, is approximately 18-mm long and 10-mm wide and lies just behind a bony ridge (termed the resident's ridge) [40] that is anterior to the posterior cartilage of the lateral femoral condyle (Fig. 1.2).

The insertion of the ACL, which is a roughly oval to triangular pattern, is located in the anterior intercondylar area of the tibia (Fig. 1.3). The insertion fans out and has been described as resembling a "duck's foot" (Fig. 1.4) [77]. The anteroposterior dimension of the insertion is approximately 18 mm, and the mediolateral dimension is 10 mm. The anterior border of the ACL is approximately 22 mm from the anterior cortex of the tibia and 15 mm from the anterior edge of the articular surface. Its center is 15–18 mm anterior to the retro-eminence ridge, which is referred to as the intercondylar eminence in anatomy textbooks (Fig. 1.5) [21, 88]. The medial and lateral tibial spines are referred to as the medial and lateral intercondylar tubercles [21]. The ACL insertion is just lateral to the tip of the medial intercondylar tubercle, with >50 % inserting anterior to the posterior edge of the anterior horn of the lateral meniscus (Fig. 1.6).

Division of ACL into Anteromedial and Posterolateral Bundles

Disagreement exists among researchers and surgeons regarding the division of the ACL into two distinct fiber bundles. While some investigators provide evidence of an anatomic and functional division, others argue that ACL fiber function is too complex to be artificially divided into two bundles. Amis et al. [3] and Colombet et al. [23] are among those who state that the anteromedial (AM) bundle functions as the proximal half of the attachment that tightens with knee flexion. In contrast, the posterolateral (PL) bundle is the distal half that tightens with knee extension. This occurs as the ACL femoral attachment changes from a vertical to a horizontal structure with knee motion. The problem is that this description of the tightening and relaxation of the AM and PL bundles represents that which occurs under no loading conditions in the laboratory. When

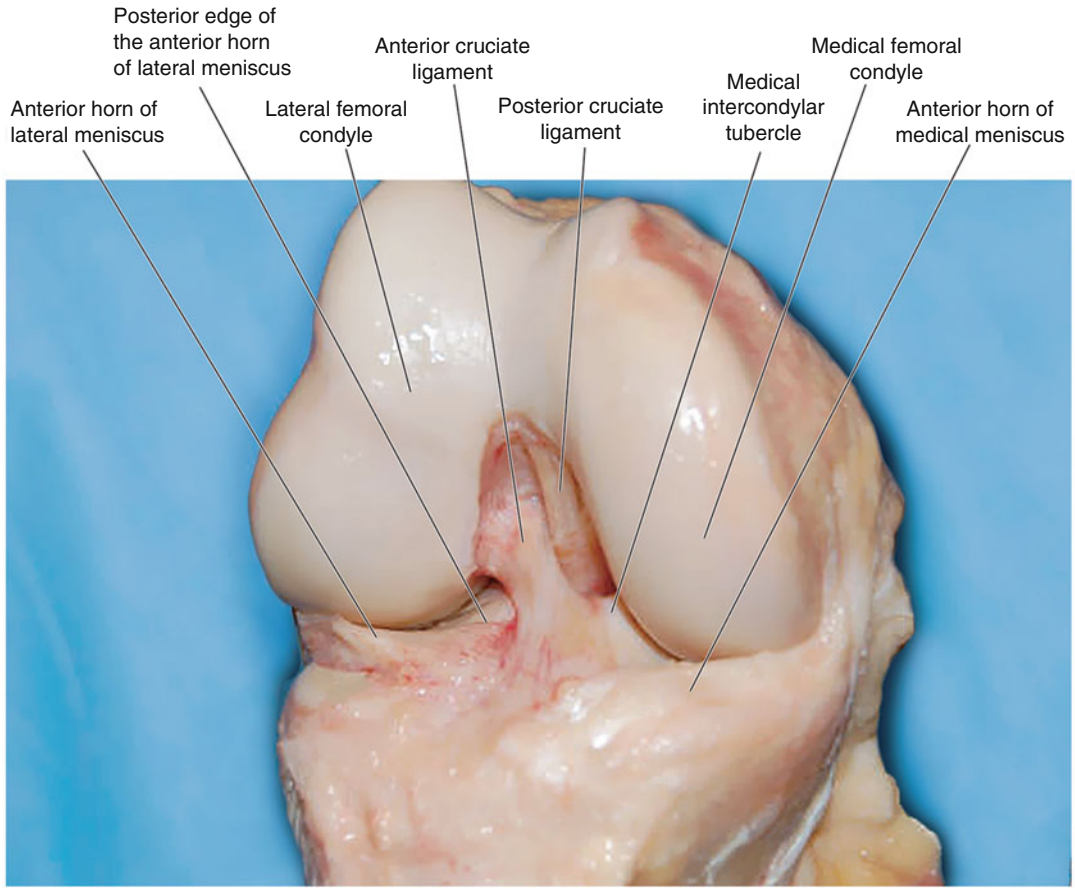

Fig. 1.1 Anterior view of the knee demonstrating the oblique orientation of the ACL originating on the medial aspect (sidewall) of the lateral femoral condyle (Reprinted with permission from Strickland et al. [89])

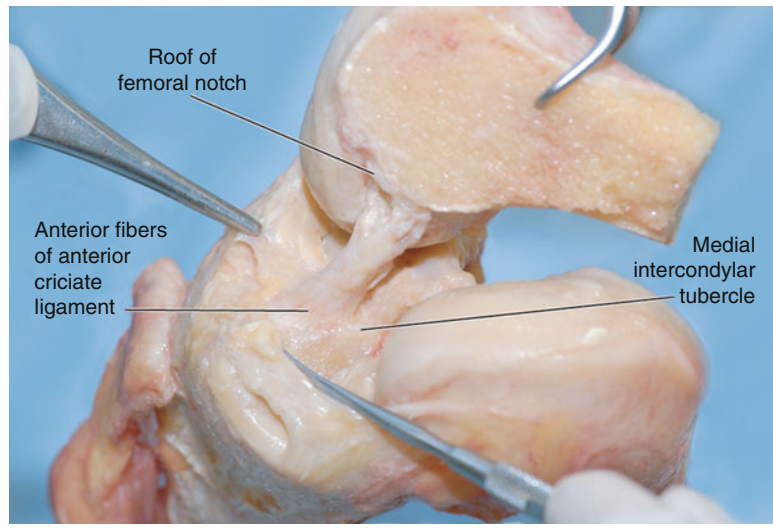

Fig. 1.2 Lateral view of the ACL. Note the distance from the posterior cartilage of the lateral femoral condyle to the ACL (Reprinted with permission from Strickland et al. [89])

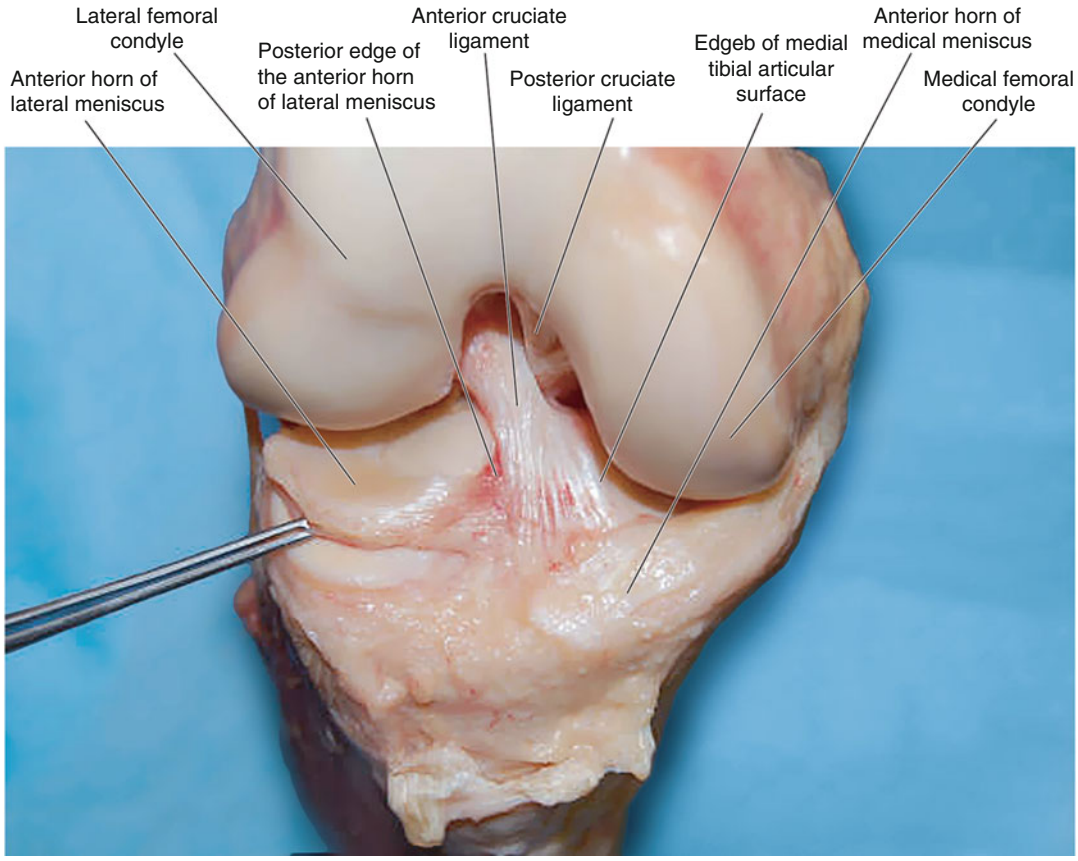

Fig. 1.3 Anterior-superior view of the knee demonstrating the ACL tibial insertion (Reprinted with permission from Strickland et al. [89])

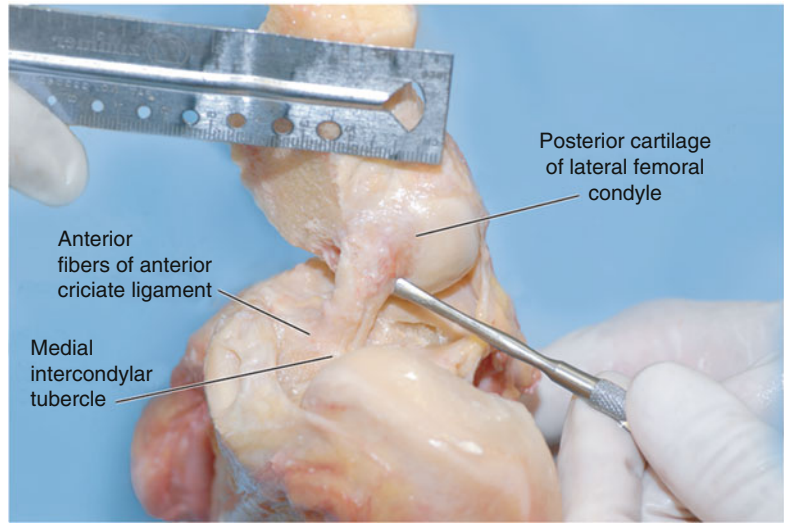

Fig. 1.4 Lateral view of the ACL. The anterior extension of the tibial insertion of the ACL is well visualized (Reprinted with permission from Strickland et al. [89])

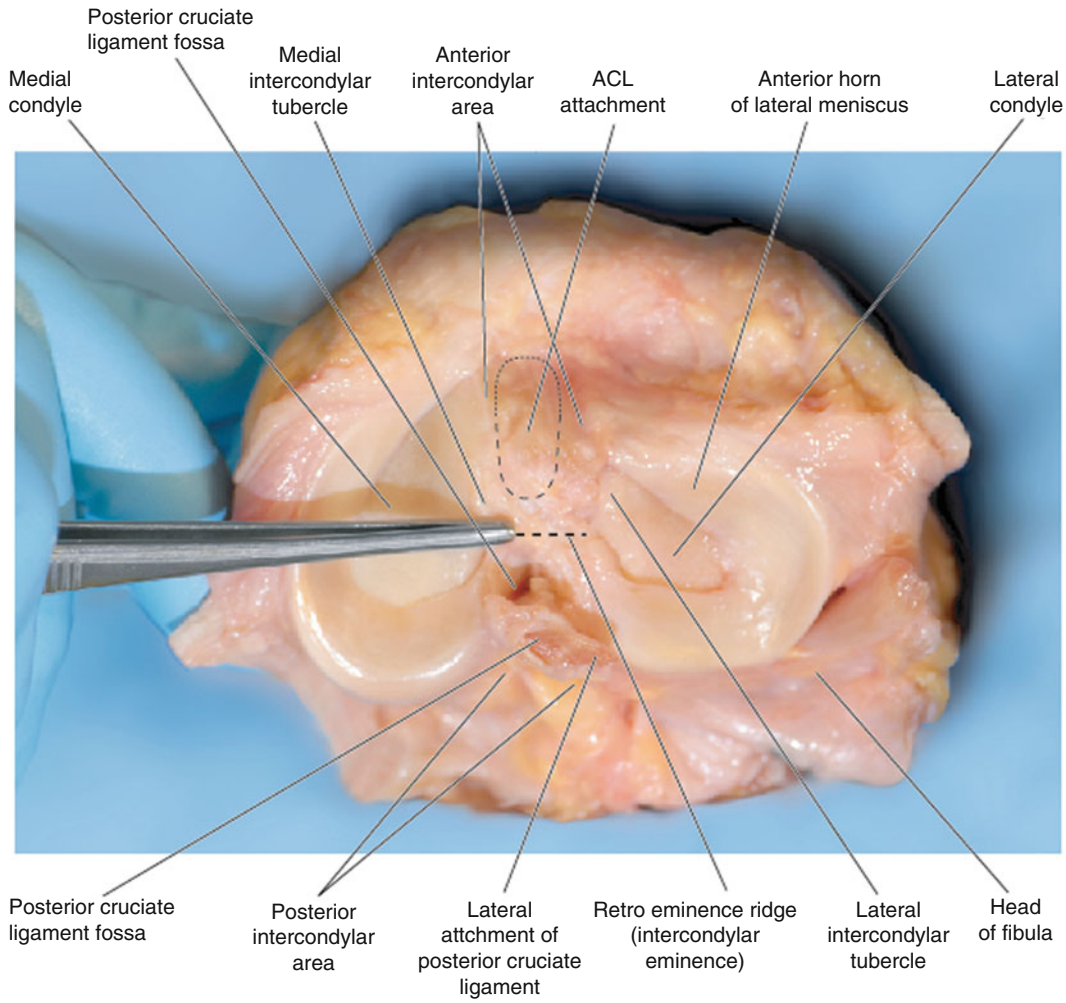

Fig. 1.5 Axial photo of the tibial plateau demonstrating the anterior insertion of the ACL. Notice the ACL's tibial insertion in relation to the medial tibial spine and the retro-eminence ridge (Reprinted with permission from Strickland et al. [89])

substantial anterior tibial loading or the coupled motions of anterior translation and internal tibial rotation are experimentally induced, the majority of the ACL fibers are brought into a load-sharing configuration [69].

The authors believe the classification of the ACL as a structure comprised of two fiber bundles represents a gross oversimplification not supported by biomechanical studies [37, 69, 71, 86]. The length-tension behavior of ACL fibers is primarily controlled by the femoral attachment (in reference to the center of femoral rotation), the combined motions applied, the resting length of the ACL fibers, and the tibial attachment locations. All ACL fibers anterior to the center lengthen during knee flexion, while the posterior fibers lengthen during knee extension. Under loading conditions, fibers in both the AM and PL divisions contribute to resist tibial displacement. The function of the ACL fibers is determined by the anterior-to-posterior direction (with the knee at extension), as well as the proximal-to-distal femoral attachment. The complex geometry and fiber function of the ACL is not restored with current reconstructive methods, regardless of graft choice or the use of single or double-bundle techniques [13, 69].

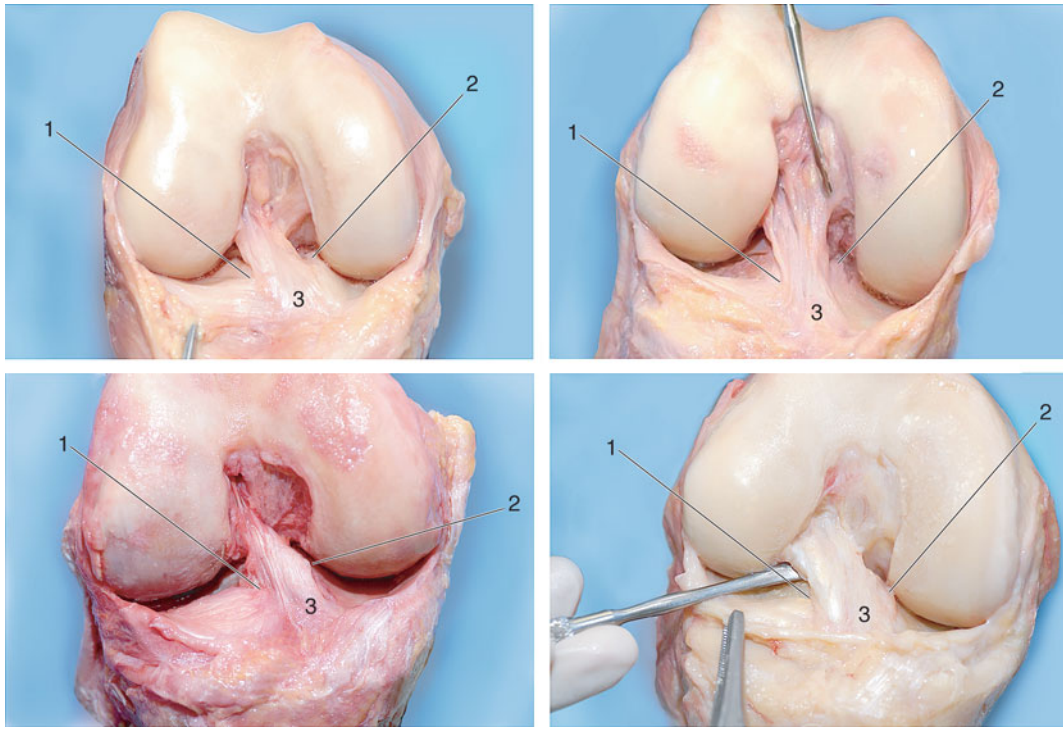

Fig. 1.6 Anterior-superior views of a series of four knees showing that >50 % of the ACL (*3*) inserts anterior to the posterior edge of the anterior horn of the lateral meniscus (*1*). Medial intercondylar tubercle (*2*) (Reprinted with permission from Strickland et al. [89])

Critical Points

ACL

- Mean length 38 mm, width 11 mm.
- Origin on medial aspect of lateral femoral condyle, 18 mm long and 10 mm wide.
- Insertion on tibia in anterior intercondylar area, oval-triangular pattern, anteroposterior dimension 18 mm, mediolateral dimension 10 mm.
- Anterior border 22 mm from anterior cortex of tibia, 15 mm from anterior edge of articular surface.
- Disagreement exists on the division of the ACL into two distinct fiber bundles: anteromedial (AM) proximal half of femoral attachment, tightens with knee flexion; posterolateral (PL) distal half of the femoral attachment, tightens with knee extension
- Reciprocal tightening and relaxation of the AM and PL bundles occurs under no anterior loading conditions.
- Under loading conditions, majority ACL fibers are in load sharing configuration.
- Characterization ACL into two fiber bundles: gross oversimplification, not supported by biomechanical studies.

Biomechanics

The ACL is the primary restraint to anterior tibial translation, providing 87 % of the total restraining force at 30° of knee flexion and 85 % at 90° of flexion (Figs. 1.7 and 1.8) [18]. The secondary restraint to anterior tibial translation is provided by the iliotibial band, mid-medial capsule, mid-lateral capsule, medial collateral ligament, and fibular collateral ligament. The posteromedial

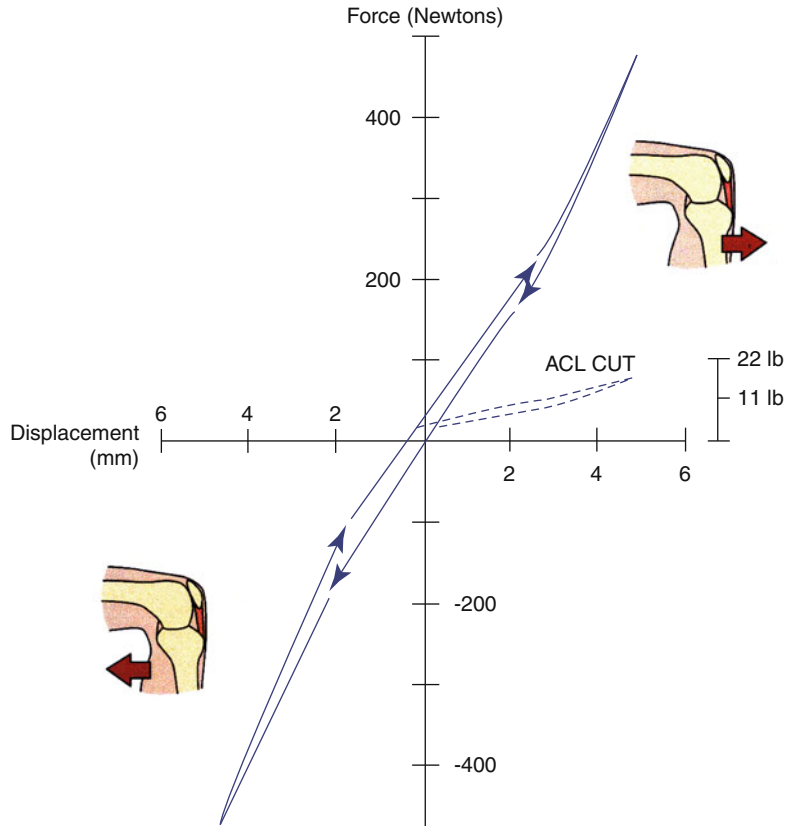

Fig. 1.7 A typical force-displacement curve for anterior-posterior drawer in an intact joint (*solid line*) and after cutting the ACL (*broken line*). The *arrows* indicate the direction of motion (Reprinted with permission from Noyes and Grood [71])

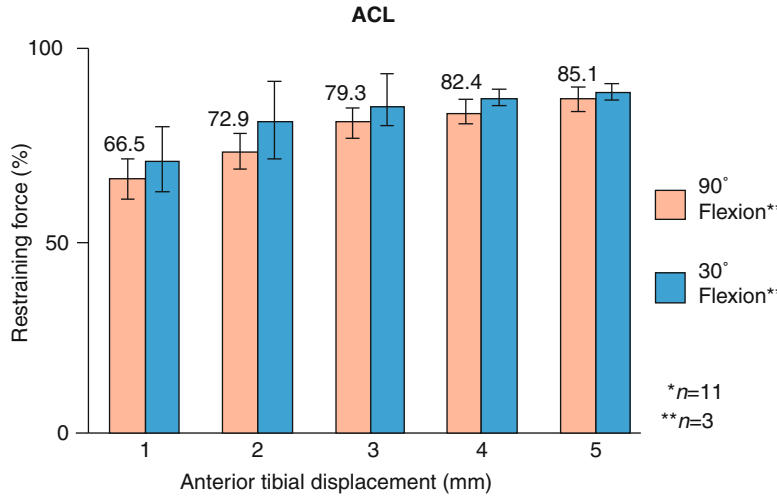

Fig. 1.8 Anterior drawer in neutral tibial rotation. The restraining force of the ACL is shown for increasing tibial displacements at 90° and 30° of knee flexion. The mean value is shown, plus or minus 1 standard error of the mean. Percentage values are given for 90° of flexion. No statistical difference was found between 90° and 30° or between 1 and 5 mm of displacement (Reprinted with permission from Noyes and Grood [71])

and posterolateral capsular structures provide added resistance with knee extension. Repeated giving-way episodes or failure to successfully reconstruct the ACL may result in loss of the secondary restraints and increased symptoms. The failure load and stiffness values of the ACL are 2,160 ± 157 N and 242 ± 28 N/mm, respectively [92]. These values decrease with age [70].

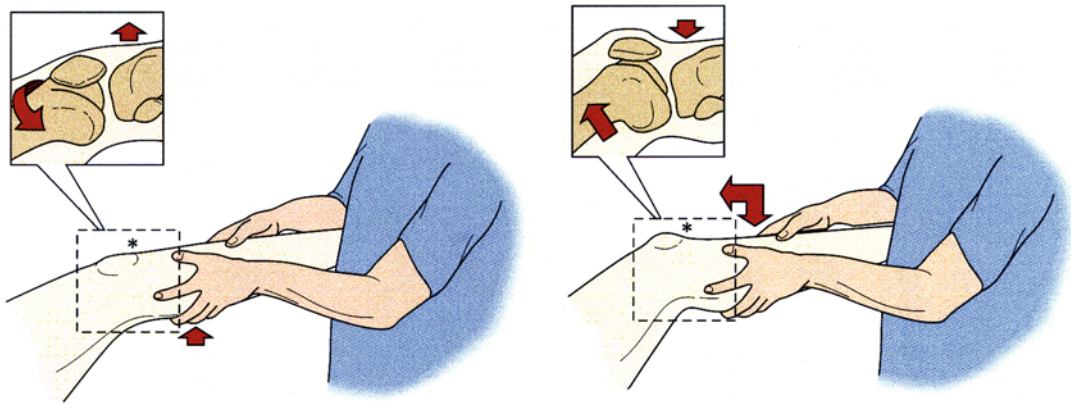

Fig. 1.9 (*Left*) Flexion-rotation drawer test, subluxated position. With the leg held in neutral rotation, the weight of the thigh causes the femur to drop back posteriorly and rotate externally, producing anterior subluxation of the lateral tibial plateau. (*Right*) Flexion-rotation drawer test, reduced position. Gentle flexion and a downward push on the leg reduce the subluxation. This test allows the coupled motion of anterior translation-internal rotation to produce anterior subluxation of the lateral tibial condyle (Reprinted with permission from Noyes and Grood [71])

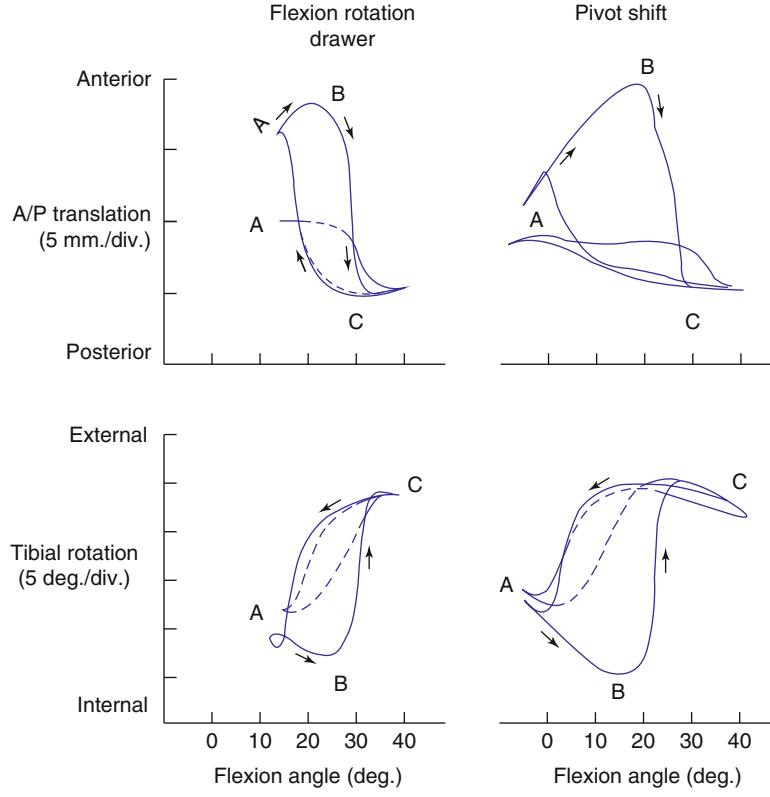

Fig. 1.10 The knee motions during the flexion-rotation drawer and pivot shift tests are shown for tibial translation and rotation during knee flexion. The clinical test is shown for the normal knee (*open circle*) and after ligament sectioning (*dotted circle*). The ligaments sectioned were the ACL, iliotibial band, and lateral capsule. Position *A* equals the starting position of the test, *B* is the maximum subluxated position, and *C* indicates the reduced position. The pivot-shift test involves the examiner applying a larger anterior translation load which increase the motion limits during the test (Reprinted with permission from Noyes and Grood [71])

The ACL limits the combined motions of internal tibial rotation and anterior translation of the tibia, as measured by the pivot-shift and/or flexion-rotation drawer tests [66, 67]. The motions that occur during the pivot-shift maneuver are shown in Figs. 1.9 and 1.10 [69]. In a knee with an ACL rupture, an increase in both anterior tibial translation and internal tibial rotation occurs as the femur drops back posteriorly and externally rotates, causing an

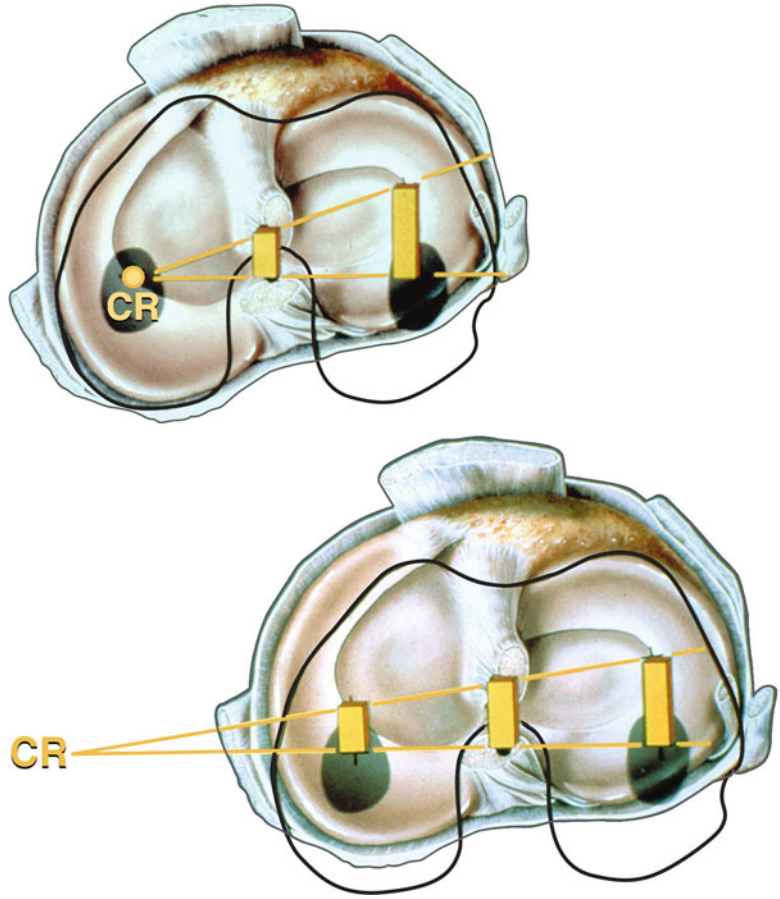

Fig. 1.11 Intact knee and after ACL sectioning: response to coupled motions of anterior tibial translation and internal tibial rotation. *Top*, Intact knee. The center of rotation may vary between the medial aspect of the PCL and meniscus border, based on the loads applied and physiologic laxity of the ligaments. *Bottom*, ACL sectioned; note shift in center of tibial rotation medially. The effect of the increase in tibial translation and internal tibial rotation produces an increase in medial and lateral tibiofemoral compartment translation (anterior subluxation). The millimeters of anterior translation of the tibiofemoral compartment represent the most ideal method to define knee rotational stability. The center of rotation under a pivot-shift type of test shifts to the intact medial ligament structures. If these are deficient, the center of rotation shifts outside the knee joint (Reprinted with permission from Noyes and Barber-Westin [69])

anterior subluxation of the lateral tibial plateau. This position is heightened as the tibia is lifted anteriorly.

The ACL also provides rotational stability to the combined motions of anterior translation and internal tibial rotation (Fig. 1.11). Knees with ACL ruptures demonstrate an increase in medial and lateral compartment translation as the center of rotation shifts from inside the knee joint to outside the medial compartment. The medial ligamentous structures influence the new center of rotation, and therefore, a combined injury to these structures results in the center of rotation shifting far medially, causing a large anterior subluxation of both compartments. These knees require surgical restoration of severely injured medial and lateral ligament structures in addition to the ACL to restore knee stability.

The ACL and posterior cruciate ligament (PCL) are secondary restraints to medial and lateral joint opening and become primary restraints when the collateral ligaments and associated capsules are ruptured. Because the cruciates are located in the center of the knee, close to the

center of rotation, the moment arms are approximately one-third of those of the collateral ligaments. Therefore, to produce restraining moments equal to the collateral ligaments, the cruciates must provide a force three times larger than that of the collaterals.

Li et al. [49] reported that, in the lateral compartment in an uninjured knee, the contact region of the femur moves in a posterior direction on the tibial plateau during squatting (from 0° to 90° of flexion). Very little displacement occurs in the medial compartment of the femur relative to the tibia. Logan and associates [52] measured the translation of the lateral and medial compartments in intact and ACL-deficient knees during a Lachman test in a vertically oriented open-access magnetic resonance imaging (MRI) scanner. The ACL-deficient knees had significantly greater mean anterior tibial translation in both compartments, with the lateral side demonstrating greater translation than the medial side. The mean anterior tibial translation was significantly greater in the ACL-deficient knee compared to the intact knee in the medial compartment (8.2 ± 1.9 and 2.5 ± 1.7 mm, respectively, $P < 0.004$) and in the lateral compartment (14.1 ± 3.6 and 4.7 ± 2.3 mm, respectively, $P < 0.0002$).

Logan et al. [51] measured tibiofemoral motion in both compartments during a weight-bearing squat in an open access MRI scanner in ten patients who had unilateral ACL ruptures. There was a significant increase in the posterior translation of the femur on the tibia in the lateral compartment in the ACL-deficient knees compared to the intact knees throughout the arc of motion ($P < 0.001$). No difference was found between knees in medial compartment translation. The authors concluded that the change in kinematics caused greater internal tibial rotation during knee flexion, which may facilitate giving way during activity. Other changes in knee kinematics and kinetics with loss of ACL function are described in Chap. 2. The reader is referred to other references for a more extensive discussion on ACL function [69, 70].

One study compared male and female cadaveric knees to determine if gender differences existed in ACL structural and material properties [19]. Ten female and ten male knees (mean age, 36 years; range, 17–50) were tested to failure. The female ACLs had lower mechanical properties (14.3 % lower stress at failure, 9.43 % lower strain energy density at failure, 22.49 % lower modulus of elasticity) compared to the male ACLs. The authors reported that the structural properties were weaker in the female specimens even after controlling for age and ACL and body anthropometric measurements.

Critical Points

- ACL primary restraint anterior tibial translation, provides 85–87 % total restraining force.
- ACL failure load 2,160 ± 157 N, stiffness 242 ± 28 N/mm.
- Limits coupled motions of internal tibial rotation and anterior tibial translation.
- Loss of ACL: increase in medial and lateral compartment translation as center of rotation shifts to outside the medial compartment.
- ACL and PCL are secondary restraints to medial and lateral joint opening, become primary restraints when collateral ligaments and capsules are ruptured.
- Female ACL appears to have lower mechanical properties than male ACL.

Common ACL Injury Mechanisms

Current Proposed Mechanisms

Knee joint stability during weight-bearing activities is influenced by the muscles, ligaments, and bony geometry which act together to resist potentially dangerous forces and external adduction moments (Fig. 1.12). At least two-thirds of ACL tears occur during noncontact situations while an athlete is cutting, pivoting, accelerating, decelerating, or landing from a jump [13, 15, 84]. Common injury circumstances have been described for both men and women, including perturbation of

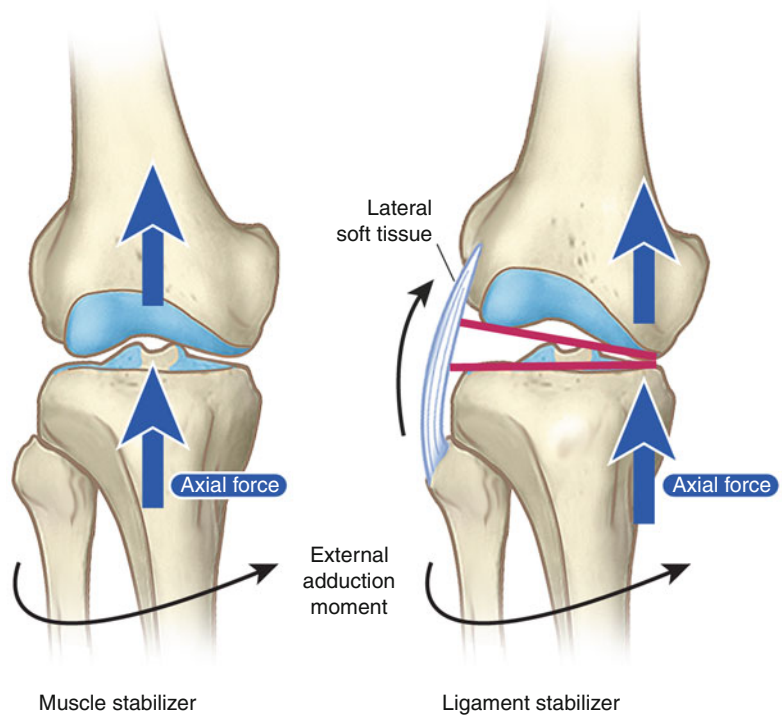

Fig. 1.12 *Left*, knee joint stability is influenced by the muscles, ligaments, and bony geometry, which act together to resist external adduction moments that are incurred during weight-bearing activities. *Right*, an abnormally high adduction moment may result in laxity of the lateral soft tissues and loss of normal lateral tibiofemoral joint contact. Termed *lateral condylar lift-off*, this phenomenon increases the potential for an ACL rupture, especially if the knee is in 30° of flexion or less (Reprinted with permission from Barber-Westin and Noyes [8])

the athlete from an opponent, reported in ≥90 % of injuries in several studies [16, 42, 45, 74]. Perturbation situations include being pushed or shoved just before the injury, avoiding a player in close proximity usually while playing offense, or attempting to avoid a collision with another player. These circumstances cause an athlete to be off-balance or lose control and alter their normal neuromuscular mechanics.

Numerous abnormal biomechanical loads producing ACL injury have been studied over the past two decades. These include [36]:

1. Anterior shear force, arising from large quadriceps contractions that occur with low knee flexion angles and lack of hamstrings muscle activation [5, 11, 12, 24, 27, 32, 48, 57]
2. Axial compression loads [47, 61]
3. Hyperextension [15, 56]
4. Valgus collapse at the knee joint [16, 45, 59, 85]
5. Internal tibial rotation [27, 58]
6. Combinations of #1–5

Markolf et al. [57] measured in vitro forces in cadaver ACL specimens during isolated and combined loading states. A high valgus or varus moment increased the risk for medial or lateral tibiofemoral joint lift-off and the potential for a knee ligament rupture. Abnormally high adduction moments may result in laxity of the lateral soft tissues and loss of normal lateral tibiofemoral contact, termed lateral condylar lift-off. These investigators reported that increases in ACL forces were greater when a valgus or varus moment was applied along with an anterior tibial translation compared to when an anterior tibial translation was applied alone. Many noncontact ACL tears have been noted to involve loads in multiple planes, as was suggested in this laboratory study.

Reduced knee flexion angles, increased hip flexion angles, valgus collapse at the knee, increased hip internal rotation, and increased internal or external tibial rotation are frequently reported at the time of or just prior to ACL injury. Debate exists regarding which of these neuromuscular mechanics are present at the time of the injury and which may occur just following the injury. Olsen and associates [74], in the first study

to use videotape analysis of sequences of ACL injuries, admitted that "whether the consistent valgus collapse observed in the videos was actually the cause of injury or simply a result of the ACL being torn is open for discussion." Hashemi et al. [35] in 2010 proposed a framework for establishing the viability of a noncontact ACL injury mechanism that included nine questions shown below:

1. What is the inciting event?
2. What are the muscle forces and joint torques/loading necessary to produce an ACL injury? Are these proposed injury forces physiological?
3. Does the proposed mechanism meet the timing requirement for ACL injury?
4. What is the role of fatigue on neuromuscular control of the muscles that span the knee, hip, and ankle joints?
5. How does sagittal and transverse kinematics of the hip as well as ankle complex kinematics alter ACL loading?
6. What is the role of the tibial plateau geometry in loading the ACL?
7. What is the effect of knee laxity on ACL injury mechanism?
8. What causes the abnormal knee kinematics during ACL injury?
9. How does the mechanism explain the existing sex-based disparity in ACL injuries?

These authors presented the mechanisms which had been discussed in the literature to date, including excessive quadriceps force producing an anterior tibial shear force, excessive joint compression mechanisms, knee abduction moments (valgus), and tibial rotation. They believed that future studies needed to address a greater number of mechanisms in multiple plans and involving not only the knee joint but the hip and ankle joints as well.

Hashemi et al. [36] proposed a theory for a noncontact ACL injury mechanism comprised of four conditions: delayed or slow co-activation of quadriceps and hamstrings muscles, dynamic ground reaction force applied while the knee is near full extension, a shallow medial tibial plateau and steep posterior tibial slope, and a stiff landing due to incompatible hip and knee flexion angles. Experimental validation of this model is required.

Effect of Muscle Forces and Knee Flexion Angle

The ACL is strained when the quadriceps are contracted with the knee near full extension. Cadaver studies [24, 48, 81] have shown that isolated quadriceps isometric and isotonic contractions increase ACL strain from 0° to 45° of flexion, with the greatest magnitude occurring at full knee extension. DeMorat et al. [24] postulated that the force caused by a high quadriceps contraction with the knee in only slight flexion could induce ACL rupture. In the laboratory, a 4,500 N quadriceps contraction in a cadaver knee in 20° of flexion resulted in a complete ACL disruption at the femoral insertion site in 6 of 11 knees and a partial ACL injury in three other knees. Fleming et al. [28] reported that the gastrocnemius muscle is an ACL antagonist when the knee is near full extension. A co-contraction of the gastrocnemius and the quadriceps loads the ACL to a greater extent than isolated contractions of these muscle groups at 15° and 30° of flexion.

The hamstring musculature is important in stabilizing the knee joint [54, 62, 65] but has a marked functional dependence on the angle of knee flexion and degrees of external tibial rotation (Fig. 1.13). These muscles actively oppose extension by stabilizing the knee posteriorly and preventing knee hyperextension and anterior subluxation of the tibia. In addition, they actively oppose external tibial rotation. The mechanical advantage of the sartorius, gracilis, and semitendinosus muscles increases as the knee goes into further flexion. For instance, at 0° of extension, the flexion force is reduced to 49 % of that measured at 90° of flexion. In addition, a quadriceps-hamstrings co-contraction cannot reduce ACL strain from full extension to approximately 20° of flexion [72, 75].

Pollard et al. [79] studied the effect of knee and hip flexion angles during the deceleration phase of a drop-jump task on knee kinematics and moments. Subjects that used less knee and hip flexion on landing demonstrated increased knee valgus angles, knee adductor moments, and vastus lateralis activity compared to athletes who landed with greater hip and knee flexion (see also

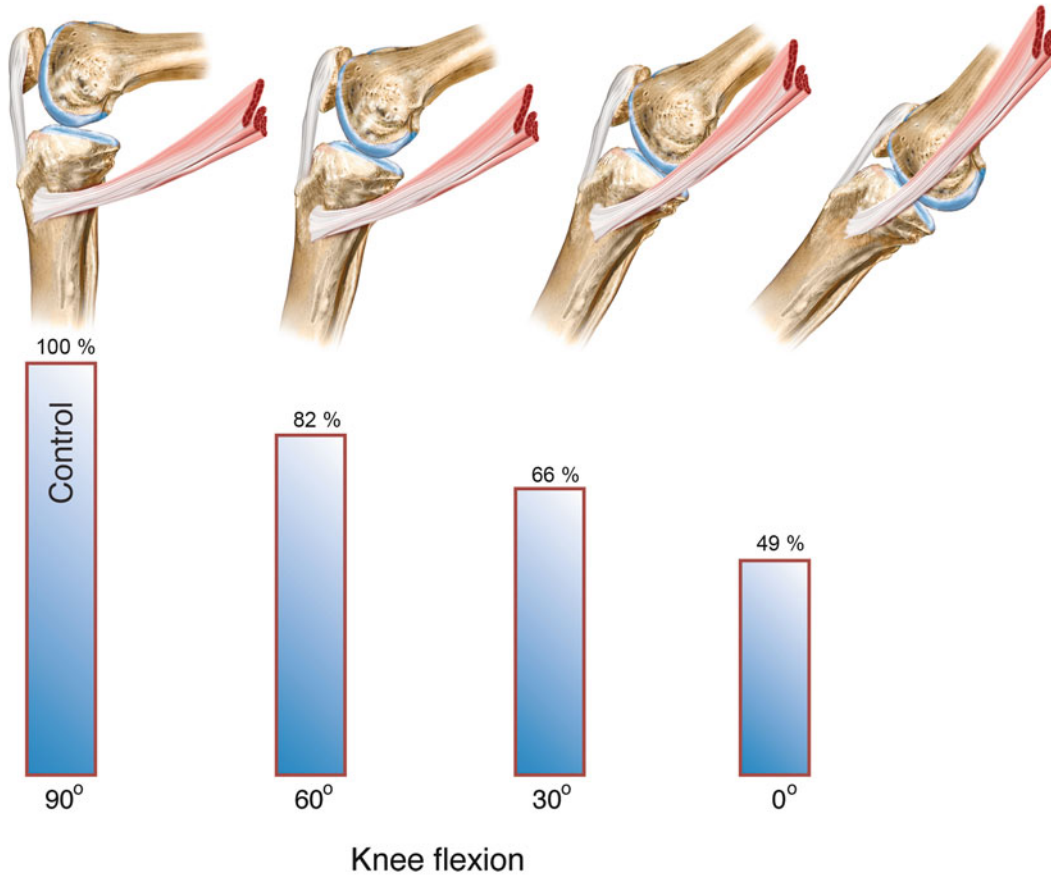

Fig. 1.13 Pes anserine (sartorius, gracilis, semitendinosus muscles) flexion forces. Stick figures show that knee extension yields a decrease in insertion course angles with respect to the tibia. Resulting loss in mechanical advantage is indicated by reduced flexion forces with extension ($P<0.05$) (Reprinted with permission from Barber-Westin and Noyes [7])

Chap. 10). This demonstrates that the quadriceps dominant pattern is influenced by the inability of the hip extensors to absorb energy when athletes land in low hip flexion angles.

Colby et al. [22] measured hamstring and quadriceps muscle activation and knee flexion angles during eccentric motion of sidestep cutting, crosscutting, single-leg stopping, and landing/pivoting in nine male and six female collegiate athletes. A high quadriceps muscle activation occurred just before foot strike and peaked in midstance. The peak quadriceps muscle activation occurred between 39° (stopping) and 53° (crosscutting) of knee flexion and averaged between 126 % (landing) and 161 % (stopping) of that measured in a maximum isometric contraction. Hamstring muscle activation was submaximal at and after foot strike. The minimal hamstring muscle activation occurred between 21° (stopping) and 34° (cutting) of knee flexion and averaged between 14 % (landing) and 40 % (crosscutting) that of a maximum isometric contraction. The knee flexion position that foot strike occurred during all four activities averaged 22° (range, 14° on stopping to 29° on crosscutting). The authors concluded that the combination of the high level of quadriceps activity, low level of hamstrings activity, and low angle of knee flexion during eccentric contractions in these maneuvers could produce significant anterior translation of the tibia.

Video Analysis of ACL Injuries

Koga and associates [42] analyzed ten ACL injury sequences from female handball and basketball players. The video sequences were quantified using a computerized 3-dimensional analysis technique that replicated the lower limb and knee joint motions that occurred during the injuries. Seven injuries occurred during cutting and three during single-leg landings. The injured player was handling the ball in all cases. The data regarding knee flexion angles, knee adduction and abduction angles, and tibial rotation demonstrated that, at initial foot contact before the injury, a neutral limb position was present. The knee abduction angle was neutral (mean 0°; range, −2° to 3°), and the tibial rotation angle was slightly external (mean, 5°; range, −5° to 12°). Knee flexion averaged 23° (range, 11–30°). The authors noted at approximately 40 ms later a mean increase in the knee flexion angle of 24° (range, 19–29°), a mean increase in the abduction (valgus) angle of 12° (range, 10–13°), and a change in tibial rotation from 5° external to 8° internal (range, 2–14°). There were "remarkably consistent descriptions of knee joint kinematics" from all ten injury situations, and the conclusion was reached that the ACL injuries most likely occurred within 40 ms from initial foot contact.

Boden et al. [16] used videotapes of noncontact ACL injuries to conduct an analysis of the position of the hip and ankle in eight female and four male athletes. The majority of subjects (96 %) had an opposing player in close proximity just before or during the injury, and most were playing offense, both of which could have caused an alteration in the normal hip, knee, and foot positions. When the injury occurred, the female subjects typically were performing a deceleration motion, whereas the male players were performing strenuous jumping and landing maneuvers. Compared to a control group analyzed during similar potential injury-type athletic motions, the subjects landed in a flat-footed position, with little ankle plantar flexion. This landing position was postulated to lead to a lack of energy dissipation by the gastrocnemius-soleus complex, thereby increasing forces to the knee. Higher hip flexion angles were noted as well for the subjects. The authors reported few differences in the injury mechanics between male and female athletes.

Krosshaug and associates [45] analyzed video footage of 17 male and 22 female basketball players who sustained ACL ruptures and reported that opponents were in close proximity in nearly all of the injury situations. Approximately one-half of the players were pushed or collided with another player just before the injury occurred, which may have altered the normal movement patterns of the subjects. Knee collapse into valgus was noted in 53 % of the female players and in 20 % of the male players. The collapse was described as a combination of hip internal rotation, knee valgus, and external tibial rotation. These authors also reported that females landed with greater hip and knee flexion than male players. However, the reliability of the study's visual inspection approach to measuring these flexion angles was questionable. Another investigation reported consistent underestimates of nearly 20° of knee flexion when comparisons were made of measurements obtained from video and the actual flexion angles that occurred during running and cutting [44].

Olsen et al. [74] analyzed videotapes of 19 noncontact ACL injuries in female team handball players. The most common injury mechanism was a plant-and-cut movement in which a forceful valgus collapse of the knee and tibial rotation (internal or external) with the knee close to full extension were noted. The foot was firmly planted on the handball court and was outside of the knee in nearly all cases. The authors acknowledged that it was unknown whether the valgus position of the knee caused the ACL injury or occurred as a result of the injury. These authors also reported that the majority of subjects were out of balance as a result of being pushed or held by an opponent or trying to evade a collision, which caused the unusually wide foot position relative to the knee and center of the body.

Authors' Proposed Mechanisms of Noncontact ACL Ruptures

From the videotaped analyses of ACL injuries, it appears that the amount of time in which an ACL