Biomechanics of Ligaments in Sports Medicine

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The motion of the appendicular skeleton is powered by the muscles that cross the various synovial joints. In turn, these joints are stabilized and guided by ligaments—bands of tough connective tissue that traverse the joints and help to limit excessive displacements between the bones. Once thought of as inert, static structures designed solely to maintain proper skeletal alignment, ligaments are now known to have many functions. Ligaments are subjected to a relatively low level of forces during repetitive activities of daily living. However, during running, throwing, or jumping the loads can be high, and loss of biomechanical integrity of the ligament may result. The high loads carried by the ligaments during strenuous activity can cause acute ruptures or tears of the midsubstance of the ligament or an avulsion injury at the insertion site.

Intense laboratory study has shown that ligaments, as well as tendons, exhibit complex and nonlinear load-elongation behavior under uniaxial tensile loads. A more complete understanding of their biomechanical function can provide insight into the mechanisms that cause ligament injury. Treatment of ligament injuries by immobilization and by controlled motion has also been studied in the laboratory. These factors can have significant effects on outcome as well as on the design of treatment and rehabilitation programs for patients with ligamentous injuries.

The goal of this chapter is to provide the reader with a basic understanding of the biomechanical properties of ligaments and how ligaments contribute to the motions of various synovial joints of the body, specifically the knee, shoulder, and ankle. The structure of ligaments and the basic concepts of biomechanics are described by using the medial collateral ligament (MCL) of the knee as an example. The effects of age, stage of skeletal maturation, immobilization, and exercise on the biomechanical properties of the MCL are covered. Major ligaments associated with the three synovial joints (i.e., knee, shoulder, and ankle) are presented in separate sections. In each section, a brief review of the anatomy is presented, followed by a description of the tensile properties and functions of the ligaments. Afterward, injuries of ligaments and current treatment options are described, together with biomechanical methods available to evaluate their outcome. The information presented in this chapter is a useful reference for clinicians who diagnose, treat, and rehabilitate patients with ligamentous injuries of the knee, shoulder, or ankle.

STRUCTURE OF LIGAMENTS
A ligament consists of a complex arrangement of extracellular matrix with randomly interspersed cellular elements. These cells, called fibrocytes, are randomly embedded throughout the matrix and are responsible for extracellular matrix production. The fibrocytes are also responsible for biologic adaptation to the mechanical environment, remodeling, and healing of injured ligaments (1,2). The extracellular matrix is predominantly a network of fibrillar collagen structures arranged parallel to the long axis of the ligament. The collagen network has an architectural hierarchy similar to that of the tendon—beginning with tropocollagen as the basic molecular component, and systematically arranged into a hierarchic structure of microfibrils, subfibrils, fibrils, and fibers (Fig. 5.1) (3). This well organized, collagenous network is responsible for the stiffness and strength of the ligament. Type I collagen is the major component of all ligament fibers; collagen types III, V, VI, X, and XII exist in small amounts (1,4,5). Small amounts of glycoproteins (e.g., fibronectin) and proteoglycans also exist as part of the extracellular matrix. The proteoglycans are hydrophilic and consequently play an important role in the interaction with the collagen fibers to yield viscoelastic properties of the ligament. Elastin, a fibrillar protein, present in small amounts, usually accounts for less than 1 of the dry weight, and contributes to the mechanical properties of ligaments.


The insertion of a ligament into bone represents a transition that is quite complex. Insertion sites are of particular importance because an abrupt transition from soft to hard tissues can create large, localized stress concentrations under applied tensile loads. Therefore, the transitional zones must be formed to minimize stress concentration. Two distinct ligament insertion sites, referred to as direct and indirect, have been identified (6). **Direct insertions** are those in which the fibers of the ligament insert directly into the bone. Morphologically, they can be divided into four distinct zones: ligament fibers, uncalcified fibrocartilage, calcified fibrocartilage, and bone. An example of a direct insertion is the femoral insertion of the MCL of the knee. **Indirect insertions**, such as the tibial insertion of the MCL, are those in which the superficial fibers blend with the periosteum and the deep fibers insert obliquely into the bone with little or no transitional zone. Indirect insertions can be found where ligaments cross the epiphyseal plate. During skeletal maturation, remodeling of the
interface of the deep fibers takes place regularly, as evidenced by the osteoclastic activities, while the attachment of the superficial fibers to the periosteum is lengthened to allow for bone growth at the epiphysis.

In most ligaments, a thin layer of tissue called “epiligament” covers the ligament (7). This layer is abundant in cells and blood vessels and may be a major source of cells needed for remodeling and healing responses. Compared with other connective tissues such as bone and skin, ligaments are poorly vascularized (7). Free nerve endings have been discovered in ligaments (8). Activation of these nerve fibers, by an increase in ligament tension, leads to appropriate muscular function so that the joint functions properly to prevent possible injury. From this finding, it is thought that ligaments play an important role in proprioception.

**BIOMECHANICS OF LIGAMENTS OF THE KNEE**

**Medial Collateral Ligament**

The medial collateral (or tibial collateral) ligament of the knee is an extraarticular structure that attaches the tibia to the femur (Fig. 5.2). It is an ideal ligament to study in the laboratory because of its accessibility, relatively uniform cross-sectional area, and reasonable length-to-width ratio. Most importantly, it is able to heal after rupture. However, what has been learned about ligaments from study of the MCL may not be completely applicable to other ligaments. In the relaxed or unloaded state, the fibrillar components of the MCL are arranged in an undulating path (described as the “crimp”) between origin and insertion.

**Anatomy and Function of the Medial Collateral Ligament**

The MCL originates from the medial epicondyle of the femur and inserts onto the anterolateral portion of the tibia, just deep to the insertion of the pes anserinus tendons. The MCL can be divided into superficial and deep layers. The superficial portion of the MCL is a distinct extracapsular structure that is considered to be in layer II of the supporting...
structures of the medial side of the knee. The anterior fibers of the superficial portion of
the MCL provide the primary restraint to valgus knee stress and appear to become taut with knee flexion of 70 to 105 degrees. The deep MCL is actually a thickening of the joint capsule and is referred to by some as the middle capsular ligament. Along with the joint capsule itself, it is a component of layer III and contributes very little to the stability of the knee joint. The MCL and its associated medial structures also act as secondary restraints, resisting anteroposterior translation.

**Tensile Properties of Medial Collateral Ligament**

One of the primary functions of ligaments is to resist tensile loads, thereby guiding joints through their physiologic range of motion. It is common practice to measure the biomechanical properties of ligaments by uniaxial tensile tests. Because of the limited length of the MCL, these tests should be done with the bones, or using the femur-MCL-tibia complex (FMTC). Typically, a nonlinear, concave load-elongation curve that represents the structural properties of FMTC is obtained (Fig. 5.3). Structural properties of ligaments are represented by parameters such as stiffness, ultimate load, ultimate deformation, and energy absorbed to failure, which are defined as follows:

- **Stiffness (N per mm):** The relationship between load and elongation measured as the slope of the linear portion of the load-elongation curve.

- **Ultimate Load or Load at Failure (N):** The highest load observed just before failure of the tissue during a tensile test. In the literature, this parameter is sometimes erroneously referred to as "tensile strength."

- **Ultimate Elongation (mm):** The maximum length a tissue can be stretched from its initial unloaded length until failure.

- **Energy Absorbed at Failure (N-mm):** The entire area under the load-elongation curve.
This load-elongation curve results from the contributions of the substance of the ligament and the bony insertions. Two regions of this curve must be distinguished. Initially, there is a nonlinear region, referred to as the “toe” region. When subjected to tensile loads, the MCL, like most soft tissues, exhibits a nonlinear load-elongation curve as well as a time-dependent response. This nonlinear response is thought to be the result of progressive straightening and stretching of an increasing number of fibers, in a process called recruitment (9). With increasing tensile load, increased stiffness is seen, and eventually a “linear” region is reached where the slope of the curve is constant. Under these loads, ligaments are stiff and can help to limit excessive displacement between the two bones. In this way the ligaments maintain joint position and alignment when large muscle forces act on the joint.

From the same test, the quality of the MCL substance (i.e., its mechanical properties), as represented by a stress-strain curve, can also be determined. The stress (σ) in a ligament is defined as the load per unit of cross-sectional area of the ligament substance. Accurate measurement of the cross-sectional area is very important and ensures that the tensile stress values are correct. A laser micrometer system has been used to determine the cross-sectional area as well as the shape of the midsubstance of the ligament (10). Strain (ε), is defined as the change in length divided by the original length. In addition, for the stress-strain curves to be valid in a uniaxial tensile test, the ligament must have a uniform cross-section along its length and an adequate length-to-width ratio.

Similar in shape to the load-elongation curve, a nonlinear stress-strain curve for an MCL is shown in Figure 5.4. Mechanical properties of the ligament are represented by parameters such as the modulus, tensile strength, ultimate strain, and strain energy density, which can be obtained from the stress-strain curve and are defined as follows:

- Modulus (N per mm$^2$): The relationship between stress and strain measured as the slope of the linear portion of the stress-strain curve.
- Ultimate Tensile Stress, or Tensile Strength (N per mm$^2$): The maximum stress
observed on the stress-strain curve before failure of the tissue.

- Ultimate Strain (% mm per mm): The strain at failure of the ligament substance.
- Strain Energy Density (MPa). The area under the stress-strain curve.

Ligaments also exhibit complex time- and history-dependent, viscoelastic behavior resulting from the interactions of collagen, proteoglycan molecules, and water with the surrounding elastin and other ground substance (11,12). Therefore, the paths of the load-elongation curve for the ligament during loading and unloading are different, forming what is known as a hysteresis loop. The area enclosed by the hysteresis loop represents the internal energy loss between loading and unloading (Fig. 5.5). Other important viscoelastic characteristics are stress relaxation, a decline in stress over time under constant elongation (Fig. 5.6), and creep, an increase in length over time under a constant load (Fig. 5.7). Ligament behavior depends on the previous loading history and the time during which the load is applied.
During walking or jogging, ligaments experience repeated cyclic stretching (13). Elastic materials would normally experience fatigue failure under these loading conditions—that is, application of repetitive loading would cause failure at a much lower value than that required to cause failure from a single application of load. As a ligament undergoes many cycles of loading and unloading in vivo, its cyclic relaxation behavior results in continuously decreasing stress (Fig. 5.8). This helps to protect the ligament from fatigue failure.

**Biologic Factors**

There is profound variation in the properties of ligaments reported in the literature. Many biologic factors and experimental procedures have been identified for these discrepancies. Specifically, biologic factors such as species, skeletal maturation, age, biochemistry, immobilization, and exercise are well known. The following section discusses some of these factors using the MCL of the knee as example.

For skeletally immature rabbits, failure of the FMTC during tensile testing occurred at the tibial insertion site,
suggesting that the tibial insertion site is the weakest link. On the other hand, in mature animals failures consistently occurred at the ligament midsubstance (14). Furthermore, structural properties of the rabbit MCL showed a dramatic increase between 6 and 12 months of age, after which the differences between the age groups diminished up to age 4 years (15). Similar trends were seen in mechanical properties of the MCL substance.

Other experiments have shown that immobilization of joints leads to significant decreases in ligament tensile properties and to stiffening of the joints (16). Laros et al. found that immobilization of the canine FMTC for up to 12 weeks reduced the ultimate load-to-body-weight ratio by approximately 27% compared with controls (17). Immobilization also led to bone resorption and disruption of ligament fibers at the MCL tibial insertion site. In rabbits and rats, stiffness and ultimate load of the FMTC were reduced by 25% to 33% after 4 to 9 weeks of immobilization (18,19). The reduction of structural properties appeared to result from changes at the insertion sites, as well as in the ligament substance, because there was a marked disruption of the deep fibers inserting into bone. Osteoclastic activity resulted in subperiosteal bone resorption for the tibial insertion but little change to the femoral insertion. This resorption correlates with an increasing occurrence of failure by tibial avulsion for the FMTC from immobilized knees.

Remobilization can reverse the deleterious effects of immobilization on the structural properties of the FMTC, but much longer time periods are required. Experimentally, 18 weeks of remobilization were necessary to reverse the detrimental effects of 6 weeks of immobilization on the structural properties of canine MCLs (17). Studies from our research center on the effects of a 9-week immobilization period revealed that, histologically, the tibial insertion of the rabbit MCL was not reestablished until after 52 weeks of remobilization (20). These data imply that, clinically, after a period of immobilization, caution must be exercised for an extended period to limit the risk of subsequent injury.

Exercise can enhance the structural properties of ligaments (e.g., stiffness, ultimate load) by about 10% to 20% (21). Tipton et al. demonstrated a 10% increase in both FMTC ultimate load and the ratio of ultimate load to body weight and a 20% increase in the energy absorbed at failure in rats that had undergone 10 weeks of treadmill training (22). Figure 5.9 represents our view on tissue homeostasis, that is, the effects of immobilization and exercise and their relation to mass and tissue properties of ligaments. Immobilization significantly compromises both the structural properties of the bone-ligament-bone complex and the mechanical properties of the ligament, with weakening being more pronounced at the insertion sites. The mechanical properties of the ligament substance return to control levels after a relatively short period of remobilization, but the insertions require a much longer period of recovery to regain their previous stiffness and strength. Therefore, the complex remains weak, and avulsion injuries are more likely during this interval. The gains in these properties resulting from exercise or increased stress are not markedly large; only moderate improvements of the properties are seen. Therefore, a nonlinear relationship exists between the stress and strain duration and mechanical properties of a ligament (Fig. 5.9).
Injury, Healing Response, and Treatment Options

Fetto and others reported that the MCL is the most frequently injured ligament of the knee and accounts for 55% of all acute knee injuries (23,24). Most isolated MCL injuries occur during sports activities such as football, soccer, baseball, and skiing.

The term healing refers to tissue repair after injury. The potential for healing varies greatly and depends mainly on the blood supply and the metabolic rate of the involved tissue. Although skin and bone, with some limitations, heal relatively quickly and predictably, ligaments are believed to have slow and limited healing potential (1). Ideal healing would lead to complete restoration of the original tissue with identical morphologic and functional characteristics, but ligaments have been shown to heal with qualities that are inferior to those of the original tissue. The healing of ligaments follows a predictable pattern, which occurs in three specific phases (7).

- Hemorrhage or Inflammatory Phase. This phase is characterized by formation of a blood clot within the damaged region and the invasion of polymorphonuclear cells and monocytes/macrophages. The monocytes remove debris and attract granulation tissue–producing reparative cells.

- Proliferative Phase. In this phase, new blood vessels are formed while fibroblasts are recruited from the local environment or circulation to produce new matrix material (mainly collagen).

- Remodeling Phase. This phase starts within weeks after the injury and can last up to several years. It is characterized by a progressive maturation of collagen fibers, which align in a longitudinal orientation in response to loads applied to the ligament.

Investigators have documented that injuries of the MCL can heal with resulting structural properties of the FMTC equal to or almost equal to those of the normal ligament, thus providing the needed function for the involved joint (25,26 and 27). Other ligaments, such as the cruciate ligaments of the knee, heal with diminished strength or do not heal at all, often resulting in increased joint laxity. In isolated ruptures of the MCL, the ligament healed macroscopically without any surgical intervention within 6 weeks (26,28). Compared with
the preinjury state, the cross-sectional area of the healed ligament was larger at 6 to 12
weeks and slowly continued to increase with time to reach structural properties of the
normal ligament (29). Even though the structural properties of the FMTC may return to
almost normal values, the mechanical properties of the healing MCL always remain inferior
when compared with normal tissue (Fig. 5.10). This occurs because the healing tissues
increase in mass to compensate for inferior quality. In some studies, the cross-sectional
area of the healed MCL was up to 2.5 times larger than that of controls after 52 weeks (30).
When severe joint laxity is associated with the ligament

injury, such as in a combined injury to the MCL and anterior cruciate ligament (ACL) of the
knee, the quality of the healed tissue (i.e., its mechanical properties) is even worse and the
increase in cross-sectional area is even more pronounced (31,32).

Histologically, healed MCL tissue differs from normal in three regards. First, the collagen
fibers are more disorganized, with more random pattern and more defects between them,
and the number of larger-diameter collagen fibrils is reduced (33,34). Second, the cell
density and vascularity of the tissue are also increased, suggesting that metabolic rates
are higher than normal (35). It takes time for the vascularity of the ligament to diminish and
even longer for the fibers to orient in the direction of applied physiologic stresses. Finally,
there are no fibrils with large diameters, and it takes up to 2 years before a small
population is observed. This lack of large-diameter collagen fibrils in the healing MCL is
important because greater stiffness and strength of the tissue are associated with the
larger-diameter fibrils (36).

Biochemical analysis of healing MCLs revealed that although the collagen content of the
tissue returns to nearly normal within 12 to 14 weeks, collagen cross-links remain at less
than 50% of normal MCL up to 1 year after injury (26,29,37). Further, the amount of type I
collagen is reduced in favor of minor collagen types, and the size of the proteoglycan
molecules is increased (35).

Clinical protocols for MCL injuries have evolved from the aggressive surgical treatment of
most MCL tears to the current emphasis on nonoperative treatment for most of these
injuries. This change has been a result of studies in experimental animals. Nevertheless,
laboratory studies continue to evolve, because there are three major variables that affect the healing response of ligaments: nonrepair versus surgical repair, immobilization versus early motion, and biologic manipulations such as the use of growth factors.

Using the MCLs of dogs and rabbits as a model, the effects of surgical repair of ligaments was compared with those of conservative treatment. A long-term study failed to demonstrate a benefit from suture repair compared with conservative treatment (25,26). The MCL had macroscopically healed after 12 weeks; varus-valgus knee laxity was slightly increased initially and returned to normal after 1 year. Structural properties of the FMTC, such as stiffness and ultimate load, were slightly diminished even at 1 year. Mechanical properties of the healed MCL remained at about 50% of normal, indicating that the tissue quality remained below normal (26). Further, in a more severe injury model (in which both MCL and ACL were injured), surgical repair of the MCL improved structural properties in the early phase (31,38) but the long-term results did not show clinical benefit from initial repair of the MCL (38).

Although immobilization has often been used in combination with ligament repair to protect the tissue from damage during the early stages of healing, its deleterious side effects, including increased joint stiffness, proliferation of fibrofatty connective tissue, and synovial adhesions, are well known (16,20). Studies in our research center as well as others have revealed that ligaments regain a higher strength after rupture when treated with passive motion instead of immobilization (39,40). Clinical studies also suggest that well-controlled protocols including early mobilization and more aggressive rehabilitation are effective in restoring joint motion without compromising stability (41,42).

Because restoration of normal tissue quality in ligament healing has yet to be accomplished under usual circumstances, several biologic approaches to enhance the quality of the healing tissue have been investigated, including cell manipulations by cytokines. Cell migration and proliferation, as well as protein and collagen synthesis, are essential characteristics of the tissue’s healing response. Endogenous growth factors that are released by inflammatory cells can act as mediators of the healing response. Growth factors such as epidermal growth factor (EGF), platelet-derived growth factor (PDGF), and transforming growth factor-β1 (TGF-β1) have been evaluated both in vitro and in vivo as to their effect of ligament healing with variable results (43,44 and 45). We found that EGF and PDGF increase fibroblast proliferation in vitro, whereas TGF-β1 increases protein synthesis (46,47). When these growth factors were combined and tested in an in vivo model, the structural properties of the healing rabbit FMTC and the mechanical properties of the healing MCL appeared to be enhanced (48). Longer-term studies are ongoing.

Advances in the fields of molecular and cell biology have led to the development of novel techniques for the delivery and production of both cells and growth factor proteins at sites of ligament injury. Biodegradable scaffolds that act to deliver both cellular elements (fibrocytes, mesenchymal stem cells) and humoral elements (growth factor proteins) to injured soft tissues are being developed and evaluated. Genetic manipulation of cells via ex vivo and in vivo gene transfer techniques is also being investigated. Issues such as cell viability and regulation of growth factor protein gene expression remain problematic and are the focus of intense laboratory study.
Anterior Cruciate Ligament

Anatomy and Function of the Anterior Cruciate Ligament

The ACL of the knee is an intraarticular, extrasynovial structure that crosses the knee joint from the medial aspect of the lateral femoral condyle to the anterior aspect of the middle portion of the tibial plateau (Fig. 5.2). The enveloping synovial membrane is thought to protect the ligament substance from the harsh environment created by the synovial fluid. The fascicular structure of the ACL allows for separation of the ligament into two “bands” (49). These have been described by many investigators as the anteromedial (AM) and posterolateral (PL) bundles, referencing the anatomic positions of the fascicular attachments of the tibial insertion (50,51,52 and 53). Several investigators have reported a functional difference between the bundles, noting that the AM bundle is taut in flexion while the PL bundle is taut in extension. The morphology and geometry of the tibial and femoral insertion sites of the ACL have also been studied. The insertions of the ACL have been described as morphologically similar to the femoral insertion of the MCL, passing through four zones before inserting directly into bone (54). Studies investigating the details of the femoral and tibial insertion sites revealed that both are relatively planar, with the femoral insertion being more semicircular and the tibial insertion more oval (53,55,56). However, the cross-sectional area of either insertion was 3.5 times greater than that of the midsubstance of the ACL.

Tensile Properties of the Anterior Cruciate Ligament

The structural properties of femur-ACL-tibia complexes (FATCs) have been characterized by several investigators (57,58,59 and 60). To assess the effects of specimen orientation with respect to specimen age on the structural properties of the human FATC, Woo et al. performed tensile testing with paired specimens in which the tensile load was applied along the axis of the ACL with preservation of the normal anatomic angles of ACL insertion (anatomic orientation) in one knee, while the tensile load in the other knee was applied along the long axis of the tibia (tibial orientation) (60). Young specimens (age 22 to 35 years) tested in the anatomic orientation were found to have higher stiffness (mean ± SD, 242 ± 28 N per mm) than those tested in the tibial orientation (218 ± 27 N per mm). The ultimate load of the young specimens in anatomic orientation was 2,160 ± 157 N, significantly greater than that of the specimens with tibial orientation (1,602 ± 167 N). There was also a significant decrease in stiffness and ultimate load as donor age increased, regardless of specimen orientation. Testing of the FATC along the anatomic axis of the ACL, maintaining the anatomic insertion angles, allows a greater portion of the fibers of the ACL to be loaded equally during tensile testing. This is substantiated by the higher values of ultimate load and linear stiffness for specimens tested in the anatomic orientation.

To determine the mechanical properties of the human ACL, early investigators tested the ligament by removing the specimen from its bony attachments. This approach induced premature specimen failure at the ligament-clamp interface due to the destructive forces
generated by the clamps. For this reason, most investigators began using FATCs, in which the insertions remain intact; this provided smooth load transmission from bone to ligament. Uniform application of force to all of the fibers of the intact ACL is extremely difficult because of the complex arrangement of fibers within the ligament substance. Therefore, some investigators have divided the ligament into bundles and tested them individually (61,62). Separating the ligament into smaller bundles allows for a more parallel orientation of the fibers in the individual units and a more uniform orientation of the fibers inserting into bone. In this manner, the tensile stress across the fibers is more evenly distributed, increasing the reliability of the measurements of mechanical properties. Newton et al. separated the AM and PL bundles of the ACL with a common tibial bone block and individual femoral bone blocks, and then performed tensile testing on the AM bundle only (62). They found that the modulus of the AM bundle was 420 ± 70 MPa. Butler et al. tested individual units from human FATCs and found that the average modulus of all units was 278 MPa and the ultimate tensile strength was 35 MPa (61). The same group later reported nonuniform properties among bundles within the ACL. The AM and PL bundles exhibited larger modulus, ultimate stress, and strain energy density values than the posterior bundle of the ACL did, with no significant differences found between the two anterior bundles (63).

**Function of the Anterior Cruciate Ligament**

A complete understanding of the contribution of the ACL to normal knee function is needed to appreciate the function of the ACL, the mechanisms involved in its injury, and the basis for ACL reconstruction. It is generally accepted that the ACL resists anterior translation of the tibia with respect to the femur. The ACL also helps to limit hyperextension (53,64). With the knee fully extended, the ACL contributes to the rotational stability of the knee, resisting both internal and external rotation of the tibia. As the knee is brought into flexion, it resists internal rotation only (53,64,65 and 66). The ACL also controls valgus rotation of the tibia, even more so than the MCL, when five degrees of freedom (DOF) motion of the tibia are measured (29). Piziali et al. found that the ACL also plays a minor role in resisting medial displacement of the tibia (66).

Early studies on the function of the ACL applied constant forces to the tibia and measured anterior translation of the tibia with respect to the femur before and after sectioning of the ACL (67,68,69,70 and 71). More recently, quantitative assessment of the force carried by the ACL in response to applied loads has gained significant attention (72,73,74 and 75). Approaches developed to examine the forces in the ligament include direct methods, which require contact with the ligament (e.g., the use of buckle transducers and implantable transducers), and noncontact methods that use strain gauges placed near the ligament insertion site, kinematic linkage systems, in-line external force transducers, or load cells implanted in the subchondral bone at the ligament insertion sites (72,73 and 74,76,77,78,79,80,81 and 82).

We have employed a robotic/universal force moment sensor (UFS) testing system that consists of a six-DOF robotic manipulator with a UFS mounted to the end effector of the robot (83,84 and 85). This testing system enables accurate determination of multiple-DOF knee kinematics as well as direct measurement of the *in situ* force in a ligament without
physical contact with the ligament. In addition, no significant dissection of the joint is necessary. The robotic manipulator is a position (or displacement) control device that records and reproduces positions in three-dimensional space (85). In combination with the UFS, which records three forces and three moments, the robotic/UFS testing system is also capable of operating under a force (or load) control mode—that is, the testing system can apply a force or moment with specified direction and magnitude.

To investigate the kinematics of intact and ACL-deficient knees and the in situ force in the intact ACL in response to externally applied loads, the end effector of the robot applies an external load to the intact knee at a chosen flexion angle while the position of the intact knee in space is recorded. The ACL is then transected and the robotic manipulator, in position control mode, reproduces the previously recorded position of the intact knee while the UFS records the force. By the principle of superposition, the vector difference in forces measured by the UFS before and after removal of the ACL represents the in situ force of the ACL (84,85 and 86). With this approach, the in situ force in multiple ligaments, under multiple loading conditions, can be studied in the same specimen, thus minimizing interspecimen variations. The changes in kinematics of the ACL-deficient knee can then be measured by applying the same external load to the specimen. In this respect, the robotic/UFS testing system mimics clinical examination maneuvers, such as the Lachman and pivot-shift tests, that rely on the detection of abnormal kinematics in response to external loads to make the diagnosis of ACL deficiency.

Studies at our research center have examined the in situ forces in human ACLs as well as the force distribution between the AM and PL bundles of the ligament in response to applied anterior tibial load as a function of knee flexion (87). In the intact knee, anterior tibial translation in response to a 110-N anterior tibial load was greatest at 60 degrees of knee flexion and least at full extension. The magnitude of in situ force in the ACL was at a minimum at 90 degrees of flexion (71 ± 30 N) and greatest at 15 degrees of flexion (111 ± 15 N). The in situ force in the AM bundle of the ACL in response to an anterior tibial load did not change significantly with respect to knee flexion angle, but that in the PL bundle followed the trend of the whole ACL and increased as the knee neared full extension. In fact, with the knee flexion angle at less than 30 degrees, the in situ force in the PL bundle was greater than that in the AM bundle. The results imply that each bundle of the ACL plays a separate but equally important role in the complex function of the ACL. Specifically, the PL bundle of the ACL probably plays an important role in knee stability when the knee is in less than 30 degrees of flexion. Therefore, restoration of normal knee function with ACL reconstruction may necessitate attention to the contribution of both bundles of the ligament.

An advantage of the robotic/UFS testing system is its ability to apply multiple external loads, mimicking clinical examinations that are used to diagnose injuries of the ACL. In one study, the clinical pivot shift test was simulated using a combined internal tibial torque and valgus torque. In the ACL-deficient knee, there were significant increases in anterior tibial translation of 103%, 61%, and 32%, when compared with the intact knee, at full extension, 15 degrees, and 30 degrees of flexion, respectively (88). These results suggest that the ACL functions to restrain anterior tibial loads as well as internal tibial torques, especially when the knee is in near-full extension.
Another factor to consider when performing functional tests is the application of muscle loads and their effect on the knee kinematics and *in situ* forces in the ACL. The robotics/UFS testing system was used to evaluate the effect of quadriceps and hamstring muscle loads on the kinematics and *in situ* force in the ACL (89). It was determined that the addition of an antagonist hamstring load (80 N) to a quadriceps load (200 N) significantly reduced both the anterior tibial translation and the internal tibial rotation. Specifically, between 15 and 60 degrees of knee flexion, both movements were significantly reduced. Accordingly, these muscle loads also reduced the *in situ* force of the ACL between those angles. This work suggests that rehabilitation of the hamstring musculature may play an important role in maximizing outcome after ACL reconstruction.

**Injuries and Treatment Options**

The incidence of acute ACL tears is estimated at 1 in 3,000 in the general population of the United States (90), while that in certain populations, such as competitive football, skiing, and soccer athletes, is even higher (91,92,93 and 94). It has been reported that 70% of all acute ACL injuries are sports related, involving both recreational and competitive athletes (95,96). Athletes who injure the ACL often experience knee instability that prevents continued participation in sports. Those who are fortunate enough to return to sporting activities often do not return to their previous level of participation.

Earlier reports detailed the natural sequelae of ACL injury, the clinical outcome of nonsurgical treatment, and the benefits of various reconstructive techniques (97,98). Despite the good results of conservative, nonsurgical management of ACL-deficient knees documented by some authors, most have reported that chronic ACL deficiency results in significant knee instability, secondary damage to other knee structures (e.g., menisci, articular cartilage, collateral ligaments), and the possibility of early onset of degenerative joint disease (99,100,101,102,103,104,105,106 and 107). Surgery to restore the function of the intact ACL is recommended in most patients in an attempt to avoid these potential long-term complications.

**Biomechanics of Anterior Cruciate Ligament Reconstruction**

ACL reconstruction is a complex issue that requires an understanding of many biomechanical factors. A graft with biomechanical properties similar to those of the native ACL is but one piece of the puzzle. Other issues, such as graft fixation and restoration of normal knee kinematics and *in situ* forces in the graft, are likely to be as important for good long-term results after ACL reconstruction surgery.

**Graft Selection**

Evaluation of the biomechanical properties of graft materials by tensile testing has been a subject of intense laboratory research. Table 5.1 summarizes structural properties, specifically stiffness and ultimate load, for the normal ACL, the bone–patellar tendon–bone (BPTB) grafts, and various hamstring graft constructs as reported in selected literature (60,108,109 and 110). Careful examination of the data reveals that there are differences in specimen type and testing methods that have profound effects on the results. Currently,
most surgeons choose either BPTB or hamstring grafts for primary ACL reconstruction. An advantage of the BPTB construct is the presence of bone blocks on each end of the graft. This allows for earlier bone-to-bone healing within the bone tunnel and contributes to initial stability of the graft (111). The bone blocks of the BPTB constructs also contribute to its superior stiffness and strength. The ultimate load of a 10-mm BPTB graft is equivalent to that of the normal ACL (60). Proponents of hamstring tendon grafts believe that donor site morbidity associated with the use of autogenous BPTB grafts is reduced. The stiffness and ultimate load of double-loop hamstring grafts are, in recent studies, greater than those of BPTB grafts. Nevertheless, problems associated with tendon fixation in the bone tunnel and relatively slow bone-tendon healing in the tunnel are potential concerns (112,113,114 and 115). Multiple-loop hamstring grafts generally have larger cross-sectional areas and, therefore, fill the bone tunnel more completely. This increases the surface area in which bone-tunnel healing can take place and possibly enhances the healing process. Use of grafts with multiple strands also allows for tensioning of each strand of the graft at a different flexion angle in an attempt to accurately reproduce the AM and PL bundles of the intact ACL.

Graft selection for ACL reconstruction surgery remains a highly debated issue. Clinicians must take into account all of the biomechanical data available as well as the potential complications of each type of graft. Ultimately, well-designed, randomized, prospective studies comparing BPTB grafts and double-strand hamstring grafts are needed to assess which type has more favorable long-term results.

**Graft Fixation**

It has been postulated that the graft-fixation construct should be stiff enough to resist the tensile forces that the graft will encounter, so that the relative motion between the graft and the bone tunnel is minimized to facilitate graft-tunnel healing. Uniaxial tensile tests and cyclic loading experiments have been used to investigate the stiffness, ultimate load, and viscoelastic behavior of graft-fixation constructs. Motion of the graft within the tunnel may also have a significant effect on the healing potential for various fixation constructs.

Investigators have studied the initial stiffness and ultimate load of various ACL replacement
grafts and graft-fixation constructs. Graft materials investigated include the central third of the BPTB, single-strand gracilis or semitendinosus, double-strand gracilis or semitendinosus, triple-strand hamstring grafts, and quadruple-strand hamstring grafts. These graft materials were fixed with devices such as cancellous screws, metal and biodegradable interference screws, suture-button and suture-post constructs, staples, press-fit technique, and soft tissue washers. Table 5.2 provides a summary of the data available for stiffness and ultimate load of BPTB grafts fixed with various devices. Kurosaka et al. compared BPTB grafts fixed with a 6.5-mm cancellous screw to similar grafts fixed with a 9.0-mm interference screw in young cadaveric knees (116). The stiffness (57.9 ± 3.9 N per mm) and ultimate load (475.8 ± 110.9 N) for the 9.0-mm interference screw were significantly higher than for the 6.5-mm cancellous screw (36.2 ± 2.9 N per mm and 208.0 ± 27.5 N, respectively) (116). Advances in biomaterials research have resulted in the production of interference screws made of biodegradable materials. Potential advantages to these screws are that their use will produce less distortion on postoperative magnetic resonance imaging scans and may eliminate the need for hardware removal once the graft is healed within the tunnel. Several investigators have compared the stiffness and ultimate load of BPTB grafts fixed with titanium versus biodegradable interference screws (117,118). In general, the stiffness and ultimate load of BPTB grafts fixed with biodegradable screws are similar to those of grafts fixed with titanium screws, but the data depend on the thread height of the screws (118). From this finding it was suggested that the design of the screw may be more important than the material from which it is made.

Fixation of soft tissue replacement grafts that lack bone blocks, such as semitendinosus, gracilis, quadriceps, and Achilles tendon grafts, have presented a challenge with respect to adequate stiffness and ultimate load of the graft-fixation construct. Available data on the ultimate load and stiffness of various hamstring grafts and soft tissue fixation methods are summarized in Table 5.3. Fixation of a double-loop semitendinosus-gracilis graft with soft tissue washers provided relatively high stiffness (29 ± 7 N per mm) and ultimate load (821 ± 219 N) (119).
The use of interference screws for fixation of hamstring grafts has also been considered.

Concerns for this method include a decrease in stiffness and strength of fixation, a
decrease in bone-tendon contact area for healing, and the potential for damaging the graft.

Several groups have compared the initial stiffness and ultimate load of various hamstring
grafts fixed with titanium versus biodegradable interference screws (118,121,122 and 123).
In general, there were no significant differences in ultimate load. Additional comparisons
showed that the stiffness and ultimate load of soft tissue grafts were less than those of
BPTB grafts fixed with the same interference screw. Simonian et al. compared placement of
the interference screw centrally (i.e., in the middle of the loops of double-loop
semitendinosus-gracilis grafts) to eccentric placement of the screw and found no difference
in ultimate load (245 ± 61 and 265 ± 48 N, respectively) (123). Nevertheless, central
placement of the screw

increases the bone-tendon contact area, which may enhance bone-to-tendon healing.

The viscoelastic behavior of graft fixation constructs plays a role in the healing of the graft
within the bone tunnel. In our research center, cyclic loading of a double-loop
semitendinous and gracilis hamstring graft fixed with a titanium button–polyester tape
device was performed to examine the motion between the graft and the osseous tunnel
(124). Significant graft tunnel motion, ranging from 0.7 to 3.3 mm, occurred during a
loading range of 50 to 300 N. This motion, occurring at the proximal fixation site (button-
polyester tape), represented approximately 90% of the total elongation of the graft-fixation
construct. Therefore, this technique for hamstring fixation results in a large amount of
motion of the graft within the tunnel and could potentially interfere with bone-tendon
healing within the tunnel.

Current methods of both BPTB and soft tissue graft fixation do not adequately reproduce
the stiffness and ultimate load of the intact ACL. A graft fixation construct that is too stiff
may limit full range of motion in the ACL-reconstructed knee, whereas inadequate stiffness
of the graft fixation construct may fail to stabilize the knee, leading to compromised results.
Inability to reproduce the ultimate load of the intact ACL may lead to catastrophic loss of
fixation if the reconstructed knee is subjected to a traumatic event before graft-tunnel
healing is complete. Few studies report the behavior of graft-fixation constructs subjected
to cyclic loading. Further work in this area will allow for an understanding of how grafts and graft-fixation constructs behave when subjected to the repetitive, low-intensity loads encountered during rehabilitation.

**Functional Testing of Anterior Cruciate Ligament Reconstructions**

The robotic/UFS testing system can be used to obtain the kinematics of the ACL-reconstructed knee and the *in situ* force in the ACL replacement graft. Kinematics of the intact knee and the *in situ* force in the intact ACL are determined as described previously. Briefly, an ACL-reconstructed knee is tested on the robotic/UFS testing system in response to external loads, and the kinematics of the ACL-reconstructed knee are recorded. The graft is released, and the robotic manipulator reproduces the previously recorded motions of ACL-reconstructed knee. The vector difference of the forces recorded by the UFS before and after release of the replacement graft represents the *in situ* force in the ACL replacement graft. The kinematics of the ACL-reconstructed knee in response to external loads are also measured and can be compared with those of the intact knee.

Using this method, the variables of the ACL reconstruction can be evaluated under applied loads. The effects of distal, central, and proximal (anatomic) tibial tunnel fixation were evaluated in the porcine model by the robotic/UFS testing system (125). In this study, the site of graft fixation in the tibial tunnel was found to have a significant effect on the kinematics of the ACL-reconstructed knee. Proximal (anatomic) fixation produced the most stable reconstructed knee. Increases in anterior tibial translation and internal tibial rotation were noted as the tibial fixation was moved distally. *In situ* forces in the replacement graft were highest when proximal fixation was employed. Therefore, proximal graft fixation in the tibial tunnel may be the best choice, because the results were closer to those of the intact knee.

The biomechanical relationships of the intact and reconstructed ACL and the medial meniscus have been defined with the use of the robotic/UFS testing system. Deficiency of the ACL results in increased *in situ* forces in the medial meniscus when the knee is subjected to an anterior tibial load (126). This may help explain the high incidence of medial meniscus tears in chronic ACL-deficient knees. In addition, absence of the medial meniscus in an ACL-reconstructed knee results in an increase in the *in situ* forces encountered by the ACL replacement graft (127). The increased forces encountered by the ACL replacement graft in the medial meniscus–deficient ACL-reconstructed knee may account for the increased incidence of graft failure in meniscus-deficient knees. The effects of medial meniscus transplantation on knee kinematics and *in situ* forces in the intact ACL and in ACL replacement grafts are currently being evaluated. The robotic/UFS testing system is also being used to directly compare BPTB and double-loop hamstring grafts in human cadaveric knees. A reconstruction using each type of graft will be performed in the same specimen, allowing for direct comparison of the two graft types. The kinematics and *in situ* forces in the replacement grafts in response to both an anterior tibial load and a simulated pivot-shift test will be compared. The results of this study will contribute further information to the ongoing debate concerning graft choice for primary ACL reconstruction.
**Posterior Cruciate Ligament**

**Anatomy and Function of the Posterior Cruciate Ligament**

The posterior cruciate ligament (PCL) of the knee originates from the lateral surface of the medial femoral condyle and inserts on the posterior aspect of the tibial plateau (Fig. 5.2). Like the ACL, the PCL is an intraarticular but extrasynovial structure. The PCL is oriented almost vertically when the knee is extended and becomes more horizontal as the knee is flexed. Based on anatomy, the PCL has been divided into two “bundles,” anterolateral (AL) and posteromedial (PM), each with distinct insertions onto the tibia (128). The nomenclature used refers to the anatomic location from femoral origin to tibial insertion (53). These bundles have been shown to possess unique anatomic and biomechanical properties, as well as specific geometry at the bony insertions. Functionally, the AL and PM bundles have different patterns of tension, depending on the angle of knee flexion. The AL bundle shows increasing tension with increasing knee flexion, while tension in the PM bundle decreases (128). The anterior and posterior meniscofemoral ligaments (MFL), known respectively as the ligaments of Humphrey and Weisburg, originate from the lateral meniscus and insert anteriorly and posteriorly to the PCL on the medial femoral condyle; they are also considered to be part of the PCL complex. The presence of these ligaments is variable, with some knees having one, both, or none. The importance of the MFLs has not been well characterized, but their reasonably high stiffness and strength properties suggest that they aid in stabilization of the lateral meniscus (129).

In our research center, the geometry of the PCL, including the shape and size of the ligament and its insertion sites, was studied in detail (55,128,130). The AL bundle was found to be two times larger in cross-sectional area than the PM bundle (128,131). The mean total cross-sectional area of the MFLs was approximately 22% of the entire PCL. The femoral and tibial insertion sites of the PCL were complex and were three times larger than the midsubstance of the PCL. The femoral insertion was typically shaped like a half-moon, whereas the tibial insertion was rectangular in configuration (130).

Anatomically, the posterolateral structures of the knee (PLS) are not components of the PCL, but functionally they have been shown to interact with the PCL to provide posterior knee stability and resistance to external tibial rotations (132,133,134 and 135). There are two major components of the PLS: the lateral collateral ligament and the popliteus complex, which includes the popliteus muscle-tendon unit, the popliteofibular ligament, and various popliteotibial and popliteomeniscal fascicles (136,137,138 and 139).

**Tensile Properties of the Posterior Cruciate Ligament**

The tensile properties of the human PCL have been investigated by several authors (57,130,131,140). When the femur-PCL-tibia complex (FPTC) from cadaveric knees was tested with the knee at 45 degrees, the linear stiffness was 204 ± 49 N per mm and the ultimate load was 1,627 ± 491 N (140). In our research center, the structural properties of the AL and PM bundles of the PCL and the meniscofemoral ligaments were measured (130). The AL and PM bundles were separated with their respective femoral bone-blocks...
and potted in polymethyl methacrylate; the tibia and lateral meniscus were left intact so that the tensile loads could be applied along the anatomic axis of the PCL. The cross-sectional areas of the three components were measured with a laser micrometer (10). The average cross-sectional area of the midsubstance of the AL bundle was found to be two times that of the PM bundle. Uniaxial tensile tests revealed that the stiffness of the AL bundle was $120 \pm 37$ N per mm, which was significantly higher than that of the PM bundle ($57 \pm 22$ N per mm) or the MFL ($49 \pm 18$ N per mm). The ultimate load for the AL bundle was $1,120 \pm 362$ N, that of the PM bundle was $419 \pm 128$ N, and that of the MFL was $297 \pm 141$ N. Because of its superior stiffness and ultimate strength, it is believed that the AL bundle should be the focus of reconstructive procedures (130).

**Functional Testing of the Posterior Cruciate Ligament**

To gain data on force or tension in the PCL when external loads are applied to the knee, a number of methods have been developed, both direct (e.g., buckle transducers, pressure probes) and indirect (73,76,80,141). However, there are limitations of direct methods. Contact with the ligament is required, and forces can be measured only in the segment of the ligament in which the transducers are placed. Other investigators have used indirect methods to measure forces in knee ligaments. For example, the lengths of various segments of the ligament are measured and the force in the ligament is then calculated from the load-elongation curve (82,142). However, the *in situ* forces in the ligament may not be repeated as the ligament is being tensile tested. More recently, Markolf et al. developed a method to determine the *in situ* forces in the PCL by rigidly fixing a load cell to the subchondral bone at the femoral insertion of the PCL (74,81,143).

We have used the robotic/UFS testing system to evaluate the *in situ* forces in the PCL. During passive flexion-extension of the knee, these forces ranged from $6 \pm 5$ N at 30 degrees of flexion to $15 \pm 3$ N at 90 degrees. Also, the distribution of the *in situ* forces between the AL and PM bundles in response to a 110-N posterior tibial load were determined in nine cadaveric knees (144). Under these conditions, the *in situ* force in the PCL increased with knee flexion, ranging from 36 N at 0 degrees to 112 N at 90 degrees of flexion. No significant differences between the bundles could be determined. These findings are consistent with those of other investigators (141). Further, in response to an 80-N isolated hamstring load, the *in situ* force in the PCL increased with knee flexion, from $12 \pm 5$ N at 0 degrees to $80 \pm 20$ N at 90 degrees of flexion (145). With the addition of a 200-N quadriceps load, these forces decreased significantly, by 23 to 31 N at 30 to 90 degrees of knee flexion. These results suggest the importance of quadriceps rehabilitation during the postoperative period after PCL reconstruction. Strengthening of the quadriceps may decrease the *in situ* forces that the newly reconstructed PCL graft encounters.

The effects of a popliteus muscle load on knee kinematics and *in situ* forces in the PCL also were investigated (146). Under a 110-N posterior tibial load, the addition of a 44-N popliteus load significantly reduced the *in situ* force in the PCL by 36% at 30 degrees of knee flexion. The popliteus muscle load also significantly reduced posterior tibial translation of the PCL-deficient knee by 1 to 3 mm.

In addition to the information on structural properties of the FPTC, determination of its *in
situ forces provides data on knee joint kinematics and contributes to a better understanding of the functional role of this ligament. Traditionally, cutting studies are used to examine the contributions of the PCL and other soft tissue structures to knee stability (67,132,133,135,147). In these studies, external loads are applied to the tibia while motion of the tibia with respect to the femur is allowed. The difference in kinematics between the intact and the PCL-deficient knee represents the contribution of the PCL. These cutting studies have demonstrated that the primary function of the PCL is to restrain posterior tibial translation (67,132,133,135,147). In our research center, the PCL has been shown to work in conjunction with the PLS of the knee to provide knee stability (132,133,134 and 135). In response to a 100-N posterior tibial load, sectioning of the PLS resulted in small increases in posterior tibial translation, compared with sectioning of the PCL. However, combined sectioning of the PCL and PLS resulted in posterior tibial translation up to three times that of the PCL alone. Further, the PLS and PCL work together to restrain external and varus rotations. In response to a varus moment of 21 Nm or an external tibial moment of 5 Nm, small increases in rotations occurred after sectioning of the PCL alone. Significantly larger increases in both rotations were observed with combined deficiency of the PCL and PLS (132,133).

Studies which evaluate the effect of knee flexion angle on function of the PCL have revealed that the PCL provides more restraint to posterior tibial translation, varus rotation, and external rotation when the knee is at higher flexion angles. The PLS, on the other hand, contribute to restraining posterior translation, varus rotation, and external rotation when the knee is in near-full extension (133). These findings are consistent with the observation that, in response to posterior tibial loads, the PCL is more taut with the knee in flexion but the PLS is more taut with the knee near extension (53,148,149). Studies have also found that PLS deficiency results in significant increases in external tibial rotation and posterior tibial translation in response to an external tibial moment or a posterior tibial load, respectively (81,134). Further, the in situ forces in the PCL were up to six times higher in PLS-deficient knees compared with the intact knees for each loading condition. These results suggest that there is a biomechanical interdependence between the PCL and PLS and that they function together to limit posterior tibial translation and external tibial rotation in response to externally applied loads.

**Injuries and Treatment Options**

In the general population, PCL injuries account for 3% to 23% of all knee ligament injuries (150,151,152,153 and 154). Among a population of trauma patients this number may be as high as 40% (155). In the past, isolated injuries to the PCL have been considered relatively benign and have traditionally been treated nonoperatively (156,157,158 and 159). However, in some studies, patients have developed increased instability and degenerative changes over time (158,159 and 160). Meanwhile, surgical management of isolated PCL injuries has not enjoyed the success of ACL reconstruction, with a high number of patients experiencing residual knee instability (151,161,162,163,164 and 165). Clinical outcomes of PCL reconstruction remain inconsistent, and the long-term prognosis of surgically treated PCL injuries remains unsatisfactory (166). More than 60% of PCL injuries involve other soft tissue structures of the knee, most frequently the PLS (155,167). Nonoperative treatment of these injuries has universally failed, and surgical management without addressing the PLS
injury has also yielded poor results (164,165 and 166,168).

We have evaluated the effect of PLS deficiency on PCL reconstruction using the robotic/UFS testing system. In a combined PCL/PLS injury, PLS deficiency resulted in an increase in posterior tibial translation after PCL reconstruction. External tibial rotation increased up to 14 degrees, while varus rotation increased up to 7 degrees. In situ forces in the PCL graft also increased significantly (by 22% to 150%) for all loading conditions (169). These results also suggest that the PLS should be addressed surgically at the time of PCL reconstruction.

**Biomechanics of Posterior Cruciate Ligament Reconstruction**

Surgical variables for PCL reconstruction involve highly controversial issues: (a) choice of which bundle to reconstruct when a single bundle graft is used; (b) onlay technique versus tunnel fixation of the tibial portion of the graft; (c) placement of the tibial and femoral tunnels; (d) position of the tibia relative to the femur at the time of graft fixation; and (e) the possible use of double-bundle PCL reconstruction techniques. Because of its superior stiffness and strength, the AL bundle of the PCL has been the focus of reconstructive procedures (130). Because the AL bundle exhibits increasing tension with knee flexion, tensioning and fixation of the graft are performed with the knee in flexion to avoid graft impingement, which could occur with the knee in extension. Theoretically, large in situ forces in the graft could result in either graft failure or decreased range of motion.

The optimal knee flexion angle and position of the tibia in the anterior-posterior direction at the time of graft fixation are other important factors affecting PCL reconstruction. Some authors have advocated fixation of the graft with the knee in flexion, and others have suggested full extension or lower-knee flexion angles (170,171). The application of an anterior tibial load to reduce posterior sag before graft fixation has also been advocated by some (170). In our research center, the effect of knee flexion angle and applied anterior tibial load at the time of graft fixation on the knee kinematics and in situ forces in a single-bundle PCL reconstructed knee were evaluated (172). Fixation of the graft with the knee at full extension overconstrained the knee and resulted in higher in situ forces than those found in the intact PCL in response to a 134-N posterior tibial load. Fixation of the graft with the knee in flexion with a 134-N anterior tibial load applied at the time of graft fixation closely reproduced the intact knee kinematics and the in situ force in the intact PCL.

A number of studies have focused on the effects of tunnel placement within the insertion site of the PCL. Although the tension in the PM bundle does not change significantly as the knee is moved through its range of motion, the AL fibers represent the bulk of the PCL (128,173,174). Some authors have advocated placement of the tunnel in the location of the PM bundle, but it has been shown that this graft placement is ineffective in restoring normal joint laxity (170,175,176 and 177). More recent studies suggest that anterior location of the femoral tunnel (in the anatomic location of the AL bundle insertion) more closely restores intact knee biomechanics when compared with isometric graft placements (170,173,177). Variations in tibial tunnel position were found to have less effect on graft
behavior than variations in the femoral tunnel placement (174,178).

A double-bundle PCL reconstruction technique has received much attention because it tends to mimic the complex functional anatomy of the PCL (179,180,181 and 182). Use of Achilles tendon or BPTB for the AL bundle and hamstring tendons for the PM bundle restored the kinematics of the PCL-reconstructed knee, in response to a 134-N posterior tibial load, to the level of the intact knee. The *in situ* force in the double-bundle PCL graft was not significantly different from that in the intact PCL between full extension and 30 degrees of knee flexion. At greater knee flexion angles (60 to 120 degrees), the *in situ* force in the graft was significantly less than in the intact PCL (183). These results suggest that the double-bundle reconstruction may have an advantage over a single-bundle reconstruction.

**BIOMECHANICS OF LIGAMENTS OF THE SHOULDER**

The shoulder has the greatest range of motion of all joints in the body, and this motion is distributed between three diarthrodial joints—the glenohumeral (GH), the acromioclavicular (AC), and the sternoclavicular joints—and the scapulothoracic joint. In this section, the anatomy and function of these joints and their surrounding ligaments are addressed. Specific injuries, their treatment options, and the biomechanical basis for treatments of injuries to the GH and AC joints are also discussed.

**Acromioclavicular Joint**

**Anatomy and Function of Acromioclavicular Ligaments**

The AC joint is the articulation between the distal end of the clavicle and the acromion process of the scapula (Fig. 5.11). The capsule of the AC joint surrounds this diarthrodial joint, which is incompletely divided by an articular disk. The AC joint capsule has distinct thickenings that are referred to as the superior, inferior, anterior, and posterior AC ligaments (184). Anteroposterior stability of the AC joint is thought to be rendered by the AC ligaments, while vertical stability is attributed to the coracoclavicular (CC) ligaments (184,185,186 and 187). The CC ligaments are strong ligaments whose fibers run from the anteroinferior surface of the clavicle to the base of the coracoid process of the scapula. They consist of two components: the conoid ligament and the larger, longer, and stronger trapezoid ligament.

*FIGURE 5.11.* Normal anatomy of the acromioclavicular joint. (From Matsen RA, Rockwood CA. *The shoulder.* Philadelphia: WB Saunders, 1998, with permission.)
We examined the \textit{in situ} forces in ligaments of the AC joint using the robotics/UFS testing system mentioned previously (188). The results of this study suggested that the trapezoid and conoid ligaments play a major role in limiting excessive AC joint translations in both the superior and posterior directions. The directions of the \textit{in situ} force in the trapezoid and conoid ligaments were different. Therefore, they should not be treated as single ligament during reconstruction procedures, even though they carry similar magnitudes of force during most modes of loading. Comparison of the data obtained in this study to previous one-DOF experiments reveals a significant difference in the magnitude of the force in each structure (185, 187). This suggests that kinematic constraints placed on the AC joint during loading are important and the role of individual ligaments is affected by coupled motions that occur during loading.

The effect of AC joint capsule disruption on AC joint kinematics and the resultant \textit{in situ} forces in the CC ligaments were also investigated (189). Disruption of the AC capsule resulted in increases in anterior (6.4 mm) and posterior (3.6 mm) translation when a 70-N anterior or posterior force was applied through the distal clavicle. Capsule transection did not significantly affect superior translation. Under applied anterior loading, the \textit{in situ} force in the conoid and trapezoid ligaments increased significantly with transection of the AC capsule. Posterior loading of the clavicle in the deficient state of the AC capsule resulted in a significant increase in the \textit{in situ} force of the trapezoid ligament only. These results suggest that disruption of the AC capsule alone causes abnormal AC joint kinematics.

**Injury and Treatment Options**

The most common cause of AC joint injury is a direct force produced when the patient falls onto the point of the shoulder with the arm at the side in an adducted position. The force is directed inferiorly and medially to the acromion, which can result in mild, moderate, or severe injury to the AC ligaments, injury to the sternoclavicular joint, or fractures of the shaft of the clavicle (184). A fall on the adducted arm can also create an indirect superior force that is transmitted through the humeral head to the acromion process of the scapula. If the force is large enough, it may result in a fracture of the acromion, rupture of the AC ligaments, or even a superior dislocation of the GH joint.

Injuries to the AC joint can be classified according to the degree of injury to the AC and CC ligaments (Fig. 5.12) (190, 191). The majority of AC joint injuries are type I, II, or III injuries and do not require surgery (184). Although optimum treatment of type III injuries, in which both the AC and CC ligaments are disrupted, remains controversial, in most patients excellent functional results can be obtained with nonoperative management (192). Some speculate that younger, more active patients with more severe injuries may benefit from operative stabilization. Types IV, V, and VI AC joint injuries are often treated surgically by open reduction of the AC joint itself and reconstruction of the CC ligaments. Options for reconstruction include augmentation with suture bands, synthetic loop augmentation through drill holes in the base of the coracoid and through the clavicle, CC fixation with
transfer of the coracoacromial ligament, use of an AC hook plate, and AC reconstruction with clavicular corticotomy (184).

Glenohumeral Joint

Anatomy and Function of Glenohumeral Ligaments

The GH joint is formed by the articulation of the large, spherical humeral head with the smaller glenoid surface of the scapula. The GH capsuloligamentous complex consists of anterior and posterior components. The anterior component comprises the anterior band of the inferior glenohumeral ligament (AB-IGHL), the superior glenohumeral ligament (SGHL), and the middle glenohumeral ligament (MGHL). The posterior band of the IGHL (PB-IGHL) and the rest of the posterior capsule form the posterior portion of the GH capsuloligamentous complex. The GH ligaments are composed of discrete collagenous bands within an interwoven collagen mesh that forms the joint capsule (Fig. 5.13) (193). The GH capsule is similar in composition to other joint capsules in the body and contains types I, II, and III collagen. Because of the orientation of the ligaments, portions of the capsule reciprocally tighten and loosen as the GH joint rotates. The SGHL and the coracohumeral ligament (CHL) are usually described together because their anatomic courses are parallel. These ligaments form the region of the GH capsule referred to as the “rotator interval,” which constitutes the triangular space between the anterior border of the supraspinatus tendon and the superior border of the subscapularis tendon (193,194 and 195).

FIGURE 5.12. Types of acromioclavicular joint injuries. Types I through III are the most common. (Adapted from Matsen RA, and Rockwood CA. The shoulder. Philadelphia: WB Saunders, 1998, with permission.)

FIGURE 5.13. The anterior band of the inferior glenohumeral ligament (AB-IGHL), the posterior band of the IGHL (PB-IGHL), the superior glenohumeral ligament (SGHL), and the middle glenohumeral ligament (MGHL) are discrete collagenous bands within the joint capsule. (From Warner JP. The gross anatomy of the joint surfaces, ligaments, labrum, and capsule. In: Matsen FA, Fu FH, Hawkins RJ, eds. The shoulder: a balance of
Through most ranges of motion, the GH ligaments and capsule are relatively lax, acting mainly as checkreins to limit extreme rotations or translations of the joint surfaces in relation to one another (194,196,197). However, the primary stabilizer of the GH joint is the concavity-compression effect. This observation is consistent with the fact that the capsule, ligaments, and glenoid labrum are structurally less robust than ligaments of the knee and therefore less likely to withstand large forces during joint motions (195,198). The stabilizing effect of the GH ligaments and joint capsule has been assessed by measuring their elongation during various joint motions with the use of experimental techniques such as radiography, mercury and Hall effect strain gauges, electromagnetic tracking devices, metallic beads, and computational models (199,200,201,202,203 and 204). Turkel et al. found that the SGHL was important in preventing inferior subluxation of the GH joint at 0 degrees of abduction (202). Many authors have suggested that the CHL plays a major role in inferior stability of the GH joint (199,201,203). O’Connell and associates found that the MGHL works together with the CHL to restrain external rotation and serves as a secondary restraint to both inferior and anterior instability (200,201 and 202,205). The IGHL has been reported to function as a primary stabilizer for anterior instability in a fashion analogous to a hammock supporting the humeral head in the glenoid during abduction and rotation of the GH joint (202,203,206).

Forces in the GH ligaments and joint capsule have also been measured with the use of buckle transducers and differential variable reluctance transducers (207). We evaluated the in situ force distribution in the GH capsule during anterior-posterior loading using the robotics/UFS testing system (208). The results showed that the GH capsule carries no load when the humeral head is centered on the glenoid; however, once an anterior or posterior load is applied to the joint, the capsuloligamentous structures carry force (SGHL-CHL at 0 degrees of abduction, 26 ± 16 N; AB-IGHL at 90 degrees of abduction, 30 ± 21 N).

**Tensile Properties of the Glenohumeral Ligaments**

A summary of the structural and mechanical properties from selected literature can be found in Table 5.4 and Table 5.5. Previous studies in our research center have documented the tensile properties of the CHL and SGHL (195). The CHL was found to have twice the stiffness of the SGHL and three times the ability to withstand tensile loads. The CHL also
absorbed six times the amount of energy to fail but only elongated 1.5 times as much as the SGHL during tensile testing. The values obtained for stiffness and ultimate load of the CHL were 150% of those reported for the coracoacromial ligament (209).

<table>
<thead>
<tr>
<th>Ligament (Reference)</th>
<th>Modulus (MPa)</th>
<th>Tensile Strength (MPa)</th>
<th>Ultimate Strain (%)</th>
</tr>
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<tbody>
<tr>
<td>Anterior Band (199)</td>
<td>38.7 ± 18.1</td>
<td>5.2 ± 2.7</td>
<td>8.3 ± 3.2</td>
</tr>
<tr>
<td>Axillary Pouch (199)</td>
<td>30.3 ± 10.6</td>
<td>5.6 ± 1.9</td>
<td>15.1 ± 5.7</td>
</tr>
<tr>
<td>Posterior Band (199)</td>
<td>41.9 ± 12.5</td>
<td>5.5 ± 2.0</td>
<td>9.9 ± 5.3</td>
</tr>
<tr>
<td>Anterior Capsule (210)</td>
<td>41.9 ± 20.6</td>
<td>15.7 ± 8.1</td>
<td>—</td>
</tr>
<tr>
<td>Posterior Capsule (210)</td>
<td>67.0 ± 22.5</td>
<td>21.2 ± 5.7</td>
<td>—</td>
</tr>
<tr>
<td>Superior Capsule (210)</td>
<td>42.2 ± 13.8</td>
<td>12.7 ± 5.6</td>
<td>—</td>
</tr>
<tr>
<td>Inferior Capsule (210)</td>
<td>31.5 ± 9.4</td>
<td>8.0 ± 3.2</td>
<td>—</td>
</tr>
</tbody>
</table>

IGHL, inferior gleno-humeral ligament.

The modulus of the anterior band of the human IGHL complex was reported to be greater than the modulus of the posterior band (198). The tensile strength of the anterior and posterior bands and the axillary pouch ranged from 5.9 ± 1.7 to 8.4 ± 2.2 MPa. The strain at failure was found to be greatest for the axillary pouch and least for the posterior band. It was hypothesized that these results reflect the variability in the functional roles of the different portions of the IGHL complex.

The anatomic, structural, and mechanical properties of four different sites (anterior, posterior, superior, and inferior) of the GH joint capsule were examined by Itoi et al. (210). They found the thickness of the posterior capsule (1.0 ± 0.4 mm) to be less than that of the anterior (1.8 ± 0.3 mm), superior (1.6 ± 0.4 mm), or inferior (1.5 ± 0.3 mm) section. The posterior capsule exhibited the greatest ultimate stress and modulus, and the superior section had the least strength. No significant differences in ultimate load could be
demonstrated. The authors postulated that the greater ultimate stress for the posterior capsule explained the low incidence of posterior shoulder dislocations.

When the stiffness of the human ACL was compared with that of the SGHL and CHL, significant differences were found. The SGHL and CHL had approximately 10% the stiffness of the ACL (211). Ultimate load also differed significantly, with values for the SGHL and the CHL only 5% and 15% of the ACL value, respectively. The modulus of the human IGHL complex was only 10% of the modulus measured in rabbit MCL, and the tensile strength was only 15%. These differences suggest that the soft tissues at the shoulder do not function in the same capacity as those at the knee joint.

**Injury and Treatment Options**

Athletes who participate in overhead sports such as throwing, swimming, tennis, volleyball, and weightlifting subject their shoulders to repetitive stresses when the arm is in extreme positions of motion. Repetitive overuse of the shoulder may cause cumulative injury to the capsule, GH ligaments, and glenoid labrum, resulting in GH instability or the inability to maintain the humeral head centered in the glenoid fossa. Clinical cases of instability can be characterized according to the circumstances under which they occur, the degree of instability, and the direction of instability. Instability may arise from an episode in which an injury occurs to the bone, rotator cuff, labrum, capsule, or a combination of these structures.

**Anterior Dislocation**

Anterior GH dislocation accounts for almost 85% of all shoulder dislocations (212). The usual mechanism of injury is a combination of shoulder abduction, extension, and external rotation that challenges the anterior capsule and ligaments, the glenoid rim, and the rotator cuff. The humeral head is displaced anteriorly with respect to the glenoid and translates inferiorly to the coracoid process. A common feature of traumatic anterior dislocations is avulsion of the anterior-inferior GH ligaments and capsule from the glenoid rim (Bankart lesion). This avulsion injury often does not heal and contributes to recurrent traumatic instability.

Although clinical studies have shown a direct relation between traumatic anterior shoulder instability and a capsulolabral injury, electromyographic and histologic studies suggest a more complicated lesion (213,214 and 215). In biomechanical studies, a Bankart lesion did not significantly affect GH joint stability during various loading conditions and arm positions (216,217 and 218). Using the dynamic shoulder testing apparatus in our research center, we found that GH translations during active abduction and external rotation were minimally affected by disruption of the anterior-inferior capsulolabral complex (219). Simulated rotator cuff muscles maintained “ball and socket” kinematics during these motions, with small translations of the humeral head occurring in all three directions.

**Posterior Dislocation**

Posterior dislocations may leave the humeral head in a subacromial (behind the glenoid and beneath the acromion), subglenoid (behind and beneath the glenoid), or subspinous
(medial to acromion and beneath the spine of the scapula) location. The subacromial dislocation is the most common one. The incidence of posterior dislocations is estimated to be 2% of all shoulder dislocations, but the diagnosis is frequently missed (220). Posterior dislocation may result from axial loading of the adducted, internally rotated arm or from violent muscle contractions or convulsive seizures. Fractures of the posterior glenoid rim and the proximal humerus are common in traumatic posterior dislocations of the shoulder.

**Inferior Dislocation**

Inferior dislocation may be produced by a hyperabduction force that causes abutment of the neck of the humerus against the acromion process, which levers the humeral head out inferiorly. The humerus is locked, with the humeral head below the glenoid fossa and the humeral shaft pointing overhead. Severe soft tissue injury occurs with this dislocation, including avulsion of the supraspinatus, pectoralis major, or teres minor muscles.

**Treatment Options**

The optimal method to surgically stabilize the joint and reestablish the delicate balance between mobility and stability after a capsulolabral injury continues to be a controversial topic in sports medicine. Surgical stabilization of the GH joint is considered if instability repeatedly compromises shoulder comfort or function despite a reasonable trial of rotator cuff and scapular stabilizer strengthening and coordination exercises. The procedures for anterior instability include tightening and realigning of the subscapularis tendon and reinforcement of the anterior capsule (Putti-Platt operation), reattachment of the capsule and glenoid labrum to the glenoid rim (Bankart repair), capsulorrhaphy, augmentation of the bony anterior glenoid rim, and transfer of the tip of the coracoid process with its muscle attachments (Bristow procedure) (212,221).

Surgical intervention for recurrent posterior dislocation of the shoulder also remains controversial because of the high rates of failure after such surgical procedures as posterior staple capsulorrhaphy or anterior capsular shift (222,223).

**Biomechanical Testing of Surgical Reconstructions**

Several studies have evaluated the biomechanics after surgical repair of lesions in the capsule, ligaments, and labrum. Janevic et al. measured humeral head translations, contact areas, and contact force magnitude at the extremes of motion after correction of anterior GH instability (217). They found that the translations and contact after repair did not duplicate the normal joint mechanics. Klein et al. compared two repair techniques after a simulated Bankart lesion in cadaveric specimens using a shoulder testing apparatus (224). They measured strain in the IGHL, torsional resistance as an indication of instability of the joint, and load to failure in order to compare traditional and suture anchor repair techniques. The results showed that repair of the defect restores strain and rigidity to control conditions with traditional techniques, providing greater load-to-failure values.

Speer et al. evaluated the effect of superior and medial anterior capsular shift strategies on
multidirectional GH motion induced by repetitive loading after a simulated Bankart lesion (218). They found the two shift procedures to be equivalent in decreasing anterior GH translation. However, for posterior and inferior translations at 45-degree elevation, the superior shift decreased translations more than the medial shift did.

Although the function of the capsuloligamentous complex in the normal joint is becoming more clear, it is evident that the biomechanics of current instability repair procedures remain poorly understood.

**BIOMECHANICS OF LIGAMENTS OF THE ANKLE**

**Lateral Ankle Ligaments**

Anatomy and Function of the Lateral Ankle Ligaments

Ankle sprains are among the most common sports-related injuries of the musculoskeletal system (225,226). They account for approximately 40% of all athletic injuries. In an epidemiologic study, the overall incidence of ankle sprains in the general population was found to be 7 per 1,000 person-years (227). The vast majority of these injuries are sprains of the lateral ligaments that result from inversion of the plantar-flexed foot.

The major ligamentous structures on the lateral side of the ankle include the three fasciculi of the lateral collateral ligament: the anterior talofibular ligament (ATFL), the posterior talofibular ligament (PTFL), and the calcaneofibular ligament (CFL). The ankle capsule is also present laterally but contributes little to the stability of joint. The ATFL and PTFL are thickenings of the capsule itself, whereas the CFL is a discrete extracapsular structure. A cadaveric study performed by Burks et al. revealed that the ATFL averages 7.2 mm in width and inserts into the talus 18 mm proximal to the subtalar joint and just distal to its superior articular surface (Fig. 5.14) (228). It originates 10 mm from the tip of the lateral malleolus. The CFL originates adjacent to the origin of the ATFL, approximately 8 mm proximal to the tip of the fibula, and courses in a posteroinferior direction at an average angle of 133 degrees from the axis of the fibula when the ankle is in a plantigrade position. The CFL inserts onto the calcaneus approximately 13 mm distal to the subtalar joint. The PTFL originates on the posteromedial aspect of the distal fibula and passes posteriorly to the talus. It is rarely disrupted with inversion injuries.

![FIGURE 5.14](272x213) Schematic drawing of the normal anatomic relationships of the lateral talofibular ligament (ATFL), the calcaneofibular ligament (CFL), and the bony architecture of the ankle.
The motion about the ankle arises from the articulation of four bones; tibia, fibula, talus, and calcaneus. The position of the ankle and motion about the ankle joint are defined geometrically by three axes (229). The normal range of motion at the tibiotalar joint is from approximately 20 degrees of dorsiflexion to 50 degrees of plantiflexion. The bony architecture of the talar dome within the ankle mortise contributes significantly to the stability of the ankle. The shape of the talar dome within the ankle mortise allows for more motion when the ankle is plantarflexed, rendering it less stable. During an inversion injury to the ankle, the anterolateral joint capsule is disrupted initially, followed by the ATFL and CFL sequentially, depending on the magnitude of the injury. Because the CFL spans both ankle and subtalar joints, injury to this ligament results in increased subtalar motion as well as ankle instability.

**Tensile Properties of the Lateral Ankle Ligaments**

The ligaments of the ankle have received little attention in the literature compared with those of the knee and shoulder. Uniaxial tensile tests of the human ATFL, CFL, and PTFL with their bony complexes revealed that the PTFL was the most stiff (163 ± 56 N per mm), followed by the ATFL (142 ± 79 N per mm) and the CFL (127 ± 43 N per mm) (230). The values for ultimate load to failure were approximately 418, 139, and 346 N, respectively (Table 5.4) (230,231). It was concluded that the ankle joint has the least amount of ligamentous support anteriorly. This is consistent with the finding that the ATFL is the most frequently injured ligament of the ankle during inversion ankle sprains (229).

**Functional Testing of the Lateral Ligaments of the Ankle**

The function of the lateral ligaments of the ankle have been investigated with the use of radiographic techniques to measure changes in ankle stability after experimentally induced ligament injuries (232,233). Others have measured talar displacement and rotation after selective sectioning of the ATFL, CFL, PTFL, and combinations of these ligaments (234,235,236,237 and 238). However, these studies have reported conflicting results. Strains of the ATFL and CFL during physiologic motion of the ankle were measured using Hall effect strain transducers in cadaveric ankles (239). In the neutral position, neither the ATFL nor the CFL was strained. In 10 degrees of dorsiflexion the CFL was strained, approximately 1%, and the ATFL was relaxed. In 30 degrees of ankle plantiflexion the ATFL was strained, approximately 2%, and the CFL was relaxed. The addition of supination and internal rotation/supination did not significantly change the strain in the ATFL. The strain in the CFL increased significantly when the ankle was placed in supination and external rotation. These results suggest that the lateral ankle ligaments work synergistically: when one is strained the other is relaxed, and vice versa, throughout the range of plantar-dorsiflexion tested. Most published literature supports this concept of lateral ankle stability. The ATFL plays a primary role when the ankle is in plantiflexion, and the CFL is important in stabilization when the ankle is dorsiflexed. Strain in the ATFL, CFL, PTFL, anterior tibiofibular, and posterior tibiofibular ligaments were also measured with the use of  

http://gateway.ut.ovid.com/gw2/ovidweb.cgi
mercury-filled Silastic strain gauges (240). Strain in the ATFL increased as the ankle was moved from dorsiflexion to plantiflexion. Inversion and internal rotation moments increased ligament strain, especially when the ankle was in plantiflexion. Strain in the CFL was generally small at all positions of plantar-dorsiflexion. Strain values decreased as the ankle was moved from 20 degrees of dorsiflexion to 20 degrees of plantiflexion, with a slight increase as the ankle was moved to 30 degrees of plantiflexion. Inversion or external rotation increased while eversion or internal rotation decreased strain in the ligament throughout the entire range of plantar-dorsiflexion. The results emphasize the importance of the ATFL in limiting internal rotation and inversion, especially when the ankle is plantarflexed.

The relatively low strain in the CFL supports the concept that it does not play an independent role in ankle stability but is important in limiting inversion when the ankle is held in dorsiflexion. In the clinical setting when the ATFL is disrupted, the CFL may become even more important for restraining talar inversion.

Although the anterior drawer test is a common clinical examination maneuver used to evaluate the status of the ATFL, there is controversy about its usefulness in the evaluation of the integrity of the ATFL (229,238,241,242,243 and 244). It has been suggested that the anterior drawer test should be performed with the ankle between 10 and 20 degrees of plantiflexion and that an excessive magnitude of force may not be necessary to diagnose a disruption of the ATFL (245). The forces in the ATFL and CFL and the ankle kinematics during simulated anterior drawer and talar tilt tests were measured with the use of buckle transducers and an instrumented spatial linkage (246). In the intact ankle, the largest forces in the ATFL were recorded with the ankle in plantiflexion, whereas the largest forces in the CFL were observed in dorsiflexion. An isolated injury to the ATFL did not significantly change the kinematics of the ankle or the force in the CFL in response to the two simulated clinical examinations. However, when both of the ligaments were disrupted, a significant increase in both anterior translation, up to 7 to 9 mm, and internal rotation, up to 6 to 9 degrees was observed in response to simulated anterior drawer testing. Simulation of a talar tilt test in the ATFL/CFL deficient ankle could result in significant increases of up to 10 degrees of supination. These results suggest that the largest laxity increase after an isolated ATFL injury would be observed when testing the ankle in plantiflexion. With the addition of a CFL injury, testing the ankle in dorsiflexion may be more appropriate.

**Injuries and Treatment Options**

The majority of lateral ligament sprains of the ankle can be managed nonoperatively with good results. Early controlled motion with the use of a functional brace, which allows limited plantar-dorsiflexion while preventing inversion, has been demonstrated to be advantageous compared with casting or surgical repair (247). However, up to 20% of patients do experience symptoms of functional instability, such as recurrent inversion sprains, pain, or difficulty walking on uneven ground (248,249,250,251 and 252). Symptoms may be controlled in some patients with a rehabilitation program aimed at improving proprioception and strengthening of the lower leg muscles. (253) Bracing is also effective in improving functional symptoms of instability.
Surgical reconstruction is reserved for patients who continue to suffer multiple recurrent inversion injuries despite adequate rehabilitation and bracing. Operative techniques can be divided into four general categories: (a) repair of the attenuated ligaments without augmentation (Bröstrom procedure) (248); (b) procedures that prevent inversion of the foot, such as tenodesis of the base of the fifth metatarsal to the lateral malleolus using the peroneus brevis tendon (Evans procedure) (254); (c) procedures that prevent inversion of the foot through reconstruction of the ATFL, such as directing the peroneus brevis tendon through bone tunnels in the distal fibula and talus to replace the ATFL (Watson-Jones procedure) (255); and (d) procedures that reconstruct both the ATFL and CFL, such as using half of the peroneus brevis tendon or a free fascia lata graft to replace the injured ATFL and CFL (Chrisman-Snook procedure). The Bahr procedure, in which the peroneus brevis tendon is routed through the calcaneus, fibula, and talus to anatomically reconstruct the ATFL and CFL has also been described (Fig. 5.15) (256,257).

**Biomechanical Testing of Lateral Ankle Ligament Reconstructions**

A successful reconstruction of the lateral ankle ligaments should fulfill three criteria. First, the placement of the grafts should approximate the normal ligament anatomy. Second, the graft material used should have tensile properties similar to those of the intact ligaments. Finally, the replacement grafts should have in situ forces and force distribution similar to those of the intact ligaments when the ankle is subjected to externally applied loads.

A summary of the biomechanical properties of commonly used autografts for lateral ligament reconstruction is shown in Table 5.6 (231). The ultimate loads for the peroneus brevis (258.2 N) and split peroneus brevis (258.8 N) tendons are significantly greater than that of the ATFL (231 N) but not significantly different from the CFL (307 N). The ultimate load for the fourth toe extensor tendon (130.1 N) is significantly less than that of the CFL but not significantly different from the ATFL.

**TABLE 5.6. CROSS-SECTIONAL AREA, ULTIMATE LOAD, AND LINEAR STIFFNESS OF THE LATERAL**
Several of the reconstructive procedures have been evaluated to assess their effect on joint motion when the ankles are subjected to externally applied loads (256, 257, 258 and 259). In cadaveric ankles, the Chrisman-Snook and Evans procedures allowed more motion than intact ankles but significantly restricted subtalar motion; the Watson-Jones reconstruction controlled anterior talar translation and internal rotation but was less effective in controlling talar tilt and also restricted subtalar motion. A new, more "anatomic" reconstruction was proposed; it returned internal rotation, anterior talar translation, and talar tilt to near-normal levels but did not restrict subtalar motion (256). Hollis et al. also used cadaveric ankles and subjected them to anterior-posterior loads and supination-pronation moments. In the ATFL/CFL-deficient state, all three reconstructions increased stability during supination-pronation loading. Anterior-posterior translation was stabilized by the Evans and Chrisman-Snook procedures, but the Watson-Jones reconstruction had little effect. Subtalar motion was restricted by all three reconstructions, most notably with the Chrisman-Snook technique (258). Anterior drawer and talar tilt tests were performed on cadaveric ankles with a clinically used testing jig. The Bröstrom, Watson-Jones, and Chrisman-Snook procedures reduced talar tilt and anterior translation when compared with the combined ATFL/CFL-deficient state (259).

Under simulated anterior drawer and talar tilt testing, the forces in the intact ATFL and CFL and their replacement grafts were measured with the use of buckle transducers and were found to change with ankle joint motion with the Bröstrom repair, the Watson-Jones reconstruction, and the new "anatomic" reconstruction technique (257). All three procedures reduced the ankle joint laxity that is observed after sectioning of the ATFL and CFL. The Watson-Jones technique restricted joint motion, as reported previously (256, 258). Analysis of the forces in the ligaments and grafts during anterior drawer and talar tilt testing revealed that, unlike the Watson-Jones technique, the forces and force patterns observed in the Bröstrom and "anatomic" techniques resembled those observed in the intact ankle.

High clinical success rates, with 85% to 100% good to excellent results, have been reported for ankle ligament reconstructions, both augmented and nonaugmented, anatomic and nonanatomic (260, 261, 262 and 263). Large, prospective, randomized clinical studies are necessary to determine whether the "anatomic" reconstruction techniques offer...
significant long-term benefits, in terms of functional stability and progression to ankle osteoarthritis, when compared with the “nonanatomic” procedures.

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