Ulnar collateral ligament injury in the overhead athlete: diagnosis and treatment

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Pain on the medial aspect of the elbow may result from instability caused by acute or chronic ulnar collateral ligament (UCL) disruption or attenuation. In 1946, Waris [1] first recognized and described UCL tears, particularly isolated anterior oblique ligament injury, in 17 javelin throwers. Subsequent to this, early reports suggested that this is a rare lesion; however, more recently, as a result of better understanding of the problem and ability to diagnose injury to the UCL, it is clear that this injury is not uncommon in athletes.

Overhead athletes, such as those who participate in baseball (particularly pitchers), tennis, football (quarterbacks), volleyball, ice hockey, and water polo subject their elbows to major valgus forces, which is detailed in the articles in this issue on throwing biomechanics by Drs. Kibler and Loftis et al. These forces are responsible for many problems in the thrower’s elbow, starting with injury to the UCL. Continued throwing in the UCL-injured elbow results in repetitive valgus force producing secondary lesions, in turn producing a variety of symptoms that may arise. This article briefly reviews the anatomy and biomechanics of UCL injury, focusing specifically on the areas affected by the forces of throwing; discusses the pathophysiology of throwing injuries; details the history, physical examination, and ancillary tests to confirm the diagnosis of ulnar collateral ligament injuries; and lists the treatment options available. More detailed discussions of the anatomy and biomechanics of throwing, and of the history, examination, and imaging of elbow injuries are presented elsewhere in this issue.
Functional anatomy and biomechanics

Although the anatomy and biomechanics of the elbow have been detailed elsewhere in this issue, a concise review of the pertinent anatomy and biomechanics as it relates to the UCL will initiate this article.

Ligamentous anatomy—medial

The UCL complex, also known as the medial collateral ligament complex, consists of three ligaments (Fig. 1): the anterior oblique, posterior oblique, and transverse ligaments. The anterior oblique ligament (AOL) is a thick, discrete ligament with parallel fibers arising from the medial epicondyle and inserting into the medial coronoid process, and is the most important of the ligaments. The AOL is functionally divided into anterior and posterior bands. Although it had been hypothesized that an isometric fiber exists within the AOL [2], recent research has disproved this concept [3]. The fan-shaped posterior oblique ligament (POL) is a thickening of the capsule that is best defined with the elbow flexed at 90°. The POL also originates from the medial epicondyle and inserts onto the medial margin of the semilunar notch. The transverse ligament arises from the medial olecranon and inserts into the inferior medial coronoid process.

Biomechanical forces

The magnitude and degree of force transmitted across the elbow joint varies based upon specific factors, which include loading configuration and angular orientation of the joint (degree of elbow flexion). A magnitude of elbow force three times body weight has been speculated to exist in certain functions [4,5]. Activities of daily living necessitate approximately 50% body weight transmitted across the joint, with maximal loads noted at about 90° of flexion [6,7].

Fig. 1. Ulnar collateral ligament complex of the elbow. (From Safran MR. Elbow injuries in athletes. Clin Orthop 1995;310:260; with permission.)
The athlete, however, is most often exposed to severe, chronic, repetitive valgus stresses. Although bony articulation contributes significantly to resisting these stresses with the elbow near full extension (flexed less than 20°) or flexion (greater than 120°) [2,8,9], the major restraint to valgus stress between these two ranges is the ulnar collateral ligament complex. The anterior half of the anterior oblique ligament (known as the anterior band of the AOL) is taut (and therefore functioning as a checkrein) from full extension to 85° of flexion, whereas the posterior band of the anterior oblique ligament is taut with flexion beyond 55°. The anterior band of the anterior oblique ligament is the most important stabilizer of the UCL complex with respect to resisting the forces associated with throwing. The posterior oblique ligament functions with the elbow flexed beyond 90° [8–11]. The tightening of the UCL complex with increasing flexion is due to the cam effect of the hinge joint. The transverse ligament, because it has its origin and insertion on the same bone, has no apparent contribution to joint stability. With division of the UCL, studies have shown that the greatest instability of the elbow is at 70° [12,13]. The radiocapitellar joint is a secondary stabilizer to valgus stress (approximately 30%) [14]. The flexor-pronator muscle group has its common tendon of origin at the medial epicondyle. This muscle group is thought to be an important dynamic stabilizer to the medial elbow. The flexor carpi ulnaris lies directly over the UCL, and is felt to be the primary dynamic contributor to valgus stability. The flexor digitorum superficialis (FDS) may also support valgus stability in greater degrees of extension, because the FDS takes part of its origin off the UCL [15,16]. The wedge shape of the olecranon central ridge within the trochlea provides additional varus-valgus stability with the addition of muscular joint compression forces across the elbow (from the muscles that cross the joint and are contracting during activity).

**Pathophysiology**

Valgus stress from throwing produces tensile forces to the medial elbow. Chronic tensile/distraction stress is initiated by the repetitious high-velocity nature of overhead sports (e.g., baseball pitch, tennis serve, javelin throw, football pass, hockey slap shot, and volleyball spike), and often predisposes the elbow to overuse syndromes [17]. Many of these sports activities require similar motion and mechanics: rapid forceful extension of the elbow, frequently accompanied by valgus stress and pronation of the supinated forearm. The normal valgus carrying angle of the elbow may predispose the medial aspect of the elbow to overuse injuries. The velocity, power, and repetitious nature of the throwing motion all contribute to the ensuing microtrauma, particularly during the late cocking and acceleration phases of the throwing cycle [17]. Biomechanical studies have estimated that the medial elbow shear forces exceed 300 N, compressive forces exceed 900 N during throwing, and elbow extension occurs at up to 2500° per second [17,18]. In tennis, elbow extension velocities occur at approximately 1300° per second [19]. Valgus stress applied to the elbow during the acceleration
phase of throwing is 64N-m [17,18], and is more than 60N-m with the tennis serve [20,21]. These estimated forces exceed the known cadaveric ultimate tensile strength of the UCL (33 N-m) [22].

The forces at the elbow due to repetitive valgus stress result in (1) traction of the medial sided structures (Fig. 2), (2) compression of the lateral side of the elbow, and (3) medially directed shear posteriorly (Fig. 3). Because throwing imparts valgus stress to the elbow, the medial elbow is subjected to tensile forces, resulting in tensile injury to the soft tissues of the medial elbow (UCL, ulnar nerve, flexor-pronator musculotendinous complex) while bony tissues of the lateral compartment (radiocapitellar joint) are inundated with compressive forces. The exception to this is the immature athlete, where the physis is the weakest link in the adolescent musculoskeletal system and thus is most susceptible to injury [23]. As elaborated by Jonas et al in another article in this issue, the medial epicondylar apophysis is most susceptible to inflammation and injury in the thrower’s elbow.

Fig. 2. Valgus stress to the elbow imparts a tensile force to the medial elbow, resulting in injury to the ulnar collateral ligament. Other medial structures, such as the flexor-pronator muscles, can be injured due to repeated eccentric contraction and while attempting to provide dynamic stability to the medial elbow. The ulnar nerve is susceptible to injury due to these tensile forces, as well as to compression within a narrowed cubital tunnel secondary to the scarring within the ligament and osteophytes. With continued valgus stress, the radiocapitellar joint is compressed, which may result in chondromalacia, osteophyte formation, and eventually, loose bodies. (From Safran MR. Injury to the ulnar collateral ligament: diagnosis and treatment. Sports Med Arthrosc Rev 2003;11:17; with permission.)
Effects on medial elbow

Medial elbow structures are susceptible to injury with repetitive throwing. The UCL is particularly at risk for injury due to repetitive tensile (valgus) overload, as seen with overhead throwing sports, and valgus stress, with golfing and hockey slap shots. With UCL injury, tensile forces are then imparted to other structures of the medial elbow, including the flexor-pronator musculotendinous unit and the ulnar nerve.

As noted earlier, the primary dynamic stabilizer to valgus stress is the flexor-pronator muscle mass, particularly the flexor carpi ulnaris and flexor digitorum superficialis [15]. With repetitive valgus stress as applied by throwing, this muscle mass may fatigue, imparting increasing stress to the UCL, and producing microtraumatic ligamentous injury resulting in stretching of the ligament (see Fig. 2). As also noted above, the UCL is the most important static stabilizer to valgus stress to the elbow in the arc between 30° and 120° of elbow flexion, greater than the range of motion needed from the elbow during throwing [2,17]. As a result, the UCL is particularly at risk for microtears or frank tears from excessive valgus force during throwing and overhead sports, depending on the magnitude of force and rate of loading of the ligament.

Furthermore, fatigue of the medial muscles may result in inflammation and injury to the musculotendinous unit of the medial elbow, as demonstrated by
Cicotti et al in an article in this issue. This may result in medial epicondylitis in adults and medial epicondylar apophysitis or avulsion in the skeletally immature thrower, as shown in the articles in this issue by Jonas et al and Cicotti et al.. An athlete may be even more susceptible to these problems if there is concomitant laxity of the UCL due to repeated injury resulting in increased dependence on the secondary or dynamic stabilizers. Furthermore, there are also reported cases of flexor-pronator muscle avulsion injuries associated with UCL tears [24,25]. Chronic traction injury to the UCL may result in chronic thickening of the ligament or traction osteophytes at its insertion on the ulna. As is discussed below and in the article in this issue by Ahmad and Elattrache, chronic UCL laxity may result in shear forces at the posteromedial elbow that may produce osteophytes and loose bodies in the posterior compartment of the elbow.

Ulnar nerve pressures have been found to be elevated to three times more than normal with the elbow flexed at 90° and the wrist extended, the position of late cocking and early acceleration [26,27]. The intraneural pressure elevation is felt to be the result of physiologic stretching of the nerve combined with compression by the flexor carpi ulnaris aponeurosis [28]. With further elbow flexion, wrist extension, and shoulder abduction, as occurs with throwing, this pressure can be elevated to as much as six times the resting normal intraneural pressure [27,29]. Furthermore, it has been found that the ulnar nerve elongates an average 4.7 mm with elbow flexion, and can be displaced by more than 7 mm by the medial head of the triceps [30,31]. As a result of these factors, the cubital tunnel narrows 40% to 55% with elbow flexion, resulting in compression of the ulnar nerve [26,30]. The cubital tunnel may be encroached further by a variety of associated pathologies, including osteophytic spurs, loose bodies, synovitis, thickening of the arcuate ligament, chronically inflamed/thickened UCL, or calcification of the UCL, all potentially resulting in further compression of the ulnar nerve [31–33].

Traction neuritis may be compounded by the valgus forces associated with throwing. With an incompetent or lax UCL, the medial joint gaps open with valgus stress, imparting a tensile stress to the nerve as it is stretched beyond its normal elongation. Professional baseball and tennis players commonly have fixed elbow flexion and valgus deformities, which are static malalignments that further predispose them to ulnar nerve problems. The end result of this excessive traction is fibrosis from direct injury, and possibly ischemia of the nerve due to prolonged or repeated elevation of pressures and stretching injury [28].

**Effects on the lateral elbow**

Valgus stress to an elbow with an attenuated UCL may result in compression of the lateral elbow, particularly the radiocapitellar joint, functioning as a secondary stabilizer. In the adult, valgus stresses in the face of an incompetent UCL may result in a radiocapitellar overload syndrome (see Fig. 2). The repetitive forces to the radiocapitellar joint can result in radial head abutment against the capitellum. This chronic, repetitive radiocapitellar joint force may result in chondromalacia, followed by cartilage and then bony degeneration.
Continued radiocapitellar compression may eventually result in osteochondral fracture and the production of intra-articular loose bodies. In the skeletally immature athlete, this may be clinically manifest as capitellar osteochondritis dissecans, possibly as a result of interruption of the subchondral blood flow due to repeated compressive forces [34,35].

**Effects on the posterior elbow**

Throwing forces may also result in the olecranon being repeatedly and forcefully driven into the olecranon fossa. There typically is a valgus stress that causes shearing, resulting in the olecranon impinging against the medial wall of the olecranon fossa. Increased valgus laxity that results from UCL attenuation may result in increased shear forces to the posteromedial elbow (see Fig. 3). This may be further accentuated by the bony hypertrophy of the distal humerus and proximal ulna, as is commonly seen in those participating in overhead sports [36,37]. The hypertrophy of the distal humerus functionally decreases the size of the olecranon fossa, making less shear translation necessary to result in bony impingement. Furthermore, the hypertrophied proximal ulna also decreases the free space between the olecranon fossa and the olecranon. In addition, the fixed valgus deformity seen in advanced throwing athletes also places the medial olecranon closer to the wall of the olecranon fossa. Thus, less translational shearing (and therefore UCL laxity) is needed before the olecranon comes in contact with the distal humerus, resulting in posterior impingement and valgus extension overload. Additionally, the repeated high extension velocities may result in impaction of the olecranon tip within the fossa. The tip of the olecranon, which is intra-articular, causes local inflammation, eventually resulting in chondromalacia and osteophyte formation. With continued impingement and shear forces, these osteophytes may break off and become loose bodies within the joint [38]. Loose bodies may cause mechanical blocking to flexion or extension, or may produce a synovitis resulting in an effusion and stiff elbow. As noted in the article in this issue by Ahmad and Elattrache, these forces may also result in other olecranon injury, including stress reaction, stress fracture, or apophyseal injury in the skeletally immature ball player [39–41].

**Ulnar collateral ligament injury diagnosis**

**History**

Evaluation of the athlete who has a suspected injury of the UCL includes assessment of the individual’s sport and level of participation, hand dominance, and affected elbow. For throwers, the style, velocity, accuracy, and occurrence of symptoms during the specific throwing phases should be recorded. For baseball pitchers, types of pitches used, pitch count, number of innings pitched, and types of pitches affected by the elbow pain are also documented.
Athletes susceptible to UCL injuries include baseball players (particularly pitchers), javelin throwers, water polo players, volleyball players, and tennis players, as well as golfers, volleyball players, arm wrestlers, wrestlers, hockey players, and gymnasts. Athletes who have an acute UCL rupture complain of sudden pain, with or without a popping sensation that occurs during throwing, while also noting an inability to continue throwing after the injury. Symptoms of ulnar nerve irritation may also occur after an acute UCL tear, due to the hemorrhage and edema from the ligament injury irritating the nearby nerve.

Athletes who have chronic valgus instability as a result of complete disruption or attenuation of the UCL have pain or soreness along the inner elbow with throwing. Overuse is the leading cause of UCL injuries in the throwing athlete [42]. The athlete may give a history of repeated bouts of medial elbow pain during and after throwing that often has responded to conservative management. These athletes are unable to throw the ball at over 75% velocity due to this pain [42]. This is often recounted as a loss of “zip” or “pop” on the ball. The athlete may note, however, that there is a single episode of “giving way” or sudden severe medial elbow pain, which is distinct and isolated. This represents the final insult to the ligament. Injury to the UCL has been described as having four stages: (1) edema and inflammation, (2) dissociation of ligamentous fibers, (3) calcification, and (4) ossification in chronic cases. The most common reported complaint by the athlete is pain during the acceleration phase of throwing; the second most common complaint is pain at ball release or point of impact in hitting the ball [24]. Due to the chronicity of the instability, the athlete may also have a number of other symptoms as a result of secondary injury via the mechanism described above. These symptoms include ulnar nerve irritation, medial epicondylitis, or symptoms of loose bodies. The athlete who has ulnar neuritis may complain of symptoms similar to those in the general population who have ulnar neuritis; however, the earliest ulnar nerve symptoms in the athlete are medial joint-line pain or clumsiness or heaviness of the hand and fingers associated with, or exacerbated by, throwing or overhead activity. The athlete may complain of numbness and tingling of the little and ring fingers, at first usually with overhead activity only. Medial elbow pain may radiate along the ulnar side of the forearm to the hand. Ulnar nerve symptoms may improve or disappear with rest, but recur with resumption of activity, often with increasing frequency, and earlier when returning to throwing. Those athletes who have symptomatic loose bodies may complain of catching or locking. They also may note needing to manipulate the elbow to release or unlock it.

Physical examination

General examination includes inspection, palpation, and motion of both upper extremities, in addition to a thorough neurovascular examination. The examination should include assessment of the shoulder and scapula, because pain or dysfunction of these areas may result in altered throwing mechanics and potentially in UCL injury, as is well described in this issue in the article by Kibler.
Documentation of the presence of the palmaris longus is important for those who may need reconstruction of the UCL.

On physical examination, athletes who have injury to the ulnar collateral ligament have point tenderness 2 cm distal to the medial epicondyle. Those who have acute UCL injury have medial elbow pain and laxity with valgus stress. The absence of pain with resisted wrist flexion, and the location of pain slightly posterior to the common flexor muscle origin help differentiate UCL injury from flexor-pronator muscle injury (though these two problems may occur simultaneously). Ecchymosis may develop in the medial joint line and proximal forearm 2 to 3 days after an acute UCL injury. With a torn or stretched UCL, the medial joint gaps open with valgus stress, and the ulnar nerve is stretched. Long-standing fixed flexion and valgus deformities, as frequently seen in baseball pitchers and tennis players, are static deformities that predispose to traction neuritis, as discussed above [43,44].

The key to the diagnosis of UCL injury is examination to determine the functional integrity of the ligament; however, many descriptions exist on how to best test the ligament. Testing for UCL laxity has been classically described to be performed with the humerus stabilized and a valgus force applied to the flexed (30°) elbow (Fig. 4). In the setting of an acute UCL rupture, care should be taken to assess for flexor pronator muscle avulsion as well. Norwood et al [25] found the flexor-pronator muscle group ruptured in all 4 of their patients who had acute UCL tears, whereas Conway and coworkers [24] found only a 13% incidence of flexor-pronator muscle rupture near its medial epicondylar origin. Athletes who
have UCL injury and flexor-pronator muscle-tendon injury are tender at the medial epicondylar origin of the muscle, and have pain that is increased with resisted wrist flexion. Complete flexor-pronator musculotendinous rupture is associated with weakness of wrist flexion.

The “milking maneuver” produces a valgus stress to the joint in flexion [45]. The affected elbow is flexed beyond 90°, and the opposite hand of the patient is placed under the elbow being tested to grasp the thumb of the affected hand, thereby exerting a valgus stress of the affected elbow (Fig. 5). The UCL is palpated by the examiner for tenderness and joint space opening during this maneuver [45,46]. This technique of examination is felt to be more sensitive at 90° flexion, based on the increased valgus rotation of the elbow at 90°, as compared with 30° when comparing with the contralateral, normal, control elbow [2].

The author uses a modification of the milking maneuver, because in his experience, the patient’s elbows are frequently flexed greater than 120° to perform this test. At this angle of elbow flexion, the contribution of the bony anatomy makes evaluation of the ligament less sensitive (Fig. 6). The author’s modification has the athlete place the arm not being examined under the elbow being examined. This places the shoulder in adduction and maximal external rotation, eliminating external rotation as a confounding motion, a problem with the classic tests for elbow valgus laxity. The subject’s elbow is then held at 70° of flexion, the position of greatest valgus laxity when the UCL is sectioned in a

Fig. 5. Milking maneuver. The patient’s contralateral extremity helps lock the shoulder of the elbow being examined. (From Safran MR, Caldwell GL, Fu FH. Chronic instability of the elbow. In: Peimer CA, editor. Surgery of the hand and upper extremity. New York: McGraw-Hill; 1996. p. 471; with permission.)
The examiner then uses one hand to pull down on the thumb of the elbow being examined, imparting a valgus stress on the elbow. At the same time, the examiner’s other hand is used to palpate the medial joint line to feel for gapping or increased space of the medial joint.

Due to the fact that the center of the varus-valgus axis is more medial than the midline, valgus stress testing of an elbow with an attenuated or torn UCL results in less medial joint gapping than if the lateral ligamentous complex is injured. Medial joint space opening is usually only a few millimeters with complete, isolated UCL injuries. This may explain why studies by experienced clinicians have noted the ability on physical examination to detect valgus elbow laxity preoperatively of 26% to 82% [47,48].

Recently, another test was described to detect UCL insufficiency—the Mayo valgus stress test described by O’Driscoll and Lawton (Fig. 7) [49]. In this examination, the athlete’s shoulder is placed in an abduced and externally rotated position. The elbow is then taken through its flexion-extension range of motion while imparting a valgus force to the elbow. For those athletes who have UCL insufficiency, pain is usually felt at a specific and reproducible point within the flexion arc of 80° to 120°. It is felt that this reproduces the pain of throwing in the athlete, because the shearing force applied to the attenuated ligament is similar to the force and mechanics of throwing.

Other clinical signs to evaluate the UCL deficient elbow have been studied. Pain with valgus stress testing has been found to be present in 26% to 53% of patients undergoing surgery for UCL insufficiency [47,48], and tenderness along
the UCL has been noted to be present in up to 80% of those undergoing UCL reconstruction [48].

**Imaging studies**

A variety of imaging studies can be obtained that may help confirm the diagnosis. Studies that have been reported to be of value include plain radiographs with or without valgus stress, arthrograms, computed tomography (with and without radiographic contrast), and magnetic resonance imaging (with and without intra-articular contrast). Recently, dynamic ultrasound has been studied and reported to be of benefit in the evaluation of the ulnar collateral ligament.

Using plain radiographs, a small avulsion fracture fragment occasionally can be identified, confirming the diagnosis of an avulsion of the UCL. Plain radiographs are useful to identify secondary findings suggestive of chronic UCL insufficiency, including ossification of the ligament (18%) [50], loose bodies in the posterior or lateral compartments, marginal osteophytes about the radiocapitellar or ulnohumeral articulations, or olecranon and condylar hypertrophy. In the absence of an avulsion fragment, instability can be confirmed by stress radiographs. In the acute setting, this may be difficult without anesthesia, due to patient guarding. A commercially produced stress device has become available to apply stress to the ulnar collateral ligament of the elbow at defined, reproducible forces. This radiographic stress testing device has been reported to be 94% sensitive and 100% specific in diagnosing UCL tears [51]; however, it should be noted that increased laxity of the elbow to valgus stress (nearly 0.5 mm) can occur in the uninjured, asymptomatic, dominant elbows of professional baseball pitchers when compared
with their nondominant elbows [52]. This is in contradistinction to the lack of difference (up to 0.5 mm) in valgus laxity between the elbows of a general, nonthrowing population [53,54]. Because of the subtlety of this difference, these injuries may be underestimated and misdiagnosed initially [55]. Conway [56] presented his initial findings of stress radiographic findings in professional baseball pitchers, and found that 2 mm of relative increased laxity and 3 mm of absolute medial joint gapping were consistent with an incompetent UCL. Thompson et al reported that 88% of studied athletes undergoing UCL reconstruction had more than 2 mm of medial joint space opening as measured in radiographs taken with valgus stress applied to the elbow [48]; however, Azar and coworkers [47] noted that only 46% of their athletes who underwent UCL reconstruction had stress tests that they would define as “positive.” The gravity stress test has historically been used to identify valgus laxity of the elbow [57,58]. The test is performed with the patient supine and the shoulder brought into maximal external rotation, with the sagittal plane of the elbow parallel to the floor. With no support under the forearm, the weight of the forearm is resisted only by the flexor-pronator muscle group and the UCL. A standard anteroposterior (AP) radiograph is taken of the elbow. If the fragment of bone (in the case of medial apophyseal injury) moves distally or the joint gaps open medially, the elbow is defined as being unstable.

An arthrogram may be useful in the acute injury; however, an arthogram is of limited usefulness in the setting of chronic UCL insufficiency, because the medial capsule is rarely ruptured chronically, and thus dye leakage from the capsule would not likely be seen. Recently, a CT scan in conjunction with intra-articular contrast has been shown to be 86% sensitive and 91% specific for acute and chronic injuries to the UCL in one study of 25 baseball players [59]. One cited advantage is the ability to visualize an undersurface tear of the UCL, a relatively recently described pathologic entity [59,60]. Yet, the role of CT arthrography must still be confirmed and further defined.

MRI can be helpful in identifying torn UCL [59,61,62]. MRI is useful because it can visualize the ligament, detect chronic injury to the ligament, and show other structures that may be injured, including the insertion to the UCL, or can show radiocapitellar overload by demonstrating bony edema and chondral thinning. In a series by Timmerman and colleagues [59], the authors found that MRI was 57% sensitive and 100% specific; however, Thompson et al [48] found MRI to be positive in 79% of athletes undergoing UCL reconstruction, but “falsely” negative in 21%. Adding intra-articular contrast to the MRI improves the ability to detect UCL injuries, while maintaining the benefit of assessing other structures about the elbow. Azar and coworkers [47] noted that MRI arthrography was 97% sensitive in detecting UCL injury, including partial undersurface UCL tears. These findings must still be confirmed in other centers for assessing their true general applicability.

Recently, dynamic ultrasonography has been studied in the evaluation of ulnar collateral ligament of the elbow [63,64]. Dynamic ultrasound of the elbow in asymptomatic major league professional baseball players revealed that this mo-
dality provides a rapid means of evaluating the UCL. When the dominant elbow is compared with the nondominant, ultrasound can reveal a thicker anterior band, the ligament is more likely to have hypoechoic foci or calcifications, and this test demonstrates increased laxity with valgus stress [63,64].

**Role of arthroscopy in diagnosis**

Arthroscopy has been studied to determine its use in confirming the diagnosis of UCL injury. Its usefulness is limited, because it has been found that only 20% of the anterior oblique ligament and up to 50% of the posterior oblique ligament can be directly visualized using the arthroscope [59,65]. Nonetheless, Timmerman et al have reported that arthroscopy is the most sensitive and specific way to diagnose UCL disruptions [59]. They and others report that the benefit of arthroscopy is that the medial compartment of the elbow can be visualized while applying a valgus stress at 70° of elbow flexion. If the medial compartment gaps open—that is, the ulna moves away from the distal humerus—UCL insufficiency exists [59]. Field and Altchek [65] systematically studied the value of arthroscopy in the assessment of UCL injury in vitro, and found that rupture of the anterior bundle only resulted in 1 to 2 mm of medial joint space gapping, whereas complete release of the UCL resulted in 4 to 10 mm distance between the distal humerus and ulna when valgus stress was applied with the elbow in 70° of flexion. They stated that imaging studies may help confirm the diagnosis, but that the history and physical examination remain the mainstays for diagnosis [65].

**Treatment**

Initial treatment of UCL injury consists of rest, anti-inflammatory measures, and physical therapy. With this conservative regimen, Barnes and Tullos [43] reported that half of their throwing athletes with UCL insufficiency were able to return to throwing, whereas the other 50% required surgery. More recently, Rettig et al reported their results with nonoperative treatment of 31 throwing athletes who had UCL injuries [66]. Nearly two thirds of their study athletes were baseball pitchers. Forty-two percent were able to their previous level of competition at an average 24.5 weeks (13–54 weeks) of conservative management [66]. Conway and colleagues [24] report treating athletic patients with two cycles of 3 months of rest from throwing and rehabilitation; and stated that if pain occurs when throwing over 75% capacity, surgery is indicated. It has not been reported what percentage of these athletes have successful return to sports with this nonoperative program [24]. Rettig and coworkers’ [66] nonoperative protocol involves 2 or more months of rest from throwing, upper extremity strengthening, and bracing. When the athlete is pain-free, they initiate a throwing program that is advanced over 1 to 2 months.

Surgical treatment for UCL tears has evolved. In one of the early reports of the surgical management of UCL insufficiency, Schwab et al [11] recommended
transfer of the anterior oblique ligament anteriorly and superiority when the UCL is present but attenuated; however, this treatment has generally been abandoned because the remaining attenuated ligament is felt to be weaker due to microrupture, its transferred position is not functionally isometric compared with its natural position, and elbow extension tends to be limited by this procedure (resultant flexion contracture), which may not be acceptable in the high-level athlete. Primary repair of the ligament for acute injuries had been advocated for many years [25,43,57]. Most ligamentous avulsions were traditionally treated by reattaching the ligament to bone through drill holes, whereas midsubstance ruptures were repaired primarily [25]. The relative prevalence in UCL injury by location was reported by Conway et al [24] in 70 athletes who had acute UCL disruptions in which 87% of the injuries were midsubstance, 10% were avulsions from the ulna, and only 3% were avulsions from the humerus. They noted that although 71% of those treated with primary UCL repair reported good to excellent results, only 50% of the 14 athletes treated this way were able to return to the same level of throwing as prior to injury [24]. Azar et al [47] also noted better results with reconstruction (81% able to return to play at the same or higher level) as compared with repair (63% return to play at the same or higher level). Conway et al [24] and Jobe and colleagues [67] recommended UCL reconstruction with free tendon graft in the acute setting as their treatment of choice, except in the few patients who have surgery soon after an acute proximal UCL tear, with no ulnar nerve symptoms, and with the remainder of the ligament appearing normal. Due to the relatively consistently good results with reconstruction as reported by Jobe et al and others, most surgeons now prefer to perform UCL reconstruction for acute and chronic injuries than primary repair [46,47]. UCL reconstruction involves using a graft, usually palmaris longus, though fourth toe extensor, hamstring tendon, strip of Achilles tendon plantaris tendon and allograft (hamstring and posterior or anterior tibialis tendon) have all been used to anatomically reconstruct the UCL. This is done through tunnels in the ulna at the sublime tubercle and in the humeral epicondyle (Fig. 8). Ulnar nerve transposition may be performed in conjunction with UCL reconstruction, though there may be a significant risk of neurologic problems postoperatively to the ulnar nerve (up to 21%) [24,47].

Ligament reconstruction is usually recommended for (1) acute ruptures in high level throwers; (2) significant chronic instability; (3) after debridement for calcification within the UCL, if there is insufficient tissue to effect a primary repair in a throwing athlete; and (4) multiple episodes of recurring pain (with subtle instability) with throwing after attempts at conservative treatment.

Conway and coworkers [24] reported their results of 56 UCL reconstructions using a palmaris longus free tendon graft. At 2 to 5 years follow up, they reported 80% good-to-excellent results, with 68% of the athletes returning to their previous level of competition for more than 1 year [24]. They noted a postoperative ulnar neuropathy rate of 21% in their series, with 40% being transient (all but one patient returned to previous sports activity); however, 60% of those who had an ulnar neuropathy required a second operative procedure for the nerve (almost half returned to previous sports activity) [24]. The results of reconstruction of the
UCL were not as good when there had been a previous operative procedure performed on the elbow. Other more recent reviews reveal that 79% to 97% of athletes were able to return to their sport following UCL reconstruction, though most of these athletes had reconstruction without detachment of the flexor-pronator muscles [30, 48, 55, 68–70].

Revisions in technique to reduce complications, such as not transferring the ulnar nerve and splitting the flexor carpi ulnaris muscle mass [48, 71], elevating flexor-pronator tendon without detaching it [47, 55], using a blind-ended tunnel on the humeral side (Fig. 9) [69, 70], using single tunnels with interference fixation (Fig. 10) [72], and fixation of the graft with suture anchors onto bone as compared with tunnels [73] are too recent to allow for critical review. Reports of these modifications of technique identify complication rates of 10% or less, however, suggesting that these modifications are useful in reducing the rate of complications associated with this procedure [47, 48, 68, 69]. Furthermore, there have been no studies directly comparing reconstruction techniques. Reports of allograft reconstruction are too recent to allow for critical review. Synthetic ligament research is an area of interest, but it is too early to know if this will be successful.

Chronic UCL microtrauma occasionally attempts to heal, with calcification resulting within the ligament. These calcifications are removed with the ligament reconstruction. It is important to note, however, that these calcifications are within the ligament. Patients who have medial elbow pain and calcifications must be examined closely. It is important not to mistake these calcifications for intra-articular loose bodies. The calcifications are extra-articular, and removal of them without reconstruction of the UCL will likely render the elbow unstable.

For those athletes who have symptomatic loose bodies, the obvious treatment is removal of the loose bodies, usually performed arthroscopically, due to its
Fig. 9. The recently described docking procedure uses the same ulnar tunnels at the sublime tubercle. Small drill holes or sutures on a Keith needle can be used to pass the sutures through the epicondyle. The graft must be shortened to allow both ends to fit in the tunnel and allow for tensioning. This reconstruction only allows for a two-ply reconstruction, but is easier to tension the graft. Two recent studies have shown excellent results with minimum 2-year follow-up. (From Safran MR. Injury to the ulnar collateral ligament: diagnosis and treatment. Sports Med Arthrosc Rev 2003;11:22; with permission.)

Fig. 10. A graft that has been doubled over (or more) is placed into a single tunnel on the sublime tubercle and held in the tunnel while advancing an interference screw using an innovative screwdriver. Then the graft can be placed and fixed on the humeral side in a similar fashion. This technique has been tested in the laboratory, and though there have been some reports of success clinically, no large series with significant follow-up have been reported, due to this recent introduction of the screwdriver. (From Safran MR. Injury to the ulnar collateral ligament: diagnosis and treatment. Sports Med Arthrosc Rev 2003;11:22; with permission.)
minimal morbidity as compared with arthrotomy. It should be noted, however, that the loose bodies will likely recur if the athlete continues to participate in the sport that produced the valgus forces initially, especially concomitant if valgus laxity persists. Most studies reveal that athletes (including professional baseball pitchers) return to their activity after removal of the loose bodies. Bennett and Tullos reported their experience with removal of loose bodies via arthrotomy and found that the average professional pitcher continued to throw effectively 3 years after this procedure (range 2–11 years) [57]; however, as they also pointed out, the loose bodies will recur in those athletes who return to throwing, because pitching is an inherently destructive process [57]. Recent studies of arthroscopic removal of loose bodies confirm the usefulness of this procedure, with its low morbidity and possibly quicker return to activity, as discussed above, especially if there is no associated degenerative arthritis [74].

Rehabilitation of the UCL injured elbow and surgical management of ulnar nerve and valgus extension overload problems are discussed in detail in other articles in this issue by Wilk et al, Keefe and Litner, and Ahmad and Elattrache, respectively.

Summary

The elbow joint is a complex structure that allows for the transmission of forces from the lower extremity to the ball, racket, or other instrument or object. In many sports, it must withstand tremendous forces that are not often encountered in daily living. Significant valgus force is not common as part of the activities of daily living, as it is in sporting activities. Significant valgus force can be destructive, with medial elbow tensile forces, lateral compressive forces, and posterior shear forces. The ulnar collateral ligament is commonly injured; however, injury may not always be manifest by gross laxity or pain at the ligament. The athlete who has UCL injury may present with symptoms related to secondary overload of other areas about the elbow accentuated by UCL laxity, such as medial epicondylitis, ulnar neuritis, lateral elbow pain due to radio-capitellar overload, posterior elbow pain due to valgus extension overload, or loose bodies. The clinician must maintain a high index of suspicion for any pain about the elbow in the throwing athlete as potentially being due to UCL insufficiency, and must focus on direct management of the underlying problem, not just the symptom, because treating the symptom rarely prevents the symptoms from returning.

References


