Posterolateral Rotatory Instability of the Elbow

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Abstract

Posterolateral rotatory instability of the elbow is a three-dimensional displacement pattern of abnormal external rotatory subluxation of the ulna coupled with valgus displacement on the humeral trochlea. This pattern causes the forearm bones to displace into external rotation and valgus during flexion of the elbow. Injury to the lateral ulnar collateral ligament allows abnormal supination of the ulna on the humerus. The radial head, being locked in the sigmoid (radial) notch of the proximal ulna by the annular ligament, subluxates posterior to the capitellum. The abnormality is usually posttraumatic and presents with locking, snapping, clicking, catching, and recurrent dislocation of the elbow. The clinical diagnosis is suspected from history and confirmed by the physical examination, which includes the posterolateral rotatory instability test. This test often is best performed under fluoroscopy or general anesthesia. Usually the instability is managed with either a repair of the ligament or an isometric reconstruction using a tendon graft.

J Am Acad Orthop Surg 2004;12:405-415

Posterolateral rotatory instability (PLRI) of the elbow is a clinical syndrome first described by O'Driscoll et al[1] in 1991. The instability usually results from an injury, such as a dislocation with subsequent failure of adequate healing of the injured lateral ligamentous structures. Patients present with symptoms of clicking or locking or even recurrent instability. O'Driscoll et al[1] postulated that an insufficiency of the lateral ulnar collateral ligament (LUCL) leads to PLRI. Some have identified further soft tissues on the lateral side of the elbow that contribute to lateral elbow instability. Others question the lateral theory of elbow instability.[2,3] Treatment options range from splinting to surgical repair or reconstruction of the torn lateral structures.

Relevant Anatomy

The important ligaments on the lateral side of the elbow are thickenings of the capsule. The lateral collateral ligament complex consists of four components: the lateral (radial)[4] collateral ligament (LCL), the LUCL (ulnar part of LCL),[4] the accessory LCL, and the annular ligament (Fig. 1, A).

The LCL attaches to the lateral epicondyle and fans out to merge indistinguishably with the annular ligament. It functions as a varus restraint and stabilizes the annular ligament. Martin[5] in 1958 described some details of the LCL complex. He reported that the lateral capsule of the elbow consists of three layers. The deep layer he described as the joint capsule. The intermediate layer was the true annular ligament. The superficial layer was derived from the lateral ligament and fanned out from the lateral epicondyle to attach to the anterior and posterior aspects of the proximal ulna.

Morrey and An[6] named the LUCL and recognized its clinical significance. The LUCL is a thickening of the capsule that attaches proximally to the lateral humeral epicondyle and distally to the tubercle of the supinator crest of the ulna (Fig. 1, B). The humeral attachment of the LUCL is at the isometric point on the lateral side of the elbow and is well defined.[4] The distal attachment of the ligament is deep to the fascia surrounding the extensor carpi ulnaris and supinator muscles (Fig. 1, A). Besides stabilizing the lateral aspect of the elbow, the LUCL also acts as a posterior buttress for the radial head to prevent its subluxation.

Injury to the LUCL allows an abnormal external rotation (supination) of the ulna on the humerus. As a result of the external rotation of the ulna,

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None of the following authors or the departments with which they are affiliated has received anything of value from or owns stock in a commercial company or institution related directly or indirectly to the subject of this article: Dr. Mehta and Dr. Bain.

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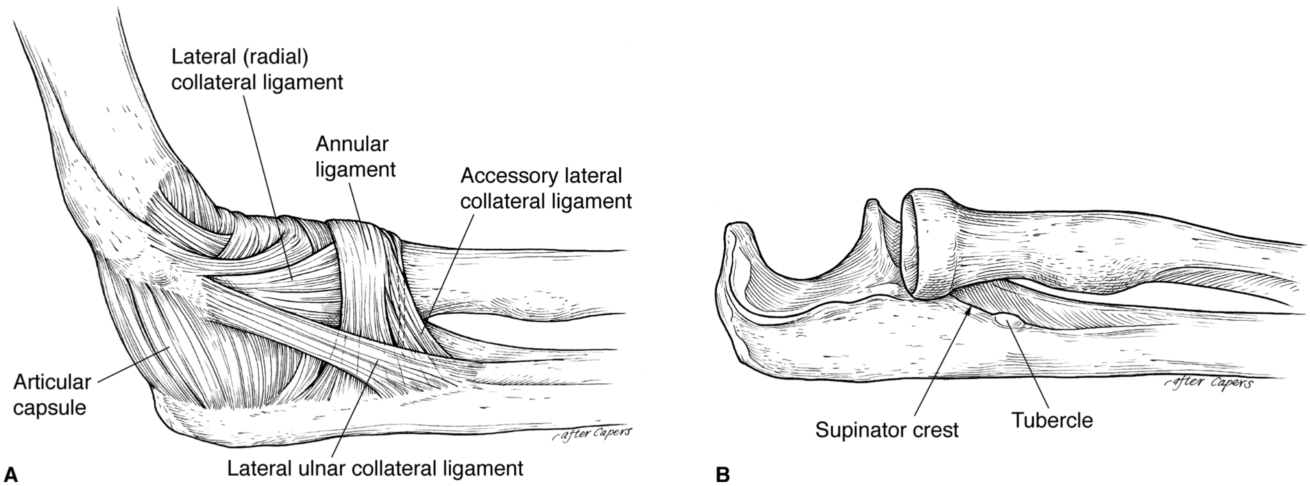


Figure 1 **A,** Lateral collateral ligament complex. **B,** Osseous anatomy of the lateral aspect of the proximal humerus. (Adapted with permission from Bain GI, Mehta JA: Anatomy of the elbow joint and surgical approaches, in Baker CL Jr, Plancher KD [eds]: *Operative Treatment of Elbow Injuries*. New York, NY: Springer-Verlag, 2001, pp 1-27.)

the radial head, which is locked into the sigmoid (radial) notch of the ulna by the annular ligament, subluxates posterior to the capitellum. On varus stressing, the LUCL is tense, and it is indistinguishable from the supinator crest. But with associated valgus, stressing the demarcation between the supinator crest and the LUCL is possible.[4] Recent studies, although confirming the presence of the LUCL, have questioned its singular role as the primary stabilizer of PLRI.[7]

The annular ligament attaches to the anterior and posterior margins of the sigmoid (radial) notch of the proximal ulna, encircling the radius but not attaching to it. The most distal aspect of the annular ligament has a smaller diameter that encircles and contains the neck to provide greater stability to the radial head.

The accessory LCL blends proximally with the fibers of the annular ligament and attaches distally to the tubercle of the supinator crest. The accessory ligament stabilizes the annular ligament during varus stress.

The medial collateral ligament (MCL) complex is a capsular thickening consisting of three components: the anterior band, the posterior band, and the transverse ligament of Cooper (Fig. 2). The anterior band is the most important and spans from the medial humeral epicondyle to the sublime tubercle on the medial aspect of the coronoid process. It is the primary stabilizer to a valgus force.[6] The posterior band spans from the medial epicondyle to the olecranon process and is a restraint against internal rotation of the ulna (on the humerus). Therefore, the three primary stabilizers of the elbow are the humeroulnar articulation, the MCL complex, and the LCL complex.[8] The secondary stabilizers are the radial head, the cap-

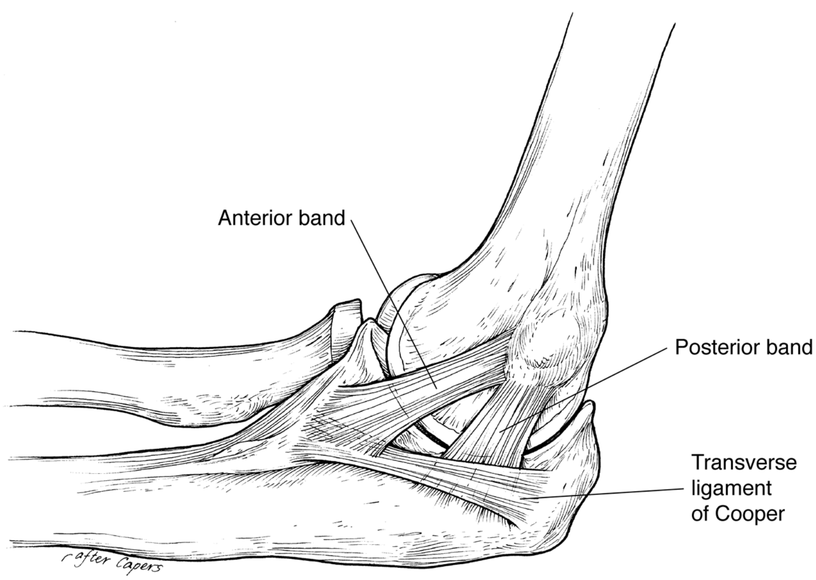


Figure 2 Medial collateral ligament complex. (Adapted with permission from Bain GI, Mehta JA: Anatomy of the elbow joint and surgical approaches, in Baker CL Jr, Plancher KD [eds]: *Operative Treatment of Elbow Injuries*. New York, NY: Springer-Verlag, 2001, pp 1-27.)

sule, and the common flexor and extensor origins.[8] If the three primary stabilizers are intact, the elbow will be stable.

Pathoanatomy of Elbow Instability

Concepts and management of elbow instability have evolved over more than a century with increased understanding of the anatomy of the elbow, the pathomechanics of elbow instability, and the management of instability. Three main concepts have emerged: central bony insufficiency, lateral ligamentous insufficiency, and medial ligamentous insufficiency. PLRI is one part of the spectrum of elbow instability and must be regarded from a global perspective of elbow instability.

Elbow instability was first recognized only in its gross form as an isolated or recurrent dislocation. The perceived etiology in most of these early cases was a shallow trochlear notch. Some reports, however, did cite ligamentous laxity, loose bodies, and epicondylar fractures as probable causes for recurrent instability. Management was largely focused in the sagittal plane, with augmentation of the coronoid process with bone blocks of diverse shapes from various donor sites.[9] Some surgeons also augmented the olecranon, whereas others provided further restraint by rerouting the biceps tendon to the coronoid or passing strips of biceps and the triceps tendon through the window between the coronoid and olecranon fossae. Today, the humeroulnar joint is thought of as a ginglymus (hinge) joint with intrinsic stability resulting from the depth of the trochlear fossa. It is one of the most congruent joints in the body, and the trochlear groove is made more stable by the olecranon and coronoid processes[10] (Fig. 1).

In elbow injuries, coronoid process fractures are common and can be regarded as an indication of a previous subluxation or dislocation. In the patient with PLRI, the coronoid process abuts the trochlea. Increasing external rotation (supination) of the ulna will push the coronoid process beneath the trochlea to allow dislocation of the elbow joint. If the coronoid is fractured, the elbow becomes more unstable, requiring less external rotation (supination) of the ulna to produce the dislocation. A large fracture-fragment of the coronoid process includes the anterior capsule and anterior band of the MCL and requires reduction and internal fixation to preserve the medial stability of the elbow.

Osborne and Cotterill[11] originated the concept of lateral ligamentous instability. They defined the essential lesion as "failure of the posterolateral ligamentous and capsular structures, torn or stretched at the time of an initial simple traumatic dislocation, to become reattached."[11] Drawing an analogy between elbow instability and recurrent anterior dislocation of the shoulder, they recommended plication of the lateral capsule with transosseous sutures to prevent recurrent instability. Others subsequently reinforced this observation, adding that the MCL also may contribute to joint instability.[12,13] Interest in the importance of the lateral aspect of the elbow was reinvigorated by the description of PLRI in 1991 by O'Driscoll et al.[1] These authors defined posterolateral instability, described the clinical syndrome, identified a clinical test to diagnose the condition, postulated an anatomic basis for it, and described a surgical procedure to address the instability.[1]

The relative importance of each ligament of the lateral complex to PLRI remains controversial. The initial report by O'Driscoll et al[1] focused on the insufficiency of the LUCL. They reported that the LUCL is the primary lateral stabilizer of the humeroulnar joint, and its deficiency is the "essential lesion"[14] that produces PLRI.

In an anatomic study of the lateral stabilizers of the elbow,[15] the LUCL was not distinguishable from the adjacent capsule in some specimens. The researchers suggested that the primary stabilizers of PLRI are a combination of the LCL and annular ligament and highlighted the role of the lateral musculature as a secondary stabilizer. In addition, two soft-tissue structures could act as secondary rotatory stabilizers: a band of extensor carpi ulnaris (ECU) fascia coursing from the lateral humeral epicondyle to the proximal ulna and the intermuscular septum separating the ECU and the extensor digitorum minimi. Thus, damage to more than one structure may be necessary to have a substantial amount of lateral instability of the elbow. This occurs with either attenuation or avulsion of both ligamentous and muscular origins from the lateral epicondyle and, as suggested by other authors,[16,17] results in compromise of both the primary and secondary restraints.

Dunning et al[18] demonstrated that when the annular ligament is intact, either the LCL or the LUCL could be transected without inducing PLRI. Therefore, the current hypothesis is that PLRI of the elbow must result from injury to other anatomic structures in addition to the LUCL. This hypothesis is based primarily on evidence from cadaveric studies and thus may differ from the actual clinical situation.[7,15]

In most elbow dislocations, the entire LCL complex and capsule are avulsed as a sheet. In cases in which the joint remains subluxated, the LCL complex is translated distally so that its proximal free edge is positioned on the articular surface of the capitellum. Therefore, the normal ligament-to-bone healing cannot occur. In some cases, new bone can be seen bridging between the epicondyle and the avulsed ligament. Recurrent instability and PLRI are most likely caused by failure of the lateral complex to heal in its anatomic position on the lateral epicondyle.

Although the medial ligamentous structures are often considered not to have a significant role in PLRI, they should be appreciated in the context of overall elbow instability. The importance of the MCL complex in elbow instability remains controversial. The MCL complex is often avulsed from its humeral attachment and remains in continuity with the periosteum.[11] It may heal, but in an attenuated position. If the joint is able to be redislocated, then a muscular tear from the humeral epicondyles is usually present.[2,3]

Eygendaal et al[19] reviewed the cases of 41 patients with an average follow-up of 9 years after a posterolateral dislocation of the elbow treated with a closed reduction and cast for 3 weeks. Fifteen patients (37%) had moderate valgus instability (<3 mm medial joint opening on stress radiographs); 3 (7%) had severe instability (>3 mm opening). The presence of medial instability was correlated with persistent pain, a worse elbow score, ectopic bone formation, and radiologic signs of degeneration. All nine patients with a positive Tinel's sign over the cubital tunnel had evidence of medial instability. Six patients had a positive lateral pivot shift test result, although no patient in the series had a recurrent dislocation. The medial ligament insufficiency is an important part of elbow instability and should not be considered in patients with PLRI.

The dynamic and positional stabilizers of the elbow include all of the muscle groups that cross the elbow, especially the anconeus, triceps, and brachialis,[10] which provide a compressive force across the joint. Cadaveric studies underestimate the clinical importance of the musculature as a dynamic stabilizer. Cohen and Hastings[15] demonstrated that the PLRI reduced spontaneously by simple forearm pronation, even when all lateral ligament restraints had been disrupted. This finding supports the clinical impression that, in some circumstances, elbow dislocations can be managed with splints that restrict forearm supination.

The pathoanatomy of elbow instability is conceptualized as a three-stage circle of soft-tissue disruption, commencing on the lateral side and extending to the anterior and then the medial aspects of the joint[20] (Fig. 3). This pattern is referred to as the circle of Horii and is analogous to the Mayfield perilunate instability pattern seen in the wrist. Stage 1 of this circle is characterized by disruption of the LUCL. This can produce joint subluxation (PLRI). With further progress of the force, stage 2 develops, with disruption of the anterior and posterior capsules. The joint is "perched" so that the coronoid is under the trochlea. Stage 3 follows with persistence and progression of the force. This stage is marked by three subcategories. In stage 3a, all of the soft tissues are disrupted, with the exception of the anterior band of the MCL. (The other tendon and muscle origins are intact, including the common flexor pronator origin.) As a result, the elbow dislocates and pivots around the intact anterior band. In stage 3b, the anterior band is also ruptured, so that the elbow is unstable to valgus force. The severity of instability necessitates immobilization of the reduced elbow in 30° to 45° of flexion. In stage 3c, the entire distal humerus is stripped of soft tissue. This leads to gross instability of the joint so that it is stable only with >90° of flexion.[8]

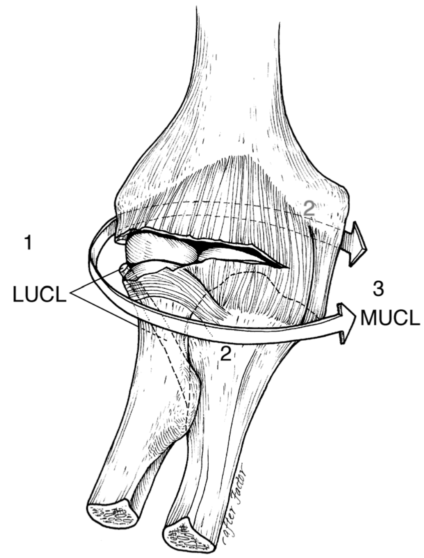


Figure 3 Three-stage circle-like soft-tissue disruption. Stage 1, LUCL disruption. Stage 2, anterior and posterior capsule disruption. Stage 3, disruption of the medial collateral ligament. LUCL = lateral ulnar collateral ligament, MUCL = medial ulnar collateral ligament. (Adapted with permission from O'Driscoll SW, Morrey BF, Korinek S, An KN: Elbow subluxation and dislocation: A spectrum of instability. *Clin Orthop* 1992;280: 186-197.)

Mechanism of Injury

PLRI typically occurs in patients who have had a history of a dislocation of the elbow that either reduced spontaneously or was treated by closed reduction.[3] However, some may have experienced either chronic elbow sprains or fractures of the radial head or the coronoid process. In most instances, the injury results from a combination of axial compression, external rotation (supination), and valgus force applied to the elbow.[20] It usually occurs as the patient falls onto the outstretched hand. This produces an axial force along the length of the arm, and a valgus force is created as the mechanical axis passes through the lateral side of the joint. The body rotates internally on the elbow (mechanically the same as forearm external rotation or supination). This combination of forces is the same as that reproduced with the posterolateral rotatory instability test.

PLRI may occur most frequently in patients with generalized ligamentous laxity[21] or varus inclinations of the distal humerus, secondary to childhood supracondylar fractures.[22] PLRI is also seen as a complication after lateral surgical approaches to the elbow.[1,23] The Kocher approach, although providing a good exposure for management of radial head fractures, violates the LCL complex, and it is imperative that the complex be repaired at the completion of the procedure.[11] PLRI of the elbow also can develop as a complication of the Boyd surgical approach, which involves release of the soft tissues from the lateral side of the proximal ulna.[24] The PLRI was a consequence of the failure to repair the lateral ligamentous complex of the elbow onto the proximal ulna.

Clinical Presentation

The patient may relate a history of having had one or more dislocations of the elbow. With each subsequent dislocation, the force required to produce instability decreases. One of our patients had 10 dislocations; the last episode occurred when the patient turned off the bedside light. Patients often learn maneuvers to reduce the joint and therefore may not require formal reduction.

Recurrent painful clicking, snapping, clunking, or locking of the elbow are the most common symptoms. These often occur in the extension half of the arc of motion, with the forearm in supination. Patients may report that the elbow feels loose or slides out of joint when they perform activities. Patients are often apprehensive about performing activities that precipitate the instability—for example, when pushing on the armrests while rising from a chair.

On initial examination, the patient will appear to have a normal elbow. It is not tender (unless there has been a recent injury), and there is usually a full pain-free range of motion. It is not uncommon to have hyperextension, particularly with the atraumatic type of instability. Applying a valgus or varus instability test does not cause pain and will not produce any significant instability, although varus stress is sometimes uncomfortable.

The clinical examination is usually unremarkable except for the posterolateral rotatory instability test.[1] This is best performed with the patient supine and the extremity over the patient's head (Fig. 4). The shoulder is fully externally rotated, which stabilizes the humerus, so that the elbow can be assessed independent of shoulder motion. The examiner then grasps the patient's forearm, which is placed in full supination. In this position, the elbow looks like a knee, and the maneuver is analogous to the pivot shift test used to assess anterior cruciate ligament instability. Starting with supination and extension, the elbow is slowly flexed while the examiner applies a slight valgus force and axial load and maintains the supination. This produces a rotatory supination torque on the forearm that can produce a rotatory subluxation of the humeroulnar joint. The ulna tilts externally on the trochlea of the humerus, and this rotation dislocates the radial head posteriorly because it is coupled to the ulna by the annular ligament. As the elbow is flexed to approximately 40°, the rotatory displacement is at a maximum. At this point, the subluxated radial head produces a posterior prominence associated with an obvious dimple in the skin proximal to the radial head.

The rotatory subluxation pivots on the anterior band of the MCL of the elbow.[8] With increased flexion, the triceps becomes taut and forces the radiocapitellar joint to reduce. The radial head translates anteriorly over the prominence of the capitellum and reduces with a sudden reduction "clunk" that is more obvious to the patient and the examiner than the initial subluxation. This clunk is the reduction of the radial head and is accentuated by the axial load that is placed across the radiohumeral joint. The reproduction of the patient's symptoms along with apprehension is also a positive test result.

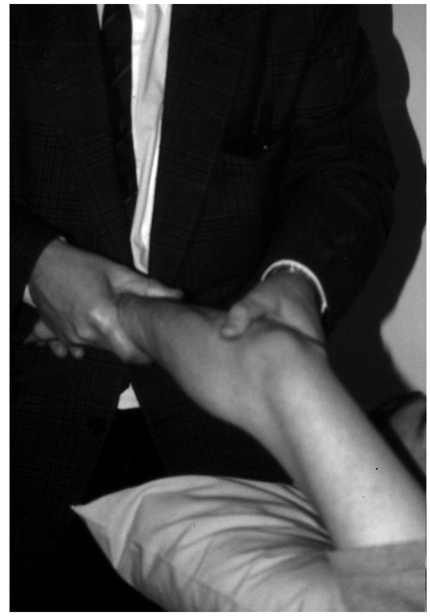


Figure 4 Posterolateral rotatory instability test maneuver, with the patient supine and arm above the head. The elbow is positioned to resemble a knee.

Patients may be apprehensive about having the provocation test performed, to the point that they refuse to be tested. Guarding to protect the elbow and limb may be either voluntary, with the patient preventing examination with the contralateral arm, or involuntary, with muscle spasm that locks the elbow and prevents normal examination. Apprehension is a symptom experienced by the patient. The subsequent facial expressions and preventive guarding are signs the examiner observes. One of three alternatives may then be adopted to aid in eliciting a positive test result. (1) Local anesthetic can be infiltrated into the joint, which will reduce the proprioceptive feedback and decrease apprehension. (2) The test can be done under fluoroscopy, so that subtle instabilities can be identified more accurately. (3) The test can be performed under sedation.

The posterolateral rotatory drawer test is similar to the Lachman test of anterior cruciate ligament integrity of the knee. The arm is positioned overhead so that it resembles a leg; the elbow is the knee. The lateral side of the proximal forearm is drawn in a posterior direction, attempting to subluxate the radial head and reproduce the dimple between the radial head and the capitellum.[21] Alternatively, the patient may display an apprehension that is highly suggestive of PLRI.

Other tests include the prone push-up and chair push-up tests described by Regan and Morrey.[25] The patient attempts to rise from the prone position or from a chair with armrests. The maneuver is attempted first with the forearms maximally pronated, then repeated with the forearms maximally supinated. If the symptoms are manifest with forearm supination but not with pronation, then the tests are positive for PLRI of the elbow.[25]

Differential Diagnosis

Valgus instability can closely mimic PLRI. To distinguish the two, the MCL complex is examined with the shoulder fully internally rotated. The assessment of the anterior band of the MCL should be done with the forearm in pronation.[1] Pronation holds the radius on the capitellum so that any medial joint space opening is the result of medial ligamentous insufficiency. The elbow is stressed in a valgus direction with the elbow in 30° of flexion and then in full extension. In flexion, the MCL is the primary stabilizer to a valgus force.[26] In full extension, the anterior capsule is taut and the olecranon is locked in its fossa, which provides stability even if the MCL has been excised.[27] Biomechanical studies have demonstrated that, in the flexed elbow, the anterior capsule, radial head, and olecranon process are all important in assisting the MCL in providing valgus stability.[26] If there is valgus instability in the flexed elbow, the MCL is incompetent. If there is also instability in the extended elbow, there is a much more extensive injury, including disruption of the anterior and posterior capsules.

Other parts of the examination include assessing for generalized ligamentous hyperlaxity and neurovascular compromise and determining whether the palmaris longus tendon is available to be used as a tendon graft.

Radiologic Evaluation

Performing the provocation tests under fluoroscopy with local anesthetic infiltrated into the joint provides the best radiologic assessment of instability. In the lateral plane, PLRI is assessed, and in the anteroposterior plane, varus and valgus instability are each assessed. Alternatively, stress radiographs can be taken while the provocation maneuvers are performed. Subtle subluxation can sometimes be seen on the nonstress radiographs in which the ulna is abnormally supinated on the trochlea, which is seen as humeroulnar joint space widening. Fractures of the coronoid process and radial head should be identified. An impaction defect in the posterolateral capitellum produced by the dislocated radial head (Hills-Sachs lesion of the elbow) also may be identified radiographically.

Management

Patients may learn to avoid the instability by performing activities with the elbow flexed to prevent subluxation. Elbow braces may be adopted, although they are cumbersome and unlikely to be used for an extended period. Most patients with PLRI will not be satisfied with a brace.

Surgery is indicated in patients with symptomatic instability of the elbow. Relative contraindications to surgery include children with open physes, concomitant arthritis of the joint, generalized ligamentous laxity, and habitual recurrent dislocations. When PLRI occurs in children, it is treated with a lateral plication instead of a reconstruction. Large coronoid process fractures may require internal fixation as part of the procedure.

Arthroscopy may be used as an adjunct to reconstruction. Arthroscopy may help identify associated joint injuries, assess the level of the instability, and débride the joint. Osteochondral injuries to the radial head and trochlea can be identified and débrided. With the arthroscope in the lateral or posterior portal, the pivot shift test can be performed and the humeroulnar joint space opening assessed.[21] The medial joint space also can be evaluated using a similar technique. The authors have used arthroscopy to classify patients with PLRI. (1) Those with isolated PLRI are managed with a lateral reconstruction. (2) Those with associated abnormal medial joint space opening are managed with a circumferential graft that reconstructs the MCL and the LUCL. We have identified a number of patients with a clinical presentation of PLRI who also have medial instability at arthroscopy. (3) In addition, we have identified patients with PLRI with associated arthritis. This is a difficult problem in which the severity of instability and arthritis requires individual assessment.

Surgical Exposure of the Lateral Capsule

The patient is positioned in the lateral position. A posterior midline incision is preferred because anatomic studies have demonstrated that a posterior incision encounters fewer nerves of smaller diameter than a lateral incision does.[28] The Kocher interval between anconeus and the ECU muscles is identified by a thin, fat strip, which can be seen through the deep fascia[24] (Fig. 5). The interval is developed to expose the elbow capsule, the lateral epicondyle, and proximal ulna. The muscles are retracted

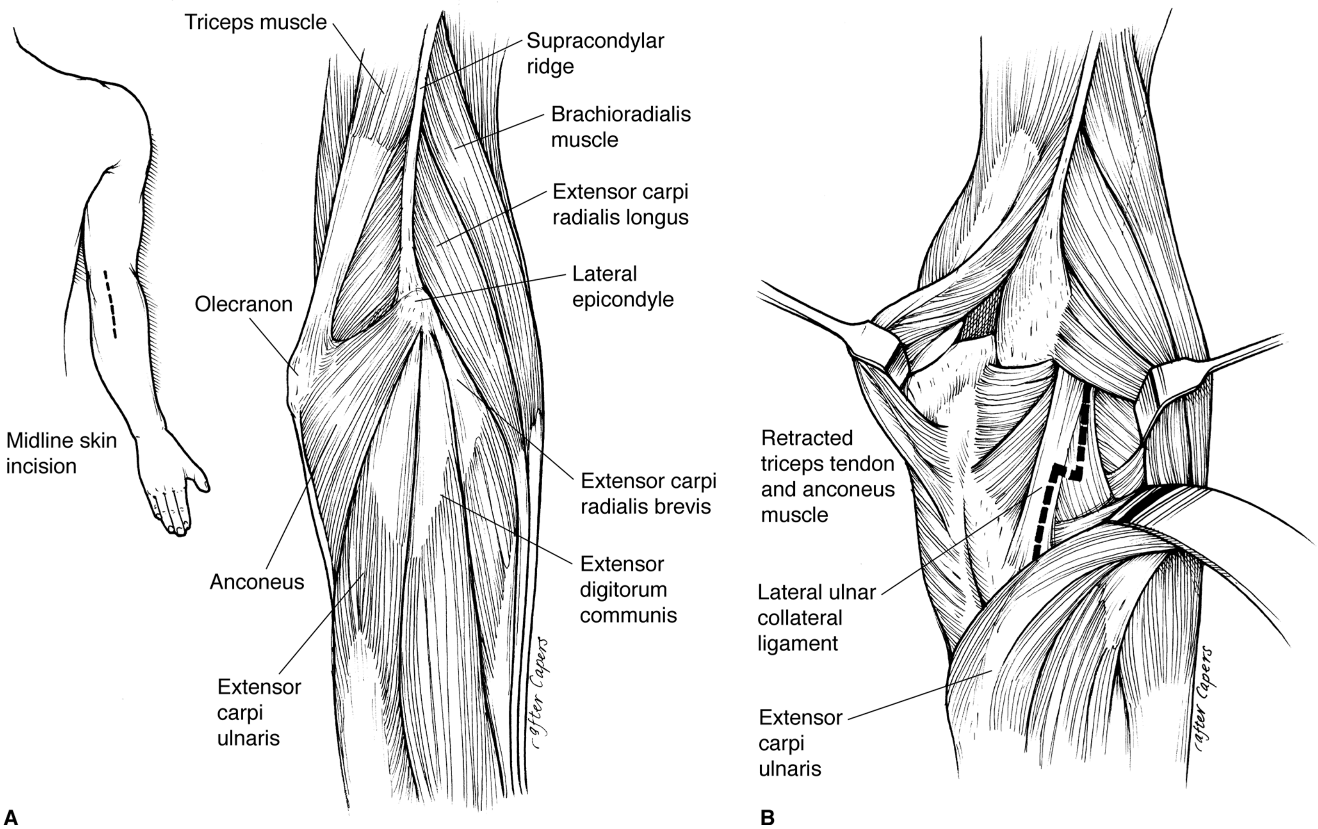


Figure 5 **A,** Through a posterior midline incision (inset), the Kocher interval between the extensor carpi ulnaris and anconeus is identified to expose the lateral joint capsule and supinator crest. **B,** Retraction of the anconeus and extensor carpi ulnaris muscles exposes the lateral capsule. (Adapted with permission from Bain GI, Mehta JA: Anatomy of the elbow joint and surgical approaches, in Baker CL Jr, Plancher KD [eds]: *Operative Treatment of Elbow Injuries*. New York, NY: Springer-Verlag, 2001, pp 1-27.)

to expose the capsule, although it may not be possible to identify the LUCL as a distinct entity.

Arthrotomy

It is important to avoid further damage to the LCL complex; iatrogenic PLRI is a serious concern in the patient with an acute injury. A lateral Z arthrotomy made anterior to the LUCL[24] minimizes intraoperative soft-tissue trauma and instability. The arthrotomy is anterior to the LUCL so that humeroulnar stability is not violated. The Z is centered over the annular ligament. This lateral arthrotomy can be extended proximally along the supracondylar ridge or distally over the annular ligament (Fig. 5, B). The Z arthrotomy is placed in the annular ligament so that it can be closed easily without undue tension (Fig. 6, A). This is particularly useful for management of radial head fractures.[24] The anterior capsular flap can be released from the humerus without violating the stability of the joint because the LUCL remains intact. Usually this provides adequate exposure to perform most lateral elbow procedures, including open release of fixed flexion deformity. At this level of exposure, only a simple soft-tissue closure is required, and stability has not been violated.

Only when extra exposure is required, such as to allow insertion of a metallic radial head prosthesis, is the posterior capsular flap released from the humerus. Once the LUCL has been violated, a transosseous ligament repair is required, as described by Osborne and Cotterill.[11] An alternative is to use suture anchors, although transosseous sutures are preferred.

Sometimes in the patient with PLRI, adequate ligamentous tissue is available for repair to reconstitute elbow stability. If adequate tissue is not present, however, a ligamentous graft is indicated to restore stability. If there are frequent spontaneous dislocations and considerable medial laxity, then a medial ligament repair also may be required.[11] The authors have used a circumferential hamstring graft in this situation.

Following an arthroscopic or open joint débridement, the LCL insertion into the lateral epicondyle is released. The LCL complex is then simply ad-

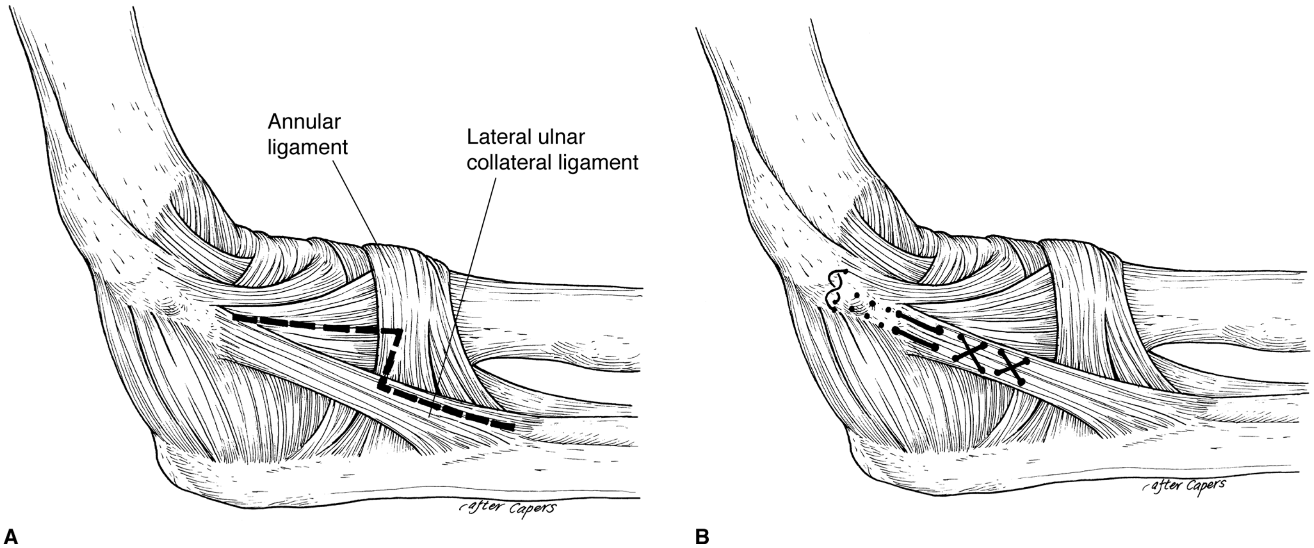


Figure 6 **A,** A Z incision in the annular ligament, anterior to the lateral ulnar collateral ligament, allows tension-free closure without compromising stability. **B,** Transosseous Bunnell suture to repair the lateral ligamentous complex allows the capsule to be advanced proximally. (Adapted with permission from Bain GI, Mehta JA: Anatomy of the elbow joint and surgical approaches, in Baker CL Jr, Plancher KD [eds]: *Operative Treatment of Elbow Injuries*. New York, NY: Springer-Verlag, 2001, pp 1-27.)

vanced and fixed to the débrided epicondyle using a Bunnell grasping transosseous suture (Fig. 6, B). Additional plicating sutures may be required in the anterior and posterior capsules.

Lateral Ligament Reconstruction

Ligament reconstruction is indicated for the patient who does not have adequate ligamentous tissue for a repair. The reconstruction technique is appealing because it is consonant with current precepts of ligament reconstruction. It is an isometric, extracapsular, anatomic reconstruction using an autogenous graft.[23] Because tightening the graft can be difficult, the authors have used a technical modification so that greater tension can be created within the graft.

The required graft length is approximately 20 cm, and if the palmaris longus tendon is used, a strip of the attached palmar aponeurosis may be required. The palmaris longus tendon is present in 85% of individuals.[29] Olsen and Søjberg[30] described using a 12 × 1-cm strip of triceps passed through humeral drill holes and fixed distally with suture anchors. Alternatively, the plantaris tendon (present in 80% of lower limbs) can be identified preoperatively with ultrasonography. When present, it is an excellent graft, given its length, and it is larger than the palmaris.[31] The fourth toe extensor tendon, a split semitendinosus tendon, or an allograft also can be used. Synthetic ligament augmentation devices have been reported, but an autogenous graft is usually preferred.

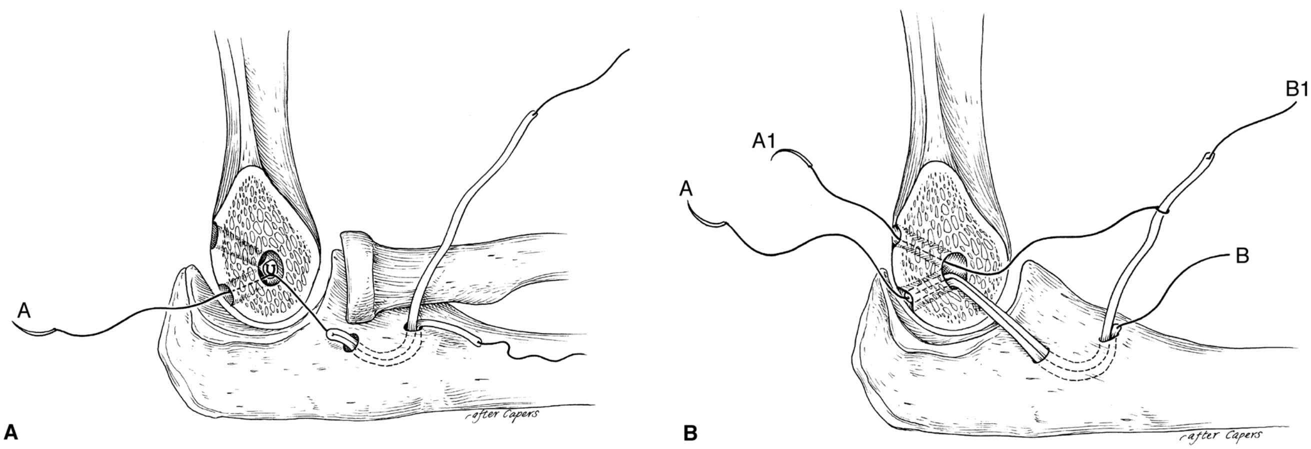
A 3.5-mm drill hole is placed into the supinator tubercle, which can be identified at the distal attachment of the lateral capsule. The second hole is drilled 1.25 cm proximal at the base of the attachment of the annular ligament to the ulna. Both holes are extra-articular, just external to the capsular attachment. A bony tunnel is created between these two holes (Fig. 7, A).

A suture is placed through the two ulnar holes, and a hemostat is attached to it. The hemostat is then placed onto the lateral epicondyle and the elbow flexed and extended. The isometric point of the humerus is the point at which the suture remains taut throughout the range of motion. This should correspond to the center of the capitellum as seen from the lateral projection.

Having identified the isometric point on the humerus, a Y-shaped tunnel is placed into the lateral epicondyle. The first hole is drilled in the lateral cortex using a 4.5 mm drill bit slightly posterior and proximal to the isometric point, so that the graft passes over the isometric point. The 3.5-mm drill is advanced through to the posterior aspect of the lateral humeral condyle (distal exit hole). The drill is withdrawn and again advanced through the isometric point but is directed proximally and posteriorly (proximal exit hole), taking care to ensure that there is an adequate bone bridge between the two posterior holes (Fig. 7, A).

Modification of Graft Fixation

A modification can be made to the technique of Nestor et al[23] that obtains



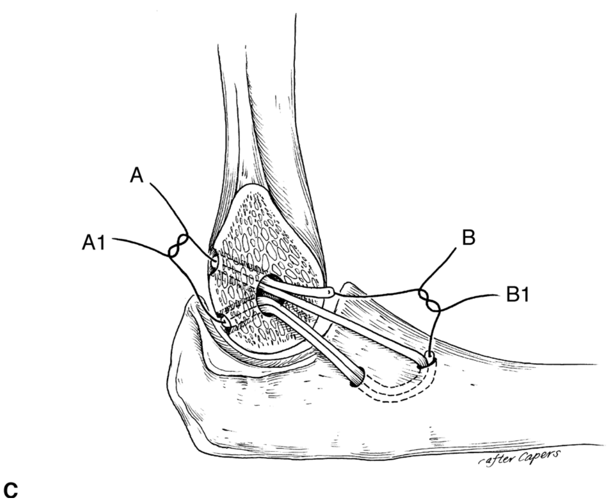


Figure 7 **A,** Location of the humeral and ulnar drill holes. The transfixing suture, A, is 5 cm from the end of the tendon and passes through the ulnar holes and distal humeral exit hole. The suture is advanced until the graft is visible through the exit hole. **B,** Suture A1 is positioned so that when it is advanced through the proximal exit hole, the graft will be taut. **C,** The two transfixing sutures (A and A1) are tied under tension over the lateral condyle. The other two sutures (B and B1) are tied under tension distally.

greater tension in securing the graft and ensures that adequate length is available because the graft does not need to pass over the posterior aspect of the lateral epicondyle. A 1/0 Ethibond suture (Ethicon, Milltown, NJ) transfixes and then encircles the graft at a point 5 cm from one end. This suture is passed through the two ulnar holes and delivers the graft. The suture is then passed through the hole at the isometric point and out through the distal exit hole until the graft is just visualized (Fig. 7, B). Sutures are placed into both free ends and tension applied. With the surgeon holding the graft in tension, the assistant moves the elbow through a full range of motion. Uniform tension of the graft while allowing full motion of the elbow indicates optimum tension.

A second transfixing suture is placed at the point where the graft, when advanced, will be just visualized at the proximal exit hole. Care is taken to ensure that the elbow is reduced and placed in full pronation at 30° of flexion. The sutures within the distal and proximal exit holes are tied under tension over the lateral condyle. The other two sutures are then tied. (Fig. 7, C). This double-tension ("double docking") technique ensures that all limbs of the graft are tight. The capsule is plicated, and the wound is closed in layers.

Postoperative Management

An above-elbow splint is applied with the forearm in midpronation and the elbow in 90° of flexion. It is removed at 1 week, and a 30° extension block orthotic splint is applied for 6 weeks. The angle can be extended, depending on the recovery. Patients who are reliable and have excellent graft fixation and stability are extended by 10° per week. Other patients may require slower mobilization. Return to contact sports is permitted after 6 months.

Other modifications include the use of a proximally based strip of the fascia of the ECU muscle and a distally based strip of triceps.[16,23,32] Augmentation with a no. 2 heavy absorbable suture or a polypropylene prosthetic ligament augmentation device (3M, St Paul, MN) also has been reported.[23] The "yolk" suture does tighten part of the tendon graft but leaves the remainder untensioned.[33]

Results and Complications

The reported results of LCL reconstruction have been very good. All patients treated with a transosseous repair in the series of Osborne and Cotterill[11] (eight patients) and Nestor et al[23] (three patients) had excellent results.

The results of ligamentous reconstruction have been mixed, partly because of the complex nature of the cases. Nestor et al[23] reported three excellent and two fair results in patients with a palmaris longus tendon reconstruction. The two patients with a synthetic augmentation device (in addition to a palmaris graft) had persistent instability, and their results were rated fair and poor respectively.

In reviewing their results, O'Driscoll et al[8] suggested that if there is no degenerative arthritis and the radial head is intact, then approximately 90% of patients have a satisfactory outcome. If the radial head is excised or there is degenerative arthritis of the humeroulnar joint, there is only a 67% to 75% satisfactory outcome. Olsen and Søjberg[30] reported that 10 of 18 cases were stable with the triceps graft. Four patients had positional apprehension to the pivot shift test. Five patients reported pain.

Regarding complications, persistent instability is the principal concern. Considerable care is required to prevent fracture of the bony bridges with the reconstruction. Cutaneous nerve injury is minimized with a posterior midline incision.[17] A fixed-flexion deformity is common but is unlikely to encroach on the functional range of motion of the joint.

Summary

A high index of suspicion should be maintained for PLRI in patients with elbow symptoms such as clicking, locking, and weakness. Performing the posterolateral rotatory instability test is diagnostic. LCL repair is usually effective in providing joint stability if adequate soft tissue exists. An isometric ligament reconstruction is recommended if inadequate ligamentous tissue is available. The surgeon should be mindful of PLRI and can use the lateral Z arthrotomy to avoid inadvertent iatrogenic instability.

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