



Diagnosis and Treatment of Posterolateral Rotatory Instability

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Posterolateral rotatory instability is caused by insufficiency of the lateral stabilizers of the elbow. Acute instability occurs as a consequence of trauma and is best treated by primary repair. Chronic instability may be a consequence of trauma, but has also been described in the setting of corticosteroid injections for lateral epicondylitis and repetitive varus load. In this setting, ligament reconstruction is typically required.

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Introduction

The lateral collateral ligament (LCL) complex plays a key role in elbow stability.¹ In most instances, this complex fails as a result of an elbow subluxation or dislocation.^{2,3} Additional causes for instability include chronic varus load, iatrogenic injury during surgery, as well as possibly following steroid injections for lateral epicondylitis.⁴

Anatomy

The lateral stabilizers of the elbow include both musculotendinous and ligamentous structures that maintain congruency of the humerus and proximal forearm. The LCL complex originates at the base of the lateral epicondyle and lateral aspect of the capitellum. In the reduced elbow, the isometric origin of the LCL is located at the intersection of a distal projection of the anterior cortex of the humerus and the longitudinal axis of the radial head.⁵ This correlates with the geometric center of the capitellum.⁶ Distally, the LCL fibers blend into the annular ligament forming a broad conjoint insertion onto the proximal ulna (Fig. 1).^{5,7} The posterior fibers of this insertion have been termed the lateral ulnar

collateral ligament, extending 2 cm along the supinator crest from the proximal border of the radial head distally.⁴ The LCL complex is covered by the supinator muscle anteriorly and the anconeus muscle posteriorly. Most superficially, the supinator muscle is covered by the common extensor origin, namely the brachioradialis, extensor carpi radialis longus and brevis, extensor digitorum communis, extensor carpi ulnaris (ECU), and anconeus.⁸

The LCL complex is the primary stabilizer of the lateral elbow.^{7,9} During axial loading of the slightly flexed elbow, the LCL complex maintains ulnohumeral and radiocapitellar congruency, especially when the forearm is held in supination. The extensor muscles and their fascial bands and intermuscular septations provide further restraint against lateral rotation of the forearm about the humerus. In supination, these muscles provide both static and dynamic support. The most proximal extensor muscle, the ECU, exhibits the best mechanical advantage in supporting the proximal forearm. In addition, the ECU has a deep fascial band that originates at the lateral epicondyle and inserts onto the ulna approximately 5 cm distal to the lesser sigmoid notch. This band tightens during supination providing secondary resistance to posterolateral rotatory instability (PLRI).⁷

Mechanism of Injury

The most common mechanism for disruption of the LCL complex involves avulsion from the humeral origin as a consequence of an elbow dislocation.^{7,10-12} Occasionally, the distal insertion of the LCL may become compromised either as

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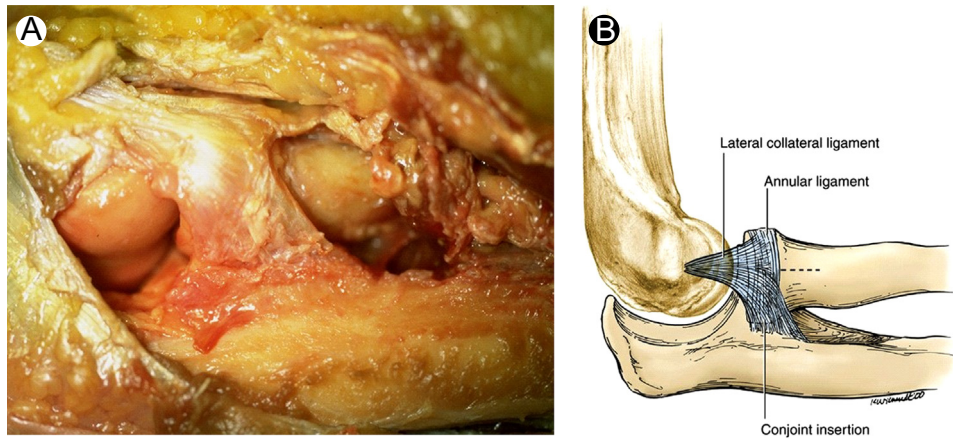


Figure 1 (A) Cadaveric specimen and (B) schematic drawing depicting anatomy of the lateral collateral ligament (LCL) and annular ligament complex in a right elbow. The overlying extensor muscles and supinator fibers have been removed. The LCL originates at the base of the lateral epicondyle and fans out blending with the annular ligament. These form a broad conjoint insertion onto the proximal ulna along the supinator crest just posterior to the proximal radioulnar joint. (Copyright: Mark S. Cohen, Chicago, IL) (Color version of figure is available online.)

an avulsion injury or as a fracture of the supinator crest.^{13,14} The prevailing theory suggests that most elbow dislocations occur as a combination of axial compressive, external rotatory, and valgus forces, in which the humerus is placed into internal rotation relative to the planted and fixed forearm.^{2,3} In this setting, disruption of elbow stabilizers proceeds in a circular path from lateral to medial.¹⁵ Less frequently, acute instability may result from a blow to the extended elbow causing an acute varus deforming force.⁴

In addition, proximal attenuation of the LCL complex has been seen in patients with chronic cubitus varus deformity, most commonly posttraumatic. It has also been described in patients with persistent lateral epicondylitis.¹⁶⁻¹⁸ Corticosteroid injections have been suggested as a possible contributing factor in these patients.¹⁹ Finally, iatrogenic injury to the LCL complex can occur during surgery for lateral epicondylitis and during procedures on the radial head.^{16,17}

Symptoms

Manifestations of LCL insufficiency may range from unremitting pain over the lateral epicondyle to frank giving way or apprehension when the elbow is axially loaded in extension and supination, such as when pushing up from a chair. However, most frequently, symptoms of chronic LCL insufficiency include painful clicking, clunking and snapping of the elbow.²⁰ Locking of the elbow in near terminal extension has also been described, especially when the forearm is in a supinated position. In this setting, the supinated radius and proximal ulna rotate laterally as a unit, off of the distal humerus in a pattern termed PLRI.

Physical Examination

In most cases of PLRI, grip strength and range of motion are usually unaffected. In some instances, a mild passive extension

deficit may be present. Frequently, palpation of the lateral elbow is mildly uncomfortable. In rare instances, a fluid collection can be observed over the lateral aspect of the elbow. This is a synovial fistula originating from the lateral joint capsule.¹⁹

In the awake patient, muscle relaxation is required to appreciate lateral elbow instability. As such, gentle manipulation of the elbow is required to avoid active muscle activation owing to guarding. With the appropriate provocative maneuvers, subluxation of the elbow can be confirmed. The elbow may be examined with the patient in either lying supine on a table or in the sitting position. In the supine position, the posterolateral rotatory apprehension (pivot shift) and posterolateral drawer tests are performed. For these tests, the examiner stands at the patient's head. The patient's shoulder is maximally flexed and externally rotated, thereby locking the humerus into a fixed position. For the pivot shift test, the elbow is held in approximately, 40 degrees of flexion. The forearm is then maximally supinated and a valgus moment with axial compression is applied. Dimpling of the skin over the radiocapitellar joint will confirm PLRI, as the ulna and radius rotate off of the distal humerus.² With the same starting position as the pivot shift test, the posterolateral rotatory drawer test is performed by applying a posteriorly directed force onto the proximal radius. Again, dimpling of the skin around the radiocapitellar joint at 40 degrees of flexion confirms PLRI. The amount of subluxation should decrease with increasing elbow flexion.

In the sitting position, one hand stabilizes the adducted humerus, with the thumb placed on the radiocapitellar joint. With the elbow in approximately 40 degrees of flexion, the examiner's opposite hand supinates the patient's forearm while applying a valgus and axial load on the elbow. Instability is appreciated as a posterolateral prominence of the radial head. This occurs as the forearm rotates away from the humerus. The joint is reduced by elbow flexion and forearm pronation. Occasionally, a palpable clunk can be perceived during reduction.

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