Preoperative and operative risk factors for failed lateral collateral ligament reconstruction

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A R T I C L E   I N F O

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Repair or reconstruction of the lateral collateral ligament (LCL) using autograft or allograft is a well-accepted treatment of posterolateral rotatory instability. The prevalence and causes for failure of LCL reconstruction are not well documented in the literature. Any approach to the assessment and management of failed LCL reconstruction must begin with understanding the risk factors for failure in the first place. Such understanding would likely make many failures preventable as well. In our experience, there are a number of identifiable preoperative risk factors concerning bony and/or soft tissue constraints for failure of LCL reconstruction. There are also operative factors such as tunnel and graft placement as well as excessive lateral condyle stripping that play a role in risk of failure. This report is an attempt to provide a systematic approach to identifying and managing the preoperative and operative risk factors. Further studies are warranted to determine the indications for, and success rates of surgical intervention in managing these risk factors.

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Repair or reconstruction of the lateral collateral ligament (LCL) using autograft or allograft is a well-accepted treatment of posterolateral rotatory instability (PLRI). Recurrent instability has been variably reported to be between 0% and 33%.[1,3-5,7-10,13,15-19] In many cases it is not clear whether the recurrence is due to unrecognized predisposing factors, incomplete or inadequate surgery, or repeat injury. This is apparent from the fact that the only study of revision LCL reconstruction reported in the literature to date documented a 27% failure rate due to recurrent instability.[1] Any approach to the assessment and management of failed LCL reconstruction must begin with understanding the risk factors for failure in the first place. Such understanding would likely make many failures preventable as well.

Several studies have suggested that osteochondral defects of the capitellum or radial head are responsible for recurring microinstability.[1,4,5,13,15,18] Three studies reported the presence of Osborne-Cotterill lesions in patients with recurrent instability.[4,5,18] Additionally, a history of previous operation[10,18] or corticosteroid injection[3,17] may be a prognostic factor. Interestingly, in 2012 Nestor et al[10] reported poor to fair outcomes in 2 patients after LCL reconstruction using a combination of palmaris longus autograft

and polypropylene ligament augmentation device synthetic graft. Tawari et al[18] also reported on the outcome of using a ligament augmentation and reconstruction system synthetic graft for LCL reconstruction. In a cohort of 10 patients, 3 experienced recurrence all of whom had a history of previous elbow surgery.

In our experience, there are a number of identifiable preoperative risk factors for failure of LCL reconstruction. The purpose of this report is to provide a systematic approach to identifying and managing the preoperative and operative risk factors.

Preoperative risk factors

Compromised bony constraints

Trauma in adults

In the setting of trauma, combined deficiencies of the bony constraints is a serious risk factor for failed LCL repair or reconstruction. Persistent or chronic instability is likely if the radial head is excised in a patient with an associated coronoid fracture. Thus, the combination of an O'Driscoll Tip subtype 2 coronoid fracture and comminuted radial head and neck fracture puts the elbow at risk of failed LCL repair or subsequent reconstruction. Persistent instability in this setting is best managed by fixation or reconstruction of the coronoid and as well as reduction and internal fixation or replacement of the radial head. If one or the other is not possible, the risk of failure increases.[14] Coronoid reconstruction can

Figure 1 Combined bony lesions can act synergistically to cause instability and increase the risk of failure of LCL repair/reconstruction. (**A** and **B**) Radiographs of a 38-year-old female with subtle abnormalities of the radial head and capitellum as well as a linear density along the lateral side of the radial neck (arrow). (**C**) 3D reconstruction showing the bony avulsion of the LCL insertion from the tubercle of the supinator crest (arrow). (**D**) 3D CT reconstruction of the humerus showing the capitellar impaction fracture (arrow) known as the Cotterill lesion. (**E**) 2D sagittal reconstruction reveals a potentially "engaging" marginal radial head fracture along with the capitellar impaction. At surgery, these were confirmed to be engaging lesions. By permission of Mayo Foundation for Medical Education and Research. All rights reserved. *LCL*, lateral collateral ligament; *3D*, 3-dimensional; *CT*, computed tomography; *2D*, 2-dimensional.

be performed using the remaining piece of the radial head if it is large enough.[2]

Another combination of bony lesions occurs when three or four small fractures, which on their own might not be a significant problem, act synergistically to compromise elbow stability. An example is the combination of a bony avulsion of the LCL insertion from the tubercle of the supinator crest along with a marginal fracture of the radial head and an engaging capitellar impaction fracture (Cotterill lesion) (Fig. 1). These sometimes are associated with small coronoid tip fractures. Marginal fractures of the rim of the radial head combined with capitellar impaction (or shear) fractures have the potential to "engage" with a lesser magnitude of posterolateral rotatory subluxation (Fig. 2). Bony avulsion of the LCL insertion, which permits minimal posterolateral rotatory subluxation on its own, is sufficient to permit "engagement" when combined with lesions of the radial head and capitellum. Johnson et al[6] showed in an experimental model that while small marginal defects in the rim of the radial head do not affect elbow stability with the LCL intact, the integrity of the radial head becomes important as a secondary constraint when the LCL is damaged. Failed LCL repair in these patients can be prevented by internal fixation of the

marginal radial head fracture (Fig. 3) and disimpaction of the capitellar impaction fracture through a small window in the posterior column. Allograft bone graft is then packed in to support the disimpacted subchondral bone (Fig. 4). The bony LCL insertion is fixed back to the supinator crest with a suture anchor prior to LCL repair (Fig. 5).

Instability in children or adults with a history of childhood injury

Every patient with recurrent or chronic instability of the elbow should be asked if the first episode of instability occurred prior to skeletal maturity, or if they ever had a childhood injury to the elbow. Two particular histories are especially important: (1) childhood dislocation and (2) pediatric supracondylar fracture. The reason is that such a patient is very likely to have dysplasia that might compromise the outcome of LCL reconstruction.

History of childhood dislocation. A patient who has had dislocations in childhood and now presents with recurrent dislocations or subluxations likely has anteromedial coronoid (and or tip) dysplasia, presumably from an unrecognized coronoid apophyseal injury when the coronoid was still predominantly cartilaginous. This results in a
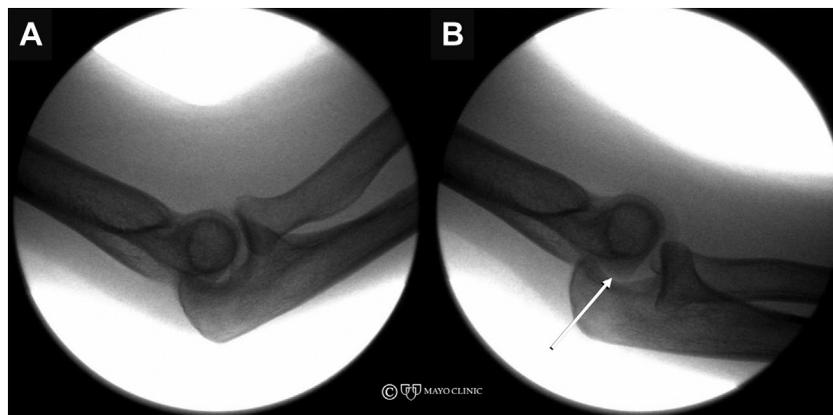
Figure 2 Posterolateral rotatory stress radiographs under anesthesia. (**A**) Lateral view without stress. (**B**) Lateral view during posterolateral rotatory drawer test shows posterolateral rotatory subluxation of the ulnohumeral joint (arrow) and radial head. By permission of Mayo Foundation for Medical Education and Research. All rights reserved.
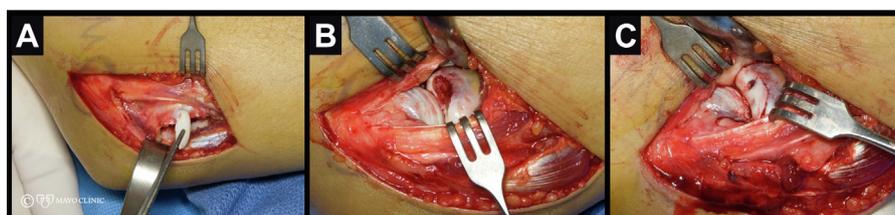

Figure 3 Internal fixation of small marginal osteochondral rim fracture of the radial head. (**A**) Free fragment retrieved from the joint. (**B**) Marginal rim defect in the radial head (**C**) ORIF with small headless screw and resorbable pin. By permission of Mayo Foundation for Medical Education and Research. All rights reserved. *ORIF*, open reduction internal fixation.

hypoplastic coronoid with anteromedial deficiency that is best appreciated on 3-dimensional computed tomography reconstruction (Fig. 6.) Without an anteromedial coronoid, the elbow is prone to subclinical varus posteromedial rotatory instability, which eventually causes attenuation of the LCL and tardy PLRI.

In such circumstances, the chronic/recurrent abnormal stresses on the developing elbow also lead to dysplasia of the trochlea, radial head, and the radial notch of the proximal radioulnar joint (Fig. 6). The trochlea, which normally is round like the letter "O" in the sagittal plane may be shaped like the letter "U." When this happens, usually the olecranon and coronoid similarly develop a "U" shape. This congruent incongruity results in the ulnohumeral joint being pried open in extension, stretching the LCL and leading to attenuation.

These same chronic/recurrent abnormal stresses on the developing radial head result in dysplasia of its shape and orientation. The anterior rim does not develop properly but becomes rounded off. The articular dish of the head is shallower than normal. Finally, the slope of the radial head becomes tilted, such that axial loads on the radial head cause it to "escape" posteriorly.

The various forms of dysplasia all result in abnormal stresses on the LCL complex. Likewise, after LCL reconstruction, such abnormal stresses will act to stretch out the reconstructed ligament or loosen its attachment sites to bone. There are no data on which to determine which dysplastic structures need to be corrected, so it is necessary to rely on principles. As stated above regarding combined bony injuries in adult traumatic instability, these dysplastic lesions also act synergistically to compromise elbow stability after LCL reconstruction. The first principle is that LCL reconstruction alone may be inadequate. The second principle is that each dysplastic lesion that can be predictably corrected should be corrected. The third principle is that the coronoid dysplasia is the most important factor but is also the most complex to correct (at least at this stage of our understanding and surgical experience).

Coronoid reconstruction has not been reported to have a high success rate, but our experience leads us to believe that it is more successful for restoring stability in the unstable elbow that is not painful or arthritic than in the painful elbow with posttraumatic arthritis due to persistent instability (Fig. 7).

Prior pediatric supracondylar fracture. Supracondylar fracture is a common pediatric injury. Closed treatment often results in varus malunion (usually with internal humeral rotation). Such varus malunion alters the biomechanical forces and moments (torsional stresses) on the developing elbow and its surrounding soft tissues in such a way as to put the elbow at risk of tardy PLRI.[12] The tip of the olecranon (and therefore the triceps insertion) is displaced medial to the long normal line of pull by the triceps (Fig. 8). This creates 2 problems.

First, triceps contraction causes an external rotational moment on the ulna leading to increased tensile stress on the lateral ulnar collateral ligament (Fig. 8). Over time, this leads to (i) medial elongation of the tip of the olecranon, (ii) hypertrophy of the tendon of the medial (deep) head of the triceps, and (iii) LCL attenuation. Second, the varus angulation causes chronic repeated stress on the LCL leading to LCL attenuation. These deforming forces act synergistically to stretch out the LCL complex over time, leading to tardy PLRI (Fig. 9). Importantly, they also act to break down any reconstruction or repair of the LCL over time.

The key to preventing failure after LCL repair/reconstruction in patients with cubitus varus from a pediatric supracondylar malunion is to understand and neutralize these deforming forces. The varus deformity is corrected by distal humeral osteotomy. The medial displacement of the triceps insertion on the elongated medial olecranon can be corrected by lateral transposition of the medial triceps tendon. LCL attenuation is corrected by LCL imbrication or reconstruction.
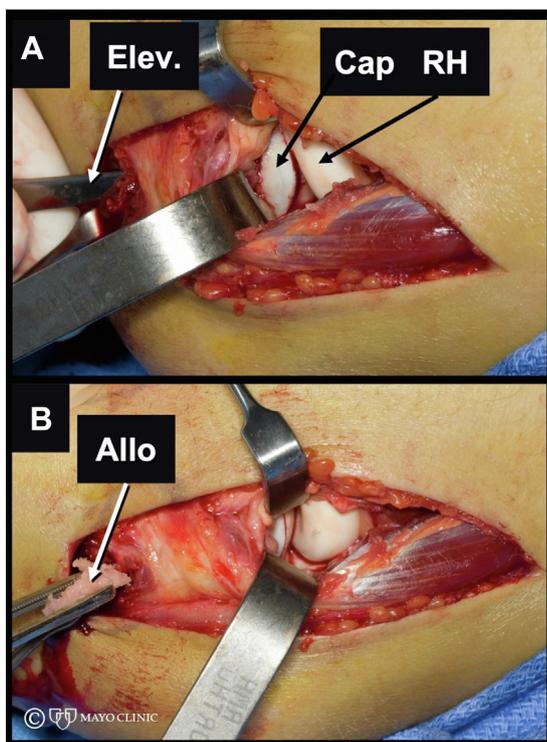
Figure 4 Treatment of the engaging Cotterill lesion shown in Figure 1. At surgery, the lesions were confirmed to be engaging. (**A**) The capitellar impaction fracture was elevated through a transosseous tunnel from the posterior aspect of the capitellum and (**B**) the tunnel packed with cancellous allograft bone graft. By permission of Mayo Foundation for Medical Education and Research. All rights reserved. *2D*, 2-dimensional; *Elev. (arrow)*, elevator; *RH*, radial head; *Cap*, capitellum; *Allo (arrow)*, allograft.

Compromised soft tissue constraints

Hyperlaxity

Hyperlaxity of the soft tissues is typically associated with hyperextension of the elbow. The lack of the normal locking mechanism, that occurs as the olecranon fits into the olecranon fossa, can increase the stress on the ligaments and compromise the results of LCL reconstruction. Although there are not enough data to know if surgery should routinely be performed differently in patients with hyperextension, it is worth considering in those who have coexisting compromise of bony constraints or who already failed LCL reconstruction.

Hyperextension can be treated by anterior capsulodesis and ulnohumeral tenodesis at the time of LCL reconstruction. There are 2 options for surgical approach. The first involves separate medial and lateral incisions for capsulodesis/tenodesis and LCL reconstruction respectively using separate allograft tendons (Fig. 10). The second, which the senior author (S.O.D.) has come to prefer, is a single lateral incision using a single continuous allograft tendon for both the capsulodesis and LCL reconstruction (Fig. 11). There are pros and cons to both, but the advantage of the latter is that, as the elbow is extended, the tension in the LCL reconstruction increases. This might mimic the sling effect of the conjoint tendon overlying the subscapularis tendon in the Latarjet procedure for shoulder instability.

Anterior capsulodesis is performed by making a transverse incision across the anterior capsule from lateral to medial (or vice versa) then performing a "vest over pants" imbrication as described for incisional hernia. The sutures are not tied until the tendon graft used for tenodesis is ready to be docked.

Ulnohumeral tenodesis is performed using an allograft (peroneus longus or semitendinosus). The graft is first passed antegrade down through the tip of the coronoid and retrieved from the lateral ulna just below the tubercle on the supinator crest, then passed through a small sagittal split created in the common extensor tendon origin and docked into the lateral humeral condyle as one would do for LCL reconstruction. The opposite end of the tendon is marked for length and docked into a tunnel in the humerus at the proximal capsular attachment site anteriorly. Length is decided based on the desired degree of intraoperative extension block with gravity alone. At this point, the optimum extension block is not known. (The senior author has generally chosen between 30°-50° extension block).

Soft tissue loss

The common extensor tendon is an important secondary constraint to elbow instability.[11] It also augments the underlying LCL complex to which it is bonded. Deficiency of the common extensor tendon can occur in tennis elbow, particularly after repeated corticosteroid injections. PLRI also can occur as an iatrogenic complication of lateral release for tennis elbow. If the tendon deficiency is greater than a certain (yet unknown) threshold, the reconstructed LCL may be subjected to excessive strain and fail. In such cases, the extensor tendon origin may need to be reconstructed as well.

Muscle imbalance combined with scarring

In our experience, the few patients with chronic instability in the setting of traumatic brachial plexus palsies have not been successful with LCL reconstruction. Two factors have been identified. First, muscle imbalance across the elbow was present, but whether it was the partial paralysis or the unopposed remaining muscles that caused a problem is not yet clear. Second, periarticular scarring was recognized to act as a tether against concentric reduction in some portions of the arc of motion.

Operative risk factors

Tunnel and graft placement

Correct tunnel placement has not yet been documented with data to predict success of surgery, but it is probably important. The graft should optimally function isometrically, but at a minimum it must resist posterolateral rotation with the elbow in the extension portion of the arc of motion. A reliable method for confirming isometricity during surgery is to pass a suture through the ulnar tunnel, then hold the suture down onto the anticipated isometric point on the humerus while flexing and extending the elbow. If it is too tight in flexion the graft will stretch out and be lax in extension. It is not known whether or not laxity in flexion is a problem.

The tendon graft should not be placed under the capsule or annular ligament. If it is, it will potentially snap, catch, and/or cause irritation against the lateral articular margin of the capitellum and/or the asymmetric annular rim of the radial head. The normal LUCL passes in an arch over and around the lateral capitellum and radial head down onto the ulna. It is not a straight line in 3-dimensional space. Failure to recognize this may result in placement of the graft in a straight line, which can lead to these complications as well as stretching of the graft and eventual recurrent instability. Prevention of these problems is best accomplished by closing the capsule (and annular ligament if it had been incised) prior to graft tensioning. A small split in the sagittal place is created in the common extensor tendon close to its origin so the graft can be passed from the extracapsular space down into the humeral tunnel for docking.
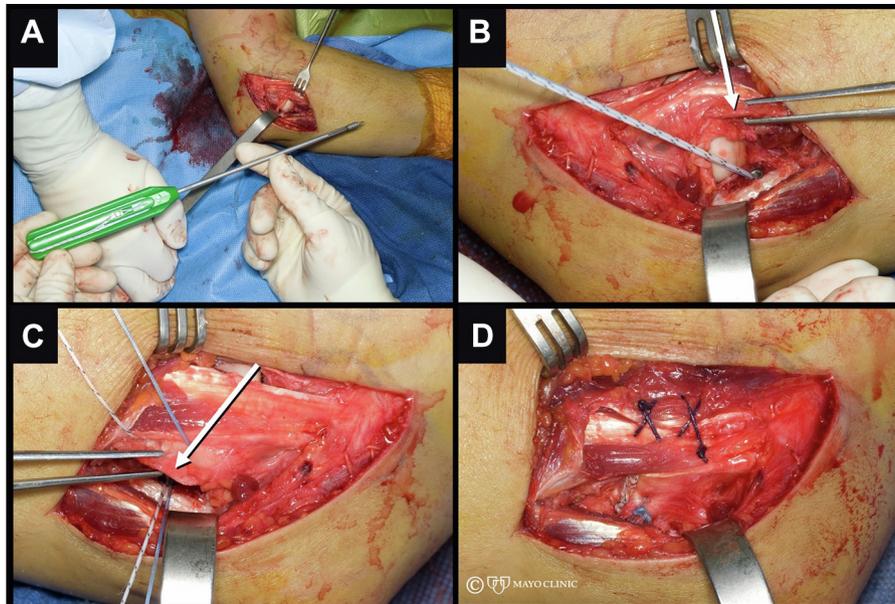
Figure 5 Repair of the LCL complex. (**A** and **B**) Suture anchor placed into fracture site on supinator crest from which the tubercle (arrow) and LCL insertion had been avulsed. (**C**) Sutures from anchor placed around avulsed tubercle (arrow) and LCL insertion. (**D**) Bony LCL insertion avulsion repair and arthrotomy closure. By permission of Mayo Foundation for Medical Education and Research. All rights reserved. *LCL*, lateral collateral ligament.
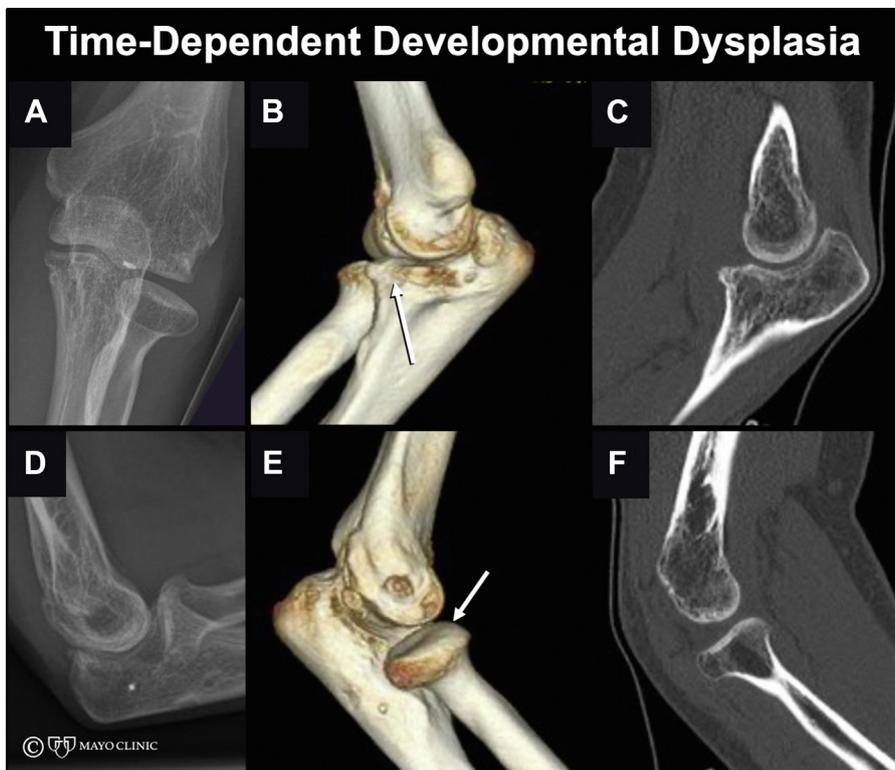

Figure 6 Time dependent developmental dysplasia refers to developmental changes that continue to evolve over time prior to completion of skeletal growth. This is an example of a 20-year-old female with recurrent dislocations. (**A**) AP radiograph reveals radiocapitellar dysplasia and a poorly defined subchondral bone lying on the coronoid, suggesting coronoid dysplasia. (**B** and **C**) Coronoid dysplasia is best appreciated on the 3D CT and 2D sagittal reconstructions. The 3D reconstruction reveals absence of the anteromedial coronoid facet (**B** arrow). (**D-F**) Radiocapitellar dysplasia, with resultant joint incongruity, is suggested on the lateral radiograph (**D**) and confirmed on the 3D and 2D sagittal reconstructions. Chronic posterior subluxation alters the radiocapitellar contact mechanics thereby disturbing proper development of the rim of the radial head. The anterior rim develops a rounded off shape (**E** arrow), which makes the articular dish of the radial head shallow and diminishes its concavity-compression contribution to elbow stability. By permission of Mayo Foundation for Medical Education and Research. All rights reserved. *AP*, anteroposterior; *3D*, 3-dimensional; *CT*, computed tomography; *2D*, 2-dimensional.
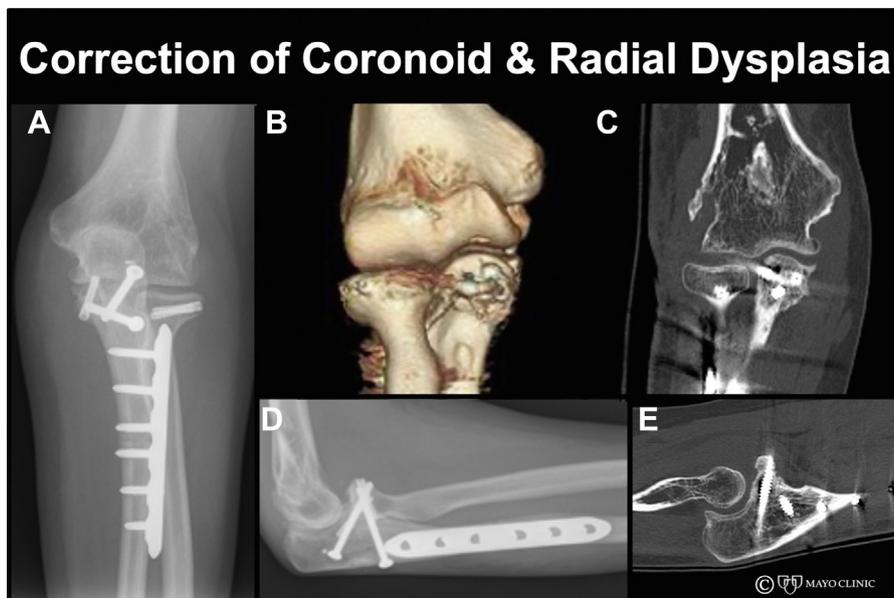
Figure 7 Radiographs and CT reconstructions obtained 4 months postoperatively on same patient in Figure 6. (**A** and **D**) Radiocapitellar congruency was restored by a combination of opening wedge partial radial neck osteotomy to deepen the articular dish of the radial head and opening wedge ulnar osteotomy to translate the radial head anteriorly. (**B**, **C**, and **E**) Coronoid reconstruction was performed with a partial radial head allograft fixed with 2 screws from posterior to anterior and one headless screw from anteromedial posterior. By permission of Mayo Foundation for Medical Education and Research. All rights reserved. *CT*, computed tomography.
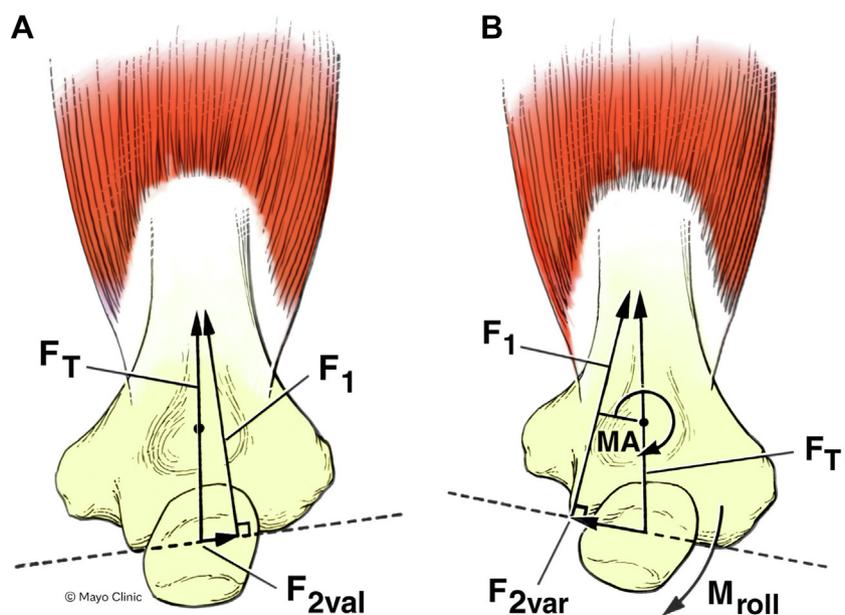

Figure 8 (**A**) In a normal elbow, with slight valgus carrying angle, the triceps force vector can be resolved into 2 perpendicular vectors. F_1 is perpendicular to the joint surface, which it compresses and stabilizes. F_{2val} create a slight valgus moment. (**B**) In patients with cubitus varus due to pediatric supracondylar malunion, the altered triceps force vector can be resolved into 2 force vectors: F_1, which is perpendicular to the joint surface, and F_{2var}, which is directed medially. This abnormal medial force vector causes external rotation of the ulna about its long axis. The offset between F_1 and the long axis of the humerus (due to the deformity at the supracondylar level) causes a moment arm through which external rotation and varus deforming torques occur with triceps contraction. By permission of Mayo Foundation for Medical Education and Research. All rights reserved. F_1, force one; F_{2val}, force two valgus; F_{2var}, force two varus; F_T, triceps force vector; $F_{T'}$, altered triceps force vector; M_{roll}, long axis; *MA*, moment arm.

Excessive lateral condyle stripping

The entire lateral soft tissue complex (tendons, capsule) contributes to elbow stability. Ideal exposure involves splitting the common extensor origin in Kaplan's interval and detaching only a few millimeters anterior to that. Further detachment of the common extensor tendon, anconeus tendon, or capsule compromises the existing constraints that would otherwise protect the LCL reconstruction and, therefore, should be avoided.

Conclusion

The prevalence and causes for failure of LCL reconstruction are not well documented in the literature. This report is an attempt to
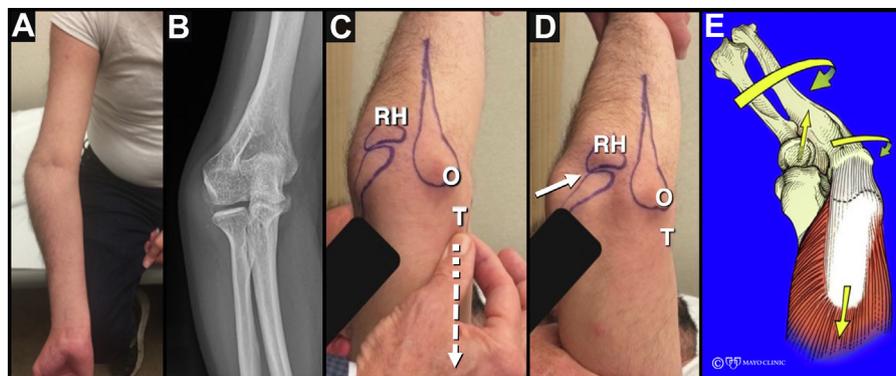
Figure 9 (**A**) Cubitus varus with (**B**) developmental dysplasia (**B**) due to long-standing deforming forces following pediatric supracondylar malunion. (**C**) Paradoxical "active" PLRI using a Transcutaneous Electrical Nerve Stimulation unit. The dysplasia from the distal humeral varus malunion causes the olecranon and radial head to rotate medially. This displacement, combined with medial elongation of the olecranon, and hypertrophy of the medial triceps tendon displaces the medial triceps medial to the joint line. Elbow extension against resistance creates an unbalanced triceps pull on the medial olecranon (dashed arrow) resulting in posterolateral rotatory subluxation, as evidenced by the dimple (**D**, solid arrow) created through the suction effect when the radial head subluxates posterolaterally. (**E**) Schematic of deforming torsional forces mechanism. With active triceps contraction (straight arrow pointing down), while extension is being resisted, deforming forces and moments cause a medial pull and external rotation torsion on the ulna about its long axis (smaller curved arrow). This not only rotates the ulna into external rotation but also causes the radial head to rotate posterolaterally off the capitellum (straight arrow pointing up). Together, they represent the initial kinematic displacement of posterolateral rotatory subluxation (larger curved arrow). Over time, these chronic forces cause attenuation of the LCL complex, including the ulnar part, leading to frank posterolateral rotatory subluxation. By permission of Mayo Foundation for Medical Education and Research. All rights reserved. *PLRI*, posterolateral rotatory subluxation; *TENS*, transcutaneous Electrical Nerve Stimulation; *O*, olecranon; *RH*, radial head; *T*, triceps; *LCL*, lateral collateral ligament.
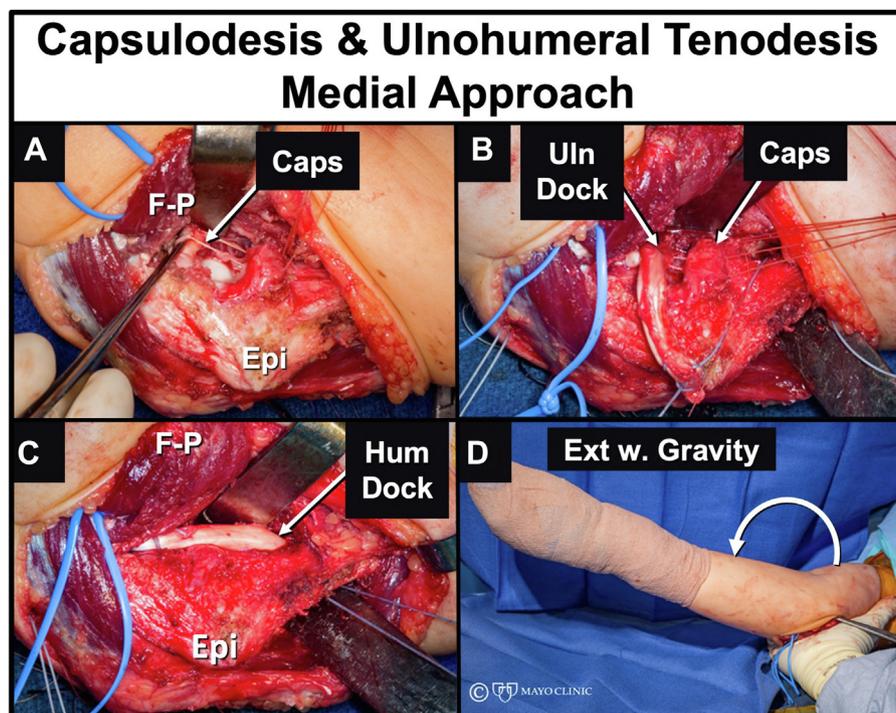

Figure 10 Anterior capsulodesis and tenodesis through a medial approach. (**A**) The flexor pronator origin is reflected from the medial epicondyle; a transverse incision is made across the anterior capsule and sutures placed for a "vest over pants" imbrication but not tied. (**B**) The allograft tendon graft is docked into a tunnel in the coronoid (Uln Dock) then the capsulotomy sutures tied, (**C**) and the tendon docked into a tunnel in the humerus (Hum Dock). (**D**) Length is decided based on the desired degree of intraoperative extension block with gravity alone (curved arrow, Ext w. Gravity). By permission of Mayo Foundation for Medical Education and Research. All rights reserved. *F-P*, flexor pronator origin; *Epi*, epicondyle; *Caps*, capsule; *Ulnar Dock*, tendon docked into a tunnel in the ulna; *Hum Dock*, tendon docked into a tunnel in the humerus; *Ext*, extensor tendon.
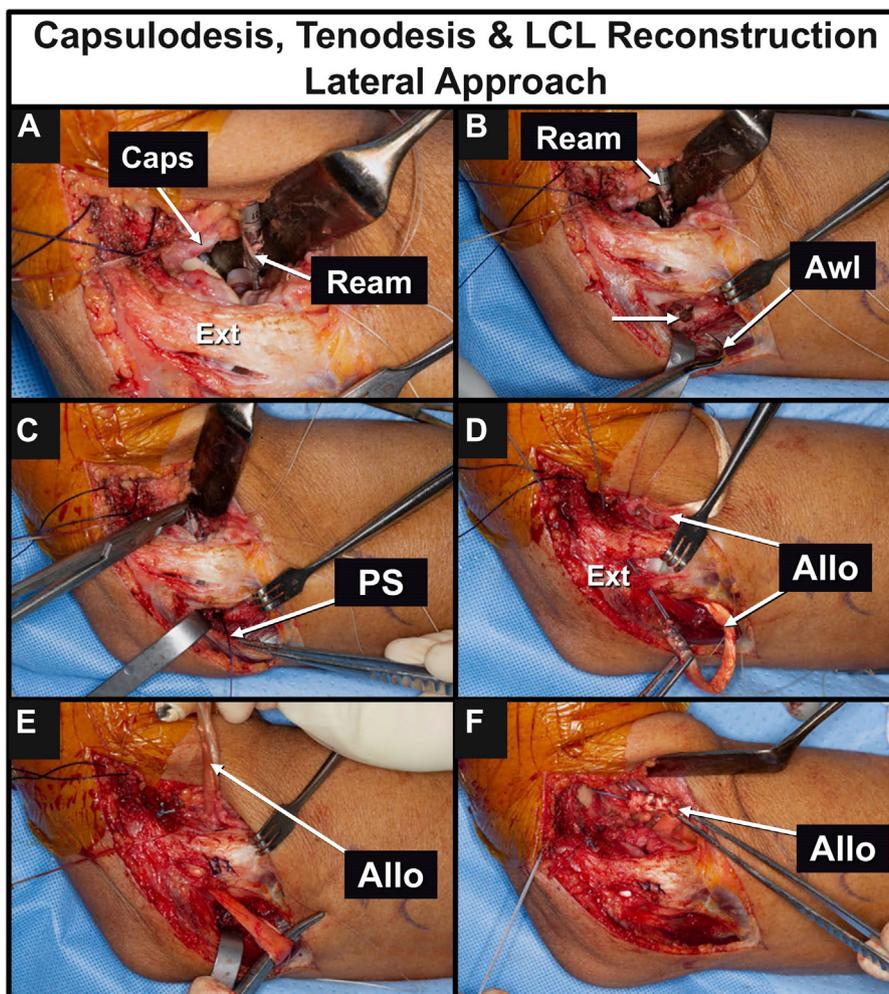
Figure 11 Combined anterior capsulodesis/tenodesis and LCL reconstruction through a lateral Kaplan/Anconeus approach above and below the common extensor tendon. (**A**) After placing sutures for a "vest over pants" imbrication of the anterior capsule, a 5 mm docking tunnel is created in the coronoid using a flexible reamer over a guide pin. (**B**) A 5 mm hole is drilled in the lateral ulnar cortex just posterior to the tubercle on the supinator crest (arrow). A Bankart awl is used to create a curve tunnel from this lateral cortical hole into the tunnel previously created in the coronoid. The reamer is seen still in the tip of the coronoid. (**C**) A passing suture is passed through the ulnar tunnel. (**D**) The graft (Allo) is first passed antegrade (solid arrow) down through the tip of the coronoid and retrieved from the lateral ulna (dashed arrow) just below the tubercle on the supinator crest, then passed through a small sagittal split created in the common extensor tendon origin and docked into the lateral humeral condyle as one would do for LCL reconstruction. (**E**) After docking the tendon in the lateral condyle, it is prestressed. (**F**) The graft is cut at the appropriate length for docking into a 5 mm tunnel in the anterior humerus just above the capsular origin. Sutures are brought out through 2 mm holes in the posterior humeral cortex. The anterior capsulodesis sutures are tied and the tendon graft docked in the humerus. The anterior capsulodesis sutures are tied and the tendon graft is docked in the humerus at the desired degree of intraoperative extension block with gravity alone. By permission of Mayo Foundation for Medical Education and Research. All rights reserved. *Ext*, extensor tendon; *Caps*, capsule; *Ream*, reamer; *Awl*, Bankart awl; *PS*, passing suture; *Allo*, allograft; *LCL*, lateral collateral ligament.

provide a systematic approach to identifying and managing the preoperative and operative risk factors. Further studies are warranted to determine the indications for, and success rates of surgical intervention in managing these risk factors.

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