Posterolateral rotatory instability is the most common type of symptomatic chronic instability of the elbow. In this condition the forearm complex rotates externally in relation to the humerus, causing posterior subluxation or dislocation of the radial head. The lateral ligament complex, radial head and coronoid process are important constraints to posterolateral rotatory instability, and their disruption is involved in the pathogenesis of this condition. The diagnosis relies on a high index of clinical suspicion, active and passive apprehension tests, and examination under anaesthesia. Surgical treatment has given consistently successful results. Open reconstruction of the lateral ligaments with a tendon graft has been the procedure of choice, with arthroscopic techniques emerging as a potential alternative.
Disruption of the lateral ligament complex may be due to trauma, to chronic attentuation or be iatrogenic. Post-traumatic disruption is the most common, as this is the first ligament to be damaged in posterolateral subluxation or dislocation of the elbow\textsuperscript{15} and is usually avulsed from its distal humeral origin.\textsuperscript{16} In traumatic posterolateral dislocation, soft-tissue disruption occurs in a circular fashion from lateral to medial (Horii circle, Stage I to III\textsuperscript{17}). In stage I injuries, the lateral ligament complex is disrupted, in stage II the anterior and posterior capsule is torn, and in stage III the medial collateral ligament (MCL) also ruptures.\textsuperscript{18}

Iatrogenic injury may follow open or arthroscopic release of the lateral epicondyle,\textsuperscript{18} or surgical approaches to the lateral side of the elbow joint and the head of the radius.\textsuperscript{19} The Kocher approach\textsuperscript{5} utilises the interval between anconeus and extensor carpi ulnaris, incising the more anterior part of the lateral ligament complex. The Wrightington approach to the radial head, which has recently been described, lifts off anconeus from the ulna with an osteotomy of the ulnar attachment of the lateral ligament complex, which is subsequently fixed back to its origin.\textsuperscript{20} This avoids incising the substance of the lateral ligament complex and allows it to be re-tensioned when it is re-attached at the osteotomy site, which could potentially minimise any posterolateral laxity. Posterolateral rotatory instability has been reported after treatment of lateral epicondylitis by serial steroid injections.\textsuperscript{21} Whether rupture of the lateral ligament complex was iatrogenic due to administration of steroid or the result of the degenerative process involving the common extensor origin was difficult to determine.

Chronic attentuation of the lateral ligament complex may occur in long-standing cubitus varus.\textsuperscript{22,23} In such chronic conditions the ligament stretches, losing its normal tension. In addition, the direction of pull of triceps is altered so that it exerts an external rotatory moment on the ulna, an important component of posterolateral rotatory instability.\textsuperscript{23} Beuerlein et al\textsuperscript{24} showed in cadaver specimens that 20° of cubitus varus causes ulnohumeral widening indicative of posterolateral rotatory instability. However, the development of clinical posterolateral rotatory instability depends not only on the absolute amount of varus deformity but also on its duration and the levels of the patient's activity. This would explain the development of symptomatic posterolateral rotatory instability in angles of cubitus varus smaller than those predicted biomechanically.\textsuperscript{23} Chronic attentuation of the lateral ligament complex may also be secondary to overuse, such as in patients with polio-myelitis who use crutches to walk. Finally, the lateral ligament complex may be inherently lax in conditions of generalised ligamentous hyperlaxity.

**Radial head and capitellum**

The radial head is a significant constraint to posterolateral rotatory instability, contributing to stability by providing osseous congruency and tensioning the lateral ligament complex. Its excision may slacken and de-function the lateral ligament complex. Following excision of the radial head in human cadavers, Jensen et al\textsuperscript{25} found a 7.1° mean increase in external rotatory laxity of the forearm relative to the humerus. Schneebberger, Sadowski and Jacob\textsuperscript{26} reported doubling of the external rotatory laxity despite intact medial and lateral ligaments. These studies are supported by the development of posterolateral rotatory instability in patients in whom the radial head was excised following a comminuted fracture.\textsuperscript{27} In the absence of the head the proximal radial stump subluxes posteriorly giving rise to symptoms of posterolateral rotatory instability.
This potential complication must be considered when dealing with fractures of the radial head, and may be avoided by arthroplasty rather than simple excision, even in the presence of an intact medial collateral ligament.

The capitellum has not been extensively investigated in the pathogenesis of posterolateral rotatory instability. However, loss of capitellar height would be expected to slacken the lateral ligament complex, causing posterolateral rotatory instability. Additionally, changes in the capitellar structure can alter the congruency of the radiocapitellar articulation, an important component of stability. Such changes may be due to a fracture, or to osteochondritis dissecans. To avoid such complications every attempt should be made to fix capitellar fractures, with excision of the fragments used as a last resort.

**Coronoid process**

The coronoid process is increasingly recognised as an important elbow stabiliser, acting as a constraint to posterior ulnohumeral displacement on axial and varus loadings. Coronoid deficiency is most commonly post-traumatic. Regan and Morrey classified coronoid fractures according to the proportion of the coronoid process involved. Stage I fractures involve the tip of the coronoid, stage II up to 50%, and stage III more than 50% of the height of the coronoid. In a biomechanical cadaver study, isolated stage I fractures did not increase posterolateral laxity, but stage II fractures with 50% reduction in coronoid height resulted in a 28% increase in external rotation of the forearm relative to the humerus. When combined with excision of the radial head, loss of 30% of coronoid height fully destabilised cadaver elbows, leading to ulnohumeral dislocation even in the presence of intact ligaments. Furthermore, in such combined injuries, when 50% of the coronoid height was lost, isolated prosthetic replacement of the radial head did not fully restore elbow stability. These laboratory observations are supported clinically by patients presenting with chronic posterolateral rotatory instability following an ununited coronoid fracture. In these cases, the coronoid process should be fixed with excision of the fragments as a last resort.

### Table 1. Clinical tests for posterolateral rotatory instability of the elbow

<table>
<thead>
<tr>
<th>Test</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lateral pivot-shift test¹</td>
<td>Patient supine, affected limb overhead. With forearm supinated, valgus and axial loading applied, elbow is flexed from full extension. In posterolateral rotatory instability as the elbow is flexed the radial head subluxes/dislocates, seen as an osseous prominence posterolaterally. With flexion beyond 40° the radial head suddenly reduces with a palpable and visible clunk. The test may also be done starting with the elbow flexed and then extending, reversing the above sequence. The test is best done under general anaesthesia for radial head dislocation and relocation to be seen.</td>
</tr>
<tr>
<td>Lateral pivot-shift apprehension test¹</td>
<td>The above manoeuvre is performed with the patient awake. The test is positive if apprehension occurs.</td>
</tr>
<tr>
<td>Posterolateral rotatory drawer test¹ seventeenth</td>
<td>Patient supine, affected limb overhead, elbow flexed 40°. Anteroposterior force is applied to the radius and ulna with the forearm in external rotation. This aims to sublux the forearm away from the humerus on the lateral side, pivoting on the intact medial ligaments. Under general anaesthesia the radial head is seen dislocating, whereas with the patient awake apprehension occurs.</td>
</tr>
<tr>
<td>Table-top relocation test²³</td>
<td>Patient performs a press-up on the edge of a table using one arm, with the forearm in supination. In the presence of instability, apprehension occurs at about 40° flexion. The manoeuvre is repeated while the examiner’s thumb presses on the radial head, preventing subluxation. The test is positive if thumb pressure relieves apprehension.</td>
</tr>
<tr>
<td>Active floor push-up sign³⁶</td>
<td>Patient pushes off the floor with elbows flexed 90°, forearms supinated and arms abducted. The test is positive if apprehension or radial head dislocation occurs as the elbow is extended.</td>
</tr>
<tr>
<td>Chair sign³⁶</td>
<td>Patient seated with elbows flexed 90°, forearms supinated and arms abducted. Patient tries to rise from the chair pushing down only with the arms. The test is positive if apprehension or radial head dislocation occurs with elbow extension.</td>
</tr>
</tbody>
</table>

---

![Fig. 2](image-url)

Photograph showing the lateral pivot-shift test. Application of external rotation, valgus and axial loading to the elbow causes apprehension or radial head subluxation/dislocation.
patients stability has been restored by bone grafting and reconstruction of the coronoid. Therefore, in dealing with acute coronoid fractures consideration must be given to the possibility of posterolateral rotatory instability and the need for surgical fixation.

Common extensor origin
The common extensor muscle mass and its strong intermuscular septa cross the lateral side of the elbow and are important static secondary constraints to posterolateral rotatory instability. This is in addition to any possible dynamic effect. Disruption of the common extensor mass has been reported in 66% of elbow dislocations undergoing surgery. Extensive release of the extensor origin in surgical treatment of lateral epicondylitis should be avoided as this may compromise elbow stability, especially if the underlying lateral ligament complex is attenuated.

Clinical presentation, investigations and diagnosis
Patients may present with a spectrum ranging from vague symptoms in the elbow to frank recurrent posterolateral dislocation. Lateral elbow pain, clicking, popping and snapping are not uncommon. Symptoms are often brought on by activities such as pushing up from a chair or doing press-ups, which place the elbow in an unstable position of external rotation of the forearm with valgus and axial loading of the elbow. A previous history of trauma or surgery on the lateral side of the elbow should be sought.

Clinical examination aims to identify signs of previous trauma or surgery, ulnohumeral alignment and range of movement. Several clinical tests for posterolateral rotatory instability have been described (Table I, Figs 2 to 5). All these place the elbow in a position of maximal instability, with a combination of external rotation of the forearm, valgus and axial loading, which try to reproduce either the symptoms or displacement of the radial head. It is important to look for coexistent valgus or varus instability as well as generalised ligamentous hyperlaxity.

Further investigations can contribute to the diagnosis, though their value is often limited. Plain radiography may show an avulsion fracture of the origin or insertion of the lateral ligament complex. It can also demonstrate the integrity of the radial head, coronoid process and capitellum, and the presence of degenerative changes. Impression fractures of the radial head or the posterior part of the capitellum, analogous to the Hill-Sachs lesion of dislocation of the shoulder, may be seen. The drop sign (an ulnohumeral distance > 4 mm on the plain lateral film of the unstressed elbow) can be indicative of residual instability following elbow dislocation. Lateral screening of the elbow during the pivot shift test may show posterior displacement of the radial head and ulnohumeral widening. The value of MRI in the diagnosis of posterolateral rotatory instability remains controversial, although Potter et al found 100% correlation between abnormal MRI findings of the lateral ligaments and clinical posterolateral rotatory instability, this has not, however, been confirmed by others who attempted to describe the lateral ulnar collateral ligament in normal and injured elbows. This may be partly because these studies tried to identify a distinct, prominent lateral
ulnar-collateral band, which anatomical studies increasingly suggest may not exist. Finally, arthroscopic examinations of the elbow may show posterior displacement of the radial head, an elongated lateral ligament complex or widening of the lateral joint space.

The diagnosis of posterolateral rotatory instability remains a clinical one with a combination of the history, active and passive apprehension tests, and examination of the elbow under anaesthesia. Apprehension is all that can be elicited because the patient is usually awake and general anaesthesia is needed to demonstrate displacement of the radial head. With the patient under general anaesthesia we initially examine for valgus and varus instability in 30° of elbow flexion to unlock the olecranon fossa. Testing for valgus instability with the forearm in supination may give a false positive result in the presence of posterolateral rotatory instability, and so we also test this with the forearm in pronation. Inability to demonstrate varus instability does not imply that the lateral ligaments are intact, as the ulnohumeral articulation is the main constraint to varus. We test for posterolateral rotatory instability using the pivot shift and posterolateral drawer tests. If these fail to demonstrate instability we screen the elbow using the image intensifier while performing the pivot-shift test.

**Management**

Management of chronic posterolateral rotatory instability of the elbow depends on the severity of the patient's symptoms. Avoiding provocative manoeuvres and bracing to limit supination and valgus loading may suffice in some, but in significantly-affected patients surgery is preferred.

Surgical management aims at re-attaching, retensioning or reconstructing the lateral ligament complex, dealing with bone deficiency of the radio capitellar and ulnohumeral articulation by replacement of the radial head or coronoid reconstruction, and correcting any varus deformity of the humerus by osteotomy. One or all of these may be necessary, depending on the underlying pathology.

Reattachment and retensioning of the lateral ligament complex with imbrication and advancement have been
used, but reconstruction with a graft may provide a more stable construct. Current techniques\textsuperscript{19,43-48} repair that part of the lateral ligament complex which passes from the lateral humeral epicondyle to the supinator crest of the ulna. A tendon graft is used, with fixation achieved by bone tunnels, anchor sutures or interference screws. Although an autograft of palmaris longus is commonly employed, the use of triceps fascia, semitendinosus, gracilis, plantaris, and synthetic devices has been reported.\textsuperscript{43,48}

In performing tendon reconstruction using bone tunnels, two drill holes are made at the ulnar supinator crest, connected by a tunnel. A large suture is passed through this tunnel and its free ends are held with a heavy artery clip on the distal humerus, where the site of entry for the graft is then drilled. Two additional humeral holes are drilled, and all three are connected by tunnels. The graft passes through the ulnar tunnel, through the isometric point of the humerus, through the humeral tunnels, and is sewn back to itself (Fig. 6), with the elbow held in 30˚ to 40˚ of flexion and full pronation.\textsuperscript{17}

Arthroscopically assisted repair of the avulsed lateral ligament complex has been described.\textsuperscript{49} Similarly, the use of arthroscopic electrothermal shrinkage of the lateral ligament complex as the sole treatment for postero-lateral elbow instability has been reported.\textsuperscript{50}

### Results of surgery

A review of the literature reveals a satisfactory outcome in most patients treated surgically, with resolution of instability, improvement of pain and maintenance of joint movement. The outcome is less good in those with degenerative changes or generalised ligamentous hyperlaxity. We identified six studies looking at isolated ligamentous repair or reconstruction in the treatment of chronic posterolateral rotatory instability (Table II).\textsuperscript{19,43-47} A series from the Mayo clinic\textsuperscript{43} described 12 patients treated by lateral ligament complex repair with humeral reattachment or imbrication and 32 by reconstruction; five had persistent instability, three treated by repair and two by reconstruction. Surgery led to relief of pain and improvement or maintenance of movement in most. The mean post-operative Mayo elbow performance score was 85; 17 patients were rated excellent, 17 good and ten fair. The results of ligament reconstruction were better than those for repair. Lee and Teo\textsuperscript{44} reported ten patients with posterolateral rotatory instability, four having lateral ligament imbrication and advancement and six tendon graft reconstruction. Post-operatively, all had a negative pivot shift test, reported no or mild pain, and retained functional movement. Olsen and Søjbjerg\textsuperscript{45} used a triceps tendon graft to reconstruct the lateral ligament complex in 18 patients. Post-operatively, 14 had a stable elbow but four continued to have apprehension on the pivot-shift test. Loss of movement of 10˚ to 15˚ was noted in only three; 13 patients had no or only occasional residual pain, and five reported moderate pain. The mean post-operative Mayo elbow score was 92. DeLaMora and Hausman\textsuperscript{19} recorded five patients treated by ligamentous reconstruction using a distally based triceps fascia strip. The pivot shift test became negative in all and the mean post-operative range of movement was 10˚ to 135˚. Eygendaal\textsuperscript{46} described 12 cases treated by ligament reconstruction using triceps tendon fixed with interference screws. All but one had a stable elbow and pain improved in most; six patients lost 5˚ to 10˚ of extension, but none lost flexion. Rizio\textsuperscript{47} performed ligament reconstruction in a skeletally immature 11-year-old with post-traumatic recurrent elbow dislocation. A fascia lata allograft was fixed using bone tunnels. As the bone tunnels were proximal to the humeral epiphysis and distal to the olecranon epiphysis no growth disturbance was seen. At follow-up there was no further instability and a full range of movement. The Sheffield Elbow Unit recently presented 11 patients treated by reconstruction of the lateral ligament in eight and of the medial in three for chronic postero-lateral rotatory instability or valgus instability.\textsuperscript{48} At a mean follow-up of three years only one patient, with generalised ligamentous hyperlaxity due to the Ehlers-Danlos syndrome, was still unstable.\textsuperscript{48}

Published techniques\textsuperscript{43,48} of ligament reconstruction report successful results in most patients, even though they only attempt to reconstruct that part of the lateral ligament complex passing from the lateral epicondyle to the supinator crest. Given our current understanding of the complex

### Table II. Studies reporting the outcome of isolated lateral ligament reconstruction for posterolateral rotatory instability of the elbow

<table>
<thead>
<tr>
<th>Study</th>
<th>Number of cases</th>
<th>Graft</th>
<th>Fixation type</th>
<th>Mean follow-up in mths (range)</th>
<th>Cases with persistent instability without further injury</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sanchez-Sotelo et al\textsuperscript{43}</td>
<td>32</td>
<td>Various autografts and allografts</td>
<td>Bone tunnels</td>
<td>69.6 (24 to 182.4)</td>
<td>2</td>
</tr>
<tr>
<td>Olsen and Søjbjerg\textsuperscript{45}</td>
<td>18</td>
<td>Triceps fascia</td>
<td>Humeral bone tunnels, ulnar suture anchors</td>
<td>44 (14 to 88)</td>
<td>4</td>
</tr>
<tr>
<td>Eygendaal\textsuperscript{46}</td>
<td>12</td>
<td>Triceps tendon</td>
<td>Interference screws</td>
<td>23 (17 to 48)</td>
<td>1</td>
</tr>
<tr>
<td>Lee and Teo\textsuperscript{44}</td>
<td>6</td>
<td>Palmaris longus, semitendinosus</td>
<td>Bone tunnels</td>
<td>27.8 (9 to 52)</td>
<td>0</td>
</tr>
<tr>
<td>DeLaMora and Hausman\textsuperscript{19}</td>
<td>5</td>
<td>Distally-based triceps fascia</td>
<td>Humeral bone tunnels</td>
<td>Not available</td>
<td>0</td>
</tr>
<tr>
<td>Rizio\textsuperscript{47}</td>
<td>1</td>
<td>Fascia lata</td>
<td>Bone tunnels</td>
<td>34</td>
<td>0</td>
</tr>
</tbody>
</table>
functional structure of the lateral ligament complex, it will be interesting to see whether more anatomical techniques of reconstruction, replacing the whole of the lateral ligament complex rather than simply its more posterior part, can further improve the results.

Arthroscopic treatment of posterolateral rotatory instability has been reported. Spanh et al described 21 patients treated with arthroscopic thermal ligament shrinkage at a mean follow-up of 2.5 years. The positive pivot-shift test became negative after surgery, and the mean Mayo elbow performance score increased from 44 to 77. There were no recurrent dislocations of the elbow or complications of thermal ablation.

The results of bony surgery in isolation or combined with ligament reconstruction are also promising. O’Driscoll et al reported 22 cases of posterolateral rotatory instability secondary to post-traumatic or congenital cubitus varus. Tardy instability developed 20 to 30 years after the deformity. In seven cases ligament reconstruction and osteotomy were performed, in ten ligament reconstruction alone, in four osteotomy alone, and in one total elbow arthroplasty. At a mean follow-up of three years, only three of the 22 patients had persistent instability. Movement was maintained or improved in all. The mean post-operative Mayo elbow score was 87. Hall and McKee reported seven patients who developed posterolateral rotatory instability following excision of the radial head for comminuted fractures. In three, replacement of the radial head and reconstruction of the lateral ligament complex was performed and their symptoms improved, the mean Mayo elbow score rising from 55 to 81.5. Okazaki et al reported three patients with chronic posterolateral rotatory instability that was attributed at least partly to post-traumatic coronoid deficiency; two had nonunion of a coronoid fracture and one a flattened trochlear notch. Coronoid reconstruction with an iliac crest or humeral bone autograft helped restore stability.

Conclusion
Disruption of the lateral ligament complex, radial head and coronoid plays an important role in the pathogenesis of posterolateral rotatory instability of the elbow. The diagnosis of this condition remains clinical, with examination under anaesthesia being useful. Open surgical reconstruction of the lateral ligaments restores stability in most patients, with arthroscopic techniques emerging as a potential alternative. Bony procedures may be needed in those with a varus deformity of the humerus or deficiency of the radial head or coronoid. Understanding the complex structure of the lateral stabilisers of the elbow and respecting their integrity during surgery may help prevent this condition in some patients.

References