Current perspectives on elbow dislocation and instability

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Abstract: Obtaining a successful outcome following a significant elbow injury can be challenging for even the most experienced specialist. What is clear is that suboptimal management has a high chance of a disappointing outcome with pain, stiffness, instability, and loss of function all commonly seen. This review will discuss the pathoanatomy and management principles of both simple and fracture dislocations of the elbow as well as resulting chronic instability. Emphasis is placed on the concept of prompt assessment, early restoration of stability by appropriate means, and immediate mobilisation to optimise outcomes.

Keywords: Elbow dislocation; fracture; elbow instability

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Introduction

The elbow is the second most commonly dislocated joint in adults. Simple dislocations have been described as those where there is no concomitant fracture other than small periarticular avulsions under 2mm in diameter. Where larger fragments are present it is classified as a fracture dislocation (1,2). The annual incidence of simple elbow dislocations is approximately 5.21 per 100,000 persons, slightly more frequent than fracture dislocations with a 53% male predominance (1). Falling from a standing height is the most common mechanism of injury (56%) while sporting activities accounted for 44% of elbow dislocation (3).

Anatomy

The elbow joint is comprised of bony, capsuloligamentous, and neuromuscular structures (Table 1).

Table 1 Elbow primary and secondary stabilizers

<table>
<thead>
<tr>
<th>Static constraints</th>
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<tbody>
<tr>
<td>Primary constraints</td>
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<tr>
<td>Unlnohumeral articulation</td>
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<tr>
<td>Anterior MCL</td>
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<td>Lateral collateral ligament complex (LLC)</td>
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<td>Secondary constraints</td>
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<tr>
<td>Radio humeral articulation</td>
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<td>Common Extensor group</td>
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<td>Common Flexor–pronator group</td>
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<tr>
<th>Dynamic stabilisers</th>
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<tbody>
<tr>
<td>Biceps</td>
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<tr>
<td>Triceps</td>
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<tr>
<td>Brachialis</td>
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<td>Anconeus</td>
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Bony structures

Medially the ulnohumeral articulation consists of the trochlea and its reciprocating greater sigmoid cavity. The olecranon forms the posterior and central sections of the cavity, accommodating the triceps insertion at its proximal aspect and continuing as the ulna diaphysis distally. The coronoid process is a fan shaped structure with 2 discrete articular facets, anteromedial and anterolateral, which are separated by a ridge that runs the length of the greater sigmoid notch. The sublime tubercle is an important element of the medial side of the coronoid where the strong anterior bundle of the medial collateral ligament inserts. Laterally the radial head forms a shallow concave surface that only covers the capitellum over an arc of 90 degrees and so the lateral compartment has greater reliance on soft tissue restraints (4-6).

Capsuloligamentous structures

The elbow is bordered by medial and lateral collateral ligament complexes (LLC). The medial collateral ligament complex (MCLC) consists of anterior (aMCL), posterior (pMCL), and transverse components. The anterior bundle originates from the antero-inferior surface of the medial epicondyle of the humerus and inserts onto the sublime tubercle (7). The posterior bundle plays a greater role in stability in the context of associated injury (8,9).

The LLC consists of the radial collateral ligament (RCL), lateral ulnar collateral ligament (LUCL), accessory lateral ulna collateral ligament (AUCL), the annular ligament (AL), and the posterolateral ligament (PL) (10). The AL encircles the radial head with attachments to the anterior and posterior margins of the lesser sigmoid notch. The RCL, LUCL, and AUCL have a common origin at the inferior surface of the lateral epicondyle at the centre of the axis of rotation. The PL arises more posteriorly. The LUCL inserts on the crista supinatoris, the RCL inserts onto the annular ligament, and the less substantial accessory collateral ligament attaches to both (10-12). The PL inserts to the margin of the greater sigmoid notch proximal to the supinator crest.

Neuromuscular structures

The biceps, brachialis, triceps, and anconeus muscles cross the elbow and provide proprioceptive feedback as well as stability through dynamic compression across the articular surfaces. The anterior capsule inserts approximately 5mm distal to the joint line, whilst the strong and broad insertion of brachialis muscle lies just distal to this reinforcing the coronoid process (Table 1). The resultant moment of the triceps, biceps, brachialis and common extensor, and flexor-pronator mass pulls the forearm bones proximally and posteriorly on to the end of the humerus (11,13).

Elbow stability

Elbow stability is derived from a combination of the osseous, capsuloligamentous, and neuromuscular structures described above. These are traditionally categorised into primary and secondary stabilisers according to their relative contributions, and static constraints or dynamic restraints according to their mechanism of action (Table 1).

Primary stabilisers

The primary static constraints include the bony ulnohumeral articulation, the LLC, and aMCL. The contrasting levels of joint congruity between the medial and lateral bony articulations explain the greater importance of the lateral soft tissue structures.

Medially, the highly congruous relationship of the trochlea and greater sigmoid cavity has been said to contribute up to 50% of elbow stability. The coronoid and olecranon together enclose the trochlea through an arc of 170 degrees producing a high degree of concavity compression. The anteromedial coronoid facet has a mean surface area of 232 mm$^2$, compared to a mean radial head surface area of 247 mm$^2$, making the anteromedial facet a vital primary varus stabiliser. The anterolateral facet has a smaller mean surface area of 142 mm$^2$ and is a second-degree valgus stabiliser acting in conjunction with the radial head (13-16).

The MCLC provides stability in valgus and posteromedial direction with the anterior bundle being the most important contributor to valgus stability throughout flexion arc. The pMCL functions primarily in providing posteromedial rotatory stability (17-19) (Table 2).

The LLC is the primary stabiliser to external rotation and varus stress. The posterolateral ligament has recently been shown to be of significant importance to posterior stability of the radial head (10). Therefore, LLC insufficiency not only causes varus and posterolateral instability but also subluxation of the radial head posteriorly (16,20) (Table 2).
Secondary stabilisers

The secondary stabilisers, which take on an important role in the context of injury to the primary stabilisers, include the radial head, the anterior capsule, and the musculotendinous structures crossing the elbow joint which have a combination of passive and active stabilising effects.

Forearm rotation has a significant impact on valgus-varus laxity with maximum laxity noted in neutral forearm rotation throughout the elbow flexion (21). The stabilising effect of biceps, brachialis, or triceps is independent of forearm rotation (17). The role of the common flexors as valgus stabilisers has been demonstrated in a cadaveric study evaluating the throwing action. The maximal valgus force generated during throwing is higher than the load to failure of the MCL illustrating the need for secondary valgus stabilisers to maintain stability. Stimulation of the FCU and FDS is able to restore stability in a MCL deficient cadaveric model (22). The muscles from the common extensor origin (CEO) have maximal tension in full pronation where they function most effectively as a varus stabiliser (17). Anconeus is assumed to act as a dynamic constraint to both varus and posterolateral instability (23-25). It follows that forearm rotation may play a role in stabilising the elbow joint in the presence of injury. Supination stabilises the MCL deficient elbow and those in which there is a large coronoid fracture, whereas pronation stabilises the LCL deficient elbow (26-28). This is supported by the clinical work of Josefsson et al. who identified disruption of the common flexor and extensor origins as an important determinant of instability after simple elbow dislocation (29,30).

Mechanism and pathoanatomy (Table 3)

The mechanisms of injury for simple and fracture dislocation of the elbow are determined by injury factors including the rate of application and direction of force, and patient factors including bone quality.

Simple elbow dislocations

Simple elbow dislocations can be grouped into posterolateral, posterior, posteromedial, divergent (paediatric injuries), and anterior dislocations (extremely uncommon without fractures). The most common direction of elbow dislocation is posterolateral. The exact mechanism remains the subject of debate.

MRI studies support a valgus hyperextension model for the more common posterolateral dislocations (31-33). The valgus hyperextension theory proposes that dislocations occur following a fall onto a hyperextended elbow. As elbow goes into hyperextension and valgus, the MCL fails first followed by the LLC, and then the common flexor origin.
(CFO). Further displacement leads to disruption of anterior capsule (AC) followed by CEO. Posterior capsule (PC) tear though rare is seen only in case with complete CEO disruption (13,31).

Schreiber et al. (34), in their study on video analysis of elbow dislocations, showed that most elbow dislocations occur as a result of hyperphysiologic valgus moment in an extended elbow with the typical arm position at the time of dislocation being forearm pronation, elbow extension, and shoulder abduction with forward flexion. This may result in a spectrum of injury (Figure 1) from grade 1 isolated medial ligament tear, grade two medial and lateral ligament, grade 3 ligamentous and CFO avulsion, and grade 4 (Figure 2) where all soft tissue stabilisers have been avulsed including the common extensor origin (31).

Posteromedial dislocations account for approximately 10% of all elbow dislocations and may follow a valgus external rotation model. Posteromedial dislocation are associated with severe soft tissue injury to the lateral ligament complex (LLC) on MRI scans (31,35).

O’Driscoll et al. (5,36) described sequential lateral to medial soft tissue disruption as “Horii circle”, beginning with partial or complete disruption of LUCL, leading to posterolateral rotatory subluxation (stage 1). Further anterior and posterior disruption leads to incomplete posterolateral dislocation (stage 2). Stage 3 describes progressive damage to the medial capsuloligamentous and musculotendinous structures resulting in complete dislocation. The valgus external rotation theory has been proposed with injury occurring as a result of a fall on a partially extended elbow. The force transmitted via the forearm is a combination of lateral rotation and valgus

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<thead>
<tr>
<th>Injury type</th>
<th>Mechanism of injury</th>
<th>Structures involved</th>
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<tr>
<td>Posterolateral rotatory (Terrible triad)</td>
<td>Valgus external rotation</td>
<td>LLC + Radial head + anterolateral facet of coronoid</td>
</tr>
<tr>
<td>Posteromedial rotatory (PMRI)</td>
<td>Varus internal rotation</td>
<td>LLC + Anteromedial facet of Coronoid + Posterior band of medial ligament</td>
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<tr>
<td>Monteggia fracture dislocation</td>
<td>Axial and Bending moment</td>
<td>Apex anterior: Ulna fracture and Radial head dislocation</td>
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<td></td>
<td></td>
<td>Apex posterior: Ulna fracture and Radial head fracture</td>
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**Table 3 Mechanism of elbow injuries**

**Figure 1** Shows the ladder concept of soft tissue involvement in an elbow dislocation. Adapted and modified from Robinson et al. [Robinson:2017bt].

**Figure 2** Magnetic resonance imaging of a post reduction posterolateral dislocation showing a grade 4 injury with complete disruption of medial and lateral structures. Large and small black arrows show disruption of common flexor origin and medial collateral ligament respectively. White arrows show disruption of lateral collateral ligament and common extensor origin.
strain. This is due to the cam effect of the lateral sloping medial side of the trochlea. The greatest displacement is on the outer side of the joint leading to stripping of the lateral ligament sleeve and tearing of the posterolateral capsule (Osborn Cotterill ligament). As a result of this, the radial head rotates backwards from the capitellar surface. This may be the model for simple posteromedial dislocation that account for 10% of simple dislocations, but can also result in fracture dislocation.

Elbow instability

Posterolateral rotatory instability (PLRI)
Chronic PLRI, characterised by posterior subluxation of the radial head over the capitellum, typically occurs after a posterior or posterolateral subluxation/dislocation, and involves injury to all or parts of the lateral primary and secondary stabilising structures (LLC, posterolateral ligament and common extensor origin). Grade 1 PLRI follows an isolated posterior ligament avulsion resulting in positive drawer test but negative pivot shift test. Grade 2 PLRI (positive drawer and pivot shift test) is as a result of an LLC avulsion with or without posterior ligament involvement. Over a period of time repetitive subluxation and spontaneous reduction of the radio-capitellar joint leads to progressive lateral degeneration (10,13,16,37,38).

Valgus loading results in MCL strain, compression of the radio-capitellar joint, and shearing forces at the posterior aspect of the elbow between the medial aspect of the tip of the olecranon and the olecranon fossa. Repetitive and/ or high valgus stress can lead to attenuation or partial/ complete tears of the MCL. The ensuing increased MCL strain can result in osteochondral capitellar lesions, loose bodies, and cartilage damage at the olecranon fossa leading to the classic finding of posteromedial osteophytes “kissing lesion”. Associated lesions may include; ulnar neuritis, flexor-pronator tendinopathy, or medial epicondyle apophysitis in skeletally immature (42-44).

Fracture dislocations
To understand the impact of fractures of the coronoid, proximal ulnar, and radial head on elbow instability a three-column proximal forearm model has been proposed with a corresponding classification system (Table 4) (16). The medial column is formed by the medial trochlea and anteromedial coronoid facet. The middle column is formed by the lateral trochlea and the anterolateral coronoid facet, and the lateral column is formed by the capitellum and radial head (Figures 3,4). A fulcrum exists between the middle and medial columns. The primary restraint to valgus collapse is therefore the lateral column with a secondary contribution from the middle column. The medial column is the only restraint to varus collapse. When the lateral column is intact the middle column has no significant stabilising role however removal of the lateral column makes the middle column essential for valgus stability (Figure 5). Osseous injuries are associated with characteristic soft tissue injuries which further contribute to instability. The key soft tissue structure in most fracture dislocations is the LLC, which in most cases is avulsed from the humeral side.

<table>
<thead>
<tr>
<th>Type</th>
<th>Bony injury</th>
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<tbody>
<tr>
<td>A</td>
<td>Anteromedial facet coronoid fracture</td>
</tr>
<tr>
<td>B</td>
<td>Bifacet Coronoid fracture</td>
</tr>
<tr>
<td>B+</td>
<td>Bifacet coronoid fracture with associated radial head fracture</td>
</tr>
<tr>
<td>C</td>
<td>Combined radial head and anterolateral facet or Comminuted radial head fracture</td>
</tr>
<tr>
<td>D</td>
<td>Diaphyseal proximal ulna fracture with dislocated intact radial head</td>
</tr>
<tr>
<td>D+</td>
<td>Diaphyseal proximal ulna fracture with associated radial head fracture</td>
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Wrightington type A
Type A injuries are medial column injuries (anteromedial coronoid facet) without injury to the middle column (anterolateral coronoid facet). These injuries are typically the results of a postero-medial fracture dislocations (19,38,45,46). As the primary varus restraint, significant damage to the medial column can lead to varus collapse and gross instability or varus posteromedial rotatory instability (VPRI) (Figures 6, 7).

The obligate injury in VPRI, characterised by posteromedial elbow subluxation, is humeral detachment of the LLC. Loss of this key primary stabiliser permits posteromedial subluxation of the elbow joint resulting in impaction of the coronoid and trochlea with a potential anteromedial coronoid facet fracture. Further progression of deformity can lead to involvement of posterior band of medial collateral ligament (pMCL) (38,47). Recent studies have shown that isolated disruption of the pMCL causes an

Figure 3 Three-dimensional computed tomography reconstruction demonstrating the three columns of the proximal forearm; lateral (radial head), middle (anterolateral coronoid facet), and medial (anteromedial coronoid facet). Adapted from Watts et al. [Watts:2019is].

Figure 4 Representative illustration of the three-column model demonstrating the natural fulcrum in between the medial and middle columns. Adapted from Watts et al. [Watts:2019is].

Figure 5 Wrightington C fracture dislocation. (A) Combined loss of the middle (anterolateral coronoid facet) and lateral (radial head) columns with disruption of the lateral ligament complex results in valgus instability. This pattern is synonymous with the terrible triad fracture dislocation and will result in posterolateral rotatory instability if not surgically stabilized; (B) restoration of the radial head and lateral collateral ligament complex will reliably restore stability in a Wrightington C fracture dislocation without the need to fix the anterolateral coronoid fracture. Adapted from Watts et al. [Watts:2019is].
increase in varus angulation and internal rotation during flexion under varus load (8,9). Hwang et al. (19) studied the role of pMCL in the VPRI and found out that pMCL has to be disrupted for gross dislocation of the elbow to occur. If VPRI is not recognised and appropriately treated, the application of varus stress may cause opening of lateral ulnohumeral joint and point loading of medial ulnohumeral joint. Over time, this increase in medial contact forces can lead to early osteoarthritis (48,49).

Wrightington B/B+
Type B are bifacet fractures (Figures 8) involving the middle and medial column. Bifacet fractures can occur in isolation as part of an extension type Monteggia fracture dislocation, or less commonly in posteromedial fracture dislocations with lateral extension of the anteromedial facet fracture. There may be an associated olecranon fracture or radial head fracture (Type B+, Figure 9). The latter results in a three-column injury with a poor prognosis if not recognised and managed appropriately. Monteggia fracture dislocations are more likely to occur in patients with osteoporotic bone and may be associated with coronoid comminution and extension to the sublime tubercle.

Wrightington Type C (Combined anterolateral facet and radial head/comminuted radial head)
A combined radial head and anterolateral facet fracture is a terrible triad injury. Loss of all valgus osseous restraints leads to instability. Isolated radial head fractures are single column injuries which are unlikely to lead to instability however highly comminuted fractures should raise the suspicion of higher energy injuries with associated damage to the medial and lateral soft tissue structures and possible resultant instability (Type C, Figure 5). These injuries may require lateral column reconstruction and soft tissue stabilisation as PLRI can occur in the presence of a minor radial head fracture with an associated avulsion of the posterolateral ligament (Osborn Cotterill Ligament) or LLC as described earlier (10,16,37).

Wrightington D/D+
Type D injuries are proximal ulna fracture dislocations in which the fracture is distal to the coronoid which remains in continuity with the olecranon. The middle and medial columns of the elbow joint remain intact (Type D). The radial head may be dislocated and intact (Type D) or

Figure 6 Illustration shows loss of the medial column (anteromedial coronoid facet) with associated proximal avulsion of the lateral ligament complex and posterior band of medial collateral ligament results in varus instability (Wrightington A fracture dislocation). This pattern is commonly seen in posteromedial rotatory fracture dislocation. Adapted from Watts et al. [Watts:2019is].

Figure 7 Wrightington A fracture dislocation. (A) Plain radiograph demonstrating an anteromedial coronoid facet fracture; (B) fixed with a coronoid plate through a medial approach. Adapted from Watts et al. [Watts:2019is].
fractured (Type D+, Figure 10). What differentiates a type D from type B injury is an intact coronoid and hence a stable ulnohumeral articulation. In a type D with an intact radial head the lateral ligament is frequently avulsed at its origin.

**Assessment**

An assessment of any significant elbow injury begins with a detailed history focussing on the mechanism of dislocation and any subjective feeling of elbow instability. The elbow should be examined for bruising over the medial or lateral side. Lateral bruising is an indicator of high-grade injury (13). Any open wounds should be managed according to local guidelines. Ligament examination in the acute setting is challenging. Reluctance to actively move the elbow may be a sign of more significant soft tissue or bony injury.

Standard anteroposterior and lateral radiographs are assessed to look for fractures and joint congruency. If there is a suspicion of a fracture, a computed tomography (CT) scan is the optimal imaging modality. Three dimensional (3D) reconstructions can be invaluable in understanding the pattern of injury particularly around the coronoid. Magnetic resonance imaging (MRI) scan can demonstrate integrity of the stabilising ligaments and tendons allowing stratification of simple elbow dislocations. When assessing an MRI scan it is important to assess the posterior and anterior bundles of MCL, flexor-pronator origin, LLC and common extensor origin. Ultrasound scanning can provide a dynamic examination of collateral ligaments and flexor and extensor tendons.

It is the practice of the senior author to reserve examination...
under anaesthesia (EUA) for simple dislocations with MRI proven soft tissue injury extending to the lateral structures or when there is clinical suspicion of high-grade soft tissue injury based on history of injury, degree of displacement, and clinical examination of soft tissue envelope. EUA involves an assessment of joint alignment under varus and valgus stress in full extension and 30 degrees of flexion, and in both pronation and supination. Re-dislocation under examination is indicative of gross instability. Mild and moderate instability are defined as less than or more than 10 degrees of joint opening respectively. Dynamic fluoroscopy has a role in delineating various grades of collateral ligament injuries and help with surgical decision-making (13,50,51).

PLRI and VPRI are clinical diagnoses. A careful history should include the position of the elbow at the time of injury if recalled. Radiographs and CT scans may reveal indirect signs of ligamentous injury such as calcification of ligaments and subluxation of the joint. Specific examination findings in PLRI may include a positive drawer and pivot shift test. Radiographs and CT imaging may reveal an avulsion fracture of the posterior aspect of capitellum (Osborne Cotterill lesion), and the drop sign (more than 4mm widening of ulnohumeral joint as seen on lateral radiograph) (52). An MRI scan will diagnose the ruptured LLC and also the presence of any osteochondral lesions.

Patients with VPRI will have positive posterolateral rotatory drawer test secondary to disruption of the LLC, which is avulsed from humeral condyle. AP radiographs show narrowing of the medial joint space, with a double crescent sign (distal displacement of medial contour of the coronoid appreciated on the lateral view radiograph) (47). Varus stress radiographs can be helpful to assess medial joint space collapse and lateral joint line widening. A CT scan with 3D reconstructions is particularly helpful in classifying any coronoid fracture but MRI best illustrates the soft tissue injury.

Patients with VEO typically present with painful throwing motion and reduced throwing velocity. A careful history can differentiate VEO from other sources of pain. Posterior elbow pain at ball release (elbow is in terminal extension) is the hallmark of VEO. By contrast, medial elbow pain at the onset of arm acceleration points to isolated MCL pathology, whereas posteromedial pain with resisted arm extension may be more likely due to triceps tendonitis.

The diagnostic test for VEO is valgus stress on the elbow at 20-30 degrees of flexion while forcing the elbow into terminal extension. This reproduces the pain over the posteromedial tip of the olecranon process experienced during throwing. Plain radiographs may show the presence of posteromedial olecranon osteophyte, olecranon stress fracture, or a loose body, however, the absence of these radiological signs does not eliminate VEO as impingement symptoms predate the formation of osteophytes and loose bodies. An MRI scan can evaluate the integrity of the MCL and show associated pathology such as loose bodies, olecranon stress fractures, and posteromedial olecranon tip osteophytes (41,53).

**Treatment**

**Simple elbow dislocation**

Stable simple elbow dislocation can be managed with early controlled mobilisation. Comparing early mobilisation
(starting after 2 days) to plaster treatment, the functional outcomes, flexion-extension arc, return to work, and extension deficits have shown to be significantly improved following early mobilisation. There were no differences in pain, relapses, instability, and ossification in the two groups. Immobilization beyond three weeks had poorer outcomes (54-57).

Traditionally non-operative management was the standard treatment for simple dislocations. Josefsson et al. (29,58) showed no statistically significant difference in surgical versus non-surgical treatment of elbow dislocation but did not stratify by grade of injury. Both groups had extension deficits with full flexion, pro-supination, and no subjective instability or re-dislocation. Subjective instability has been reported in 8% of individuals (59) but Modi et al. reported only 2.3% of patients required stabilisation surgery at one year (60). Stratification of the extent of the injury with EUA, MRI, or US may identify those patients in whom all the soft tissue stabilisers have been stripped from the humerus and may benefit from early surgical stabilisation. The degree of instability is dependent on the extent of involvement of secondary dynamic stabilisers (2,51).

Surgical decision making in acute injuries relies on any of lateral joint line gapping, common extensor disruption, or EUA evidence of instability. Moderate instability is associated with poor function and a higher need for secondary revision surgery. Surgical stabilization is recommended for cases with moderate instability (51,61). Surgery aims to downgrade the level of injury and permit early mobilisation in the supine position in an attempt to avoid instability and stiffness. The acute repair involves open or arthroscopic repair of the lateral ligaments and common extensor origin. If instability persists following lateral ligament repair, an open repair of the medial ligament and CFO is undertaken (13).

**Chronic instability**

**PLRI**

Non-surgical management of established PLRI has a limited role as most functional activities put the elbow in the position of higher risk for instability and osteoarthritis. Anconeus and extensor muscles strengthening exercises may help in limiting mild PLRI (23,25). The primary aim of treatment is to restore and maintain articular congruity. Primary repair is the first line of treatment for ligament injuries that have occurred within six weeks. Daluiski et al. (62) found no difference in the range of movements and Mayo Elbow Performance Score (MEPS) in the acute versus delayed group respectively (<30 and >30 days). Sanchez-Sotelo et al. found better functional results and MEPS in the reconstruction group compared to the ligament repair group (63).

Grade 1 PLRI can be managed arthroscopically as described by Roger Van Riet (38). This technique consists of imbrication of the LUCL from the lateral epicondyle to the soft spot portal and then the supinator crest with the use of No 2 polydioxanone suture. The suture is then doubled and tails pass subcutaneously back to the soft spot portal and tied. Open repair of the Osborne Cotterill lesion can be performed using an all suture anchor in the posterior capitellum to restore the posterior capsular ligament.

LUCL reconstruction is preferred with an autograft, in cases of chronic instability. Allograft or synthetic grafts are used in patients with hypermobility syndrome.

**VEO**

Initial management in cases with isolated VEO with no MCL symptoms is with active rest followed by an interval throwing program and the gradual return to competition.

When conservative treatment fails, surgical treatments include arthroscopic posteromedial elbow decompression and +/- MCL reconstruction. The objective of arthroscopy is to assess the posterior surface of olecranon for osteophytes and any loose bodies. Posterior osteophytes should be removed with a shaver; however, excessive olecranon resection can lead to increased tensile forces across the MCL during valgus stress and lead to further valgus instability (64,65). Up to 8 mm of the olecranon can be resected safely without an increased strain on MCL (66). Eighty five percent of athletes are able to return to competitive sports after arthroscopic management (Reddy et al. arthroscopy 2000).

Formal reconstruction of MCL (Tommy Jones Procedure) involves open surgical procedure and is limited to the athlete who wishes to return to same level of competition and has failed conservative management (67).

**Fracture dislocations**

Unlike simple dislocations the presence of an associate fracture will normally mandate surgery. The Wrightington classification categorises these injuries and provides treatment algorithms based on the previously described three-column model of fracture dislocation instability (see algorithm, Figure 11).

**Wrightington Type A**
Type A fractures may result in a very small anteromedial facet fragment. Stability may be restored by fixing the lateral ligament only, but if instability persists bony fixation is mandatory. Repair of pMCL may be required if instability remains (Figure 7). Management of patients who develop VPRI, due to neglected injuries, ranges from non-operative management to LLC reconstruction and +/- reconstruction of the anteromedial facet of the coronoid with a graft. Non-operative management is indicated in patients with no medial collapse of ulnohumeral joint space, no lateral gapping, and a firm endpoint in varus stress. Weekly radiographs are indicated for the first 3 weeks followed by another radiograph at 6 weeks interval to confirm maintenance of alignment and rule out any collapse. ROM exercises in the supine position are started early and progressive stretching is started at 6 weeks (38,48,68,69).

Surgery is recommended if subluxation or dislocation is seen, if the varus stress radiographs are positive, or clinical symptom of grading or instability is observed when flexing the elbow in the horizontal plane with the shoulder abducted 90 degrees.

Surgical treatment aims to restore elbow kinematics with the aim of preventing chronic varus instability and the onset of early osteoarthritis. Conventional open approaches require extensive anterior capsular detachment to gain exposure and compromise vascularity and fixation. Arthroscopically assisted fixation allows for anatomic repair without extensive soft tissue dissection (70). This involves arthroscopic fixation of the coronoid fracture with a threaded 1.2 mm k-wire or cannulated screw from the dorsal surface of ulna into the coronoid. This can be facilitated by a tip aiming ACL guide. The pMCL repair is needed for cases with residual instability after LCL and anteromedial coronoid facet fixation (38,48).

**Wrightington Type B**

Bifacet fractures require fixation of the coronoid process usually with lag screws for single large fragments or plates where there is comminution. If the coronoid fracture is associated with a Monteggia fracture dislocation a plate should be applied to the ulna to restore the alignment of the olecranon and ulna diaphysis. Fixation of the coronoid should not be undertaken through the dorsal ulna plate as this compromises the coronoid fixation and risks recurrent instability (Figure 8). When bi-facet fractures occur in association with a radial head fracture, as part of a direct posterior injury or flexion type (apex posterior) Monteggia fracture dislocation the priority is to address the coronoid fracture which is aided by approaching the elbow through a lateral Kaplan approach (71) typically with a cannulated screw passed from the dorsum of the ulna across the fracture using the anterior trochlea as a “mould” to ensure adequate reduction. If the coronoid is fragmented a coronoid buttress plate can be inserted through a separate medial approach (Figure 9). Where the radial head is unsalvageable the coronoid is reduced and fixed prior to radial head replacement to ensure appropriate restoration of radial length. In Monteggia fracture dislocations the coronoid and radial head can be addressed through the ulna fracture via a posterior midline approach as described by McKee et al. (72). The lateral ligament is fixed once medial and lateral columns are restored (73).

**Wrightington Type C (combined/comminuted radial head)**

In type C fractures restoration of the radial head (lateral
column), either by fixation or replacement, will restore stability without fixation of the anterolateral facet coronoid fracture (middle column) as long as the LLC injury is addressed (74, 75). The MCL may need to be fixed if instability persists.

**Wrightington D**
Comminuted radial head fractures frequently require replacement and LLC repair. Where the radial head is intact the lateral ligament is frequently torn and needs to be repaired. In all cases, restoration of anatomical ulna alignment is important to ensure congruous articulation of the radial head with the capitellum, and to restore forearm biomechanics (Figure 10).

**Rehabilitation protocol**
Early elbow mobilisation has shown to have favourable outcomes (54-57). Splints, braces, plaster of paris, or external fixators can be avoided. Immediate mobilisation with a recumbent overhead regime has been described by Schreiber et al. (76). Above head exercises are performed whilst the patient is lying in a supine position with the shoulder flexed to 90, adducted and in neutral rotation. In this position the posteriorly directed forces are minimised by decreasing the effect of gravity and allowing the triceps to function as an elbow stabiliser. When biceps hypertonia had reduced, the patients then progress to open and closed chain functional exercises.

**Conclusions**
Elbow instability varies from simple dislocation with an isolated soft tissue injury to fracture dislocation. Surgical intervention aims to restore stability to allow early range of movements. Understanding the pathoanatomy is vital for successful management of these injuries.

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**Footnote**

*Conflicts of Interest:* All authors have completed the ICMJE uniform disclosure form (available at http://dx.doi.org/10.21037/aoj-19-186). The authors have no conflicts of interest to declare.


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