Hypothesis: This study’s purpose was to provide a reproducible way for surgeons to intraoperatively assess the elbow’s lateral ulnar collateral ligament origin and determine whether there is posterolateral rotatory instability (PLRI) despite an intact common extensor origin (CEO). We hypothesized that we could recreate clinically relevant disruption of lateral supporting structures despite an intact CEO and illustrate progressive elbow PLRI.

Methods: The relationship of the lateral capsule to the capitellum articular surface was noted in 8 cadaveric upper extremities. The lateral capsule and extensor origin were sequentially sectioned at 4 stages: intact, release to the lateral epicondyle, release of the posterior capsular insertion, and release of the CEO. Posterior and lateral translation of the radial head (RH) relative to the capitellum was measured with the forearm in extension and supination.

Results: The average specimen age was 78.9 years. The lateral capsule originated within 1 to 2 mm of the capitellum articular surface. Lateral capsular sectioning to the 6-o’clock position of the lateral epicondyle created an unstable elbow with posterior and lateral RH translation. Sequential sectioning of the posterior capsular insertion created significant additional RH translation posteriorly ($P < .05$). With release of the capsule and the extensor origin, the elbow was grossly unstable.

Conclusions: The elbow’s lateral capsuloligamentous complex plays an important role in preventing PLRI. Larger degrees of elbow laxity are associated with further peel back of the capsuloligamentous complex despite an intact CEO. The surgeon must retract the extensor origin intraoperatively to assess for lateral ulnar collateral ligament and/or lateral capsule disruption to prevent a missed case of PLRI.

Level of evidence: Anatomy Study; Cadaveric Dissection

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Keywords: Elbow; capsule; LUCL; capsular insertion; PLRI; posterolateral rotatory instability
from its humeral origin of the lateral epicondyle.\textsuperscript{1,9} In some patients with an elbow dislocation or fracture-dislocation, disruption of the LUCL may occur while the extensor origin remains intact (Fig. 1).\textsuperscript{1,9} If this is not recognized, the patients are at risk of persistent posterolateral rotatory instability (PLRI). Injury to one or both of the collateral ligaments during an elbow fracture-dislocation was reported to occur 96\% to 100\% of the time.\textsuperscript{9,13} This study aims to provide an easy and reproducible way for surgeons to intraoperatively assess the status of the LUCL origin and determine whether there is PLRI. We hypothesized that we could re-create clinically relevant disruption of lateral supporting structures and illustrate progressive posterolateral instability of the elbow.

**Materials and methods**

In 8 human cadaveric upper extremities, the radiocapitellar joint was approached through an extensor-splitting approach. The lateral capsule posterior to the mid axis of the radiocapitellar joint contains the LUCL. The relationship of the lateral capsule to the articular surface of the capitellum was noted. Next, the lateral capsule and extensor origin were sequentially sectioned. The posterior and lateral translation of the RH relative to the capitellum was measured at 4 stages of sectioning: intact, release of origin and/or capsule to the lateral epicondyle, release of the posterior capsular insertion, and release of the common extensor origin (CEO). Sectioning was performed as follows: First, the extensor mechanism and musculature were retracted to expose the capsular insertion on the capitellum. Next, the capsular insertion was released to the level of the lateral epicondyle sharply using a scalpel. If the capitellum is pictured as a clock face, this would correspond to the area from the 12- to 6-o’clock position. The next level of sectioning was release of the posterior capsular insertion, from the 6- to 12-o’clock position. Finally, the CEO was released from the lateral condylar ridge. All measurements were made with the forearm supinated and extended using a digital caliper (Digimatic; Mitutoyo, Kawasaki, Japan). The position of the anterior aspect of the RH relative to the capitellum was marked using a 1.14-mm Kirschner wire while an assistant extended and supinated the forearm maximally. This was repeated in the same fashion in all specimens. The RH and capitellum diameters were measured. All dissections were performed by the same fellowship-trained hand surgeon (Fig. 2).

Posterior translation was calculated as a percentage of posterior translation of the RH relative to the capitellum. Lateral translation was calculated as a percentage of translation of the RH. Statistical analysis was performed using a 1-way analysis of variance with “interval capsular sectioning” as the factor and individual as a random effect, followed by a post hoc Tukey HSD (honestly significant difference) correction for multiple comparisons, with $\alpha$ equal to .05.

**Results**

The average specimen age was 78.9 years. There were 5 male and 3 female specimens. The average RH and capitellum diameters were 23.8 mm (range, 21.4-25.1 mm) and 22.9 mm (range, 20.1-26.6 mm) respectively. In each specimen, the lateral capsule originated within 1 to 2 mm of the capitellum articular surface. At baseline, with an intact capsuloligamentous complex, there was, on average, RH translation of 33.2\% posteriorly and 26.9\% laterally. Sequential lateral capsular sectioning led to progressive posterior and lateral translation of the RH and a progressively unstable elbow. With lateral capsular sectioning to the 6-o’clock position, directly posterior to the lateral epicondyle, there was significant additional RH translation posteriorly ($P < .05$) (Table I). With release of the capsule and extensor origin, the elbows were grossly unstable, with average posterior translation of the RH of 61.4\% and average lateral translation of 52.4\% (Fig. 3).

**Discussion**

Elbow stability is defined by its bony articulation, the collateral ligaments, the muscle origins, and the elbow capsule.\textsuperscript{2,5,7,10-12,14-16,18,20,23} The ulnar part of the lateral collateral ligament and its contribution to preventing PLRI have been extensively studied.\textsuperscript{1,5,11,14,16,18} Unfortunately, the literature does not clearly describe the surgical appearance of a disrupted LUCL, the spectrum of possible injuries to the lateral side, and a predictable method to identify the problem intraoperatively. In this study, we are describing the “naked capitellum sign,” in which the capsular insertion on the capitellum is completely disrupted despite an intact CEO. We
believe that the presence of a naked capitellum signifies an inherently unstable elbow, and failure to identify its presence will lead to chronic instability of a surgically treated elbow fracture.

O’Driscoll et al\textsuperscript{16} described the LUCL and its contribution to preventing PLRI. In addition, Cohen and Hastings\textsuperscript{5} reported, in a cadaveric study, that post-traumatic PLRI is the “result of attenuation or disruption of both the ligamentous and the muscular origins from the lateral epicondyle of the humerus.” However, the senior author’s experience is consistent with the findings of Giannicola et al\textsuperscript{9} and McKee et al\textsuperscript{13} that post-traumatic lateral elbow instability can be present in the setting of a disrupted lateral capsule, despite an intact CEO. Our study findings support and highlight these observations by showing that with disruption of the capsule to the 6-o’clock position, there was nearly 45% posterior translation of the RH.

Soft-tissue injuries nearly always occur to varying degrees during elbow fracture-dislocations.\textsuperscript{8,9,13} Giannicola et al\textsuperscript{9} reported on a series of 47 patients with complex elbow instability.

Table I  Posterior and lateral translation of radial head as percentage of capitellum and radial head diameter, respectively

<table>
<thead>
<tr>
<th>Specimen age, yr/sex</th>
<th>Posterior translation, %</th>
<th>Lateral translation, %</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Intact capsule</td>
<td>LUCL released to LE</td>
</tr>
<tr>
<td>78/female</td>
<td>21.7</td>
<td>44.8</td>
</tr>
<tr>
<td>68/female</td>
<td>40.7</td>
<td>48.4</td>
</tr>
<tr>
<td>84/male</td>
<td>49.6</td>
<td>49.8</td>
</tr>
<tr>
<td>84/male</td>
<td>31.8</td>
<td>47.2</td>
</tr>
<tr>
<td>81/male</td>
<td>24.4</td>
<td>33.0</td>
</tr>
<tr>
<td>84/male</td>
<td>44.5</td>
<td>52.7</td>
</tr>
<tr>
<td>72/female</td>
<td>28.5</td>
<td>35.3</td>
</tr>
<tr>
<td>80/male</td>
<td>24.2</td>
<td>38.5</td>
</tr>
<tr>
<td>Average</td>
<td>33.2</td>
<td>43.7</td>
</tr>
</tbody>
</table>

LUCL, lateral ulnar collateral ligament; LE, lateral epicondyle.
They found a 96% rate of ligamentous injury. They reported an injury to the lateral collateral ligament in 91% of cases, to the posterolateral capsule in 66%, and to the CEO in 21%. Similarly, McKee et al reported on 62 elbow dislocations or fracture-dislocations that required operative repair. They found a 100% rate of lateral collateral ligament injury and concomitant rupture of the CEO 66% of the time. Both of these studies show that collateral ligament injury, in the setting of an intact CEO, is exceedingly common with complex elbow injuries, and a high suspicion for LUCL and capsular injury is paramount in preventing chronic elbow instability after operative repair.

Our study has clinical relevance for the treatment of post-traumatic elbow injuries in which the presence of PLRI is unknown or suspected. Cases in which the entire LUCL complex and CEO are disrupted are easy to identify. This study brings to light situations in which cases of PLRI may be missed if the surgeon is “fooled” by an intact CEO, in the setting of LUCL complex disruption. This is avoidable by placing a retractor on the extensor origin and extending and supinating the forearm. Three findings foretell PLRI: a naked capitellum, separation of the olecranon fossa and trochlea, and posterior and lateral translation of the RH and shaft with respect to the capitellum.

Recognizing the varied faces of PLRI is especially important in the terrible-triad injury involving a coronoid fracture, RH fracture, and LUCL injury. When the capsuloligamentous injury is combined with a coronoid fracture and RH fracture, a strong repair of the LUCL complex is of paramount importance to restore stability. Conventional wisdom held that all 3 components of the terrible-triad injury had to be addressed to restore stability. Two recent studies have suggested that addressing the LUCL and RH alone restored stability and led to acceptable functional outcomes. These 2 studies reinforce the essential nature of the LUCL and capsular complex in maintaining elbow stability, as well as the need to adequately repair this structure.

There are limitations to this study. Our cadaveric sample size is small, and examination of more specimens may show anatomic variations. In addition, we attempted to replicate a situation in which a surgeon would have to assess the stability of the elbow intraoperatively; therefore, the load could not be standardized as might be accomplished with an elbow simulator, potentially making our results susceptible to observer bias. However, our study’s aim was not to precisely measure the forces across the elbow but rather to replicate the situation commonly experienced by orthopedic surgeons in the operating room.

The findings of this cadaveric study have implications for the treatment of complex elbow injuries. First, they confirm that the lateral capsule and LUCL complex of the elbow play an important role in preventing PLRI. When a surgeon explores a traumatic elbow injury, he or she should place a retractor on the posterior limb of the split extensor origin and extend and supinate the forearm. In normal instances, the lateral capsule originates within 1 to 2 mm of the articular margin of the capitellum. When the elbow is extended, even with an intact extensor origin, if the capsule has been disrupted from the articular margin—the naked capitellum sign—the complex should be repaired. Confirmation of PLRI includes separation
of the olecranon fossa and trochlea along with posterior and lateral translation of the RH if intact or of the radial shaft when the RH is shattered. These findings are magnified with further peel back of the capsule and extensor origin (Figs. 4 and 5).

**Conclusion**

We hypothesized that we could re-create clinically relevant disruption of lateral supporting structures despite an intact CEO and illustrate progressive PLRI of the elbow. With progressive sectioning of the lateral capsuloligamentous complex, the elbow became increasingly unstable, despite an intact CEO. In cases of traumatic elbow fractures and fracture-dislocations, the operating surgeon must make sure to retract an intact CEO and assess for a naked capitellum to prevent missing disruption of the capsuloligamentous complex and PLRI.

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**Disclaimer**

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**References**