The Role of the Coronoid Process, Lateral Collateral Ligament and Radial Head in Varus Stability of the Elbow

Stephen E. Fern, John R. Owen, Nicholas J. Ordyna, Jennifer S. Wayne, N. Douglas Boardman
Departments of Orthopaedic Surgery and Biomedical Engineering, Virginia Commonwealth University, Richmond, VA
jswayne@vcu.edu

Introduction: Terrible triad is a debilitating injury involving elbow dislocation with injury to the lateral collateral ligament (LCL), radial head (RH), and coronoid process (CP). Short and long term outcomes are historically poor. The role of each component in elbow stability is still emerging. Previous testing showed a decrease in the varus stability after removal of >50% of the CP (1,2), but did not account for RH injury or lateral soft tissue constraints. Studies including these structures have not done so in pure varus (3,4). This study investigates the role of each component in elbow stability.

Materials and Methods: Ten cadaveric specimens were cut mid-humerus; skin and soft tissues were left intact except for medial and lateral approaches. A custom device kept the humeral shaft horizontal and the medial epicondyle vertical on an Instron 1321 materials testing machine. (Fig. 1)

Load lines attached to Triceps, Brachialis and Biceps Tendons

Specimen angular positioning device and mounting base.

Figure 1: Custom test device keeping humeral shaft horizontal and the medial epicondyle vertical, while applying varus displacement.

LCL repair was performed using braided microfiber suture passed through a bone tunnel at the isometric point centered on the capitulum. A custom device allowed for reproducible tensioning of the LCL repair to 25N. RH repair with Biomet’s ExploR (Biomet, Warsaw, IN) modular design allowed easy transition from a resected state (stem only) to a replaced state (head plus stem). CP resection was performed per previous work (1).

For each test, tendon loads were applied and the specimen was loaded in five cycles of 1.5cm varus displacement. Each specimen was tested first in combinations of intact, cut, and repaired LCL; intact, resected, and replaced RH; and intact CP, then in combinations of four repair states: LCL deficient/RH resected (LxRx), LCL deficient/RH repaired (LxRr), LCL repaired/RH resected (LrRx), and LCL repaired/RH repaired (LrRr) at each of seven CP resection levels (intact and cut by 25%, 40%, 50%, 67%, 75%, and 100%). Each test was conducted at 30 and 90 degrees of flexion. No additional resistance to varus displacement was conferred by isolated RH replacement. This further supports our previous findings (1) about the importance of the CP in varus stability but also provides evidence for the LCL as the primary stabilizer until the coronoid fracture becomes greater than 50%. Results correlate with current clinical knowledge that Type I and some Type II fractures may be adequately treated with only repair of the lateral sided structures. However, beyond this 50% coronoid injury, it appears that the LCL and RH cannot overcome loss of varus stability. These findings provide a biomechanical basis for aggressive treatment of coronoid fractures as a component of terrible triad injury.

Results: For the load at the beginning of the 5th cycle, the LCL repair state was not different from the intact (native) LCL regardless of the RH repair status (p>0.8). However, the intact (native) RH was different from a replaced RH at this starting position (p<0.02) with the prosthesis pushing the specimen into varus.

For LrRx, the 100% cut was the only level that significantly differed from the intact coronoid (p<0.02). (Fig.2)

Discussion: This study showed that beyond a 50% loss of coronoid process neither repair of the LCL nor replacing the RH alone, and beyond 67% for both repaired, resulted in a statistically significant increase in varus stability. Further, each incremental increase in coronoid loss resulted in further loss of varus stability starting at a 67% CP at 30 and 90 degrees. No additional resistance to varus displacement was conferred by isolated RH replacement. This further supports our previous findings (1) about the importance of the CP in varus stability but also provides evidence for the LCL as the primary stabilizer until the coronoid fracture becomes greater than 50%. Results correlate with current clinical knowledge that Type I and some Type II fractures may be adequately treated with only repair of the lateral sided structures. However, beyond this 50% coronoid injury, it appears that the LCL and RH cannot overcome loss of varus stability. These findings provide a biomechanical basis for aggressive treatment of coronoid fractures as a component of terrible triad injury.

Acknowledgements: Biomet Inc. for use of the ExploR instrumentation.

Figure 2: The change in load from the start of the 5th cycle to 1.5 cm of excursion (delta load) at 30 degrees flexion.

With the RH out and the LCL unrepaired, there was no significant difference in the delta load except when comparing the intact to 100% CP. With RH repair, a difference in load resisting varus was found at 75% CP (p<0.004) and at 100% CP (p<0.0001) versus the intact state. With the RH out but with a repaired LCL, the intact CP was significantly different from a 67% and greater coronoid lesion (p<0.0001). Furthermore, a 50% CP differed significantly from the 67% CP (p<0.02) with the only repair consisting of the LCL. In the final repair state with both the RH replaced and the LCL repaired, again there was a significant decrease in load resisting varus from an intact CP to a 67% CP (p<0.0001). And again there was a significant decrease in load resisting varus as the coronoid lesion was increased to 67% CP as compared to 50% CP (p<0.01). Findings were similar at 90 degrees of flexion (data not shown).

References:

Figure 2: The change in load from the start of the 5th cycle to 1.5 cm of excursion (delta load) at 30 degrees flexion.