INTRODUCTION
The effect of a coronoid fracture on elbow stability has been of interest to orthopedic surgeons. The fracture is often located anteromedially, between the tip of the coronoid and the sublime tubercle. The mechanism of injury is thought to be varus posteromedially and ulnar posteromedial subluxation and causes the elbow to articulate incongruently under axial load and varus stress eventually producing arthritis. [1],[2] The individual roles of bony injury versus ligamentous injury in producing clinically relevant varus instability is unknown. In this investigation, cadaveric elbow specimens were tested before and after anteromedial coronoid fractures followed by serial disruptions of the LCL and MCL, to assess the role of these individual structures in stability of the elbow under varus loading.

MATERIALS AND METHODS
Ten fresh cadaveric elbow specimens were mounted in a custom testing apparatus with the humerus rigidly fixed to the device in a horizontal position and the lateral epicondyle oriented upward as in Figure 1. Each elbow served as its own control. Varus stress was produced by the weight of hand and forearm in the following test conditions: intact elbow (n=10), anteromedial coronoid fracture alone (n=10), anteromedial coronoid fracture with LCL (n=5) or posterior band of MCL disruption (n=5), and anteromedial coronoid fracture with both ligaments disrupted (n=10). Coronoid fractures were created by high-speed saw from anterior one-third of the insertion of anterior band of MCL (sublime tubercle) to the most anterior aspect of the articular surface of the proximal radioulnar joint. This was chosen to represent a typical anteromedial coronoid fracture involving approximately 20% of the coronoid. All muscles were intact with out any external load applied. Varus laxity throughout the elbow flexion-extension arc was measured in the following conditions: intact elbow (n=10), anteromedial coronoid fracture alone (n=10), anteromedial coronoid fracture with LCL (n=5) or posterior band of MCL disruption (n=5), and anteromedial coronoid fracture with both ligaments disrupted (n=10) using an electromagnetic tracking device (3Space Fastrak, Polhemus, Colchester, Vermont, USA). Sensors were rigidly fixed to the humerus and ulnar with the arm in full extension. The varus angle was defined as the angle of the ulna relative to the humerus about the medial-lateral axis of the humerus throughout the range of motion. Varus instability or laxity was defined as the amount of deviation of the ulna relative to the humerus in each condition relative to the intact condition (in degrees). Varus laxity was determined in 20 degree increments of the motion from 20 to 120 degrees of flexion. Unpaired Wilcoxon / Kruskal-Wallis Tests (Rank Sums) test was used for inter-specimen comparisons and matched pair Wilcoxon Sign-Rank test was used for intra-specimen comparison.

RESULTS
The overall result throughout the flexion–extension arc demonstrated that isolated anteromedial coronoid fractures showed an increased mean varus laxity of 1.32 degrees (SD=0.7) compared to the intact condition (p<0.004). LCL disruption further increased mean varus laxity from coronoid fracture alone by 2.31 degrees (SD=0.85) compared to posterior MCL disruption (0.77 degree, SD=0.33) (p<0.002). Disruption of both LCL and posterior MCL increased mean varus laxity of the elbow by 4.04 degrees (SD=0.5) from isolated anteromedial coronoid fracture alone (p=0.002). Mean varus laxity at each flexion angle is shown in Figure 2.

CONCLUSION
In response to varus stress from weight of the forearm and hand, elbows with isolated anteromedial coronoid fractures were found to have significant varus instability. When associated with LCL and MCL disruptions, this instability are increased statistically significantly, with the LCL having a more significant role in providing varus stability compared to the posterior band of the MCL.

REFERENCES