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Ulnar collateral ligament insufficiency affects cubital tunnel syndrome during throwing motion: a cadaveric biomechanical study

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Background: In throwing athletes, cubital tunnel syndrome and insufficiency of the ulnar collateral ligament (UCL) are common pathologic processes of the elbow. The objective of this study was to investigate the effect of UCL tears on ulnar nerve elongation in the simulated throwing position.
Methods: Eight fresh frozen cadaveric upper limbs were tested at the simulated late cocking to acceleration phase in the throwing motion using an elbow testing system. Elbow valgus laxity and ulnar nerve length and strain under 2 Nm of applied valgus torque (maximum torque in cadaveric elbow) were evaluated. Paired *t*-tests were used to compare all data between intact UCLs and UCLs after complete transection of the anterior oblique ligament. Linear regression analysis was used to investigate relationships between elbow valgus laxity and ulnar nerve strain.
Results: Elbow valgus laxity significantly increased after transection of the UCL. Ulnar nerve length after UCL transection was significantly greater than that in the intact condition at 60° ($P = .006$) and 90° of elbow flexion ($P < .0001$). In addition, ulnar nerve strain was positive (increased) at 60° and 90° of elbow flexion. Maximum ulnar nerve strain at 90° of elbow flexion was 3.9% ± 0.9% when the UCL was intact and 6.8% ± 0.7% after transection. UCL transection yielded significant positive correlation between elbow valgus laxity and ulnar nerve strain ($P = .006$; $r = .4714$).
Conclusion: Increased elbow valgus laxity due to UCL insufficiency may cause elongation of the ulnar nerve and exacerbate cubital tunnel syndrome during the throwing motion.
Level of evidence: Basic Science Study; Biomechanics

Keywords: Cubital tunnel; elbow; elongation; ulnar collateral ligament; ulnar nerve; valgus laxity

Cubital tunnel syndrome is a common pathologic process of the elbow of throwing athletes that can cause medial elbow pain, paresthesia in the small and ring fingers, decreased grip strength, and decreased pinch

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strength.[2,7,8,17,20] When symptoms are severe, cubital tunnel syndrome inhibits the athletes from throwing. Nonoperative treatment is applied in most athletes. This involves limitation or prohibition of throwing; stretching of the posterior shoulder, trunk, and hip joint; or medication.[17,20] When nonoperative treatment is ineffective, anterior transposition of the ulnar nerve is performed to facilitate return to the previous activity.[2,20]

One cause of ulnar neuritis in throwing athletes is ulnar nerve elongation due to excessive valgus stress.[7] Nerve elongation of 5% to 10% impairs intraneural blood flow,[22] axonal transport,[12] and nerve conduction.[25] When a peripheral nerve is inflamed, even minimal elongation (3%) can lead to mechanosensitivity, potentially provoking pain and other nerve symptoms.[13] In addition, repetitive excessive valgus stress during throwing motion can cause tearing of the ulnar collateral ligament (UCL), which is a common injury in overhead throwing athletes.[1,3-9,14,18,19,24] The medial joint space in the elbow spreads because of elbow valgus torque during throwing motion. When the athlete has UCL insufficiency, this widening increases, and consequently the ulnar nerve may elongate excessively. UCL injury is significantly related to poor outcomes of nonsurgical treatment for ulnar neuritis around the elbow in baseball players.[20] However, whether UCL insufficiency exacerbates ulnar nerve elongation has not been investigated thus far. The objective of this study was to investigate the effect of UCL insufficiency on ulnar nerve elongation in the simulated throwing position. We hypothesized that the increased valgus angle that results from a UCL tear will increase the elongation of the ulnar nerve.

Materials and methods

Preparation of specimens

A total of 8 fresh frozen cadaveric upper extremities (from scapula to fingers; 2 female and 6 male specimens; mean age at death, 66.4 ± 5.3 years, University of California willed body program) were used to test ulnar nerve elongation in a custom elbow testing system (Fig. 1). The elbow testing system was designed for rigid fixation of the specimen in the desired position. All specimens were confirmed to lack pre-existing disease. The specimens were stored at −20°C and thawed overnight before dissection. Once thawed, the specimens were dissected free of soft tissue, but the tendons, musculature, and hand were left intact. Each specimen was prepared and mounted to the elbow testing system in a standardized manner: the hand was fixed by using sutures placed at midfinger, allowing control of wrist positioning; the forearm was inserted into an aluminum cylinder and securely fixed by 2 threaded cross pins in the distal radius, thus centering the forearm in the cylinder; the upper arm was mounted by 2 screws through the midshaft of the humerus such that the epicondylar axis was parallel to the baseplate; and the scapula was securely fixed to a mounting plate by 3 screws and then attached to the elbow testing system with 60° glenohumeral abduction, which simulates 90° of shoulder abduction.

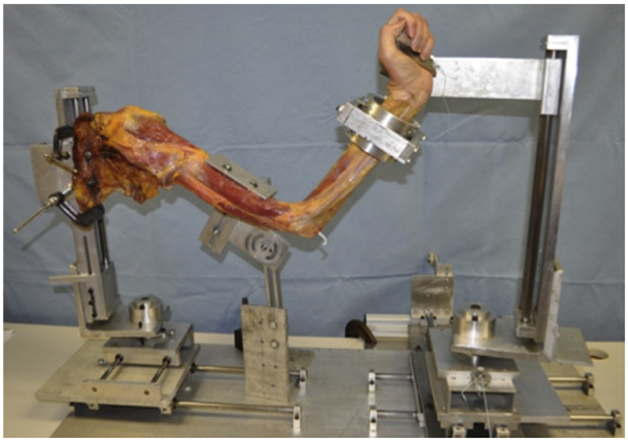

Figure 1 Elbow testing system.

A muscle-splitting approach[4] through the posterior third of the common flexor mass within the anterior fibers of the flexor carpi ulnaris was used to access the ulnar nerve in the intermuscular septum at 3 cm proximal to the arcade of Struthers, leaving the epineurium intact. The nerve was then exposed in a distal direction along the septum until the point at which it traversed the cubital tunnel, entering the forearm. Osborne's fascia was left intact. The ulnar nerve was not released out of its bed. All specimens were kept moist throughout the experiment with 0.9% saline to keep the same material properties of the ulnar nerve during testing.

Measurements

Elbow valgus laxity

Two markers on the humerus and 2 markers on the ulna were digitized by using a 3-dimensional digitizer (MicroScribe 3DLX; Revware, Raleigh, NC, USA; accuracy, 0.3 mm) at 0°, 30°, 60°, and 90° of elbow flexion. By use of position data from the 4 markers, the angle between the 2 vectors formed by the 2 markers on the humerus and the 2 markers on the ulna was calculated. During all measurements, a varus torque of 0.3 Nm was applied at the control condition to create a consistent baseline position. Valgus torque of 2 Nm was applied during measurement of the valgus angle at each condition and flexion angle. The change in valgus angle from 0.3 Nm of varus torque to 2 Nm of valgus torque was recorded as elbow valgus laxity.[21]

Ulnar nerve strain

The nerve was marked by 7 metallic beads that were sutured to the nerve tissue at 1-cm intervals along its anatomic course underneath the epineurium, starting in the intermuscular septum proximal to the arcade of Struthers (Fig. 2, *A*). The marks extended to the entrance to the cubital tunnel. The locations of the nerve marks were digitized by using a 3-dimensional digitizing system (MicroScribe 3DLX) at 0°, 30°, 60°, and 90° of elbow flexion. After completion of the tests, the entire nerve segment was dissected free, and the nerve markers were digitized. The length of

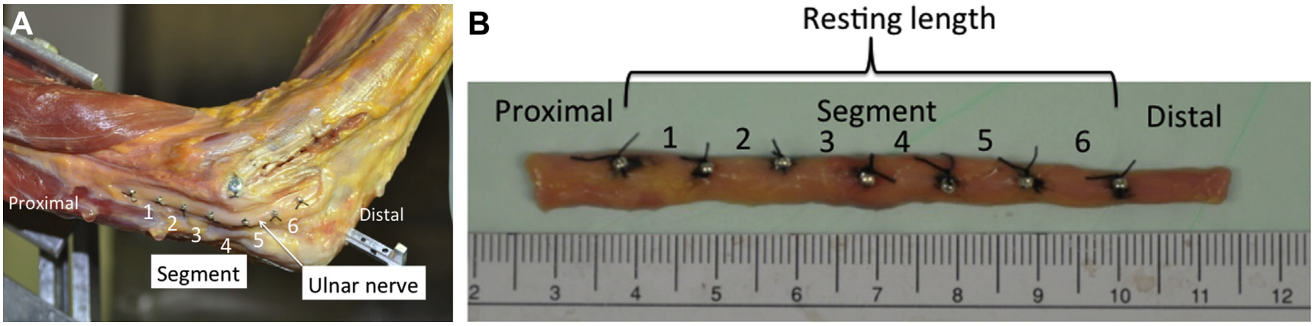

Figure 2 Suture marking on the ulnar nerve during testing (**A**) and after harvest (**B**).

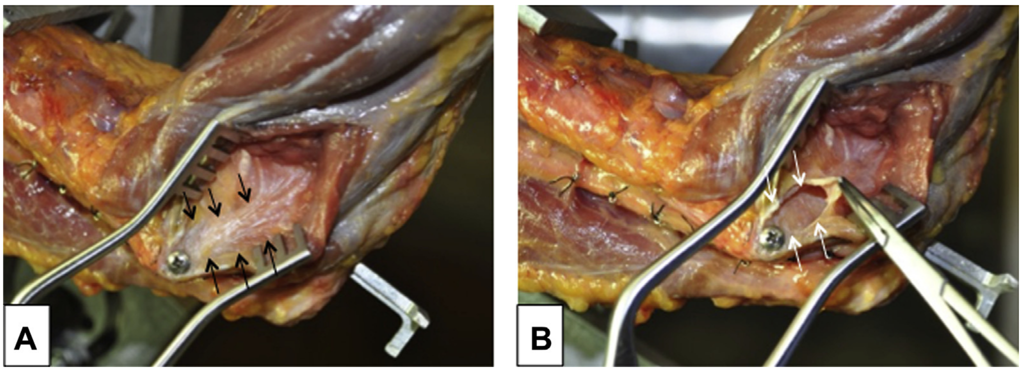

Figure 3 (**A**) Intact ulnar collateral ligament (←). (**B**) After transection of the anterior oblique ligament of the ulnar collateral ligament (*white arrows*, simulated anterior oblique ligament complete tear).

the ulnar nerve when dissected free of the specimen was defined as its resting length (Fig. 2, *B*).

The segment length of the ulnar nerve was defined as the magnitude of the vector between 2 consecutive nerve marks, which defined a nerve segment. Total nerve length was calculated by summing the lengths of all individual segments. Ulnar nerve strain (positive values represent elongation of the nerve) was calculated by subtracting resting length from length at 2 Nm of valgus torque and dividing this difference by the resting length.

Conditions

To simulate the late cocking to acceleration phase of the throwing motion, all measurements were performed at 0°, 30°, 60°, and 90° of elbow flexion while the upper limb was fixed at 60° of glenohumeral abduction, which represents 90° of shoulder abduction, 45° of forearm pronation, and 45° of wrist extension.[16] First, elbow valgus laxity and ulnar nerve strain were recorded while the UCL was intact (Fig. 3). Then, elbow valgus laxity and ulnar nerve strain were reassessed after transecting the anterior oblique ligament (AOL) of the UCL (simulated complete tear of the AOL; Fig. 3).

Data analysis

All measurements were performed twice, and the average value was used for data analyses. Paired *t*-tests were used to compare elbow valgus laxity and ulnar nerve length/strain between intact UCL and UCL after complete transection of the AOL (UCL tear model). Ulnar nerve length was compared with and without valgus torque applied. For both intact and UCL tear conditions, linear regression analysis was used to investigate relationships between elbow valgus laxity and ulnar nerve strain. A P value $< .05$ was considered to indicate statistical significance. Data were reported as means ± 1 standard deviation of the mean. Normal distribution was proved using the Shapiro-Wilks W test. All data analyses were performed by using Statistica version 6.0 software (StatSoft, Tulsa, OK, USA).

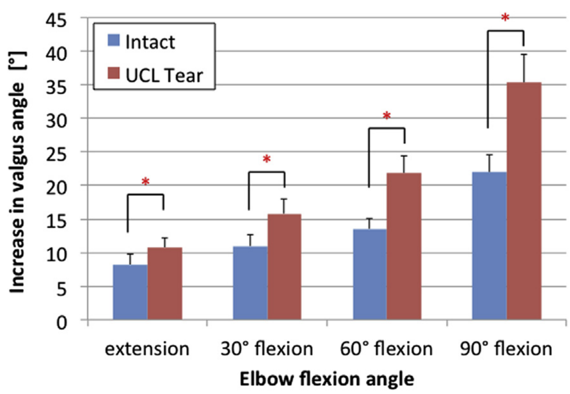

Figure 4 Comparison of elbow valgus angle between intact and torn ulnar collateral ligaments (*UCLs*) at each elbow flexion angle. *Significantly different ($P < .05$).

To determine the appropriate sample size, a power analysis was performed by using the G*Power3 statistical analysis software

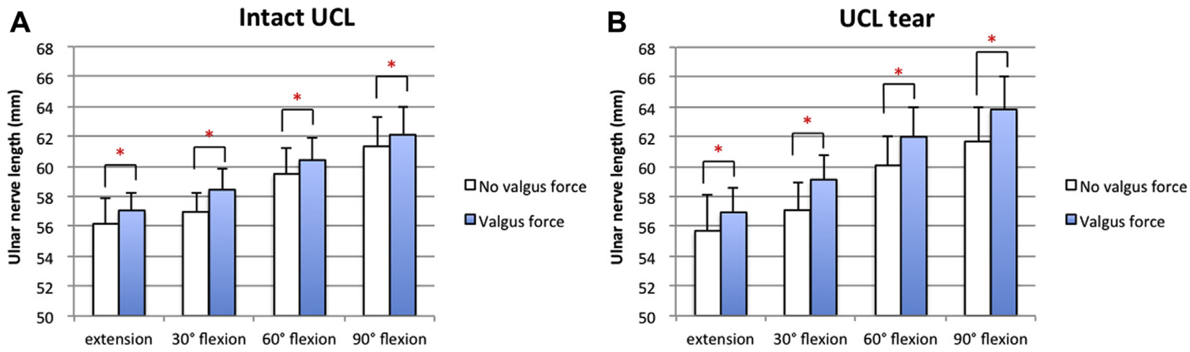

Figure 5 Comparison of ulnar nerve length with and without valgus force. (**A**) Intact ulnar collateral ligament (*UCL*). (**B**) After transection of the anterior oblique ligament of the ulnar collateral ligament. *Significantly different ($P < .05$).

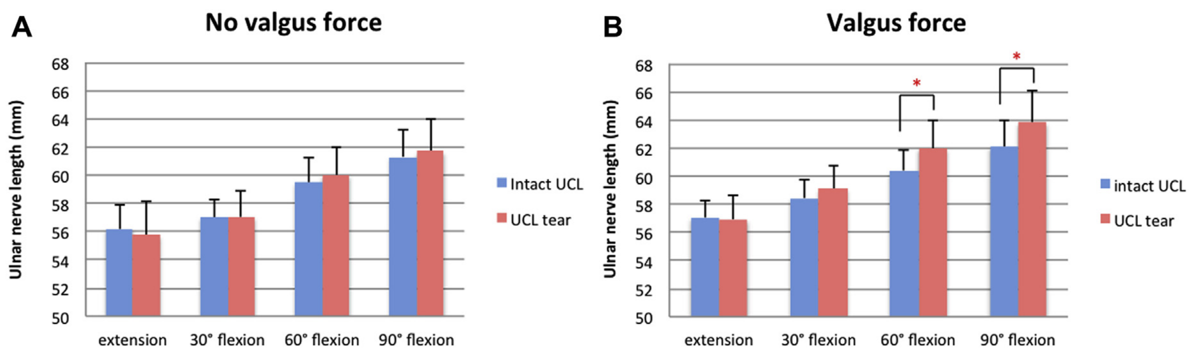

Figure 6 Comparison of ulnar nerve length between intact and torn ulnar collateral ligaments (*UCLs*) with (**A**) or without (**B**) valgus force. *Significantly different ($P < .05$).

package. Power ($1 - \beta$) was calculated by defining the sample size as 8, the level of significance (α) as .05, and the effect size as 1.29 in valgus angle and 1.20 in ulnar nerve strain at 90° of elbow flexion.

Results

The power analysis indicated that a total sample size of 8 specimens provided 80% power ($1 - \beta = 0.8$; $\alpha = .05$) to detect significant differences in valgus angle and ulnar nerve strain between intact and UCL tear conditions, assuming a power of 0.95 and 0.92.

Elbow valgus laxity significantly increased after creation of the UCL tear at all flexion angles (Fig. 4): at 0° of elbow flexion, by 3° ($P = .01$); at 30°, by 5° ($P < .0001$); at 60°, by 8° ($P < .0001$); and at 90°, by 13° ($P < .0001$). Applying 2 Nm of valgus torque significantly increased the length of the ulnar nerve compared with the control condition ($P =$ .007 to .0001; Fig. 5). Ulnar nerve length was greater after creation of the UCL tear than in the intact condition at 60° ($P = .006$) and 90° ($P < .0001$) of elbow flexion (Fig. 6). Ulnar nerve strain increased as the elbow flexion angle increased and was positive (elongated) at 60° and 90° of elbow flexion (Fig. 7). Compared with the intact condition, creation of the UCL tear increased ulnar nerve strain at 60° (by 2.6%; $P < .0001$) and 90° (by 2.9%; $P < .0001$) of elbow flexion (Fig. 7). Maximal ulnar nerve strain at 90° of elbow flexion was 3.9% ± 0.9% when the UCL was intact and 6.8% ± 0.7% for a torn UCL. Elbow valgus laxity and ulnar nerve strain showed significant positive correlation ($P = .006$; $r = .4714$) when the UCL was torn but not when it was intact (Fig. 8).

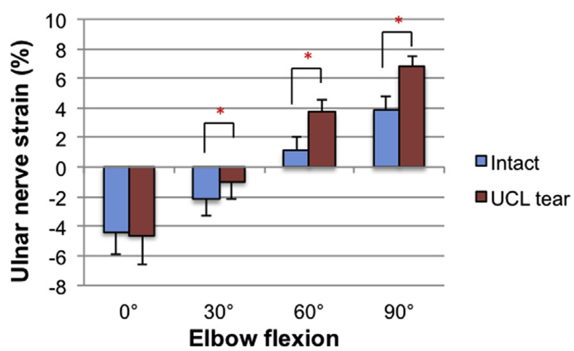

Figure 7 Comparison of ulnar nerve strain between intact and torn ulnar collateral ligaments (*UCLs*). Ulnar nerve strain (positive values represent elongation of the nerve) was calculated by subtracting resting length from length at 2 Nm of valgus torque and dividing this difference by the resting length. *Significantly different ($P < .05$).

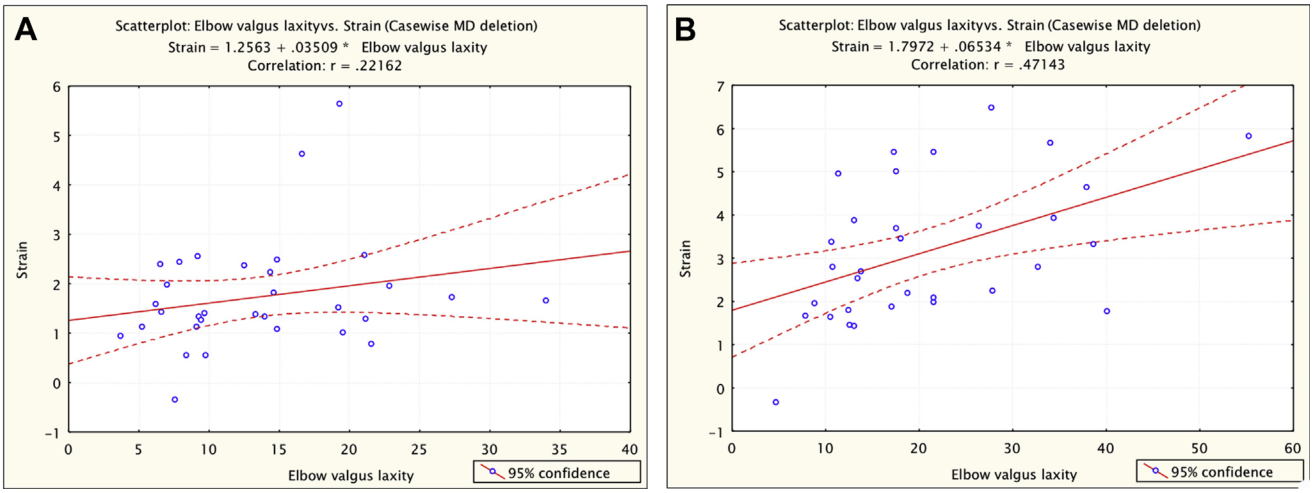

Figure 8 Linear model of ulnar nerve strain by elbow valgus laxity. The area between (– – – –) indicates the 95% confidence band for the regression line. (**A**) Intact ulnar collateral ligament (*UCL*). (**B**) Ulnar collateral ligament tear. *MD*, missing data.

Discussion

In throwing athletes, cubital tunnel syndrome[2,7,8] and UCL insufficiency[1,3-6,9,14,18,19,23,24] are common elbow pathologic processes, but the relationship between these conditions has not been investigated thus far. In this study, tearing of the UCL increased the ulnar nerve length when elbow valgus torque was applied at 60° and 90° of elbow flexion. Nerve elongation (increased ulnar nerve length) impairs intraneural blood flow,[22] axonal transport,[12] and nerve conduction[25] and consequently can cause nerve symptoms. Therefore, UCL insufficiency can exacerbate cubital tunnel syndrome during the throwing motion.

This study showed that tearing of the UCL significantly increased elbow valgus laxity, which in turn elongated the ulnar nerve. Furthermore, elbow valgus laxity was significantly positively correlated with ulnar nerve strain in the simulated UCL tear condition. Baseball throwing generates high valgus torque in the elbow joint. A previous biomechanical study estimated elbow valgus forces as high as 64 Nm during the late cocking and early acceleration phases of throwing as the elbow moves from 110° to 20° of flexion.[16] Therefore, the increased elbow valgus laxity due to UCL insufficiency may cause elongation of the ulnar nerve and exacerbate cubital tunnel syndrome during the throwing motion. In contrast, tearing of the UCL did not increase ulnar nerve length in the absence of elbow valgus torque. This result suggests that limitation and prohibition of throwing may ameliorate ulnar nerve symptoms even in patients who have UCL insufficiency.

Nerve elongation of 5% to 10% is thought to impair intraneural blood flow,[22] axonal transport,[12] and nerve conduction.[25] In our study, tearing of the UCL induced a pathologic level of ulnar nerve elongation (6.8% ± 0.7%), whereas the maximal ulnar nerve elongation in the intact UCL condition (3.9% ± 0.9%) was below the pathologic threshold. An investigation of poor outcomes after nonsurgical treatment of ulnar neuritis in adolescent baseball players revealed UCL injury as a significant negative predictor.[20] Both this biomechanical study and the clinical study in adolescent baseball players[20] suggest that UCL insufficiency is a risk factor for cubital tunnel syndrome in throwing athletes.

During joint movement, nerve gliding prevents excessive nerve elongation and tension.[15] When nerve gliding is inhibited because of any adhesion to surrounding soft tissues or any pathologic change in anatomy from repetitive throwing, the nerve can be elongated in the area of inhibition instead of gliding during joint movement.[10,11,15] In this study, elbow valgus torque increased ulnar nerve length even in intact UCLs, and the ulnar nerve at 60° and 90° of elbow flexion was longer than its resting length. Although the increase in ulnar nerve length was less in intact UCLs than in torn UCLs, any additional pathologic condition might decrease ulnar nerve gliding and consequently cause ulnar nerve symptoms in throwing athletes with intact UCLs. Furthermore, the maximal ulnar nerve strain was 3.9% ± 0.9% at 90° of elbow flexion in the intact UCL condition. When a peripheral nerve is inflamed and mechanosensitive, even 3% of nerve elongation can provoke pain and other nerve symptoms.[12] For pathologic nerve elongation, nonoperative treatment involving anti-inflammatory drugs or resting (or both) may be effective for decreasing nerve symptoms.

Our study had several limitations. First, the applied valgus torque was less than that during actual throwing motions, and we did not apply any muscle forces. The valgus torque used was the maximum that could be applied without any tearing or stretching of soft tissues in the elbow joint in the absence of any muscle forces. Therefore, we believe that the valgus torque we used here approximated excessive but nondestructive force in the cadaveric elbow joint. Second, the donors of the cadaveric specimens were older than most throwing athletes. However, although we used cadaveric elbows, none had any disease. Third, current results in our in vitro setting were not exactly the same as data of the living body because cadaveric specimens do not have muscle activation. However, the joint movement and passive effect of soft tissues were kept intact. Therefore, we believe current results were reliable and useful for understanding throwing injuries.

The strengths of our study include its direct measurement of ulnar nerve length and strain in cadaveric specimens; these factors cannot be investigated accurately in living subjects. In addition, we were able to determine the precise strain of the ulnar nerve through comparison with its resting length, which was an accurate measurement of the nerve's length after dissection and in the absence of force.

Conclusion

Tearing of the UCL significantly increased elbow valgus laxity, consequently stretching the ulnar nerve. Furthermore, elbow valgus laxity and ulnar nerve strain showed significant positive correlation in the UCL tear condition. These results suggest that increased elbow valgus laxity due to UCL insufficiency may cause elongation of the ulnar nerve and exacerbate cubital tunnel syndrome during the throwing motion. In contrast, a UCL tear did not increase the ulnar nerve length in the absence of applied elbow valgus torque. This result suggests that limitation and prohibition of throwing may decrease ulnar nerve symptoms even in patients with UCL insufficiency.

Disclaimer

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