

# Ultrasound-Detected Anconeus Epitrochlearis and Cubital Tunnel Syndrome Signs: A Clustering-Adjusted Cross-Sectional Study

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## Research Article

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# Abstract

## Purpose.

The anconeus epitrochlearis (AE) is an accessory muscle variably implicated in cubital tunnel syndrome (CuTS). This study used ultrasound to determine AE prevalence and bilaterality and evaluate its clustering-adjusted association with positive clinical signs of CuTS.

## Methods.

Fifty-two participants (104 elbows) underwent bilateral elbow ultrasound and standardized clinical assessment, including Tinel's sign and the elbow flexion test. Elbow-level data were analyzed using Generalized Estimating Equations (GEE) to account for two-elbow clustering. Effect sizes, inter-test agreement (Cohen's  $\kappa$ ), and a dose-response gradient (Spearman correlation) were also assessed.

## Results.

AE was identified in 32/104 elbows (30.8%); 26/52 participants (50.0%) had at least one AE-positive elbow, 6 (11.5%) bilaterally. After clustering adjustment, AE presence was independently associated with a positive Tinel's sign (OR = 5.46;  $p = 0.009$ ), elbow flexion test (OR = 3.52;  $p = 0.016$ ), and symptomatic status, defined as at least one positive test (OR = 3.72;  $p = 0.014$ ). AE prevalence rose monotonically with the number of positive tests, from 21.4% to 63.6% (Spearman  $r = 0.307$ ,  $p = 0.002$ ). AE presence showed 50.0% sensitivity and 78.6% specificity for symptomatic status (AUC 0.643); agreement between the two provocation tests was fair ( $\kappa = 0.38$ ).

## Conclusion.

Ultrasound reliably identifies the AE muscle, whose presence is associated with significantly higher rates of positive ulnar-nerve provocation signs in a graded, dose-dependent pattern. Whether this subclinical association, in a young and largely asymptomatic cohort, extends to clinically diagnosed CuTS requires confirmation in symptomatic populations.

## Introduction

The anconeus epitrochlearis (AE) is an accessory muscle of the posterior elbow arising from the medial epicondyle and inserting on the medial aspect of the olecranon process, spanning the cubital tunnel roof, the anatomical region through which the ulnar nerve passes. First described in cadaveric literature, AE prevalence estimates vary substantially by detection method: cadaveric dissection 5–27%, magnetic resonance imaging 8–23%, and surgical exploration 4–9%. [9, 12, 15, 16, 20]

The clinical significance of AE remains contested. Several authors have proposed a protective role, wherein the muscular roof may reduce compressive forces on the ulnar nerve compared with the relatively inelastic arcuate ligament. [21] A systematic review with meta-analysis reported AE in only ~ 4.5% of confirmed CuTS surgical cases versus ~ 14.2% of healthy controls, suggesting underrepresentation among symptomatic patients. [20] Conversely, case series document symptomatic ulnar neuropathy attributable to hypertrophied AE, particularly in athletes with repetitive elbow-flexion demands. [7, 14, 18, 19]

High-resolution musculoskeletal ultrasound provides a dynamic, radiation-free, and accessible modality for identifying AE in living subjects. Unlike cadaveric study, ultrasound enables simultaneous anatomical assessment and clinical correlation. Despite growing interest in ultrasound evaluation of peripheral nerve disorders including CuTS, [2, 4, 5, 6, 8, 13] the relationship between ultrasound-confirmed AE and standardized clinical provocation-test results has not been rigorously characterized at the elbow level, with appropriate statistical correction for the bilateral nature of data collection.

This study addresses that gap through three aims: (1) to estimate the prevalence and bilaterality of AE using ultrasound in a prospectively recruited cohort; (2) to evaluate, with appropriate clustering-adjusted methods, associations between AE presence and positive CuTS provocation tests; and (3) to characterize the relationship between AE presence and the provocation signs, including inter-test agreement, in a cohort without electrodiagnostic confirmation.

## Methods

### Study Design and Participants

This was a cross-sectional observational study conducted at the Ultrasound Laboratory, Department of Anatomy, Northumbria University, Newcastle upon Tyne, United Kingdom. Participants were recruited over a two-month period in February–March 2026. Fifty-two adult participants were prospectively recruited and underwent bilateral elbow assessment. Eligibility was open to adults of any age, sex, gender, or ethnicity; individuals with a history of elbow trauma, prior elbow surgery, or elbow deformity were excluded. Each participant contributed two elbows (left and right), yielding 104 elbow-level observations. The sample size was determined pragmatically based on participant availability within the recruitment period rather than an a priori power calculation; the achieved sample provided adequate power to detect the primary AE–provocation test associations reported here, but limited power for secondary subgroup and interaction analyses (see Limitations). The study was conducted in accordance with institutional ethical standards.

### Ultrasound Examination

All participants underwent bilateral elbow ultrasound performed by a single trained examiner using a GE ultrasound system with a 12L-RS linear-array transducer operating at 12 MHz, using the manufacturer's

"Small Parts" preset. The probe was positioned over the cubital tunnel, between the medial epicondyle and the olecranon process, with the elbow in 90 flexion. The AE muscle was identified as a distinct muscular structure bridging these landmarks, superficial to the ulnar nerve. Each elbow was classified dichotomously as AE present or AE absent (Fig. 1). Ultrasound served as the reference standard for AE detection. The ultrasound examiner was blinded to the results of the clinical provocation tests at the time of scanning. In cases where AE classification was ambiguous on initial scanning (e.g., equivocal echogenicity or indistinct muscle-fascial boundaries), repeat scanning was performed to confirm classification; this was not performed for the full cohort and does not constitute a formal reliability assessment (see Limitations).

## Clinical Assessment

### Tinel's sign

Percussion over the ulnar nerve at the cubital tunnel was considered positive if the participant reported tingling or paresthesia radiating into the ring or small fingers.

### Elbow flexion provocation test

The elbow was held in maximum flexion with the wrist extended for 60 seconds. The test was considered positive if ulnar-distribution paresthesia was reproduced. Each test was recorded as positive or negative per elbow, lateralized to left, right, or bilateral.

An elbow was classified as symptomatic if either provocation test was positive on that side, per established diagnostic convention. [3, 17] Clinical provocation testing was performed by a second examiner, independent of and blinded to the ultrasound findings, so that ultrasound and clinical assessments were conducted without knowledge of each other's results.

## Statistical Analysis

All analyses were conducted in Python (v3.11) using SciPy, Statsmodels, and Scikit-learn. Statistical significance was set at  $\alpha = 0.05$  (two-tailed). Confidence intervals were computed using the Wilson score method for proportions. All 52 participants completed the full assessment protocol (bilateral ultrasound and both clinical provocation tests), with no missing observations for any variable analyzed.

*Descriptive analysis.* AE prevalence was computed at the elbow level and participant level. Bilaterality was defined as AE present in both elbows. Subgroup comparisons by sex, side, and hand dominance used chi-square or Fisher's exact test as appropriate.

*Clustering-adjusted analysis.* Because each participant contributed two elbows, standard logistic regression treating elbows as independent would underestimate standard errors and inflate type I error. [1, 11, 22] We therefore applied Generalized Estimating Equations (GEE) with an exchangeable working

correlation structure and robust (sandwich) variance estimation, clustering on participant ID. Primary predictors were AE presence, sex, and hand dominance. Outcomes modeled were positive Tinel's sign, positive elbow flexion test, and symptomatic arm status. GEE results are reported as odds ratios (OR) with 95% confidence intervals.

*Effect-size analysis.* For AE versus non-AE comparisons, we calculated absolute risk difference (ARD), relative risk (RR), crude odds ratio (Fisher's exact), Cohen's  $h$ , number needed to harm (NNH =  $1/ARD$ ), attributable fraction among exposed (AF<sub>e</sub>), population attributable fraction (PAF), and population attributable risk (PAR).

*Association and agreement.* Because no electrodiagnostic reference standard was available, no test was treated as a diagnostic gold standard. The association between AE presence and each provocation sign was quantified using odds ratios (crude and GEE-adjusted), and agreement between the two provocation tests was assessed using Cohen's  $\kappa$ . As a secondary descriptive measure, the sensitivity and specificity of AE presence for symptomatic status (defined by the provocation tests, without reference to AE) were computed.

*Dose-response analysis.* An ordinal variable was constructed representing the number of positive provocation tests per elbow (0, 1, or 2). Spearman rank correlation was used to test whether AE prevalence increased monotonically across this gradient.

*Bilaterality analysis.* Participants were stratified into three groups (no AE, unilateral AE, and bilateral AE), and symptomatic rates were compared across groups.

*Multiple comparisons.* Given the number of secondary, subgroup, and interaction analyses performed, no formal correction for multiple comparisons was applied to these exploratory tests; the primary GEE-adjusted associations between AE and each provocation sign were prespecified, whereas subgroup and interaction findings (e.g., the hand-dominance association) should be interpreted as hypothesis-generating rather than confirmatory.

## Results

### Participant Characteristics and AE Prevalence

Fifty-two participants (26 male, 26 female; mean age  $21.6 \pm 3.8$  years; range 18–39) were included, contributing 104 elbows. Fifty-one participants (98.1%) were right-hand dominant (Table 1).

Table 1  
Participant Characteristics

| Characteristic                                                                    | Value                  |
|-----------------------------------------------------------------------------------|------------------------|
| Participants, N                                                                   | 52                     |
| Elbows assessed, N                                                                | 104                    |
| Age, years, mean $\pm$ SD (range)                                                 | 21.6 $\pm$ 3.8 (18–39) |
| Sex, male, n (%)                                                                  | 26 (50.0%)             |
| Sex, female, n (%)                                                                | 26 (50.0%)             |
| Hand dominance, right, n (%)                                                      | 51 (98.1%)             |
| Hand dominance, left, n (%)                                                       | 1 (1.9%)               |
| SD, standard deviation. Each participant contributed two elbows (left and right). |                        |

Ultrasound identified the AE muscle in 32 of 104 elbows (30.8%; 95% CI 22.7–40.2%). By participant, 26 of 52 (50.0%) had AE in at least one elbow: 20 (38.5%) had unilateral AE and 6 (11.5%) had bilateral AE. Among participants with any AE, bilateral occurrence was observed in 23.1% (6/26).

AE prevalence did not differ significantly by elbow side (right 17/52, 32.7% vs. left 15/52, 28.8%;  $\chi^2$   $p$  = 0.832), by sex (male 19/52, 36.5% vs. female 13/52, 25.0%;  $\chi^2$   $p$  = 0.288), or by hand dominance (dominant 32.7% vs. non-dominant 28.8%;  $\chi^2$   $p$  = 0.832). Table 2 and Fig. 2 summarize all elbow-level descriptive statistics.

Table 2  
Descriptive Statistics: AE Prevalence at Elbow and Participant Level

| Variable                                                                                                 | n/N    | %     | 95% CI     | p-value |
|----------------------------------------------------------------------------------------------------------|--------|-------|------------|---------|
| AE Present (all elbows)                                                                                  | 32/104 | 30.8% | 22.7–40.2% | —       |
| AE – Right elbow                                                                                         | 17/52  | 32.7% | 21.5–46.2% | Ref.    |
| AE – Left elbow                                                                                          | 15/52  | 28.8% | 18.3–42.3% | 0.832   |
| AE – Male elbows                                                                                         | 19/52  | 36.5% | 24.8–50.1% | Ref.    |
| AE – Female elbows                                                                                       | 13/52  | 25.0% | 15.2–38.2% | 0.288   |
| Participants: No AE                                                                                      | 26/52  | 50.0% | 36.8–63.2% | —       |
| Participants: Unilateral AE                                                                              | 20/52  | 38.5% | 26.5–52.0% | —       |
| Participants: Bilateral AE                                                                               | 6/52   | 11.5% | 5.4–23.1%  | —       |
| Tinel’s sign positive                                                                                    | 13/104 | 12.5% | 7.5–20.2%  | —       |
| Elbow flexion test positive                                                                              | 32/104 | 30.8% | 22.7–40.2% | —       |
| Either test positive (symptomatic)                                                                       | 34/104 | 32.7% | 24.4–42.2% | —       |
| CI, confidence interval (Wilson score method). p-values by chi-square test for side and sex comparisons. |        |       |            |         |

## Association of AE with Clinical Provocation Tests

Elbow-level test-positivity rates are shown in Table 3 and Fig. 3. Among AE-positive elbows, 25.0% (8/32) demonstrated a positive Tinel’s sign compared with 6.9% (5/72) in AE-absent elbows (Fisher’s exact OR = 4.47; 95% CI 1.27–18.8;  $p = 0.021$ ). The elbow flexion test was positive in 50.0% (16/32) of AE-positive elbows versus 22.2% (16/72) of AE-absent elbows (OR = 3.50; 95% CI 1.40–9.04;  $p = 0.006$ ).

Symptomatic status (either test positive) was observed in 53.1% (17/32) of AE-positive versus 23.6% (17/72) of AE-negative elbows (OR = 3.67; 95% CI 1.43–9.96;  $p = 0.006$ ).

Table 3  
Crude Association: Clinical Test Positivity by AE Status (Elbow-Level, N = 104)

| Outcome                                                                                                                                    | AE Present (n = 32) | AE Absent (n = 72) | Crude OR (95% CI) | p-value |
|--------------------------------------------------------------------------------------------------------------------------------------------|---------------------|--------------------|-------------------|---------|
| Tinel's sign positive                                                                                                                      | 8/32 (25.0%)        | 5/72 (6.9%)        | 4.47 (1.27–18.8)  | 0.021   |
| Elbow flexion test positive                                                                                                                | 16/32 (50.0%)       | 16/72 (22.2%)      | 3.50 (1.40–9.04)  | 0.006   |
| Symptomatic (either test +)                                                                                                                | 17/32 (53.1%)       | 17/72 (23.6%)      | 3.67 (1.43–9.96)  | 0.006   |
| OR, odds ratio; CI, 95% confidence interval by Fisher's exact test. These are crude estimates; clustering-adjusted results are in Table 4. |                     |                    |                   |         |

## Clustering-Adjusted GEE Analysis

After accounting for within-participant elbow clustering using GEE, AE remained a significant independent predictor of all three outcomes (Table 4). The adjusted OR for positive Tinel's sign was 5.46 (95% CI 1.54–19.4;  $p = 0.009$ ); for elbow flexion test, 3.52 (95% CI 1.28–9.74;  $p = 0.016$ ); and for symptomatic status, 3.72 (95% CI 1.31–10.64;  $p = 0.014$ ). Hand dominance was associated with provocation-test positivity: a positive Tinel's sign was significantly less frequent in dominant arms (5.8%, 3/52) than in non-dominant arms (19.2%, 10/52) (adjusted OR 0.22; 95% CI 0.06–0.74;  $p = 0.015$ ). A consistent but non-significant protective direction was observed for the elbow flexion test (adjusted OR 0.53;  $p = 0.064$ ) and symptomatic status (adjusted OR 0.53;  $p = 0.051$ ). Sex was not a significant modifier in any model.

Table 4

GEE-Adjusted Associations: AE Presence and Clinical Outcomes (Clustered by Participant, N = 104 elbows / 52 participants)

| Outcome / Predictor                                                                                                                      | $\beta$ (coef.) | SE    | z     | Adj. OR (95% CI)  | p-value |
|------------------------------------------------------------------------------------------------------------------------------------------|-----------------|-------|-------|-------------------|---------|
| <b>Tinel's sign positive</b>                                                                                                             |                 |       |       |                   |         |
| AE present                                                                                                                               | 1.697           | 0.647 | 2.625 | 5.46 (1.54–19.4)  | 0.009   |
| Male sex                                                                                                                                 | −0.356          | 0.664 | −0.54 | 0.70 (0.19–2.58)  | 0.592   |
| Dominant arm                                                                                                                             | −1.528          | 0.627 | −2.44 | 0.22 (0.06–0.74)  | 0.015   |
| <b>Elbow flexion test positive</b>                                                                                                       |                 |       |       |                   |         |
| AE present                                                                                                                               | 1.256           | 0.520 | 2.415 | 3.52 (1.28–9.74)  | 0.016   |
| Male sex                                                                                                                                 | −0.716          | 0.542 | −1.32 | 0.49 (0.17–1.41)  | 0.186   |
| Dominant arm                                                                                                                             | −0.635          | 0.343 | −1.85 | 0.53 (0.27–1.04)  | 0.064   |
| <b>Symptomatic (either test +)</b>                                                                                                       |                 |       |       |                   |         |
| AE present                                                                                                                               | 1.317           | 0.535 | 2.464 | 3.72 (1.31–10.64) | 0.014   |
| Male sex                                                                                                                                 | −0.485          | 0.542 | −0.89 | 0.62 (0.21–1.78)  | 0.371   |
| Dominant arm                                                                                                                             | −0.628          | 0.322 | −1.95 | 0.53 (0.28–1.00)  | 0.051   |
| GEE, generalized estimating equations; exchangeable working correlation; robust (sandwich) standard errors; clustered on participant ID. |                 |       |       |                   |         |

## Effect-Size Analysis

Table 5 presents the full battery of effect-size estimates for AE versus non-AE elbows. For symptomatic status, the absolute risk difference was 29.5 percentage points (RR = 2.25; Cohen's  $h$  = 0.619, a medium effect). The number needed to harm was 3.4, indicating that for approximately every 3–4 additional elbows with AE, one additional elbow becomes symptomatic. The attributable fraction among exposed was 55.6%, and the population attributable fraction was 17.1%.

Table 5  
Effect-Size Analysis: AE-Positive vs. AE-Negative Elbows

| Measure                                                                                                                                                         | Tinel's Sign   | Elbow Flexion Test | Symptomatic Status |
|-----------------------------------------------------------------------------------------------------------------------------------------------------------------|----------------|--------------------|--------------------|
| Risk in AE+ ( $p_1$ )                                                                                                                                           | 25.0%          | 50.0%              | 53.1%              |
| Risk in AE- ( $p_0$ )                                                                                                                                           | 6.9%           | 22.2%              | 23.6%              |
| Absolute Risk Difference (ARD)                                                                                                                                  | 18.1%          | 27.8%              | 29.5%              |
| Relative Risk (RR)                                                                                                                                              | 3.60           | 2.25               | 2.25               |
| Crude Odds Ratio (OR)                                                                                                                                           | 4.47           | 3.50               | 3.67               |
| Cohen's h (effect size)                                                                                                                                         | 0.514 (medium) | 0.589 (medium)     | 0.619 (medium)     |
| Number Needed to Harm (NNH)                                                                                                                                     | 5.5            | 3.6                | 3.4                |
| Attributable Fraction (exposed)                                                                                                                                 | 72.2%          | 55.6%              | 55.6%              |
| Population Attributable Fraction                                                                                                                                | 22.2%          | 17.1%              | 17.1%              |
| Cohen's h: small $\geq 0.20$ , medium $\geq 0.50$ , large $\geq 0.80$ . NNH = number of AE-positive elbows per additional symptomatic elbow attributable to AE. |                |                    |                    |

## Dose–Response Analysis

A statistically significant positive dose–response gradient was observed between the number of positive provocation tests and AE prevalence (Spearman  $r = 0.307$ ,  $p = 0.002$ ). AE was present in 21.4% (15/70) of elbows with no positive tests, 43.5% (10/23) with one positive test, and 63.6% (7/11) with two positive tests. This monotonic increase supports a graded anatomical–clinical relationship (Fig. 4).

## Bilaterality Analysis

Symptomatic rates differed across the three participant-level bilaterality groups (Table 6). Among participants with no AE, 26.9% (7/26) had at least one symptomatic elbow. This rose to 65.0% (13/20) among those with unilateral AE and was 50.0% (3/6) in those with bilateral AE. The highest symptomatic burden was observed in the unilateral AE group, consistent with a scenario in which one AE-bearing elbow is symptomatic against an asymptomatic contralateral control.

Table 6  
Bilaterality Analysis: Symptomatic Rates by AE Group (Participant Level, N = 52)

| AE Group                                                                                                                                                                                                               | N  | Any Symptomatic Elbow | Comment                                         |
|------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|----|-----------------------|-------------------------------------------------|
| No AE (0 elbows)                                                                                                                                                                                                       | 26 | 7/26 (26.9%)          | Baseline symptomatic rate                       |
| Unilateral AE (1 elbow)                                                                                                                                                                                                | 20 | 13/20 (65.0%)         | Highest symptomatic burden                      |
| Bilateral AE (2 elbows)                                                                                                                                                                                                | 6  | 3/6 (50.0%)           | Bilateral variant; potential partial adaptation |
| Symptomatic = at least one elbow with a positive provocation test (Tinel's or flexion). The trend across No AE → Unilateral → Bilateral is not strictly monotonic; unilateral AE confers the highest symptomatic risk. |    |                       |                                                 |

## Interaction and Dominance Analyses

No statistically significant interaction was detected between AE status and sex ( $p = 0.59$ ), AE and hand dominance ( $p = 0.83$ ), or AE and bilaterality in GEE models. Dominant AE-positive elbows had a symptomatic rate of 47.1% (8/17) compared with 60.0% (9/15) in non-dominant AE-positive elbows; this difference was not statistically significant.

## AE as a Marker of Symptomatic Status; Inter-Test Agreement

Because the cohort lacked an electrodiagnostic reference standard, AE and the provocation signs are best interpreted as correlated markers rather than as diagnostic tests against a gold standard (see Limitations). Evaluated as a predictor of symptomatic status (defined by the provocation tests, without reference to AE), ultrasound-detected AE showed a sensitivity of 50.0%, specificity of 78.6%, PPV of 53.1%, and NPV of 76.4% (Table 7); because AE is a binary marker, its corresponding AUC (0.643) equals its balanced accuracy and reflects modest discrimination above the 0.50 chance level. Agreement between Tinel's sign and the elbow flexion test was fair (Cohen's  $\kappa = 0.38$ ; 95% CI 0.15–0.60). We do not present ultrasound AE detection as the best-performing diagnostic test: because AE presence would define part of any AE-inclusive composite target, such a comparison would be circular.

Table 7

Association Between Ultrasound-Detected AE and Positive Clinical Provocation Signs (Per Elbow; N = 104 elbows / 52 participants)

| Provocation sign                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                 | Crude OR (95% CI) | GEE-adjusted OR (95% CI) | p (adj) |
|--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|-------------------|--------------------------|---------|
| Tinel's sign positive                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                            | 4.47 (1.27–18.8)  | 5.46 (1.54–19.4)         | 0.009   |
| Elbow flexion test positive                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                      | 3.50 (1.40–9.04)  | 3.52 (1.28–9.74)         | 0.016   |
| Either test positive (symptomatic)                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                               | 3.67 (1.43–9.96)  | 3.72 (1.31–10.64)        | 0.014   |
| Crude ORs by Fisher's exact test; adjusted ORs from GEE (exchangeable correlation, robust SEs, clustered on participant, adjusted for sex and hand dominance). Agreement between Tinel's sign and the elbow flexion test was fair (Cohen's $\kappa$ = 0.38; 95% CI 0.15–0.60). <i>Secondary descriptive measure:</i> AE presence as a marker of symptomatic status: sensitivity 50.0%, specificity 78.6%, PPV 53.1%, NPV 76.4%; AUC 0.643 (equal to balanced accuracy for a binary marker). Diagnostic-accuracy metrics are not reported against a disease reference standard because none (e.g., electrodiagnostic confirmation) was available. |                   |                          |         |

## Discussion

This cross-sectional study provides the first comprehensive, clustering-adjusted analysis of the association between ultrasound-detected AE and standardized clinical provocation tests for cubital tunnel syndrome. Several findings merit emphasis.

**AE prevalence and bilateral pattern.** The observed AE prevalence of 30.8% exceeds commonly cited figures from cadaveric (5–27%) and MRI (8–23%) series, though it falls within the upper range of published estimates. The high prevalence in this cohort may reflect the young, athletic demographic (mean age 21.6 years) and the sensitivity of high-resolution ultrasound as a detection method. Notably, 50% of participants had at least one AE-positive elbow, and bilateral occurrence was found in 11.5%, accounting for 23.1% of AE-positive participants. This bilateral proportion is consistent with known bilateral anatomical variants.

**Clinical correlation and clustering-adjusted effects.** The key methodological advance in this analysis is the use of GEE to account for the bilateral data structure. Previous studies analyzing AE and clinical outcomes have frequently treated elbows as statistically independent, inflating effective sample size and underestimating standard errors. [1, 11, 22] Our GEE-adjusted odds ratios (Tinel OR = 5.46; flexion OR = 3.52) were consistent in direction and magnitude with crude Fisher's estimates, providing confidence that the observed associations are robust to statistical correction (Fig. 5). AE presence was independently associated with both provocation tests after adjustment for sex and dominant hand, which themselves showed no significant effects, reinforcing that AE carries independent explanatory weight.

**Dose-response gradient.** The monotonic increase in AE prevalence across the spectrum from 0 to 2 positive tests (21.4% → 43.5% → 63.6%; Spearman  $r$  = 0.307,  $p$  = 0.002) provides evidence of a biologically plausible gradient. Dose-response patterns are considered one of Bradford Hill's canonical

criteria for causal inference in observational research. While cross-sectional design precludes causal claims, this pattern strengthens the argument that AE presence is not merely incidentally associated with ulnar-nerve irritability but may mechanistically contribute to it, possibly through dynamic compression during elbow flexion or increased tissue bulk within the cubital tunnel. [10]

**Contrast with prior literature.** Our findings appear to contrast with systematic-review data suggesting AE is underrepresented among CuTS surgical patients, potentially implying a protective role. Several explanations reconcile these observations. First, our participants are predominantly young and asymptomatic at baseline (not CuTS surgical candidates), so AE detected by provocation tests represents subclinical irritability, not confirmed neuropathy. Second, electrodiagnostic confirmation was not performed, and provocation tests have inherent false-positive rates. Third, hypertrophy, a proposed mechanism for symptomatic AE, may be more prevalent in our active cohort. Our data do not support classification of AE as universally protective.

**Interpretation as correlated markers.** We deliberately refrain from presenting ultrasound AE detection as the best-performing diagnostic test. Because AE presence would define part of any AE-inclusive composite target, and because this cohort had no electrodiagnostic reference standard, formal diagnostic-accuracy comparisons would be circular and are not interpretable here (Fig. 6). The clinically informative finding is that AE presence is associated with a higher rate of positive provocation signs (Tables 2–3), with a graded dose–response relationship that motivates, but does not by itself establish, a role for ultrasound in the workup of suspected CuTS.

**Effect-size interpretation.** An NNH of 3.4 for symptomatic status (meaning approximately 3–4 elbows with AE must be examined to encounter one additional symptomatic elbow) indicates a substantial association burden. Cohen’s  $h$  of 0.619 represents a medium effect by conventional thresholds. The attributable fraction among exposed of 55.6% suggests that, in AE-positive arms, over half of the excess in provocation-positive presentations is statistically associated with AE presence, although causal attribution cannot be established from cross-sectional data.

**Hand dominance.** An unexpected secondary observation was that dominant arms were less likely than non-dominant arms to exhibit positive provocation signs, reaching significance for Tinel’s sign (adjusted OR 0.22;  $p = 0.015$ ) with a consistent protective direction for the flexion test and symptomatic status. This runs counter to the intuition that the dominant, more heavily used limb would show greater ulnar-nerve irritability. One speculative explanation is posture- and loading-related: individuals frequently rest weight on the non-dominant elbow or sleep with it flexed, which may sustain ulnar-nerve pressure on that side. This finding should nonetheless be interpreted with caution: it rests on a small number of positive tests (13 positive Tinel’s signs across 104 elbows), the crude association did not reach significance, and no correction was made for multiple comparisons. We therefore regard it as hypothesis-generating, warranting confirmation in larger, prospectively designed cohorts.

## Limitations

Several limitations warrant acknowledgment. First, diagnosis of CuTS was based solely on clinical provocation tests without electrodiagnostic confirmation (nerve conduction studies or electromyography); accordingly, “symptomatic” designates a positive provocative response and should not be equated with confirmed ulnar neuropathy. Second, the sample is predominantly young (mean 21.6 years) and nearly universally right-hand dominant, limiting generalizability to older or occupationally diverse populations. Third, the cross-sectional design precludes causal inference; it is not possible to determine whether AE predisposed to symptoms or whether symptomatic individuals were selected in ways that increased AE detection. Fourth, AE hypertrophy, a proposed causative mechanism, was not quantitatively assessed; future studies incorporate AE cross-sectional-area measurement would strengthen mechanistic inference. Fifth, despite GEE adjustment for clustering, the sample of 52 participants (26 AE-positive) limits power for interaction analyses and multivariable adjustment. Sixth, the ethnic composition and referral context of this cohort were not explicitly controlled for and may represent an uncharacterized source of selection bias. Seventh, formal intra- and inter-observer reliability of ultrasound-based AE classification was not assessed across the full cohort; repeat scanning was performed only in individual cases judged ambiguous at the time of examination, rather than as a systematic, blinded reliability protocol (e.g., a prespecified subsample re-scanned by the same or a second examiner with a reported agreement statistic). The reproducibility of AE detection in unambiguous cases therefore remains unquantified, and future studies should formally establish this given AE's variable echogenicity relative to surrounding fascia. Future prospective studies should integrate electrodiagnostic data, quantitative ultrasound (nerve and muscle cross-sectional area), reliability testing, and longitudinal follow-up to fully characterize AE's role in CuTS pathophysiology.

## Conclusion

Musculoskeletal ultrasound reliably identifies the anconeus epitrochlearis muscle and reveals a prevalence of 30.8% in this cohort, with bilateral occurrence in 11.5% of participants. When clustering of elbows within participants was appropriately accounted for using Generalized Estimating Equations, AE presence remained independently and significantly associated with a positive Tinel's sign (adjusted OR = 5.46), a positive elbow flexion test (adjusted OR = 3.52), and symptomatic status (adjusted OR = 3.72). A dose–response relationship between the number of positive provocation tests and AE prevalence supports a graded anatomical–clinical association. These findings identify ultrasound-detected AE as a candidate anatomical correlate of ulnar-nerve irritability. Before ultrasound AE assessment can be recommended for the routine clinical workup of cubital tunnel syndrome, prospective studies in symptomatic patients, incorporating electrodiagnostic confirmation and quantitative imaging, are needed.

## Declarations

**Ethics Approval.**

The study was conducted in accordance with the ethical principles of the Declaration of Helsinki. Ethical approval was obtained from the Northumbria University Research Ethics Committee (Approval No. 7087).

### **Consent to Participate.**

Written informed consent was obtained from all participants before data collection.

### **Competing Interests.**

The authors declare no competing interests.

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## **Author Contribution**

S.J. conceived and designed the study. F.A., A.B., M.F., Y.S., and M.K. performed data collection and management. V.S. and A.P. performed the data analysis. S.J. wrote the main manuscript text. All authors reviewed the manuscript.

## **Data Availability**

The datasets generated during the current study are available from the corresponding author on reasonable request.

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# Figures

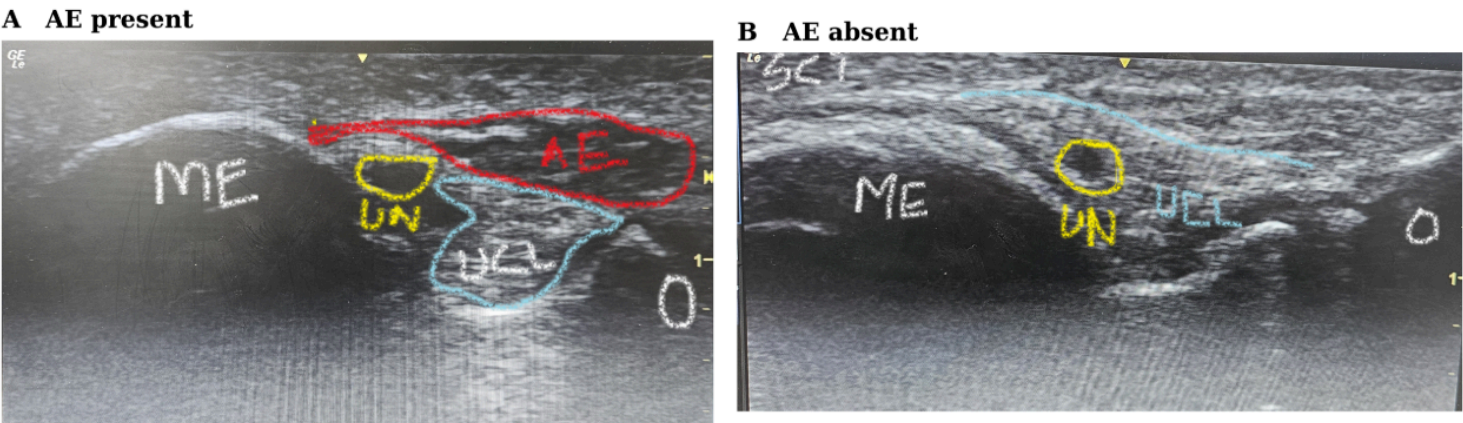


Figure 1

Representative musculoskeletal ultrasound images of the medial elbow. **(A)** AE-positive elbow, showing the anconeus epitrochlearis (AE) bridging the medial epicondyle (ME) and olecranon, with the ulnar nerve (UN) and ulnar collateral ligament (UCL) shown for anatomical reference. **(B)** AE-negative elbow from a different participant, showing the medial epicondyle (ME), ulnar nerve (UN), and ulnar collateral ligament (UCL), with no accessory muscle identified in the expected AE location

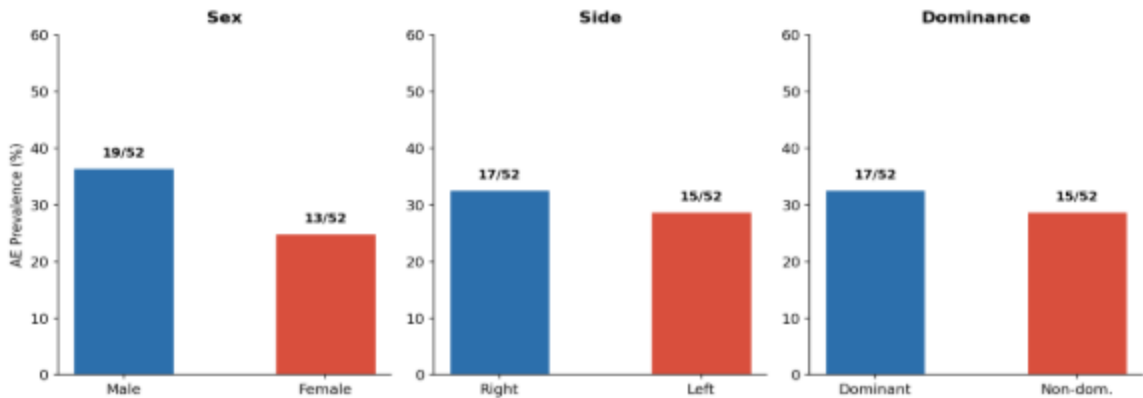


Figure 2

AE muscle prevalence stratified by sex, side, and hand dominance. No significant differences were observed across subgroups (all  $p > 0.05$ )

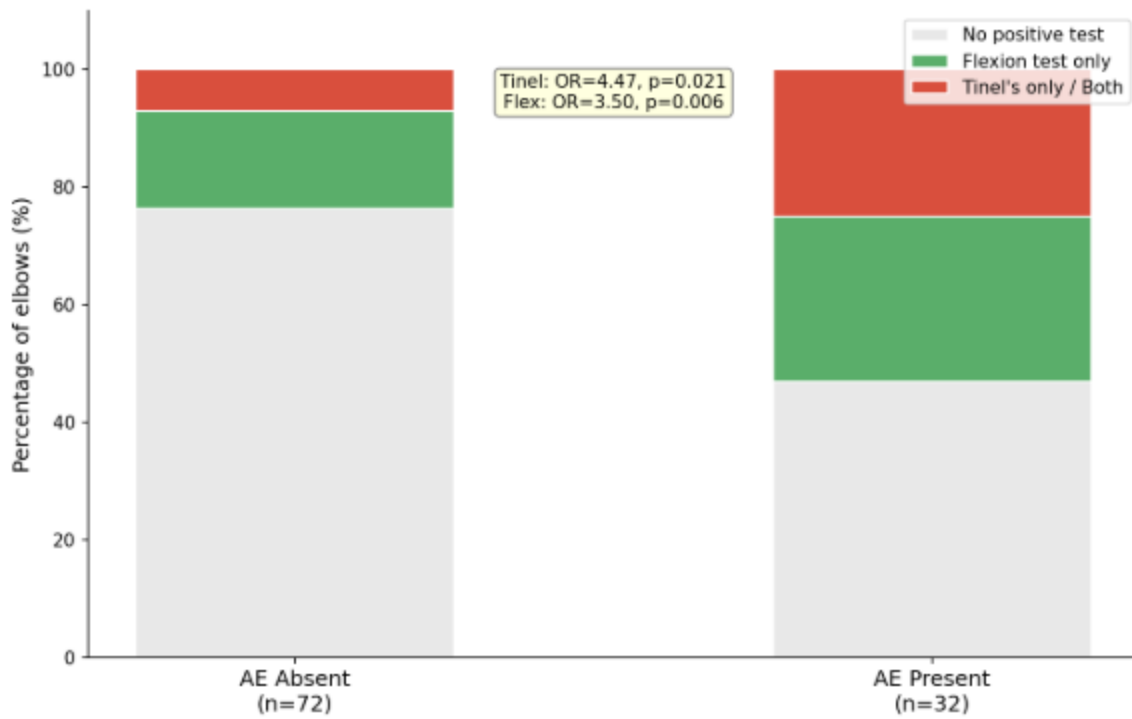


**Figure 3**

Clinical test positivity rates by AE status. AE-positive elbows demonstrated significantly higher rates of both Tinel’s sign (OR = 4.47, p = 0.021) and elbow flexion test positivity (OR = 3.50, p = 0.006)

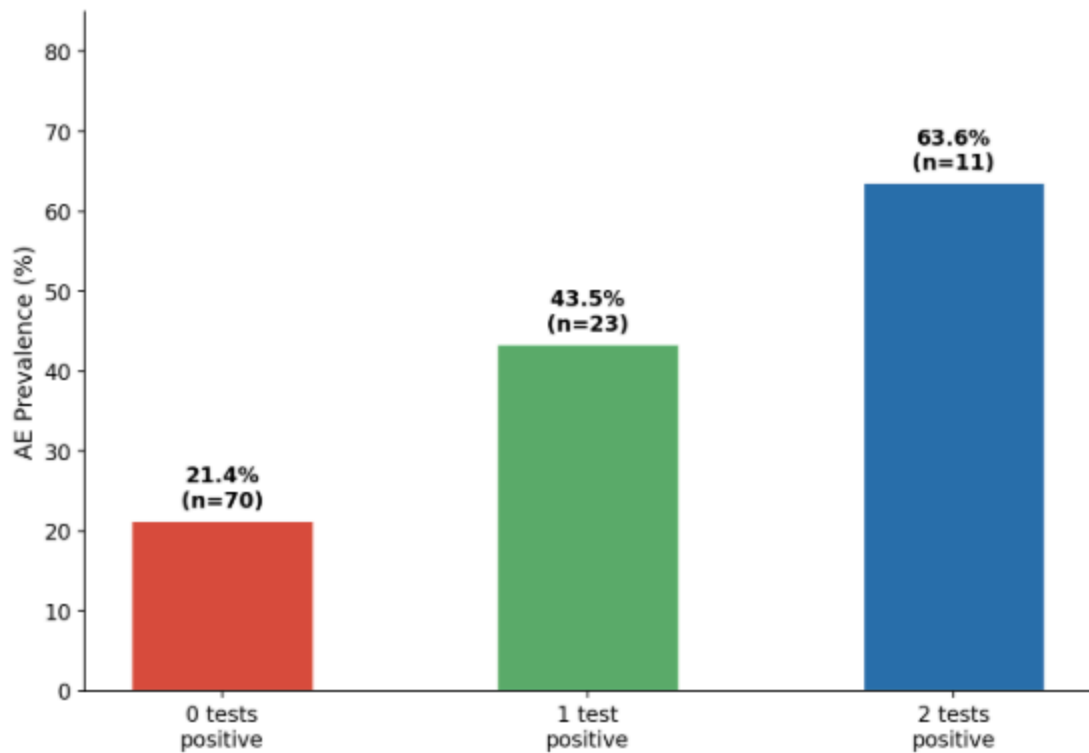


**Figure 4**

Dose-response gradient: AE prevalence by number of positive provocation tests (0, 1, or 2). Monotonic increase supports a graded anatomical-clinical relationship (Spearman  $r = 0.307$ ,  $p = 0.002$ )

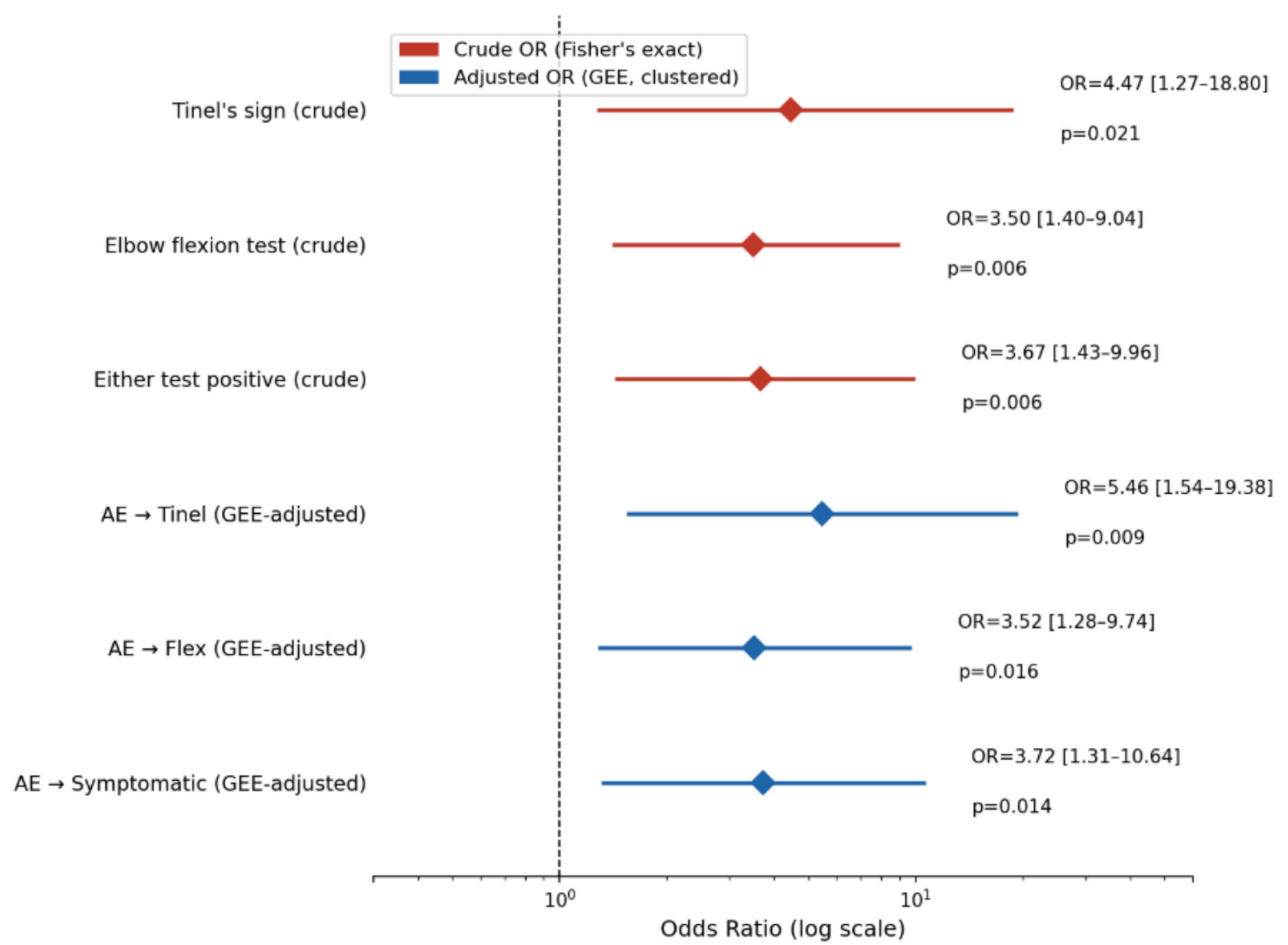


Figure 5

Forest plot of crude (Fisher's exact) and GEE-adjusted odds ratios for the association between AE presence and positive clinical signs. All estimates favour increased risk with AE presence; dashed line = null (OR = 1.0)

**A AE presence vs. symptomatic status**

|               |            | Symptomatic status |              |
|---------------|------------|--------------------|--------------|
|               |            | Symptomatic        | Asymptomatic |
| Ultrasound AE | AE present | 17                 | 15           |
|               | AE absent  | 17                 | 55           |

Sensitivity 50.0% · Specificity 78.6% · PPV 53.1% · NPV 76.4%  
Balanced accuracy (AUC) = 0.643 (0.50 = chance)

**B Inter-test agreement**

|              |         | Elbow flexion test |           |
|--------------|---------|--------------------|-----------|
|              |         | Flexion +          | Flexion - |
| Tinel's sign | Tinel + | 11                 | 2         |
|              | Tinel - | 21                 | 70        |

Cohen's  $\kappa$  = 0.38 (fair agreement)  
Observed agreement 77.9% · n = 104 elbows

**Figure 6**

AE as a marker of symptomatic status, and inter-test agreement. **(A)** Cross-classification of ultrasound-detected AE against symptomatic status ( $\geq 1$  positive provocation test) across all 104 elbows, with derived marker performance: sensitivity 50.0%, specificity 78.6%, PPV 53.1%, NPV 76.4%, and balanced accuracy (AUC) 0.643 (0.50 = chance). Because AE is a binary marker, the AUC equals its balanced accuracy. **(B)** Agreement between Tinel's sign and the elbow flexion test (Cohen's  $\kappa$  = 0.38; observed agreement 77.9%), indicating fair concordance