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Cubital tunnel perfusion in different postures—An anatomical investigation

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Abstract

Introduction/Aims: For cubital tunnel syndrome, the avoidance of predisposing arm positions and the use of elbow splints are common conservative treatment options. The rationale is to prevent excessive stretching and compression of the nerve in the cubital tunnel, as this mechanical stress impedes intraneural perfusion. Data regarding those upper extremity postures to avoid, or whether elbow flexion alone is detrimental, are inconsistent. This study aimed to assess perfusion and size changes of the cubital tunnel during different postures in an experimental cadaver setup.

Methods: Axillary arteries in 30 upper extremities of fresh cadavers were injected with ultrasound contrast agent. High-resolution ultrasound of the cubital tunnel was performed during five different arm postures that gradually increased tension on the ulnar nerve and caused cubital tunnel narrowing. Contrast enhancement within the tunnel was measured to quantify perfusion. Cubital tunnel cross-sectional area was measured to detect compression.

Results: Increasing tension significantly reduced perfusion. When isolated, neither shoulder elevation, elbow flexion, pronation, nor extension of wrist and fingers impaired perfusion. However, combining two or more of these postures led to significant decreases. Significant narrowing of the cubital tunnel was seen in full elbow flexion and shoulder elevation.

Discussion: Combinations of some upper extremity joint positions reduce nerve perfusion, but isolated elbow flexion does not have a significant impact. We hypothesize that elbow splints alone may not influence cubital tunnel perfusion but may only prevent direct compression of the ulnar nerve. Advising patients about upper extremity postures that should be avoided may be more effective.

KEYWORDS
contrast-enhanced ultrasound, nerve entrapment, perfusion, posture, ulnar neuropathy

Abbreviation: CSA, cross-sectional area.

1 | INTRODUCTION

Cubital tunnel syndrome is the second most common compression mononeuropathy.[1] Symptoms are caused by two factors: direct compression of the ulnar nerve within the cubital tunnel and tension on the nerve as it is stretched during upper extremity movement.[2-5] Both factors impede intraneural perfusion.[6-9] If untreated, persistent entrapment of the nerve leads to focal nerve damage, resulting in hypesthesia, paresis, and muscle atrophy.[3]

Various approaches to conservative treatment are found in contemporary literature, but optimal conservative treatment of cubital tunnel syndrome is unclear.[10,11] Local perineural injection of corticosteroids has shown no benefit,[12,13] ultrasound therapy and low-level laser therapy,[14] as well as nerve gliding exercises[15,16] have been investigated only in small cohorts. The current mainstays of conservative treatment are splints to inhibit elbow flexion and patient education to avoid predisposing upper extremity postures. It remains unclear which measure is most effective at reducing symptoms.[10,17,18]

The influence of upper extremity postures on nerve compression and tension has been shown in previous studies,[2,19-21] but their influence on cubital tunnel perfusion has not yet been investigated. The aim of this study was to assess cubital tunnel perfusion in different upper extremity postures that cause tension on the nerve and cubital tunnel narrowing with direct compression of the structures within.

2 | METHODS

This study was approved by the institutional review board of our institution (EK 2256/2016). Non-embalmed cadavers were randomly selected as cadavers became available to the study center. Before their death, body donors had signed informed consent, approving the use of their bodies for educational and research purposes. Cadavers with medical records of any kind of neuromuscular disease, previous surgery to the upper extremities, or signs of trauma were excluded.

Bodies were severed at the upper cervical spine and the level of the diaphragm, preserving the continuity of the ulnar nerve to the spinal cord. At room temperature, the cadaver was placed supine and the axillary artery was cannulated using an 18-gauge peripheral intravenous catheter (Becton Dickinson, Franklin Lakes, New Jersey). Brachial veins and brachial plexus were left unaltered. The intact veins allowed for as much drainage of contrast agent/saline as possible to prevent any accumulation in the upper extremity.

The upper extremity was placed and fixed according to posture 1 (Table S1 and Figure S1), avoiding undue force, while leaving the cadaver supine. In this posture, ultrasound contrast agent was injected into the axillary artery for 30 seconds at a constant pressure of 140 mmHg (190.33 cmH_2O); the perfusion pressure was achieved by suspending the infusion bottle from an intravenous pole of the appropriate height. We used a mixture of 5 mL of SonoVue (Bracco International BV, Amsterdam, The Netherlands) diluted in 500 mL of normal saline. This contrast agent is based on gas-filled microbubbles that stay within the blood vessels and do not pass into the extravascular space. Thus, contrast enhancement is only visualized from blood vessels. We used this contrast enhancement as a surrogate for perfusion.

A 9-MHz linear ultrasound probe (9L-D; General Electric, Boston, Massachusetts) was positioned in a transverse plane, perpendicular to the ulnar nerve at the cubital tunnel. It was held between the tip of the medial epicondyle and the posterior tip of the olecranon. All scans were performed using a standard ultrasound system (Logiq e9; General Electric). Ultrasound still images were stored at 0, 5, 10, 15, 20, 25, and 30 seconds.

The contrast agent within the arm was then destroyed and thus rendered invisible to ultrasound. This was achieved by bursting its microbubbles with ultrasound of a high mechanical index (mechanical index refers to the mechanical / thermic forces of ultrasound waves an investigated tissue is exposed to). The upper extremity posture was then changed to posture 2 and the full procedure with contrast agent injection and destruction was repeated until data from all postures were collected.

Postures were designed as described in previous studies.[2,22] The focus was put on adding joint positions that cause elongation of the ulnar nerve and thus create tension; less emphasis was placed on creating postures of everyday usage.

After examination in all five postures, the cubital tunnel was dissected and inspected by a neuropathologist and a neurosurgeon to rule out any pathologies or scar tissue affecting the results.

In each ultrasound still image, a region of interest (Figure FIGURE S2) containing the cubital tunnel was defined. The outline was put at the surface of the medial epicondyle, the olecranon, and Osborne ligament. In this region of interest, containing the ulnar nerve, its accompanying vascularization, and the surrounding gliding apparatus, we measured contrast enhancement as a ratio of pixels showing contrast enhancement vs pixels with no enhancement using ImageJ software (National Institutes of Health, Bethesda, Maryland). This served as a surrogate for perfusion. The cross-sectional area (CSA) of the cubital tunnel was measured in all postures to detect direct compression.

Statistically the cumulative effect of all individual upper extremity joint postures on cubital tunnel perfusion was estimated by calculating the area under the curve for each posture according to the trapezoidal rule. Mean area under the curve values of contrast enhancement were analyzed with a mixed linear model, including cadavers as levels of a random effect and arm postures as fixed within-subject factors. P values corresponding to pairwise comparisons were adjusted using the Bonferroni correction. GraphPad Prism 7.04 (GraphPad Software, San Diego, California) was used to generate graphs. A two-sided $P < .05$ was considered statistically significant.

3 | RESULTS

Thirty upper extremities of six male and nine female cadavers were examined. Their mean age was 81.40 (range, 51.42-94.31) years. Five extremities (16.67%) did not show homogeneous contrast enhancement of the nerve or surrounding tissues in any posture. We assumed disseminated postmortem thrombosis or occlusion of a large proximal

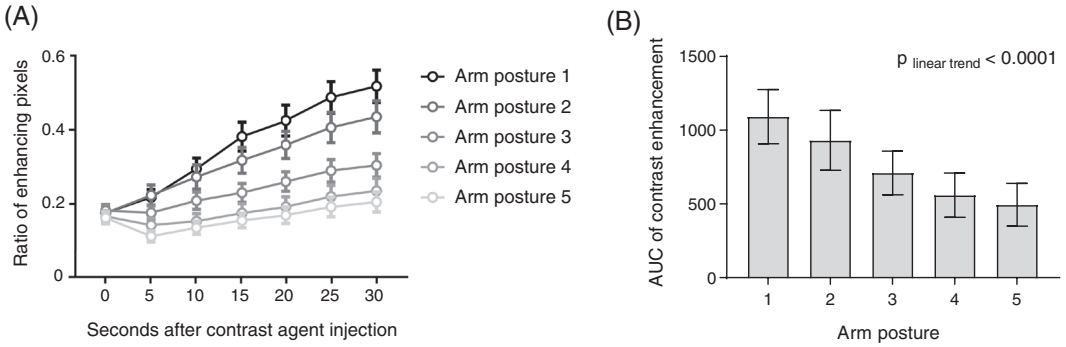

FIGURE 1 Course of contrast enhancement. A, Comparison of the time course of enhancement in the five different arm postures. The y axis shows the ratio of pixels with contrast enhancement vs pixels without enhancement, serving as a surrogate for perfusion. B, Area under the curve results for contrast enhancement in the five different arm postures

TABLE 1 Direct comparison of arm postures using mean area-under-the-curve values of contrast enhancement

Arm posture vs arm posture	Fold difference	*P* value[a]
1 vs 5	0.45	<.0001[b]
1 vs 4	0.53	<.0001[b]
1 vs 3	0.70	.0002[b]
1 vs 2	0.88	1
2 vs 5	0.51	.0001[b]
2 vs 4	0.60	.002[b]
2 vs 3	0.79	.185
2 vs 1	1.13	1
3 vs 5	0.65	.001[b]
3 vs 4	0.76	.091
3 vs 2	1.27	.185
3 vs 1	1.44	.0002
4 vs 5	0.85	1
4 vs 3	1.31	.091
4 vs 2	1.66	.0002[b]
4 vs 1	1.88	<.0001[b]
5 vs 4	1.17	1
5 vs 3	1.54	.001[b]
5 vs 2	1.95	<.0001[b]
5 vs 1	2.21	<.0001[b]

Note: Difference in area under the curve (AUC) for posture 1 vs posture 5 is 0.45, as shown in line 1. This means that the AUC of posture 1 multiplied by 0.45 equals the AUC of posture 5.
[a]*P* values adjusted using Bonferroni correction.
[b]Significant increase or decrease ($P < .05$).

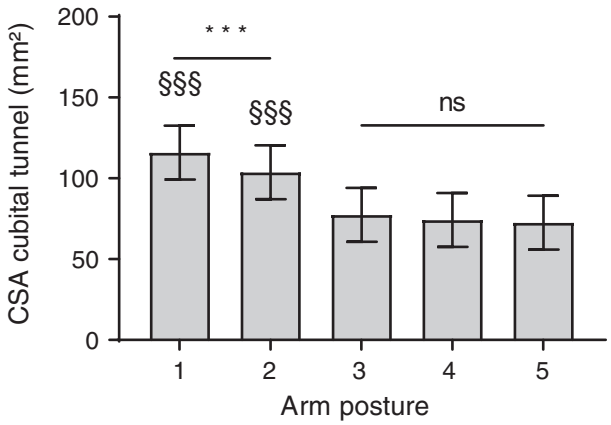

FIGURE 2 CSA (given as mm^2) of the cubital tunnel in arm postures 1 to 5. The CSAs of the cubital tunnel in postures 1 and 2 differed significantly from each other ($^{***}P < .001$) and were higher than in postures 3, 4, and 5 ($^{§§§}P < .001$ each). Thus, maximum elevation of the shoulder and maximum flexion of the elbow led to a significant decrease of the CSA. No significant difference was found between postures 3, 4 and 5 (ns; 3 vs 4, $P = .380$; 3 vs 5, $P = .175$; 4 vs. 5, $P = .631$). Abbreviation: CSA, cross-sectional area; ns, not statistically significant

vessel and excluded these specimens, resulting in 25 arms eligible for statistical analysis.

Contrast agent signal at 0 seconds was almost equal throughout all cadavers and postures (Figure 1), indicating adequate comparability. A slight decrease of signal within the initial seconds in arm postures 3 to 5 was found to be a technical artifact in all arm postures.

Mean area-under-the-curve values of contrast enhancement decreased significantly from posture 1 to posture 5 ($P < .0001$; Figure 1), indicating that increasing tension correlates with reduced perfusion.

None of the individual joint positions (shoulder elevation, full elbow flexion, pronation, extension of wrist and fingers) alone caused a significant decrease in perfusion (Table 1). However, when compared with posture 1 representing maximum relaxation, shoulder elevation plus elbow flexion (posture 3) impeded perfusion significantly. Adding more tension (postures 4 and 5) further decreased the perfusion.

CSA of the cubital tunnel in postures 1 and 2 differed significantly from each other. The CSAs of postures 1 and 2 also were significantly higher than in postures 3, 4, and 5. No significant difference was found between postures 3, 4, and 5 (Figure 2). Thus, maximum shoulder

elevation and maximum elbow flexion led to a significant decrease in CSA of the cubital tunnel. The minimum CSA was 27 mm^2, while a maximum of 181 mm^2 (standard deviation = 33 mm^2) was measured.

4 | DISCUSSION

Our results show that cubital tunnel perfusion decreased significantly when tension increased. No singular joint position caused a significant decrease in perfusion. However, a combination of two or more joint positions did cause a significant decrease.

Elbow flexion alone did not significantly reduce perfusion, but it caused a significant narrowing of the cubital tunnel. A significant decrease in CSA was also found with elevation of the shoulder. We assume the impact of shoulder movement is due to tension of the triceps brachii muscle on the tendinous structures of the cubital tunnel.[23] The CSA measurements in this study are in line with previously published data.[19] Another cadaver study[2] investigated tension on the ulnar nerve in upper extremity postures. The greatest increase in tension on the ulnar nerve at the elbow was found during combined shoulder abduction (110°), elbow flexion, pronation, as well as wrist and finger extension. This is in line with our results, resulting in a minimized perfusion in posture 5.

In entrapment neuropathies, chronic low pressure on the nerve causes neural thickening due to edema within and around the nerve. If persistent, this is followed by fibrosis, myelin turnover, and ultimately the loss of axons. Exposure to high pressures may cause direct arterial ischemia.[24] Longstanding compression of the ulnar nerve fibers thus may lead to irreversible neural damage with sequential hypesthesia, atrophy, and contractures.[3] Omejec and Podnar[25] postulated two distinct mechanisms behind cubital tunnel syndrome. In patients whose work involves strenuous use of the arms, the ulnar nerve is entrapped under the ligamentous roof of the cubital tunnel that becomes thickened over years. The authors differentiated this from compression of the nerve in the retroepicondylar groove by external structures, affecting individuals not performing heavy manual labor. In the former, more severe symptoms and sonographic nerve constrictions were found, and surgery was advocated. In the latter, there were no nerve constrictions and conservative treatment was suggested.

In this study we examined the ulnar nerve within the cubital tunnel below the Osborne ligament; thus, our data contribute mainly to knowledge about the mechanism of true ligamentous nerve compression. In our specimens, we found direct compression by full elbow flexion and shoulder elevation, but neither of these postures alone reduced blood flow. We therefore hypothesize that permanent compression within a narrowed cubital tunnel in patients may not impair perfusion of nerve and surrounding tissue, even with the elbow flexed. Further narrowing of the tunnel by shoulder abduction or additional tension by joint movements is necessary to significantly reduce the blood flow. This in part contradicts Omejec and Podnar,[25] who hypothesized that tension due to arm postures is of little pathophysiological relevance, as provoking activities were not different in their two patient cohorts with or without direct nerve compression. According to our data, patient education may still be useful for these individuals.

This conclusion is limited by the fact that all but one of our cadaver specimens resembled "healthy patients," as they did not show sonographic signs of cubital tunnel syndrome. It is unknown whether body donors had symptoms of ulnar nerve dysfunction or nerve conduction abnormalities.

Another limitation is that our postures resembled a stepwise increase of tension, starting with shoulder abduction and elbow flexion. Thus, tension was mostly combined with compression, and individual effects on perfusion are hard to differentiate. This should be considered in future studies. Nevertheless, comparison of postures 3 and 5 shows a significant decrease of perfusion by increasing tension, without changing the cubital tunnel diameter.

Future studies should also include postures that resemble situations of daily living, such as reading a book or the prolonged use of a mobile phone, as the postures used in our study and similar work may be rare in daily living.

A study in healthy subjects[26] recorded nerve conduction velocities in postures similar to postures 1, 2, and 3 in this study. A significant difference in conduction velocity was found between postures 1 and 3. Moreover, with the elbow flexed, addition of shoulder abduction caused significant changes of conduction velocities along the limb (although this was nonsignificant across the elbow). These changes in conduction velocity resemble our findings regarding perfusion and, in our opinion, underline the relevant impact of tension. Nerves may adapt to extremity movements by their partly elastic properties and by longitudinal gliding facilitated by the surrounding gliding apparatus.[2,27-29] Adaptation is not possible in cases of nerve entrapment syndromes, postoperative scar formation, or degradation of the gliding apparatus.[2,28,30] This resulting tension on the nerve may be detrimental.

Clinical evidence on the best strategy for conservative treatment in cubital tunnel syndrome is scarce and controversial,[11,17,18] and there are no consistent recommendations about which specific postures to avoid. Shah et al[17] reported successful treatment in 88% of mild to moderate cases with a combination of rigid night splinting and patient education for 3 months. A randomized trial[18] showed that, using patient education, 89.5% of patients with mild to moderate cubital tunnel syndrome improved after 6 months, whereas night splinting or nerve gliding exercises did not add any benefit. According to our findings, elbow splints may only prevent direct compression of the nerve. However, when full elbow flexion is made impossible by a splint, cubital tunnel perfusion may be less susceptible to the compromising effects of shoulder elevation, pronation, or the extension of wrist and fingers. It remains speculative whether impairment of ulnar nerve perfusion due to certain postures, as shown in this study, is a relevant mechanism behind the clinical success of patient education.

We chose an experimental setup with cadaver specimens for two reasons. First, in a cadaver, it is possible to optimize tissue contrast enhancement that would have been of inferior quality in volunteers

due to a lower concentration of contrast agent. Second, we could exclude local pathology by dissection of the entire region, a process that would have been impossible in volunteers.

This study has limitations. Due to the experimental setup with cadavers, our results may only serve as a surrogate for the clinical situation in patients. Further clinical research is necessary to evaluate and verify our findings in both healthy volunteers and patients with cubital tunnel syndrome. Simultaneous nerve conduction studies may reveal subclinical effects of the postures applied. Moreover, including patients who had recently undergone simple ulnar decompression may help distinguish the effects of external compression and posture-related elongation of the nerve. Another limitation is the advanced mean age of the body donors and their uniform race, which limits the generalizability of our data to the general population. Degenerative changes of the elbow joint as a cause of nerve compression appeared not to influence our data; measurements of the mean CSA of the cubital tunnel were comparable with data from a study using MRI.[19] Simultaneous analysis of pressure, tension, and perfusion within the cubital tunnel would have added further insights. However, inserted probes would have altered the perineural connective tissue and subsequently its perfusion.

In conclusion, in this study we found that one joint position alone that puts the ulnar nerve under mechanical stress does not significantly reduce perfusion in a cadaver setting. Only the combination of at least two specific upper extremity joint positions will lead to diminished perfusion. Postures causing perfusion impairment, such as postures 3 to 5, should be avoided based on our data. Further clinical research is necessary to validate the findings of this anatomical study.

CONFLICT OF INTEREST

The authors declare no potential conflicts of interest.

ETHICAL PUBLICATION STATEMENT

We confirm that we have read the Journal's position on issues involved in ethical publication and affirm that this report is consistent with those guidelines.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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SUPPORTING INFORMATION

Additional supporting information may be found in the online version of the article at the publisher's website.

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