



Reinnervation assessment using a modified nerve conduction study protocol following end-to-side anterior interosseous nerve transfer in advanced cubital tunnel syndrome: A case series and proof of concept study

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KEYWORDS

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Summary *Introduction:* Supercharge end-to-side (SETS) anterior interosseous nerve (AIN) transfer augments motor recovery in advanced cubital tunnel syndrome (CuTS). However, standard electrodiagnostic studies cannot differentiate between native ulnar nerve regeneration and reinnervation via AIN transfer. We aimed to establish a proof-of-concept for a modified nerve conduction study (NCS) protocol by employing median nerve stimulation to electrophysiologically isolate and evaluate AIN-mediated motor recovery.

Methods: We retrospectively analyzed 13 patients with advanced CuTS who underwent AIN SETS transfer (mean follow-up duration: 1.6 years). Postoperative NCS was performed by stimulating the median nerve at the elbow and recording compound muscle action potentials (CMAPs) from the first dorsal interosseous (FDI) and abductor digiti minimi (ADM). Contralateral measurements were obtained at the follow-up for comparative reference.

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Results: All 13 patients demonstrated FDI CMAPs upon median nerve stimulation. Among the 8 patients with longitudinal data, 6 showed a progressive increase in the median nerve-stimulated CMAP amplitudes over time. The mean median nerve-stimulated FDI CMAP was 3.1 mV on the operative side versus 2.3 mV on the contralateral side; however, the difference was not statistically significant. Notably, no median nerve-stimulated CMAP responses were recorded from the ADM in any patient.

Discussion: This modified NCS protocol demonstrated median nerve-stimulated CMAP at the FDI, confirming reinnervation after AIN SETS transfer. The selective CMAP recovery in the FDI, but not in the ADM, reflects a site-specific reinnervation pattern resulting from AIN coaptation to FDI-specific ulnar fibers. Therefore, future prospective studies in larger cohorts are warranted to refine this protocol as a standard clinical assessment.

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Cubital tunnel syndrome (CuTS) is the second most common compressive neuropathy of the upper extremity.^{1,2} In advanced cases, chronic axonal degeneration leads to irreversible intrinsic muscle atrophy and substantial functional loss.³ Standard surgical treatments, including in-situ decompression, medial epicondylectomy, and anterior transposition of the ulnar nerve, have been used to alleviate mechanical compression and facilitate axonal regeneration.^{2,4} Although effective in stopping ongoing nerve damage, these procedures have notable limitations when used in severe cases.³ Native ulnar axons regenerate slowly (approximately 1 mm/day) often requiring over six months to reach the intrinsic hand muscles.⁵ During this period, the denervated muscles remain vulnerable to motor endplate degeneration and irreversible atrophy, even after successful decompression. Supercharge end-to-side (SETS) nerve transfer was introduced to mitigate this delay. It provides early axonal input during ulnar regeneration, which helps in preserving motor end-plates and supporting reinnervation. By providing an early source of axons closer to the target muscles, the anterior interosseous nerve (AIN) SETS transfer procedure preserves motor endplates while the native ulnar axons regenerate.⁶ Furthermore, the first dorsal interosseous (FDI) muscle is known to exhibit more consistent and robust recovery than the abductor digiti minimi (ADM) after AIN SETS transfer.⁷⁻⁹

However, objectively validating the specific contribution of the transferred AIN remains a challenge. Moreover, conventional nerve conduction studies (NCS) cannot distinguish between native ulnar nerve recovery and AIN-mediated reinnervation. Prior studies relied on subjective measures, such as pinch strength or patient-reported outcomes, making it difficult to isolate the effect of AIN SETS from decompression alone.⁶⁻⁹ Although Robinson et al. proposed the use of volitional maneuvers to identify AIN-to-ulnar crossover,¹⁰ its reliance on patient effort and the associated technical difficulty limit its clinical reproducibility. To address this limitation, we developed a modified, purely objective NCS protocol based on evoked compound muscle action potentials (CMAPs). The protocol involves stimulating the median nerve at the elbow while recording CMAPs from the FDI and ADM muscles, both of which are innervated by the ulnar nerve.

We hypothesized that this protocol would detect the presence of reinnervation and reveal a site-specific pattern,

thereby distinguishing true neural recovery from potential artifacts such as volume conduction. This exploratory study presents a proof-of-concept for the neurophysiological validation of AIN SETS transfer, thereby evaluating its feasibility and longitudinal outcomes in patients with advanced CuTS.

Methods

Study design and patient selection

This retrospective case series and proof-of-concept study was reported in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines and the completed STROBE checklist is provided as [Supplementary Material](#). We retrospectively reviewed patients who were diagnosed with CuTS and underwent surgery at the authors' institution, a tertiary referral hospital, between January 2022 and December 2024. The indication for AIN SETS was advanced CuTS with clinically apparent atrophy of the FDI muscle. Electrodiagnostic findings included reduced CMAP amplitudes in the FDI and ADM muscles with or without spontaneous activity (fibrillation potentials or positive sharp waves). Patients demonstrating increased motor unit action potentials (MUAPs) with reduced recruitment were also considered as candidates, based on the electrodiagnostic criteria described in a previous study.¹¹ All patients underwent standard cubital tunnel release with concurrent AIN SETS, which was performed by a single hand surgeon. Thirteen patients who underwent follow-up examinations via our new post-surgical NCS protocol after AIN SETS were included in this analysis.

AIN SETS procedure

Following ulnar nerve decompression, the AIN, which supplies the pronator quadratus, was transected and coapted to the ulnar motor branch using an end-to-side technique on the volar-radial side.^{6,12} The ulnar motor branch was identified using a nerve stimulator without additional dissection at the wrist level, in accordance with the techniques described in recent studies.

Median nerve stimulation postoperative NCS protocol

To evaluate the specific contribution of AIN-to-ulnar nerve reinnervation, we employed a modified NCS protocol. The median nerve was stimulated just below the antecubital fossa, proximal to the AIN origin. Supramaximal stimulation was applied to ensure maximal axonal recruitment and CMAPs were recorded from the FDI and ADM muscles. Standard electrode placement and belly-tendon montage were used to ensure reproducibility. Identical measurements were also obtained from the asymptomatic contralateral limb to serve as a comparative electrophysiological reference.

Initially, the protocol also included needle electromyography (EMG) to evaluate volitional MUAPs from the ADM and FDI during finger abduction and forearm pronation, an action that activates the AIN.¹⁰ Separate needle EMG recordings during finger abduction alone and combined finger abduction with pronation were required to assess AIN innervation of the intrinsic muscles. However, it was discontinued owing to inherent subjectivity of effort-dependent maneuvers and technical difficulty in isolating dynamic forearm movements without introducing movement artifacts. To ensure inter-examiner reliability and objectivity, we transitioned to a purely evoked-response NCS protocol.

We postulated that owing to the “babysitting” mechanism and time required for axonal regeneration and synaptic engagement, at least 6 months following surgery is required for the signs of reinnervation to become evident.^{9,13} Therefore, patients were examined 6 months postoperatively. Initially, we intended to examine patients 6-, 12-, and 18-months after surgery, but the timing and frequency of checks varied based on patient availability and preference. Patients were included if they underwent at least one follow-up using this protocol.

Clinical assessment

Functional outcomes were categorized using the modified McGowan grading system, which classifies CuTS severity based on sensory and motor deficits: Grade I indicates minimal symptoms, Grade IIA indicates mild motor weakness without atrophy, Grade IIB indicates intrinsic muscle atrophy and weakness, and Grade III indicates severe motor and sensory impairment.¹⁴

Statistical analysis

Owing to the exploratory nature of this proof-of-concept study and heterogeneous follow-up intervals, formal inferential statistical analyses were limited. Descriptive analysis was prioritized to characterize the longitudinal electrophysiological changes within individual subjects.

Results

Patient characteristics

Thirteen patients underwent the AIN SETS surgery. There were 8 men and 5 women, with a mean age of 62.4 ± 13.6

Table 1 Demographic and surgical characteristics of the patients (n = 13).

Characteristic	Value
Sex	Male: 8 (61.5%), Female: 5 (38.5%)
Mean age $\pm$ SD, year	62.4 ± 13.6 (range 28-79)
BMI $\pm$ SD, kg/m ²	26.0 ± 7.1
McGowan stage	IIB: 10 (76.9%), III: 3 (23.1%)
Surgical side	Right: 10 (76.9%), Left: 3 (23.1%)
Etiology	Compression with OA: 9 (69.2%) Trauma: 3 (23.1%) Pure compression: 1 (7.7%)
Additional surgical procedures	Epicondylectomy: 6 (46.2%) Transposition: 2 (15.4%) Debridement arthroplasty: 2 (15.4%)

BMI, body mass index; OA, osteoarthritis.

years (range, 28-79 years). The mean duration of symptoms was 1.6 years (range, 0.5-5 years). Patients with McGowan stage IIB-III CuTS were included. Ten patients had stage IIB and 3 had stage III. The dominant limb was affected in 10 patients. The mean body mass index (BMI) was 26 ± 7 kg/m². The most common etiology was compressive neuropathy owing to elbow osteoarthritis, observed in 9 patients (69%). Three patients (23%) had trauma-related ulnar neuropathy, and 1 (8%) had idiopathic compression without osteoarthritis or trauma. In addition to ulnar nerve decompression at the elbow, medial epicondylectomy was performed in 6 patients (46%), nerve transposition in 2 (15%), and combined debridement arthroplasty with nerve transposition in 2 (15%). The demographic profiles, surgical laterality, etiologies, and adjunct procedures performed on these patients are summarized in [Table 1](#).

Clinical outcomes

All 13 patients reported improvement in sensory symptoms following surgery. At the latest follow-up visit, 5 patients reported a subjective improvement in motor function. Favorable outcomes were more commonly observed in younger patients, those who experienced a shorter duration of symptoms prior to surgery, and cases of CuTS secondary to trauma rather than primary compression neuropathy. However, given the small sample size, these trends were not statistically significant.

Postoperative NCS outcomes

Thirteen patients were followed up at least once using the new postoperative NCS protocol. [Table 2](#) describes the patients' demographic data and the preoperative and final follow-up NCS FDI CMAP values. Preoperative FDI CMAP was low, 0.8 mV, whereas at the final postoperative follow-up, the CMAP of the ulnar nerve was 1.9 mV. The mean duration of the last follow-up NCS was 13 months. Despite the resolution of the conduction block, the ulnar nerve-stimulated FDI CMAP did not recover sufficiently. Contrastingly, the mean median nerve-stimulated FDI CMAP was 3.1 mV,

Table 2 Electrophysiological Outcomes Following Anterior Interosseous-to-Ulnar Motor Nerve Supercharge End-to-side Transfer.

Patient number	Sex/ Age, year	Final follow-up period (mo.)	Preoperative ulnar nerve-stimulated FDI CMAP (mV)	Final postoperative ulnar nerve- stimulated FDI CMAP (mV)	Serial postoperative median nerve- stimulated FDI CMAP (mV) <i>Affected side</i>	Final postoperative median nerve- stimulated FDI CMAP (mV) <i>Unaffected side</i>
1	M/67	25	0.3	0.6	4.1→4.1 ($\Delta 0.0$)	3.1
2	F/77	12	2.9	4.6	2.3→2.6 ($\Delta +0.3$)	1.8
3	M/75	16	0.5	0.5	1.9	1.5
4	M/66	24	0.1	1.8	2.4→3 ($\Delta +0.6$)	2
5	M/28	12	1.3	3.4	2.6	2.9
6	F/57	18	0.4	2.7	3.8→6.3 ($\Delta +2.5$)	1.5
7	M/64	12	0.5	0.2	1.6→3.4 ($\Delta +1.8$)	3.6
8	M/52	12	0.6	3.5	1.2→2.4 ($\Delta +1.2$)	1.6
9	M/64	15	1.4	2.3	3.2	1.8
10	F/49	11	1	1.7	2.2→4.1 ($\Delta +1.9$)	3.8
11	F/68	12	0.7	1.7	1.7→2.2 ($\Delta +0.5$)	4.5
12	F/79	6	0	0.1	1.4	1.9
13	M/65	6	0.7	1.7	3.4	0.2
Average (mV)			0.8 ± 0.8	1.9 ± 1.4	3.1 ± 2.2	2.3 ± 1.2

NCS; nerve conduction study; CMAP, compound muscle action potential; FDI, first dorsal interosseous; mo., month; mV, millivolt; Δ = final postoperative minus first postoperative median nerve-stimulated FDI CMAP. One patient (Patient 6, Case A) had three serial measurements; the serial column reports the first and last values.

which was higher on the contralateral side's 2.3 mV, though this difference was not statistically significant. (Table 2) Additionally, among the 8 cases observed more than twice under the new protocol, 6 showed an interval amplitude increase in the median nerve-stimulated FDI CMAP, whereas 2 remained stationary. Crucially, this response was consistently absent or negligible in the ADM across all patients, supporting the specificity of the FDI reinnervation.

Case presentation: Case A

A 57-year-old woman presented with an advanced right CuTS. Physical examination revealed classic signs of advanced CuTS, including positive Tinel's sign, elbow flexion test, scratch collapse test, Egawa test, and Froment's sign. Additionally, sensory changes in the ulnar nerve distribution, visible atrophy of the FDI, and an abduction deformity were observed. The range of motion of the elbow was nearly complete. She underwent AIN SETS transfer along with ulnar nerve decompression and medial epicondylectomy. Preoperatively, the motor NCS showed severely reduced CMAP in the FDI and ADM muscles upon ulnar nerve stimulation.

Although the ulnar nerve conduction block resolved quickly (velocity increased from 23.9 to 53.3 m/s), the ulnar nerve-stimulated FDI CMAP remained stagnant for 12 months. However, the median nerve-stimulated FDI CMAP demonstrated an early and progressive increase, starting at 6 months (3.8 mV) and reaching 6.5 mV at 18 months. Table 3 presents comprehensive electrophysiological data, including ulnar nerve velocity and CMAP amplitudes over time. The ADM showed no consistent response to median nerve stimulation. These findings are consistent with FDI reinnervation via the median nerve, although there was no

evidence of ADM reinnervation.¹³ The patient noted subjective improvement, noting that "the space between the fingers became fuller," and reported satisfaction with the outcome. Figure 1 displays representative CMAP waveforms at 18 months postoperatively, whereas additional pre- and postoperative NCS/EMG tracings for Case A are shown in Supplementary Figures 1-5.

Discussion

Unlike previous studies that relied on subjective functional measures,^{7,9,11,15} our protocol provides objective, electrophysiological evidence of AIN-mediated reinnervation. Notably, FDI CMAP with median nerve stimulation was consistently observed in most patients and demonstrated an increase over time, thereby supporting the hypothesis of physiological FDI reinnervation via the AIN, independent of native ulnar nerve recovery.

In the FDI, median nerve-stimulated CMAP was greater on the operative side than on the unaffected side; however, the difference did not reach statistical significance. The detection of median nerve-stimulated CMAPs on the unaffected side suggests potential volume conduction from thenar muscle activation rather than true reinnervation.¹⁶ However, an increase was observed in CMAP amplitude on the operative side in patients who underwent serial examinations; however, no increase was not observed on the unaffected side.

However, in the ADM, low-amplitude CMAPs observed after median nerve stimulation were similar to those observed on the unaffected side, and no interval increase was identified on serial examinations. This indicates that volume conduction rather than genuine reinnervation is

Table 3 Longitudinal electrophysiological changes in the first dorsal interosseous and abductor digiti minimi muscles of case A (case 6 in Table 2) following anterior interosseous-to-ulnar motor nerve supercharge end-to-side transfer.

Time point	Muscle	Ulnar nerve velocity above the elbow(m/s)	Ulnar nerve velocity below the elbow(m/s)	Ulnar nerve-stimulated CMAP (mV)	Median nerve-stimulated CMAP (mV) <i>Affected side</i>	Median nerve-stimulated CMAP (mV) <i>Unaffected side</i>	Interpretation of bold values
Preoperative	FDI	23.9	43.7	0.4	NA	NA	Severe lesion with conduction block at elbow (23.9 m/s)
POD 6M	FDI	53.3	52.9	0.3	3.8	NA	Conduction block resolved after CutR (53.3 m/s). Persistent loss of ulnar nerve-evoked CMAP (0.3 mV), but Median nerve-evoked CMAP (3.8 mV) indicates AIN-mediated reinnervation.
POD 1Y	FDI	50.5	56.4	0.4	4.9	1.8	Sustained reinnervation (4.9 mV) with persistent loss of ulnar nerve-evoked CMAP (0.4 mV).
POD 1Y6M	FDI	52.2	56.6	2.7	6.5	1.5	Increase in CMAP amplitude (6.5 mV) and delayed improvement of ulnar nerve function from CuTR (2.7 mV)
Preoperative	ADM	27.9	44	2.8	NA	NA	Severe lesion with conduction block at elbow (27.9 m/s)
POD 6M	ADM	55.8	54.6	2.9	0.4	NA	Conduction block resolved after CutR (55.8 m/s). Low-amplitude CMAP (0.4 mV) likely owing to volume conduction from adjacent muscles
POD 1Y	ADM	53.3	52.6	3.0	NR	NR	No evidence of sustained reinnervation (NR)
POD 1Y6M	ADM	57.8	59.2	3.8	NR	NR	No AIN SETS reinnervation (NR) with delayed improvement of ulnar nerve function from CuTR (3.8 mV)

POD, postoperative day; M, months; Y, year; CMAP, compound muscle action potential; FDI, first dorsal interosseous; ADM, abductor digiti minimi; mV, millivolt; ms, millisecond; NA, not assessed; NR, no response; AIN, anterior interosseous nerve; CutR, cubital tunnel release.

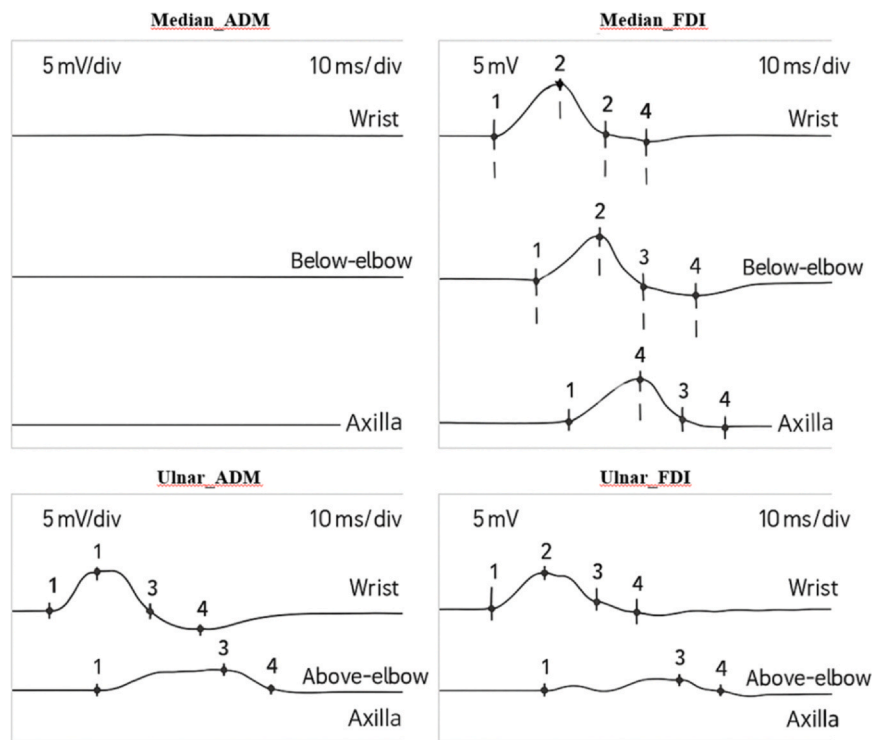


Figure 1 Compound muscle action potential (CMAP) waveforms of the abductor digiti minimi (ADM) and first dorsal interosseus (FDI) muscles at 18 months postoperatively. CMAPs were recorded after median and ulnar nerve stimulation. Stimulation of the median nerve elicits a clear response in the FDI but not in the ADM, indicating successful reinnervation via anterior interosseous nerve (AIN) supercharge end-to-side (SETS) transfer. In contrast, ulnar nerve stimulation evokes minimal response in the FDI and a preserved response in the ADM, suggesting selective reinnervation of the FDI by the transferred AIN, with limited native ulnar recovery.

attributable to AIN SETS in the ADM.¹⁶ The consistently stronger reinnervation of FDI compared with ADM may reflect anatomical and physiological factors.^{4,7,17,18} A previous study demonstrated differential recovery patterns between FDI and ADM after AIN SETS and suggested that the internal fascicular topography of the ulnar motor branch and direction of AIN coaptation may influence preferential intrinsic muscle recovery, even though this relationship remains inconclusive.⁴ In the present study, the AIN was coapted to the volar-radial aspect of the ulnar motor branch, which may have oriented donor axons toward the presumed FDI fascicular territory rather than ADM fascicles. FDI also receives denser corticospinal input and plays a more prominent role in fine motor control than ADM, which could contribute to its selective reinnervation following nerve transfer.¹⁷ Thus, the lack of serial increase in ADM response indicates that meaningful AIN-mediated ADM reinnervation is unlikely and supports a degree of spatial specificity in our recordings. However, this finding should not be interpreted as definitive exclusion of volume conduction, particularly given the possibility of thenar far-field contamination of the FDI signals. Accordingly, we relied primarily on the time-dependent increase in median-stimulated FDI CMAP on serial studies, rather than on the absence of ADM responses alone, to support ongoing reinnervation.

This study supports the potential clinical role of AIN SETS as an active reinnervation strategy for advanced CuTS.^{6,11}

By introducing median nerve axons into the ulnar motor fascicle, the AIN SETS promotes earlier FDI reinnervation compared to native ulnar regeneration across the elbow, which typically exceeds 6 months. Moreover, the end-to-side coaptation preserves the structural integrity of the ulnar nerve, allowing dual innervation and reducing surgical damage.^{12,18} The longitudinal profile of Case A illustrates the practical utility of our protocol as a proof-of-concept. Although immediate recovery of ulnar nerve conduction velocity indicated successful decompression, the concurrent increase in median nerve-stimulated FDI CMAP on serial studies supports the interpretation that the AIN transfer functionally contributes to ongoing native ulnar recovery.¹⁸

Nevertheless, some limitations must be acknowledged. First, the sample size restricts the statistical power and generalizability of the findings. Additionally, the follow-up duration was relatively short and varied among the patients, which limited standardized time point comparisons and potentially underestimated late reinnervation. Second, although our targeted NCS protocol was intended to allow the selective assessment of AIN-derived responses, potential confounding from anomalous innervation or cross-over connections, including Martin-Gruber anastomosis, could not be entirely excluded because the protocol was not applied preoperatively to establish the baseline responses. Additionally, supramaximal stimulation at this proximal forearm level may increase the risk of activating crossover

fibers and inadvertent co-stimulation of adjacent nerves, including the ulnar nerve, which cannot be fully excluded in this retrospective setting. Importantly, Martin-Gruber anastomosis may preferentially contribute to the FDI; therefore, a median-stimulated FDI CMAP alone cannot reliably exclude this anatomical variation. Therefore, we emphasize the longitudinal pattern: the median-stimulated FDI CMAP showed interval changes on serial studies, which is more consistent with the ongoing reinnervation than with a static anatomical variant. However, without preoperative screening, this possibility cannot be completely ruled out.

In conclusion, this study indicates that AIN SETS transfer may enable meaningful reinnervation of FDI in advanced CuTS. Furthermore, the median nerve stimulation NCS protocol demonstrated the potential to provide objective longitudinal evidence of reinnervation, suggesting its clinical value for surgical planning and outcome assessment. This finding should be regarded as a proof-of-concept, illustrating the viability of a modified median nerve-stimulation NCS protocol for the objective monitoring of reinnervation. Further studies are needed to validate this protocol in larger, prospective cohorts, with standardized follow-up intervals and concurrent functional testing. Such efforts may help more precisely define the therapeutic value, optimal timing, and patient selection criteria for AIN SETS in the treatment of advanced CuTS.

Ethical approval

Not required.

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Declaration of Competing Interest

None declared.

Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at [doi:10.1016/j.bjps.2026.07.018](https://doi.org/10.1016/j.bjps.2026.07.018).

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