Tremor and peripheral nerve entrapment

Case report

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A patient is described in whom pain, paresthesias, weakness, and resting tremor gradually developed 8 years after an ulnar nerve transposition. Electromyography revealed that the tremor occurred at 4 to 5 Hz, was abolished by voluntary muscle contraction, and was localized to ulnar-innervated muscles. Ulnar nerve conduction was locally slowed at the elbow; therefore, ulnar neurolysis was performed and a fusiform neuroma-in-continuity was found. Mechanically tapping the neuroma elicited repetitive discharges at 4 to 5 Hz in the intrinsic muscles of the hand; these discharges were abolished by anesthetic block proximal to the neuroma. Although the pain, paresthesias, and weakness were abolished by the neurolysis, the tremor persisted. Possible neurophysiological mechanisms underlying the appearance of tremor with peripheral nerve entrapment are discussed.

KEY WORDS • tremor • nerve entrapment • ulnar nerve • neurolysis

ALTHOUGH tremor is often associated with central nervous system (CNS) disease, it has been described in patients with peripheral nerve disease and no evidence of CNS dysfunction.1,7 Tremor due to peripheral neuropathy has usually been seen in acquired or hereditary polyneuropathy. Only a few cases of tremor in association with nerve injury or entrapment have been described.2 We present the case of a patient with localized tremor accompanying peripheral nerve entrapment and we outline the effects of surgical release on that tremor.

Case Report

This 38-year-old male carpenter presented with pain, paresthesias, weakness, and tremor in the left hand. Eight years before, he had fallen and struck his left elbow causing the immediate onset of left hand paresthesias and weakness. Over the ensuing month he had developed marked atrophy in the intrinsic muscles of the hand and underwent anterior subcutaneous transposition of the left ulnar nerve at the elbow, with complete resolution of weakness, atrophy, and paresthesias.

One year before his present admission he noted the gradual reappearance of pain and paresthesias in the fourth and fifth digits and ulnar aspect of his left hand. He also developed weakness of the hand and a tremor of the fourth and fifth digits. There was no new trauma to the extremity.

On physical examination, there was decreased pinprick sensation in the fifth digit and ulnar aspects of the fourth digit and of the palm and dorsum of the left hand. No clinically detectable weakness or atrophy was noted in the left hand or forearm. At rest, rhythmic twitching of the left fourth and fifth digits was noted when the forearm was supinated and not supported; this tremor abated when the forearm was pronated with wrist and hand supported. No tremor or ataxia was noted with voluntary movement or in the other upper extremity. Electrodiagnostic studies revealed a normal left ulnar motor distal latency at 8 cm of 2.9 msec to the abductor digiti minimi muscle (ADM). Conduction velocity for the forearm segment was 51.8 m/sec, with a significant decrease in amplitude (8.0 to 5.7 mV) and increase in dispersion of the compound muscle action potential. Inching technique revealed focal slowing and a decrease in amplitude between 4 and 12 cm distal to the medial epicondyle. The left ulnar sensory compound nerve action potential recorded from the fifth digit was small (5 μV) relative to the left median sensory
FIG. 1. Electromyographic recording of the tremor activity obtained from surface electrodes overlying the abductor digiti minimi muscle. Horizontal bar: 500 msec, vertical bar: 1 mV.

FIG. 2. Intraoperative photograph of the neuroma showing a fusiform neuroma-in-continuity (arrow). This neuroma of the previously transposed left ulnar nerve was located just ventral to the medial epicondyle of the humerus.

FIG. 3. Electromyographic activity in response to mechanical tapping of the left ulnar neuroma recorded intraoperatively with surface electrodes overlying the abductor digiti minimi manus muscle. Horizontal bar: 500 msec, vertical bar: 500 μV.

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potential (12 μV) recorded from the second digit, although distal latencies were normal (3.5 msec, ulnar; 3.0 msec, median at 14.0 cm).

Needle electromyography of the left ADM, first interosseus dorsalis, flexor carpi ulnaris, and triceps brachii muscles did not reveal any positive waves or fibrillations. On voluntary recruitment, the interference pattern was mildly reduced in the ADM but was normal in the other muscles. Motor-unit amplitudes, durations, and configurations were normal except for moderately large (4.5 mV) first-order motor units in the ADM.

Rhythmic, nearly synchronous discharges of three to five motor units were recorded with surface electrodes overlying or with a needle electrode within the left ADM (Fig. 1). These discharges accompanied clinical tremor at a frequency of about 4 Hz and stopped with minimal voluntary contraction of the muscle.

Operation. Because of the recurrent ulnar symptoms and the confirmatory electrodiagnostic studies, an ulnar neurolysis was scheduled. At surgery, an incision was made over the medial antecubital fossa, and the previously transposed left ulnar nerve was dissected. A fusiform neuroma-in-continuity was found ventral to the medial epicondyle of the humerus (Fig. 2). The nerve was further constricted as it passed under the tendinous insertions of the flexor carpi ulnaris muscle. On intraoperative recording, the motor conduction velocity across the neuroma was 30 m/sec. Mechanical tapping of the neuroma produced repetitive synchronous discharges of 1 to 4 motor units recordable from the ADM (Fig. 3). Such repetitive discharges occurred at a regular frequency of 4 to 5 Hz for three to five discharges, then stopped spontaneously. These discharges were not elicited on tapping the nerve proximal or distal to the neuroma. Epineural injections of lidocaine hydrochloride (2%) proximal to the neuroma abolished both the tremor and the repetitive discharges in response to tapping of the neuroma. The ulnar nerve was freed from fibrous tissue encircling it at the level of the medial epicondyle, and a fibrous band was sectioned where the ulnar nerve passed between the two heads of the flexor carpi ulnaris muscle.

Postoperative Course. After surgery, the patient described complete resolution of the weakness and paresthesias. However, at the 1-year follow-up examination the resting tremor still persisted, indistinguishable from the preoperative status with a frequency of 4 to 5 Hz.

Discussion

Several observations suggest that the tremor in this patient resulted from peripheral nerve entrapment. First, the tremor was localized to muscles innervated by the left ulnar nerve; this nerve was entrapped in the proximal forearm, both on nerve conduction studies and on direct observation at surgery. Second, no other tremor was present clinically or electromyographically. Third, no other evidence of CNS disease was present either historically or on clinical examination.
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Although the tremor seemingly resulted from peripheral nerve entrapment, it required central input proximal to the entrapment site as evidenced by its disappearance following an anesthetic nerve block. Also, the presence of tremor at rest but primarily with the forearm supinated suggests that stretch reflexes from the ulnar-innervated finger flexor and/or lumbricales muscles may be contributing factors. The absence of rhythmic contractions with passive stretch distinguishes these discharges from clonus, and it implies that central facilitation is necessary to bring out these rhythmic discharges.

The neurophysiological mechanism underlying the tremor in peripheral nerve disease is not known. Said, et al., have suggested that the tremor of peripheral neuropathy may result from minimal weakness and impairment of the stretch reflex. Alternatively, the stretch reflex may become hyperactive; such increased reflex excitability has been described in animals following partial nerve injury or other procedures that reduce sensory input. This increased gain in the reflex pathway would magnify the likelihood of tremor oscillations. Hypothetically, a slowly progressive peripheral nerve entrapment, involving some but not all large-diameter sensory axons, might reduce sensory input to stretch reflex pathways but preserve some reflex connections. The resulting decrease in sensory input might then cause increased synaptic excitability to develop in those afferent fibers that are still functioning.

The repetitive discharges obtained in response to mechanically tapping the neuroma had a frequency identical to the tremor and were abolished by a proximal anesthetic nerve block; thus, these may represent discharges in focally demyelinated afferent fibers at the site of entrapment with orthodromic conduction to the cord and elicitation of hyperactive stretch reflexes. Muscle contraction from this synchronous reflex discharge would then be followed by relaxation and, thus, stretch of muscle spindles with another afferent volley which would excite a second reflex discharge because of synaptic hyperexcitability, similar to the mechanism proposed for tremor in this patient.

Although apparently uncommon, it appears that resting tremor can accompany a partial entrapment of a peripheral nerve. Observations in our present case suggest that releasing the entrapment may not abolish the tremor, even though pain, paresthesias, and weakness are relieved.

References


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