Measurement of Intraoperative Nerve Conduction Velocities During Anterior Interosseous Nerve Decompression

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In 1918, Tinel[1] described an isolated motor palsy of the anterior interosseous branch of the median nerve. In its complete form, the syndrome presents as weakness during forearm pronation with an inability to flex at the interphalangeal (IP) joint of the thumb and the distal interphalangeal (DIP) joint of the index finger. Because of presumed variations in the innervation of the long-finger flexor digitorum profundus (FDP), the syndrome may also present with variable weakness or inability to flex at the DIP joint of the long digit.[2]

While the syndrome remains well documented in the literature, its pathophysiology remains controversial. In 1948, Parsonage and Turner[3] reviewed 136 cases of "neuralgic amyotrophy" and identified 6 patients with anterior interosseous nerve (AIN) involvement. As 5 of the 6 patients had associated shoulder girdle weakness, the authors believed the syndrome represented an abnormality of the anterior horn cells. Subsequently describing 2 cases that responded to conservative treatment, Kiloh and Nevin[4] hypothesized that the syndrome was due to a localized interstitial neuritis of the AIN. Later authors also ascribed the syndrome to viral or inflammatory neuritis and suggested nonoperative management with expectant recovery as the norm.[5]

On the other hand, Fearn and Goodfellow[6] ascribed the syndrome to a compressive neuropathy and reported that a fibrous band from the humeral origin of the pronator teres compressed the nerve. The patient had complete recovery after division of the constricting band. Other authors have also described external compression related to a multitude of anatomical causes resulting in a compressive neuropathy.[7,8]

The disparity in opinions on the pathophysiology of AIN syndrome has led to difficulty in recommendations for surgical indications. In an effort to provide more accurate prognosis for patients and further define the pathophysiology of this syndrome by quantifying the functional effects of compression on the nerve, we conducted intraoperative nerve conduction velocity (NCV) studies during surgical release in 3 patients.

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Case Series

After providing informed consent regarding the risks of intraoperative NCV assessment and the potential benefit of more accurate prognosis, patients underwent general endotracheal anesthesia and external neurolysis of the AIN. A sterile tourniquet was used for the operative exposure and was released after visualization of the nerve above the pronator tunnel and distal to the flexor digitorum superficialis (FDS) arcade. Strict hemostasis was maintained with bipolar electrocautery. Nerve-to-nerve conduction velocity was measured before surgical release of any impinging structures. The NCV study was repeated after release of each potential impinging structure evident under loupe magnification. The stimulating amplitude and the distance between the stimulus and the recording electrode were maintained as constants throughout the procedure.

Case 1

This patient was a right-hand–dominant female in her early 40s with a 6-month history of right thumb weakness. She described a progressive loss of thumb IP joint flexion and flexion of the DIP joint of the index finger during the first 2 months. She also described no viral prodrome and denied complaints of shoulder girdle symptoms or distal paresthesias. On physical examination, she displayed minimal forearm atrophy with absent function of the flexor pollicis longus (FPL) and FDP to the index finger. Sensation to light touch was unaffected. A magnetic resonance imaging (MRI) study performed 5 months after the onset of symptoms demonstrated no evidence of proximal impingement or a supracondylar process.

The patient was scheduled for release of the right AIN 6 months from the onset of her original symptoms. At the time of surgery, an incision was made transversely across the elbow crease and carried distally approximately 4 to 5 cm. The median nerve was dissected and exposed proximally 6 to 7 cm above the elbow, with no evidence of impingement. The nerve was also exposed distal to the FDS arch. Intraoperative nerve-to-nerve conduction velocity then revealed a prerelease amplitude of 40 mV. After release of the fascia from the humeral head of the pronator teres, the NCV was repeated, but no change was recorded. The AIN was then sequentially released distally through

the fibrous origin of the FDS arcade. At this point, the NCV was increased in amplitude to 200 mV. The decision to forgo further surgery was made, and the wound was irrigated and closed.

Two and a half weeks after surgery, the patient was noted to have a complete return of function to the FPL and FDP to the index finger, along with remission of her minimal preoperative forearm atrophy. This result was noted during independent examinations by 2 consulting surgeons. The patient returned to work without limitations.

Case 2

This patient was a left-hand–dominant male in his late 20s with a 7-month history of absent active flexion of the thumb IP joint and the DIP joint of the index finger. The symptoms began with an episode of right arm swelling and pain that was alleviated with continuous elbow flexion. This led to a flexion contracture of the right elbow, which subsequently resolved with physical therapy. However, the patient continued to notice atrophy and wasting of forearm musculature and experienced numbness and tingling in the index finger, long finger, and thumb over the next several months. An MRI study performed to evaluate the right forearm was negative for any intra-articular findings or the presence of a supracondylar process of the humerus but showed signal changes consistent with AIN syndrome. The patient subsequently returned to the clinic complaining of increased right forearm pain. On examination, he exhibited a positive Tinel sign over the pronator teres and lacertus fibrosus and displayed marked atrophy of the forearm with absent function of the FPL and the FDP to the index finger and weak pronation with the elbow in maximum flexion. Sensation to light touch was maintained in the tips of all digits.

The patient was scheduled for AIN release 6 months from the onset of original symptoms. At the time of surgery, an incision was made over the right median nerve just distal to the antecubital fossa, and the skin was elevated. The median nerve was visualized proximally 6 to 7 cm above the elbow, with no evidence of nerve impingement noted. Intraoperative NCV was then performed, stimulating the median nerve at the level of the elbow. Initial NCV readings revealed a latency of 1.3 m/s and an amplitude of 44 mV. The median nerve was then sequentially decompressed distally, with corresponding NCV readings, starting at the level of the pronator teres. NCV results were virtually unchanged until decompression of the FDS arch was achieved. At this point, the nerve conduction latency decreased to 0.74 m/s, and the amplitude increased to 105 mV. The decision to forgo further surgery was made, and the wound was irrigated and closed.

Two weeks after surgery, the patient exhibited full elbow range of motion (ROM) and began to actively flex the thumb IP joint. However, persistent numbness remained in the distribution of the median nerve. At 6 weeks, the previous numbness had subsided, and full active flexion of the thumb IP joint had returned, as did a faint return of active flexion in the index-finger IP joint. Three months after surgery, forearm atrophy and difficulties with pronation had mostly resolved, along with the return of full function in the FPL and FDP.

Case 3

This patient was a right-hand–dominant female in her early 40s with a 3-month history of spontaneous left elbow pain, inability to completely extend the elbow, generalized hand weakness, and paresthesias in the distribution of the median nerve. She exhibited a positive Tinel sign over the lacertus fibrosus and had a positive flexion test. She also specifically described a progressive decrease in active flexion of the thumb IP joint and index-finger DIP joint. Peripheral NCV and electromyogram (EMG) were essentially normal, with the exception of no voluntary motor unit potentials in the FPL and FDP to the second digit. Three months after the onset of symptoms, the patient demonstrated a recovering pronator syndrome but had residual AIN deficits and slight forearm atrophy. On examination, she had absent function of the FPL and FDP to the index finger and weakness in forearm pronation but maintained light touch sensation to the tips of all digits.

She was scheduled for AIN release 3 months from the onset of original symptoms. The surgical procedure was similar to that described for case 2. The median nerve was visualized proximally above the elbow, with no compressive lesions found. Intraoperative NCV was performed stimulating the median nerve at the level of the elbow. Initial NCV readings revealed a latency of 1.7 m/s and an amplitude of 10 mV. The median nerve was sequentially released distally, including a fibrous band at the level of the pronator teres muscle, which was noted to be constricting the nerve with forearm supination. At this point, the intraoperative NCV readings showed a decrease in latency to 1.1 m/s and an increase in amplitude to 45 mV. Decompression of the median nerve was continued distally along its course through the FDS arch; however, the NCV results remained unchanged. The decision to forgo further surgery was made, and the wound was irrigated and closed.

Two weeks after surgery, the patient showed no signs of improvement. At 6 weeks, she exhibited slight flickering of the FDP to the index finger and an occasional flicker of the FPL. However, at 5 months, the patient had a complete return of FPL function, with full FDP function to the index finger returning by 7 months. The patient's final visit was 9 months after surgery, at which time all forearm atrophy had resolved, full elbow ROM had returned, and pinch/grip strength was 80% as compared with the contralateral side.

Discussion

The pathophysiology of AIN syndrome is obviously multifactorial. Anatomical compression of the nerve by fibrous bands and physiologic block of nerve function resulting from inflammation and other means remain common etiologies. AIN syndrome often is a sequelae of trauma[9]; it remains the most common nerve injury after supracondylar humerus fractures in children. However, the majority of

AIN syndromes resulting from trauma subsequently resolve. In one series of 34 children with Monteggia fractures and subsequent AIN palsies, all patients regained full function 4 to 17 weeks after injury.[10] The etiology of these palsies may have been directly related to trauma, constrictive dressings used during subsequent fracture treatment, or both.

The causes of spontaneous AIN palsies remain difficult to determine. If the syndrome is secondary to neuritis of either the AIN or the brachial plexus, then observation is indicated. However, if the syndrome is a compressive neuropathy, then operative decompression may be necessary. Multiple cases document the anatomical causes of AIN compression, including fibrous bands from or within the pronator teres, the FDS, or the lacertus fibrosis. In addition, compression from thrombosed vessels, enlarged bicipital bursae, and tumors has also been reported.[7]

There have been reports in the literature of "hourglass constrictions" of the anterior and posterior interosseous nerves that result in spontaneous onset of AIN syndrome.[11] Nagano[12] described 31 cases of spontaneous AIN syndrome and believed that the incidence of entrapment neuropathy was "very low." He subsequently performed interfascicular neurolysis on 23 patients who did not show any motor recovery by 3 months after onset. No external compression was found along the course of the nerves; however, he noted an hourglass-like fascicular constriction in 22 of the 23 patients. Nagano indicated that the "basic abnormality is this hourglass-like fascicular constriction,"[12] which may result from the mechanical torsion experienced by the fascicles during flexion-extension of the elbow or pronation-supination of the forearm.[13] The difficulty in interpreting this study, however, is that it relies on the subjective interpretation of evidence of external compression. As with any operative decompression, there is a subjective element that represents sufficient external compression to result in the interruption of normal nerve function. However, the ability to associate objective data, such as NCV, with subjective observation strengthens a surgeon's ability to offer a more accurate prognosis to the patient.

All 3 patients in this case series had neurolysis of visible adhesions performed under 5.0 loupe magnification. Based on the electrophysiologic studies in these 3 patients, there is a subgroup of patients with true compressive neuropathy who will respond well to operative decompression. In case 1, the patient's recovery of motor function within 3 weeks of decompression despite a motor palsy of 3 to 6 months' duration remains difficult to explain. The cause may be vulnerability of this pure motor nerve to any external compression. Certainly, improved NCVs after decompression indicate no cellular disruption requiring repair.

Timing of operative decompression was not addressed in this study. The patients in cases 1 and 2 were both unable to perform their respective vocations before surgery and were extremely pleased by the operative outcomes and subsequent rapid recovery. Whether this recovery was fortuitous and would have occurred in any event cannot be determined from the results of this study; however, the speed of the recovery after decompression cannot be underestimated in patients such as these.

Last, intraoperative NCV has a valuable role. In each of our cases, initial nonoperative treatment was based on the assumption that the syndrome may be secondary to neuritis or hourglass constrictions, both of which did not respond to decompression alone. To provide these patients with an accurate prognosis and determine the necessity of a more invasive procedure, such as intrafascicular neurolysis, the NCV studies were performed. In each case, these studies implied to the operative surgeon that decompression alone would result in functional recovery without the need for further surgery. Thus, intrafascicular neurolysis was not performed. This prevented the subsequent discovery of any possible hourglass constrictions, though their absence would not be unexpected based on the compressive cause of AIN syndrome found in these patients.

Conclusions

The objective data obtained from intraoperative NCV studies can be beneficial to the surgeon performing nerve decompression procedures. These studies permit more precise neurolysis of adhesions and are used to determine, intraoperatively, the need to continue with a more invasive procedure.

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This paper will be judged for the Resident Writer's Award.