18 Proximal Median Neuropathy

Proximal median neuropathy is distinctly uncommon compared with median entrapment at the carpal tunnel. Differentiating between median neuropathy at the wrist and more proximal entrapments can be difficult based on clinical grounds alone, especially in mild cases. Electrodiagnostic (EDX) testing plays a key role in localizing the lesion in these unusual cases, especially if the lesion results from trauma or compression.

DETAILED ANATOMY AT THE ANTECUBITAL FOSSA

As the median nerve descends in the upper arm, it runs medial to the humerus and anterior to the medial epicondyle. In a minority of individuals, a bony spur originates from the shaft of the medial humerus just cephalad to the medial epicondyle. A tendinous band known as the *ligament of Struthers* stretches between the spur and the medial humeral epicondyle. In the antecubital fossa, the median nerve travels adjacent to the brachial artery (Figure 18–1). As it enters the forearm, it runs first beneath the *lacertus fibrosus*, a thick fibrous band that runs from the medial aspect of the biceps tendon to the proximal forearm flexor musculature. In most individuals, the median nerve then runs between the two heads of the pronator teres (PT) muscle to provide innervation to that muscle. In many individuals, there are fibrous bands within the two heads of the PT muscle. The *anterior interosseous nerve* then is given off posteriorly, approximately 5 to 8 cm distal to the medial epicondyle, after the median nerve passes between the two heads of the PT. As the median nerve runs distally, it passes deep to the flexor digitorum sublimis (FDS) muscle and its proximal aponeurotic tendinous edge, known as the *sublimis bridge*.

ETIOLOGY

Median neuropathy in the region of the antecubital fossa has been described as a consequence of external compression from casting, trauma, venipuncture, and compressive mass lesions, including tumor or hematoma. Rare cases of brachial artery puncture and subsequent hematoma formation have led to compartment syndromes and subsequent injury of the proximal median nerve.

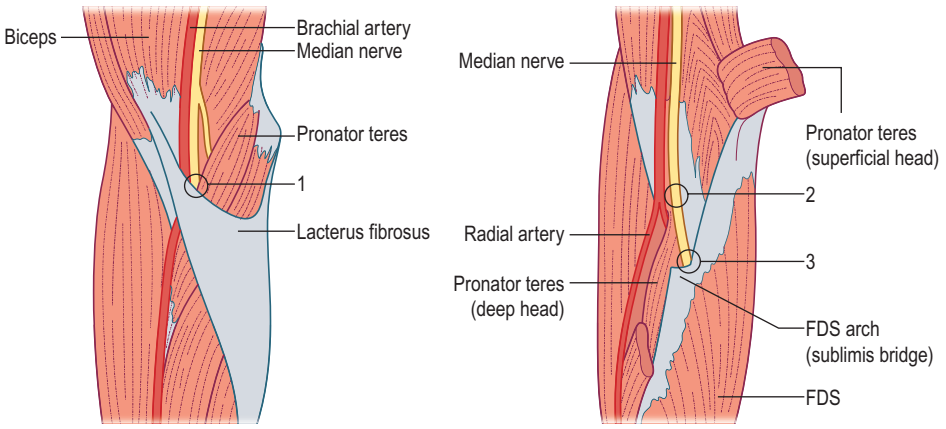

FIGURE 18–1 Median nerve anatomy in the region of the antecubital fossa, and potential sites of entrapment. **Left:** In the antecubital fossa, the median nerve travels adjacent to the brachial artery. As it enters the forearm, it runs first beneath the lacertus fibrosus, a thick fibrous band that runs from the medial aspect of the biceps tendon to the proximal forearm flexor musculature. In most individuals, the median nerve then runs between the two heads of the pronator teres. **Right:** Superficial head of the pronator teres sectioned to show the underlying median nerve. As the median nerve then runs distally, it passes deep to the flexor digitorum sublimis (FDS) muscle and its proximal aponeurotic tendinous edge, known as the sublimis bridge. The pronator syndrome refers to several potential sites of entrapment that occur in the region of the antecubital fossa: (1) lacertus fibrosus, (2) within the pronator teres muscle, and (3) the sublimis bridge. (Adapted with permission from Dang, A.C., Rodner, C.M., 2009. Unusual compression neuropathies of the forearm, part II: median nerve. J Hand Surgery (AM) 34 (10), 1915–1920.)

DOI: 10.1016/B978-1-4557-2672-1.00018-0

In addition, several sites of proximal median entrapment have been reported (Figure 18–1). All are uncommon, and some remain controversial. The four major potential sites of entrapment are as follows:

- Median nerve entrapment may occur at the ligament of Struthers in the distal upper arm, where both the median nerve and brachial artery pass between this ligament and the humerus.
- More distally in the region of the antecubital fossa, the median nerve may become entrapped beneath a hypertrophied lacertus fibrosus.
- Further distally, the median nerve may become entrapped in the substance of the PT muscle, especially in individuals who have additional fibrous bands running through that muscle.
- More distally, the median nerve may become entrapped beneath the sublimis bridge of the FDS muscle.

CLINICAL

The clinical syndromes of proximal median neuropathy depend on the underlying etiology and lesion site.

TRAUMATIC LESIONS

In patients with traumatic lesions, there usually is an obvious, acute disturbance of median motor and sensory function. Significantly, sensory disturbance in proximal median neuropathy is noted in the entire median territory, including the thenar eminence, as well as the thumb, index, middle, and lateral ring fingers. This feature clearly distinguishes proximal median neuropathy from carpal tunnel syndrome (CTS), in which sensation over the thenar eminence is spared. Sensory loss over the thenar eminence occurs as the palmar cutaneous branch, which innervates the thenar eminence, leaves the median nerve proximal to the carpal tunnel. Depending on the site of the lesion, weakness may affect some or all of the proximal median-innervated forearm muscles, including the PT, FDS, flexor digitorum profundus (FDP) to digits 2 and 3, flexor carpi radialis (FCR), flexor pollicis longus (FPL), and pronator quadratus (PQ), as well as the distal median-innervated muscles, including the abductor pollicis brevis (APB), opponens pollicis (OP), and first and second lumbricals. Weakness of the FDP to digits 2 and 3, FDS and FPL often leads to a characteristic high median neuropathy posture, whereby the individual is unable to flex the thumb, index, and middle fingers (Figure 18–2).

ENTRAPMENT SYNDROMES

The symptoms and signs in the proximal median nerve entrapment syndromes are fairly nonspecific. Typically, there is pain or discomfort in the region of the entrapment.

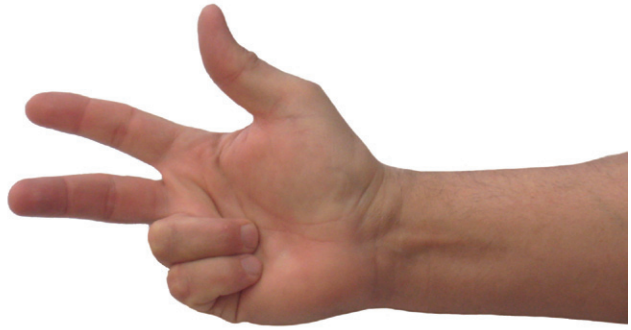

FIGURE 18–2 High median neuropathy hand posture. A complete high median neuropathy results in a classic hand posture when the patient attempts to make a grip: the patient is unable to flex the thumb, index, and middle fingers.

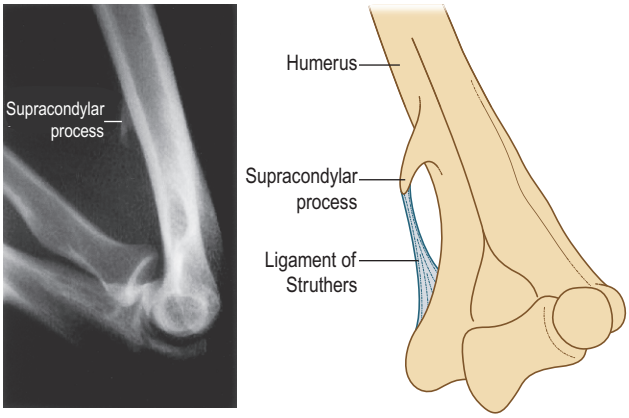

FIGURE 18–3 Ligament of Struthers. Rare individuals have a supracondylar bony spur from which a tendinous band (ligament of Struthers) runs to the medial epicondyle. The median nerve and brachial artery travel under this ligament. The supracondylar process can be demonstrated by plain bone X-ray films.
(Adapted from Struthers, J., 1854. On some points in the abnormal anatomy of the arm. Br Foreign Med Ch Rev 13, 523–533.)

Unlike CTS, the symptoms are not exacerbated at night. The two major syndromes include (1) proximal entrapment of the median nerve at the ligament of Struthers and (2) median nerve entrapment more distally, either beneath the lacertus fibrosus, in the substance of the PT, or beneath the sublimis bridge (Figure 18–1). The latter three entrapment sites usually are referred to collectively as the *pronator syndrome*. Strictly speaking, the term may be reserved for nerve entrapment within the substance of the PT muscle proper. However, entrapment at any of these last three locations usually produces a similar clinical syndrome.

Ligament of Struthers Entrapment

Entrapment at the ligament of Struthers is a very rare syndrome whereby the median nerve is entrapped by a tendinous band running from the medial epicondyle to a bony spur on the distal medial humerus (Figure 18–3). The

prevalence of such a supracondylar bony spur is approximated at 1 to 2% of the population. The syndrome is characterized by pain in the volar forearm and paresthesias in the median-innervated digits, which are exacerbated by supination of the forearm and extension of the elbow. The radial pulse also may be attenuated with these maneuvers, as the brachial artery also runs with the median nerve under the ligament of Struthers. A bony spur may be palpable at the distal humerus. Weakness of the PT and other median-innervated muscles may occur, and subtle sensory loss may be noted in the median distribution, including the thenar eminence.

Pronator Syndrome

Although rare, the pronator syndrome occurs more often than entrapment at the ligament of Struthers. The PT muscle may be enlarged or firm, with a Tinel's sign over the site of entrapment. Pain may radiate proximally and often is aggravated by using the arm, especially with repeated pronation/supination movements. Specific maneuvers that may produce symptoms of pain in the forearm and paresthesias in the median-innervated digits depend on the site of entrapment (Figure 18–4): resisted pronation with the elbow in extension (for the PT); resisted flexion of the proximal interphalangeal joint of the middle finger (for the sublimis bridge); and resisted flexion of the elbow with the forearm in supination (for the lacertus fibrosus). The sole finding of increased pain with these maneuvers is an unreliable sign, unless it is accompanied by median nerve territory paresthesias. Significant weakness or wasting of median-innervated muscles is rare, but mild weakness of the FPL and APB is not uncommon, with occasional involvement of the FDP to digits 2 and 3 and the OP. The pronator teres muscle is usually spared. There may be occasional paresthesias radiating into the median-innervated digits, with subtle impairment of sensation in the median nerve distribution, including the thenar eminence.

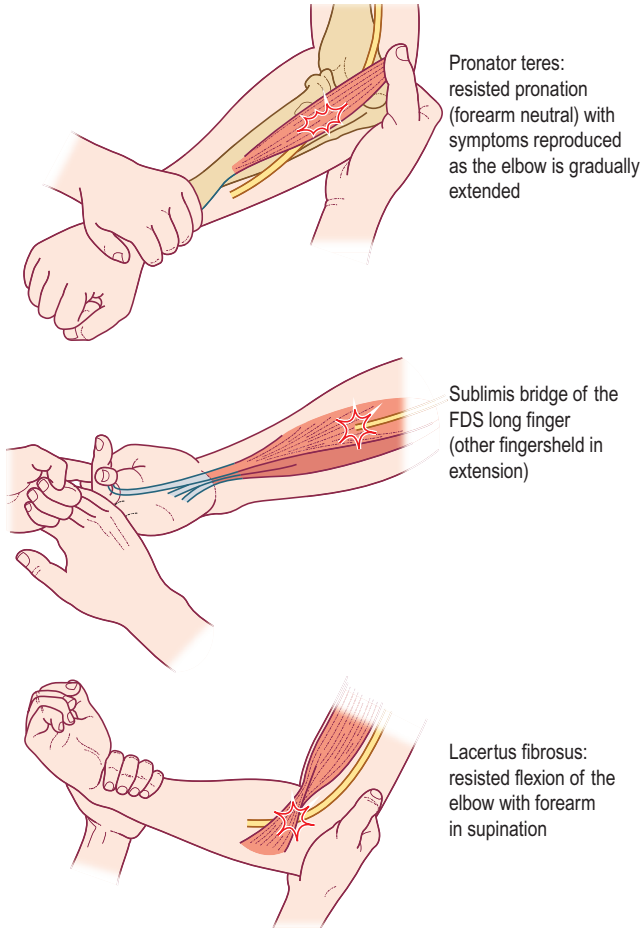

FIGURE 18–4 Provocative maneuvers for pronator syndrome. Different provocative maneuvers may reproduce symptoms associated with the pronator syndrome, depending on the site of entrapment: pronator teres, sublimis bridge (arch of the flexor digitorum sublimis [FDS]), and lacertus fibrosus. Note: many consider these maneuvers to be unreliable and nonspecific. The sole finding of increased pain with these maneuvers is especially unreliable unless the pain is accompanied by median paresthesias.
(From Omer, G.E., Spinner, M., 1980. Management of peripheral nerve problems. WB Saunders, Philadelphia.)

ANTERIOR INTEROSSEOUS NERVE SYNDROME

The anterior interosseous nerve, the largest branch of the median nerve, leaves the main trunk of the median nerve just distal to the PT to innervate three muscles: FPL, FDP to digits 2 and 3, and PQ. It carries deep sensory fibers to the wrist and interosseous membrane, but it carries no cutaneous sensory fibers. Clinically, patients present with the inability to flex the distal phalanx of the thumb, index, and middle fingers, with weakness of pronation. Weakness of the PQ is best demonstrated with the elbow flexed to avoid the contribution of the PT, which is not involved in anterior interosseous syndrome. With the elbow flexed, the PQ is the primary muscle to pronate the arm; with the elbow extended, the PT is the primary muscle to pronate the arm. There is no sensory loss. A characteristic compensatory posture occurs when the patient attempts to make an "OK" sign and is unable to flex the distal thumb and index fingers. Compensatory hyperextension of the distal interphalangeal joint of the index finger and interphalangeal joint of the thumb then occurs (Figure 18–5). Anterior interosseous neuropathy (AIN) has been reported to occur following fractures and crush injuries. In addition, it can rarely occur as an entrapment neuropathy, but more often it is a variant presentation of brachial neuritis, a full discussion of which is found in the section on brachial neuritis in Chapter 30, including the electrophysiologic evaluation.

Occasionally, it may be difficult to recognize an AIN. In some patients, the slip of the FDP to digit 3 is supplied by the ulnar nerve, leaving middle finger flexion intact despite an AIN. The situation is more complicated when an AIN occurs in combination with a Martin–Gruber anastomosis (MGA). In MGA, there is an anomalous

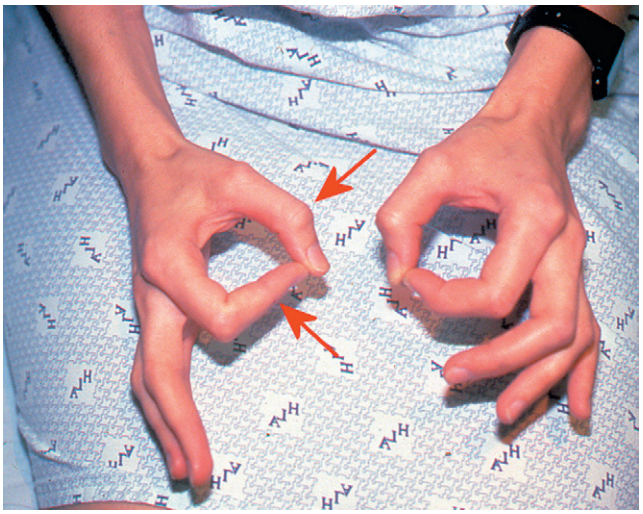

FIGURE 18–5 Anterior interosseous neuropathy. Lesions of the anterior interosseous nerve result in weakness of the flexor pollicis longus, flexor digitorum profundus (to digits 2, 3, or both), and pronator quadratus. Patients characteristically are unable to make an "OK" sign (i.e., form a circle with thumb and index finger). The thumb and index finger are unable to flex at the interphalangeal joints and the distal interphalangeal joints, respectively (arrows).

cross-over of median-to-ulnar fibers. Occasionally, the median fibers that cross over run in the anterior interosseous nerve. Thus, a patient with an AIN also can develop weakness of some ulnar-innervated intrinsic hand muscles, if an MGA is present and the cross-over fibers run in the anterior interosseous nerve.

DIFFERENTIAL DIAGNOSIS

In cases of acute trauma or injury, the clinical differential diagnosis is limited and usually straightforward. For the entrapment syndromes in the region of the antecubital fossa, however, the differential diagnosis is extensive because the symptoms often are vague. For example, local orthopedic problems may present in a similar fashion. Median neuropathy at the carpal tunnel also may give rise to diagnostic confusion. Patients with CTS can present with vague pain or heaviness in the forearm associated with median paresthesias, similar to symptoms in the proximal median entrapment syndromes. Additionally, patients with cervical radiculopathy may present with radiating pain associated with paresthesias into the hand. In cervical radiculopathy, however, there usually is a history of neck pain that radiates into the arm. Examination in cervical radiculopathy may reveal weakness outside the median territory, as well as decreased biceps, brachioradialis, or triceps reflexes.

ELECTROPHYSIOLOGIC EVALUATION

The purpose of nerve conduction studies and electromyography (EMG) in suspected proximal median neuropathy is (1) to demonstrate that median nerve abnormalities are proximal to the wrist and (2) to exclude a lesion higher in the brachial plexus or cervical nerve roots. However, the EDX evaluation may be complicated by the fact that the electrophysiology in true cases of proximal median entrapment often is normal or nonspecific, despite what one might expect on theoretical grounds.

Nerve Conduction Studies

Nerve conduction studies should include routine median motor studies stimulating the median nerve at the wrist and antecubital fossa, recording at the APB (Box 18–1). If there is a question of entrapment at the ligament of Struthers, proximal stimulation should also be performed at the axilla. Routine ulnar motor and sensory studies should also be performed to exclude a coexistent polyneuropathy. Sensory nerve conductions of median-innervated digits should always be performed, recording the most symptomatic digit(s), especially if numbness or paresthesias have been observed on clinical examination. If values are borderline or just slightly above the upper limits of normal, comparison with the contralateral side should be done. *Finally, in all suspected median neuropathies, it is imperative to perform at least one of the median-versus-ulnar comparison studies across the wrist to exclude median neuropathy at the wrist. If values are borderline or just slightly above the upper limits of normal, a second median-versus-ulnar comparison*

Box 18–1. Recommended Nerve Conduction Study Protocol for Proximal Median Neuropathy

Routine studies:

1. Median motor study recording abductor pollicis brevis, stimulating wrist, antecubital fossa, and axilla
2. Ulnar motor study recording abductor digiti minimi, stimulating wrist, below groove, and above groove
3. Median and ulnar F responses
4. Median sensory response, recording digit 2 or 3, stimulating wrist (bilateral studies suggested)
5. Ulnar sensory response, recording digit 5, stimulating wrist
6. Median and ulnar palm-to-wrist mixed nerve studies using identical distances of 8 cm

The following patterns suggest possible proximal median neuropathy:

1. Reduced median compound muscle action potential and/or sensory nerve action potential amplitudes with distal latencies that are either normal or only slightly prolonged (never in the demyelinating range) and no significant slowing of the median palm-to-wrist latency compared with the ulnar
2. Either conduction block/temporal dispersion or marked conduction velocity slowing between the wrist and antecubital fossa, or between antecubital fossa and axilla, with normal or only slightly prolonged distal latencies on median motor studies
3. Prolonged median F responses despite a relatively normal distal compound muscle action potential amplitude and distal latency

study should be done to look for median neuropathy at the wrist.

A lesion of the median nerve that results in wallerian degeneration, regardless of the lesion site, will result in decreased compound muscle action potential (CMAP) and sensory nerve action potential (SNAP) amplitudes distal to the lesion. Often the distal latencies will be slightly prolonged, and conduction velocity will be mildly slowed because of dropout of the fastest-conducting axons. However, although such findings are abnormal and indicate a median nerve lesion, they do not localize the lesion. If there is focal demyelination at the ligament of Struthers, one might expect to see either focal slowing or a drop in CMAP amplitude (i.e., conduction block or temporal dispersion) between the antecubital fossa and the axilla sites. If there is a focal lesion in the region of the antecubital fossa, there may be a conduction block between the wrist and antecubital fossa sites. Although such findings can be expected on theoretical grounds, in fact they rarely occur.

Electromyographic Approach

The EMG usually is more gratifying than the nerve conduction studies in patients with a suspected proximal median neuropathy (Box 18–2). The distal median muscles (APB) should always be studied. However, the critical part of the study consists of careful examination of several median-innervated muscles proximal to the carpal tunnel. These muscles include the PT, FCR, FDS, FDP to digits 2 and 3, FPL, and PQ. If any of these muscles are abnormal (evidence of denervation or reinnervation), the problem must be proximal to the wrist. If the lesion is at the level of the ligament of Struthers, EMG abnormalities may be noted in all the median-innervated muscles, including the PT. In the pronator syndrome, EMG abnormalities have been reported most often in the FPL and FDP to digits 2 and 3, less often in the FDS and APB, and only rarely in the PT, because the compression site most often occurs distal to its innervation. If any of the proximal median-innervated muscles are abnormal, other muscles innervated by the same myotomes as the proximal median muscles, but supplied by different nerves, should be sampled to exclude a more proximal lesion of the brachial plexus or cervical nerve roots. At a minimum, one non-median C6–C7 innervated muscle (e.g., triceps) and one non-median C8–T1 innervated muscle (e.g., first dorsal interosseous) must be checked.

A few technical points are important to keep in mind. The proximal median-innervated muscles that are most accessible and easiest to sample are the PT, FPL, and FCR. In all suspected proximal median nerve lesions, the PT and at least one of these other muscles should be sampled. Two proximal median-innervated muscles that are somewhat more difficult to study on a routine basis are the FDP to digits 2 and 3 and the PQ. The FDP has two slips: a median-innervated slip to digits 2 and 3 and an ulnar-innervated slip to digits 4 and 5. The ulnar-innervated slip is superficial and very easy to study. The median slip is deep, however, and thus is much more difficult to localize. Likewise, the PQ is a deep muscle that lies beneath the extensors to the thumb and index finger, making it technically more difficult to study.

Box 18–2. Recommended Electromyographic Protocol for Proximal Median Neuropathy

Needle examination of:

1. Median muscle distal to the carpal tunnel (abductor pollicis brevis)
2. At least two median muscles proximal to the carpal tunnel, including the pronator teres and one of the following: flexor pollicis longus, flexor carpi radialis, flexor digitorum sublimis

If the abductor pollicis brevis is abnormal:

3. Test at least two other non-median, lower trunk/C8–T1 innervated muscles (e.g., first dorsal interosseous, extensor indicis proprius, flexor digitorum profundus to digits 4 and 5) to exclude a lower brachial plexopathy, polyneuropathy, or C8–T1 radiculopathy

If the proximal median muscles are abnormal:

4. Test at least one non-median C6–C7 and C7–C8 innervated muscle (e.g., triceps, extensor digitorum communis, extensor indicis proprius) to exclude a more proximal brachial plexopathy or cervical radiculopathy

Note: If nerve conduction studies show a non-localizing median neuropathy, electromyography can only localize the lesion to at or above the take-off to the most proximally affected median-innervated muscle. For example, an abnormal flexor carpi radialis with a normal pronator teres does not necessarily localize the lesion to the median nerve between those two muscles, but only to at or proximal to the take-off to the flexor carpi radialis muscle. Although this may seem counterintuitive, remember that there are proximal lesions that spare some of the muscles distal to the lesion while affecting others.

EXAMPLE CASES

Case 18–1

History and Examination

A 24-year-old man noticed numbness of the right thumb, index, and middle fingers. The numbness was noted after removal of a cast that had been in place for 6 weeks following wrist fusion because of trauma.

Examination showed wasting of the thenar eminence. Thumb abduction was moderately weak. Wrist flexion was difficult to evaluate because of the effects of surgery. Hypesthesia was present over the thumb, index, and middle fingers, as well as over the thenar eminence.

Summary

The clinical history and examination both are suggestive of a median nerve lesion. Given the history of trauma to the wrist and subsequent surgery, median neuropathy at the wrist seems a likely diagnosis. However, the finding of hypesthesia over the thenar eminence should alert one to a more proximal lesion because that area should be spared in median nerve lesions at the carpal tunnel.

CASE 18–1. Nerve Conduction Studies

Nerve Stimulated	Stimulation Site	Recording Site	Amplitude Motor = mV; Sensory = μV			Latency (ms)			Conduction Velocity (m/s)			F-wave Latency (ms)		
			RT	LT	NL	RT	LT	NL	RT	LT	NL	RT	LT	NL
Median (m)	Wrist	APB	0.4	8.6	≥4	6.1	3.8	≤4.4				NR	28	≤31
	Antecubital fossa	APB	0.3	8.4		10.6	7.6		44	52	≥49			
Ulnar (m)	Wrist	ADM	11.2		≥6	3.0		≤3.3				27		≤32
	Below elbow	ADM	11.2			6.3			60		≥49			
	Above elbow	ADM	11.1			9.6			61		≥49			
Median (s)	Wrist	Index finger	4	22	≥20	3.1	3.0	≤3.5	54	56	≥50			
Ulnar (s)	Wrist	Little finger	24		≥17	2.9		≤3.1	62		≥50			
Median (mixed study)	Palm	Wrist	9		≥50	1.6		≤2.2	62		≥50			
Ulnar (mixed study)	Palm	Wrist	23		≥15	1.6		≤2.2	62		≥50			
Mixed difference						0.0		≤0.3						

m = motor study; s = sensory study; RT = right; LT = left; NL = normal; NR = no response; APB = abductor pollicis brevis; ADM = abductor digiti minimi.
Note: All sensory and mixed latencies are peak latencies. All sensory and mixed-nerve conduction velocities are calculated using onset latencies. The reported F-wave latency represents the minimum F-wave latency.

CASE 18–1. Electromyography

Muscle	Insertional Activity	Spontaneous Activity		Voluntary Motor Unit Action Potentials				
		Fibrillation Potentials	Fasciculations	Activation	Recruitment	Configuration		
						Duration	Amplitude	Polyphasia
Right APB	↑	+3	0	NL	↓↓	+1	+1	+3
Right FDI	NL	0	0	NL	NL	NL	NL	NL
Right EIP	NL	0	0	NL	NL	NL	NL	NL
Right PT	NL	0	0	NL	↓	+1	+1	+1
Right triceps brachii	NL	0	0	NL	NL	NL	NL	NL
Right FCR	↑	+1	0	Fair	↓	+1	+1	+1
Right FDS	↑	+1	0	NL	NL	NL	NL	NL
Right biceps brachii	NL	0	0	NL	NL	NL	NL	NL

↑ = increased; ↓ = slightly reduced; ↓↓ = moderately reduced; APB = abductor pollicis brevis; FDI = first dorsal interosseous; EIP = extensor indicis proprius; PT = pronator teres; FCR = flexor carpi radialis; FDS = flexor digitorum sublimis.

Proceeding to the nerve conduction studies, the right median motor study is strikingly abnormal. The amplitude is markedly decreased, with moderate prolongation of the distal motor latency and moderate slowing of conduction velocity. The distal latency of 6.1 ms strongly suggests the possibility of demyelination between the wrist and the recording site. Indeed, the distal latency of 6.1 ms is near the unequivocally demyelinating range. Although this degree of slowing might represent true demyelination, it could also represent marked dropout of the medium- and fastest-conducting fibers, secondary to severe axonal loss. If severe axonal loss is present, one would expect the distal CMAP amplitude to be very low and the needle EMG of that muscle to be strikingly abnormal. In this case, both are true. The distal median CMAP amplitude is very low at 0.4 mV, suggesting that there has been severe axonal loss. This is confirmed by the EMG of the APB, which shows prominent active denervation and reinnervation with reduced recruitment of motor unit action potentials (MUAPs), which are signs of axonal loss.

The right median F responses are absent. Theoretically, this finding could represent a proximal median lesion; however, a distal CMAP amplitude this low usually results in an absent F response, but not because of a proximal lesion. Because the F response normally is only

1 to 5% of the distal CMAP amplitude, F responses in the setting of such a low CMAP amplitude are commonly absent or difficult to obtain.

The ulnar motor study and the ulnar and radial sensory responses are completely normal, which suggests that the problem is limited to the median nerve. The median sensory and palm-to-wrist mixed-nerve studies also show a normal latency with a low amplitude. Comparison of the median and ulnar palm-to-wrist mixed latencies reveals no significant difference. These studies strongly argue against a median nerve lesion at the wrist.

In summary, the nerve conduction studies demonstrate a severe median neuropathy involving motor and sensory fibers. The neuropathy does not appear to be localized to the wrist for the following reason: although the median sensory and mixed nerve amplitudes are low, their latencies are not slowed. The distal motor latency to the APB is moderately prolonged, but that may be attributable to severe axonal loss, which cannot be localized to the wrist.

On needle EMG, the right APB is floridly fibrillating with moderately reduced recruitment of long polyphasic MUAPs. This provides additional EMG confirmation that the APB has undergone significant axonal loss. Because of the abnormal APB, the FDI is sampled next to exclude a C8–T1 radiculopathy or, more importantly, a widespread brachial plexopathy, in light of the abnormal median sensory and mixed-nerve potentials. The right extensor indicis proprius (EIP) also is checked for the same reason. The findings of a normal FDI and EIP with such an abnormal APB argue strongly against a lesion of the lower trunk of the brachial plexus or the C8–T1 roots, and suggest strongly that the problem is limited to the median nerve.

The more proximal median muscles also are denervated, including the FCR and FDS. The PT and FCR also show evidence of reinnervation. The biceps brachii and triceps brachii are sampled to exclude a C6 or C7 radiculopathy or brachial plexopathy as the cause of the changes in the proximal median-innervated muscles.

Because these muscles are normal and all of the EMG abnormalities are limited to median-innervated muscles spanning several myotomes (C6–T1), an electrophysiologic impression can be made.

IMPRESSION: *There is electrophysiologic evidence consistent with a severe right median neuropathy at or proximal to the take-off to the PT muscle.*

The etiology of the high median neuropathy in this case most likely was the cast, which was too tight for the patient and caused chronic compression of the median nerve at the antecubital fossa. Although the patient sustained significant trauma to his wrist and underwent wrist fusion surgery, there is no definite electrophysiologic evidence of focal median slowing at the carpal tunnel. If median motor, sensory, and F response studies alone had been performed, along with an EMG limited to the APB and FDI muscles, a misdiagnosis of median neuropathy at the carpal tunnel could easily have been made, perhaps leading to an inappropriate median nerve decompression at the wrist.

The clinical clues that more extensive studies were called for included (1) numbness over the thenar eminence, which is not found in CTS, and (2) the fact that the numbness was appreciated after removal of the cast for wrist fusion, and not after the initial trauma.

Case 18–2

History and Examination

A 25-year-old man was shot in the left arm two months previously. The bullet entered the posterior arm and exited anteriorly in the region of the mid biceps. The patient complained of persistent numbness of the thumb, index, and middle fingers and poor dexterity of the hand.

Examination showed marked wasting of the thenar eminence. Thumb abduction and opposition were markedly weak. There was moderate weakness of thumb, index, and middle finger flexion at both the distal interphalangeal and proximal interphalangeal joints. Arm pronation was mildly weak. The remainder of the motor examination was normal, as were the reflexes. There was sensory loss over the left thumb, index, and middle fingers and the thenar eminence.

Summary

The history and examination are consistent with a proximal median nerve lesion. Although nerve conduction studies and EMG generally are most useful in determining localization, in this case there is little doubt where the lesion is (i.e., the bullet hole). The nerve conduction studies and EMG serve additional important roles in determining the severity of the lesion and hence the prognosis in this type of case where there has been severe trauma.

There is no response from the APB muscle stimulating the left median nerve. The sensory response from the left median nerve also is absent. The left ulnar motor study and the contralateral right median motor and sensory nerve conduction studies are completely normal. These findings strongly suggest an isolated lesion of the left median nerve. Several sensory conductions, including studies of the left ulnar, radial, and lateral antebrachial cutaneous nerves, are normal. These also tend to preclude a more proximal plexus lesion. The complete absence of median motor and sensory responses makes the diagnosis of a median neuropathy inescapable; however, the nerve conduction results are of no value in localizing the lesion.

On EMG, the APB is floridly fibrillating, and no MUAPs can be activated. The nerve conduction finding of an absent median motor response and the EMG finding of florid fibrillation potentials with no MUAPs in the APB suggest that there is no axonal continuity to the distal median muscles. Assessment of the more proximal

CASE 18–2. Nerve Conduction Studies

Nerve Stimulated	Stimulation Site	Recording Site	Amplitude Motor = mV; Sensory = μV			Latency (ms)			Conduction Velocity (m/s)			F-wave Latency (ms)		
			RT	LT	NL	RT	LT	NL	RT	LT	NL	RT	LT	NL
Median (m)	Wrist	APB	6.2	NR	≥4	4.2	NR	≤4.4				29	NR	≤31
	Antecubital fossa	APB	6.0			7.9			54		≥49			
Ulnar (m)	Wrist	ADM		9.0	≥6		2.9	≤3.3					28	≤32
	Below elbow	ADM		8.9			6.4			57	≥49			
	Above elbow	ADM		8.7			8.1			59	≥49			
Median (s)	Wrist	Index finger	24	NR	≥20	3.4	NR	≤3.5	56	NR	≥50			
Ulnar (s)	Wrist	Little finger	22	23	≥17	2.9	3.0	≤3.1	62	64	≥50			
Radial (s)	Forearm	Snuffbox		35	≥15		2.4	≤2.9		65	≥50			
Lateral antebrachial cutaneous(s)	Elbow	Forearm		19	≥10		2.9	≤3.0		62	≥55			

m = motor study; s = sensory study; RT = right; LT = left; NL = normal; NR = no response; APB = abductor pollicis brevis; ADM = abductor digiti minimi.
Note: All sensory and mixed latencies are peak latencies. All sensory and mixed-nerve conduction velocities are calculated using onset latencies. The reported F-wave latency represents the minimum F-wave latency.

CASE 18–2. Electromyography

Muscle	Insertional Activity	Spontaneous Activity		Voluntary Motor Unit Action Potentials				
		Fibrillation Potentials	Fasciculations	Activation	Recruitment	Configuration		
						Duration	Amplitude	Polyphasia
Left APB	↑	+3	0	None				
Left FPL	↑	+2	0	NL	↓↓↓	+3	+1	+1
Left PT	↑	+3	0	NL	↓↓	−1/+1	NL	+1
Left FCR	↑	+3	0	NL	↓↓	−1/+1	NL	+1
Left FDI	NL	0	0	NL	NL	NL	NL	NL
Left biceps brachii	NL	0	0	NL	NL	NL	NL	NL
Left triceps brachii	NL	0	0	NL	NL	NL	NL	NL

NL = normal; ↑ = increased; ↓↓ = moderately reduced; ↓↓↓ = markedly reduced; APB = abductor pollicis brevis; FPL = flexor pollicis longus; PT = pronator teres; FCR = flexor carpi radialis; FDI = first dorsal interosseous.

median muscles, including the PT, FCR, and FPL, also shows fibrillation potentials. More importantly, however, MUAPs are also present. In the PT and FCR, there is a combination of brief and long-duration MUAPs, with moderately to markedly reduced recruitment.

The biceps and triceps are also checked to exclude a coexistent C6 or C7 radiculopathy or upper/middle trunk plexopathy that could account for the abnormalities in the proximal median-innervated muscles. The FDI is checked to exclude a coexistent C8–T1 radiculopathy. These muscles are all normal.

An electrophysiologic impression now can be formed.

IMPRESSION: *There is electrophysiologic evidence consistent with a severe left median neuropathy at or proximal to the take-off to the PT muscle. No axonal continuity to the APB muscle can be demonstrated. A repeat study is recommended in 2 to 3 months to further assess axonal continuity.*

In this case, the localization of the lesion is obvious, marked by the bullet, which clearly went through the median nerve in the region of the upper arm. This is a severe lesion that has led to marked denervation in most of the median-innervated muscles. Of note, there is a combination of brief and long-duration polyphasic MUAPs in two of the proximal median-innervated muscles. The brief-duration, polyphasic motor unit potentials might lead one to question whether there is a coexistent myopathy. The answer clearly is no. These

MUAPs represent early reinnervation, or so-called nascent motor units. The key to differentiating nascent motor unit potentials from myopathic motor unit potentials is the presence of decreased recruitment in neuropathic conditions, whereas recruitment is normal or early in myopathic conditions.

The surgeon in this case was considering a tendon transfer to provide function to the left thumb. The question arises whether serial EMGs would be helpful in this regard. Although EMG is a very useful diagnostic tool, in general it has little role in following a patient's improvement. One exception to this rule includes cases in which one is trying to document axonal continuity of a nerve. In the case under consideration here, there has been a severe traumatic lesion of the median nerve with complete denervation of the APB, and no evidence of axonal continuity to the APB on the initial EMG study. A repeat study several weeks or months later would be useful to look for evidence of early reinnervation in the APB before consideration of tendon transfer surgery. After trauma and wallerian degeneration, axonal regrowth occurs at approximately 1 mm per day. Thus, a lesion in the region of the arm might not reinnervate the APB for several months to a year. In this case, if the APB were to become reinnervated, the first change on EMG would be the presence of nascent motor unit potentials that recruit poorly, along with fibrillation potentials. The appearance of nascent motor unit potentials would be a clear indication to delay the surgery and observe further in the hope that continued axonal regeneration would obviate the need for surgery.

Suggested Readings

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