



February 1998 EMG Case-of-the-Month

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HISTORY

A 51-year-old man has a 2 month history of right forearm pain and weakness in the right thumb, without loss of sensation. The onset was gradual. He denies problems in other limbs. He complains of difficulty opening jars. There is no history of recent trauma to the hand or arm. The patient is a farmer and an avid guitar player.

- **Prior to continuing, please develop a differential diagnosis, and list each diagnosis in order of likelihood.**
- **Is there any additional information from the clinical history that might be helpful in clarifying your differential list or changing its order of priority?**

COMMENTARY I

The differential diagnosis is quite broad and includes isolated nerve entrapments (median, ulnar, or radial); musculoskeletal problems of the forearm or wrist (lateral/medial epicondylitis or DeQuervain's tenosynovitis); or a more proximal nerve injury of the brachial plexus, cervical root, or anterior horn cell. The lack of sensory complaints makes an anterior horn cell injury or pure motor peripheral nerve injury more likely among the possible neurogenic causes. Patients with musculoskeletal problems (e.g., tendinitis) usually present with pain, but often have no other associated sensory complaints.

HISTORY, continued

The patient is in good health with no history of diabetes, thyroid disorder, or connective tissue disorders. Family history and review of systems were non-contributory.

- **If necessary, revise your differential diagnosis based on the additional clinical history.**
- **On what details of the physical examination do you think you should focus at this point?**

COMMENTARY II

The lack of other medical disorders that would predispose to a peripheral neuropathy or hereditary neuropathy makes these diagnoses less likely.



PHYSICAL EXAMINATION

The physical examination reveals normal sensation in the upper limbs. Muscle stretch reflexes are also normal (2+/4 symmetrically). Strength testing is normal except for weakness of the right forearm pronation (4/5) and finger flexion (0/5) of the distal phalanges of the thumb and index fingers. There is no atrophy, and muscle bulk appears normal and symmetric. There is no pain with resisted wrist extension or flexion. Finkelstein and Spurling maneuvers are negative.

- **At this point, review your differential diagnosis, and revise as appropriate.**
- **Are there additional observations on physical examination that might be helpful in narrowing your differential list?**

COMMENTARY III

The weakness of the finger flexors and forearm pronation is consistent with an anterior interosseous nerve injury, although a more proximal injury could result in a similar presentation. Rupture of the flexor tendons of the digits should also be considered, given their profound weakness in combination with the normal sensory examination. Although possible, tendon rupture is highly unlikely given the history of no trauma and gradual onset.

PHYSICAL EXAMINATION, continued

When asked to make an "OK" sign with the right hand (i.e., a circle with the thumb and index finger), he forms a triangular shape instead of a circle. He can form a circle easily with the left thumb and index finger.

- **If necessary, revise your differential diagnosis based on the additional physical examination results.**
- **Are there laboratory or other tests that could help you in your differential diagnosis?**

COMMENTARY IV

The inability of the patient to form a circle when making the "OK" sign is highly suggestive of a right anterior interosseous nerve injury, but could also be seen with a more proximal lesion of the median nerve. Weakness in the flexor pollicis longus translates into the inability to flex the distal phalanx of the thumb while weakness of the flexor digitorum profundus (lateral heads) translates into inability to flex the distal phalanx of the index and middle finger.

Thus, when asked to make the "OK" sign, a person with a AIN syndrome is unable to flex the distal phalanges, and they are forced into hyper-extension, creating the triangular shape of the thumb and index finger that is characteristic of this syndrome. Rupture of the flexor tendons may also present similarly.



ELECTROPHYSIOLOGIC DATA

ELECTROMYOGRAPHY										
n = normal incr = increased decr = decreased 0 = absent 1+ = minimal 4+ = maximal crd = complex repetitive discharge fasc = fasciculation potential myk = myokymic discharge myt = myotonic discharge nmt = neuromyotonic discharge p wave = positive sharp waves fib = fibrillation potentials recr = recruitment amp = amplitude dur = duration poly = polyphasic potentials										
R/L	MUSCLE	INSERTION		SPONTAN		VOLUNTARY				
		activ	p wave	fib	other	recr	amp	dur	poly	effort
R	cervical paraspinals	n	0	0	0	n	n	n	n	n
R	deltoid	n	0	0	0	n	n	n	n	n
R	biceps brachii	n	0	0	0	n	n	n	n	n
R	triceps	n	0	0	0	n	n	n	n	n
R	extensor carpi radialis	n	0	0	0	n	n	n	n	n
R	pronator teres	n	0	0	0	n	n	n	n	n
R	flexor carpi radialis	n	0	0	0	n	n	n	n	n
R	flexor pollicis longus	incr	sust	4+	0	0	-	-	-	n
R	pronator quadratus	incr	sust	3+	0	0	-	-	-	n
R	flexor carpi ulnaris	n	0	0	0	n	n	n	n	n
R	flexor digitorum profundus (medical heads)	n	0	0	0	n	n	n	n	n
R	abductor pollicis brevis	n	0	0	0	n	n	n	n	n
R	FDI (hand)	n	0	0	0	n	n	n	n	n

SENSORY NERVE CONDUCTION									
nr = no response									
NERVE	LATENCY (ms)			AMPLITUDE (µV)			CONduc VEL (m/s)		
	R	L	Norm	R	L	Norm	R	L	Norm
median sensory from wrist to index finger	3.4	3.5	<3.7	18	19	>20	54	52	>49
ulnar sensory from wrist to 5 th digit	3.6	-	<3.5	12	-	>10	52	-	>49



MOTOR NERVE CONDUCTION									
nr = no response									
NERVE	LATENCY (ms)			AMPLITUDE (mv)			CONDUCT VEL (m/s)		
	R	L	Norm	R	L	Norm	R	L	Norm
median (wrist) to thenar	3.5	-	<4.4	10.4	-	>4	-	-	-
median (elbow) to thenar	7.6	-	-	9.9	-	-	53	-	>49
ulnar (wrist) to hypothenar	2.8	-	<3.5	11.4	-	>5	-	-	-
ulnar (below elbow) to hypothenar	6.4	-	-	10.8	-	-	57	-	>49
ulnar (above elbow) to hypothenar	8.7	-	-	10.2	-	-	50	-	>49

F-WAVE								
# = number of stimuli P = persistence CD = chronodispersion F:M = ratio of average F-wave amplitude to M-wave amplitude								
R/L	NERVE	#	LATENCY (ms)			CD (ms)	P (%)	F:M (%)
			min	mean	max			
R	median	10	28.2	-	-	-	-	-
R	ulnar	10	27.4	-	-	-	-	-

The nerve conduction studies are normal, but the needle examination reveals denervation of the flexor pollicis longus and the pronator quadratus. No other C8 or lower trunk innervated muscles are involved, and the paraspinal muscles are also normal.

- **On the basis of both the clinical and electrodiagnostic evaluations, formulate your final impression. List the most likely diagnosis followed by other possibilities that are not excluded by the data. Eliminate those diagnoses not supported by the data.**
- **What other diagnostic procedure are needed?**



DIAGNOSTIC IMPRESSION: COMMENTARY

This is a case of right anterior interosseous nerve (AIN) syndrome, with typical presentation and physical examination findings. The EMG data confirm a right anterior interosseous nerve injury. The essentially normal sensory evoked responses argues against a brachial plexus lesion or a more proximal median nerve entrapment. The median sensory response is borderline small, but is symmetric; it is unlikely to hold any clinical significance.

The possibility of a root or anterior horn cell lesion (C7-8) is effectively ruled out by the needle examination. The only muscles involved are innervated by the anterior interosseous branch of the median nerve. More proximal branches of the median nerve are normal. Other muscles in the same root or cord level are normal. The findings of denervation in muscles innervated by the anterior interosseous nerve branch make the alternative of multiple tendon ruptures very unlikely.

The anterior interosseous nerve is the largest branch of the median nerve, separating from the superficial branch approximately 5-8 cm distal to the lateral epicondyle. It supplies motor fibers to the flexor pollicis longus, flexor digitorum profundus to the thumb and index finger, and the pronator quadratus. It also supplies sensory fibers for deep pain and proprioception in the radiocarpal, radioulnar, intercarpal, and carpometacarpal joints, but without cutaneous distribution.

The causes of AIN syndrome include direct trauma (rare), external compression (e.g. forearm cast), internal pressure from excessively muscular forearms (pronator teres and forearm flexors), anomalous fibrous bands, isolated neuritis of the AIN, or a proximal lesion in the median nerve, with predominance of AIN fiber abnormality. Many believe the most common cause is acute idiopathic neuralgic amyotrophy.

Treatments include relative rest, NSAIDs, steroid injections in the area of the pronator teres heads, and immobilization of the arm in supination. If due to trauma, appropriate surgical repair is indicated. AIN syndromes are almost always axonopathies and are frequently quite profound. Recovery results from nerve regeneration, and it is often more than 6 months before the earliest signs of clinical recovery become evident. If no improvement occurs in 6 months, surgical exploration may be considered, although many subjects show spontaneous improvement without surgical intervention. Findings at surgery are often non-specific or normal.

The patient was followed non-operatively for 6 months and did not show any signs of improvement. At that time he decided, in consultation with his neurosurgeon, to undergo a surgical exploration of the nerve. Intraoperative nerve stimulation showed active conduction in the AIN (though markedly reduced compared to more proximal median nerve branches). Therefore nerve grafting was not pursued.

An adhesion was constricting the median nerve between the two heads of the pronator teres, and this was lysed. The anterior interosseous nerve did not appear gray or edematous. Within 6 months post surgery, the patient noted improvement in distal finger and thumb flexion; EMG revealed volitional motor units in the pronator quadratus and flexor pollicis longus.

Interestingly, one year later the patient reported similar symptoms in the left forearm with weakness of the left thumb. Electrodiagnostic evaluation revealed an isolated left anterior



interosseous mononeuropathy. Subsequent surgical exploration revealed an otherwise healthy appearing anterior interosseous nerve. The patient did improve following neurolysis without nerve grafting.

SUGGESTED READING

1. Wertsch JJ. AAEM Case Report #25: Anterior Interosseous Nerve Syndrome. *Muscle Nerve* 1992; 15:977-983.
2. Kern RZ, Alex A. Anterior interosseous neuropathy: motor conduction studies. *Muscle Nerve* 1989; 10:763.
3. Rask MR: Anterior interosseous nerve entrapment: (Kiloh-Nevin syndrome). *Clin Orthop* 1979; 142: 176-181.
4. Miller-Breslow A, Terrono A, Millender LH. Nonoperative treatment of anterior interosseous nerve paralyseis. *J Hand Surg* 1990A; 15:493-6.