

A case report is presented of a 33-year-old woman who awoke with distal paresthesias, mild incoordination, and progressive weakness. Examination 3 days later demonstrated weakness of the extremities, which was greater in distal muscles than in proximal ones, mild facial weakness, distal vibratory loss, and areflexia. Electrodiagnostic studies provided evidence of an acquired demyelinating polyradiculoneuropathy of recent onset. Motor conduction studies revealed abnormal temporal dispersion and partial conduction block. Preserved sural responses with abnormal median sensory conduction studies supported the diagnosis of Guillain-Barré syndrome, as did subsequent cerebrospinal fluid examinations documenting increasing total protein, identification of preceding cytomegalovirus infection with increasing serum convalescent titer, and progressive clinical improvement after a brief plateau. The role of electrodiagnosis in establishing the diagnosis and prognosis in Guillain-Barré syndrome is reviewed.

Key words: Guillain-Barré syndrome • demyelinating polyneuropathy • GBS

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AAEE CASE REPORT #4: GUILLAIN-BARRÉ SYNDROME

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Guillain-Barré syndrome or acute idiopathic polyradiculoneuritis is a disorder of unknown etiology involving the peripheral nervous system. Although established diagnostic criteria¹ are descriptive, electrodiagnostic findings are often characteristic and important in both establishing the diagnosis and predicting clinical outcome.

CASE REPORT

Clinical History. A 33-year-old registered nurse awoke with a "pins-and-needles" sensation and accompanying numbness in her hands. Upon arising, she became aware of mild incoordination and weakness of her lower extremities. The weakness progressed to involve her upper extremities, and she was hospitalized by her family physician 3 days later.

She previously had been in excellent health with the exception of a 4-day hospitalization 2 weeks earlier for a flu-like syndrome consisting of

mild fever, cervical adenopathy, malaise, and diffuse arthralgias and myalgias. A precise diagnosis had not been established, but her symptoms resolved. She was taking no medications and used alcohol socially.

Physical Examination. There was diffuse weakness of all 4 extremities, distal greater than proximal and involving the upper more than the lower limbs [Medical Research Council (MRC) grade 3 distally and 4 proximally]. There was mild facial weakness. Muscle tone was decreased and vibratory sensation was diminished in the distal lower extremities. Muscle stretch reflexes were absent. No pathologic reflexes were present. The remainder of the clinical examination was normal.

Laboratory Evaluation. Routine studies of blood and urine gave normal results. Acute viral titers demonstrated a cytomegalovirus titer of 1:8, an Epstein-Barr titer of 1:32, and a herpes virus titer of 1:64. Rheumatoid factor and antinuclear antibody were negative. Urine porphobilinogen, delta-aminolevulinic acid, and heavy metal screens were unremarkable. Heterophile agglutination was negative.

Lumbar puncture revealed clear, colorless cerebrospinal fluid (CSF) with a total protein of 45 mg% and an increased IgG fraction (15%). CSF glucose was normal, and there was no pleocytosis. CSF cultures were sterile.

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ELECTRODIAGNOSTIC EXAMINATION

Methods. Nerve conduction studies were performed using standard techniques of supramaximal percutaneous stimulation and surface recording. Amplitudes of compound muscle action potentials (CMAPs) were measured from baseline to negative peak and were reported for stimulation at distal and proximal sites; conduction velocity was measured in the forearm or leg segment. Evidence of abnormal temporal dispersion was estimated by comparing proximal and distal CMAP amplitudes, recognizing that the measure may reflect some combination of conduction block as well as temporal dispersion.⁶ Proximal to distal amplitude ratios less than 0.7 were considered abnormal, as were negative phase duration increases exceeding 20%.¹ F response latencies were measured as the minimal latency in a series of F responses following distal (wrist or ankle) motor nerve stimulation. Sensory nerve action potential (SNAP) amplitudes were measured peak to peak. Skin temperatures were recorded and maintained above 32°C for all recordings using an infrared heater. Needle electromyography was performed using a standard concentric needle electrode. Insertional activity was characterized subjectively. Spontaneous activity at rest (fibrillation potentials and/or positive sharp waves) was graded from 0 to 4+:

- 0 = no fibrillation
- 1+ = persistent single trains in at least 2 areas
- 2+ = moderate numbers in 3 or more areas
- 3+ = many in all areas
- 4+ = filling the baseline in all areas

Motor unit action potential (MUAP) recruitment, amplitude, duration, and configuration were estimated subjectively.

Nerve Conduction Studies. Initial motor and sensory conduction studies of the upper and lower extremities, performed at the referring institution 5 days after the onset of symptoms, revealed borderline-prolonged distal latencies, a reduced median CMAP amplitude, and reduced median SNAP amplitude with normal sural SNAP recordings. Needle electromyography revealed only decreased recruitment.

Electrodiagnostic studies performed after transfer to our institution (3 weeks after onset of symptoms) are shown in Table 1. SNAPs could no longer be recorded using conventional methods of surface stimulation and recording. Evoked CMAPs were of reduced amplitude and temporally dispersed, with prolonged distal latencies and

reduced conduction velocities in nerves tested. F response latencies were markedly prolonged and difficult to obtain.

Needle Electromyography. Needle electromyography demonstrated a moderate to severe decrease in recruitment without evidence of either increased insertional activity or abnormal spontaneous activity in extremity muscles. A slight increase in insertional activity was recorded in paraspinous muscles.

Repeat needle electromyography 2 weeks later at the nadir of her illness (5 weeks after onset) was unchanged.

Interpretation. The clinical findings suggested a predominantly motor polyneuropathy of relatively acute onset. The electrodiagnostic evaluation demonstrated a moderately severe demyelinating polyradiculoneuropathy of recent onset. The findings, taken together, were consistent with those expected in the Guillain-Barré syndrome.

CLINICAL COURSE

There was progression of weakness (MCR grading 0 to 3) involving facial, bulbar, and extremity muscles with a concomitant deterioration of respiratory function over approximately 7 days, prompting transfer to our institution. Admission to the intensive care unit was necessitated by a tachyarrhythmia and fluctuating blood pressure. Assisted ventilation was not required.

Repeat CSF examinations demonstrated progressively increasing total protein (200 mg% in week 3). Repeat rheumatoid factor and ANA titers were transiently elevated and immune complexes were present in abnormal quantities as measured by the Raji cell assay. Convalescent serums demonstrated a marked increase in the cytomegalovirus titer to 1:256 without change in other titers. Throat swab eventually produced a positive culture of cytomegalovirus.

Therapeutic plasma exchange, the current treatment of acute Guillain-Barré syndrome,³¹ was considered but not performed because of clinical stabilization shortly after admission to our hospital. Unequivocal improvement was noted 2 weeks later, 5 weeks after onset of her initial symptoms. She was discharged after a 2-month hospitalization. She was able to ambulate with assistance. Follow-up evaluations demonstrated progressive improvement, and she had only mild symptoms of fatigue and signs of distal weakness 4 months later.

Table 1. Electrodiagnostic studies performed 3 weeks after onset of symptoms.

Nerve conduction studies											
Stimulate	Record	Amplitude (mV or μ V)			Conduction velocity (msec)			Distal latency (msec)			
		Right	Left	Normal	Right	Left	Normal	Right	Left	Normal	
Ulnar M											
Wrist	Hypothenar	3.5	5.1	6–16				4.8	4.4	1.8–3.5	
Below elbow		2.8	2.1		44	46	49–71				
Above elbow		2.7	2.1		51	49					
Ulnar F response	Hypothenar							51	49	<31	
Median M											
Wrist	Thenar	3.1		4–18				5.8		2.4–4.4	
Elbow		2.0			47		49–70				
Median F response	Thenar							53		<31	
Peroneal M											
Ankle	EBD	1.2		2–12				11.0		3.3–6.1	
Below fibula		0.3			30		41–57				
Above fibula		0.2			32						
Peroneal F response	EBD							74		<45	
Median S wrist	Index	NR	NR	>20				NR	NR	2.5–3.7	
Sural S calf	Ankle	NR		6–47				NR		3.2–4.2	

Needle Electromyography					
Muscle	Insertional activity	Spontaneous activity		Voluntary motor unit action potentials	
		Fib.	Fasc.		
L. biceps brachii	Normal	0	0	mod ↓ #, nl A,D, %P	
L. FDI (hand)	Normal	0	0	mod ↓ #, nl A,D, %P	
L. vastus medialis	Normal	0	0	sl ↓ #, nl A,D, %P	
L. anterior tibialis	Normal	0	0	mod ↓ #, nl A,D, %P	
L. FDI (pedis)	Normal	0	0	sev ↓ #, nl A,D, sl ↑ %P	
L. Parasp.-mid lumbar	Few Unsust. + waves, CRD	0	0	—	

Note: NR = no response, M = motor, S = sensory, CRD = complex repetitive discharge, A = amplitude, D = duration, + = positive waves, %P = percent polyphasic MUAPs, # = number of MUAPs, ↑ = increased, ↓ = decreased. Temperatures: right forearm = 34.5°C, right palm = 33.0°C, left forearm = 33.5°C, right ankle = 32.5°C.

DISCUSSION

Guillain-Barré syndrome is a nonfamilial inflammatory demyelinating disease of peripheral nerve that may be associated with extensive secondary axonal and even anterior horn cell degeneration.⁵ Antecedent events are common and include infections (viral, mycoplasmal, and chlamydial), immunization, malignant disease, and surgery.⁴ In our patient, convalescent serums were consistent with a recent cytomegalovirus infection. The etiology of Guillain-Barré syndrome has not been established, but immunologic mechanisms almost certainly are involved.⁹ Although a full range of clinical features exists,²⁹ cardinal features are characterized by a symmetric, rapidly progressive quadriparesis frequently involving bulbar and respiratory muscles, associated with absent or markedly reduced muscle stretch reflexes, and elevated CSF protein.^{4,5,16,29,33} Although a variety of cranial nerve palsies may occur,⁷ facial mononeuro-

pathies are most common, being described in at least half of the patients.⁴ Despite frequent sensory symptoms, objective sensory loss is infrequent.⁷ Dysautonomia is common and results in bowel and bladder impairment, cardiac dysrhythmias, labile heart rate and blood pressure, and impaired thermoregulation.³²

The interval from onset to peak impairment is approximately 2 weeks in 50% of the patients,⁷ and patients who progress for more than 4 weeks should have alternative diagnoses considered. Mechanic ventilation is required in about 30% of patients and usually is initiated within 18 days after onset (mean of 10 days).³ Complete recovery, when defined as return to all previous activities, occurs in approximately 50% of patients.³³ Death occurs in 2–6% of patients, and 7–22% are left with a substantial neurologic impairment.^{19,27,29,33}

A variety of electrodiagnostic findings are reported, reflecting the temporal changes that occur

in response to cumulative multifocal demyelination and axonal degeneration.¹ Studies reported for 49 patients evaluated during the first 3 weeks of illness¹⁷ demonstrated no abnormality of conduction in 14%, conduction velocity less than 70% of the normal mean in 61%, and prolonged distal latencies only without substantial abnormality of conduction velocity in 25%. In a subsequent study of 114 patients with a clinical diagnosis of Guillain-Barré syndrome,²¹ marked slowing of conduction velocity or an abnormally prolonged distal latency consistent with demyelination was found in one or more nerves in about 50% of patients, and abnormalities of sensory conduction were reported in 75% of patients.

Similar electrodiagnostic results were reported for 30 patients evaluated during or before the third week of involvement: 3 (10%) had no abnormality of motor conduction, 14 (47%) had reduced conduction velocities with proportionally prolonged distal latencies, 7 (23%) demonstrated prolonged distal latencies only, 3 (10%) had reduced CMAP amplitude only, and 3 (10%) had completely absent CMAPs.¹ Minimal criteria suggestive of segmental demyelination were present in about 50% of patients during the first 2 weeks of illness. This increased to about 85% during the third week. Follow-up evaluation of patients having only prolonged distal latencies demonstrated sequential slowing of conduction velocity and/or partial conduction block. The slowest motor conduction velocities recorded were in the range of 15–25 m/sec. SNAP amplitudes were abnormal in 16 of 20 patients evaluated.

Studies performed very early when the diagnosis may be unclear often demonstrated only delayed or absent F responses.¹ Subsequent examinations demonstrated evidence of segmental demyelination including evidence of abnormal temporal dispersion and partial conduction block (e.g., Fig. 1), best demonstrated in motor nerves.¹⁸ The pronounced temporal dispersion is useful in distinguishing acquired from familial demyelination¹⁸ and can be explained by the variable amount of demyelination of individual axons resulting in a marked increase in the range of conduction velocity and block of conduction in some fibers. When obtainable, F response latencies may be greater than expected from distal conduction velocities, indicating proximal involvement.²¹ Uniform electrodiagnostic criteria for segmental demyelination in motor nerves have not had widespread application. Minimal criteria such as described above¹ may result in overestimation of the presence of demyelination in suspected Guillain-Barré syndrome. Use of more restrictive criteria (Table 2) will reduce false-positive studies.

Sensory evoked potentials may remain normal but are absent in the median or ulnar nerves in 50% of patients¹⁴ and abnormal in 76% of patients.²¹ Interestingly, SNAPS may be abnormal or absent in certain nerves (e.g., median and ulnar) while normal in others (e.g., sural).^{1,24} During the first 3 weeks of illness, almost 50% of patients will have an abnormal median sensory response but normal sural nerve conduction studies.¹ This finding in association with the appropriate clinical picture is characteristic of the

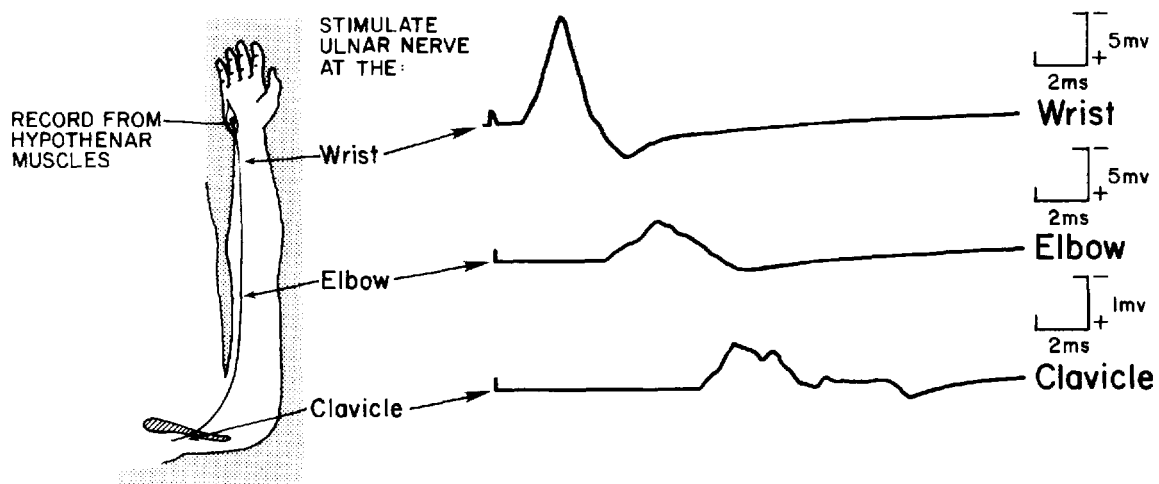


FIGURE 1. Abnormal temporal dispersion and partial conduction block of ulnar compound muscle action potential, recorded from hypothenar muscles of patient with Guillain-Barré syndrome. Modified from Albers JW: Electromyography in the prognosis of nerve injury. *American Academy of Neurology Special Course No. 22: Clinical Electromyography*, 1980.

Table 2. Criteria suggestive of demyelination in the electrodiagnostic evaluation of Guillain-Barré syndrome.

Demonstrate at least three of the following in motor nerves (exceptions noted below):

1. Conduction velocity less than 90% of lower limit of normal if amplitude exceeds 50% of lower limit of normal; less than 80% if amplitude less than 50% of lower limit of normal (2 or more nerves).^a
2. Distal latency exceeding 115% of upper limit of normal if amplitude normal; exceeding 125% of upper limit of normal if amplitude less than lower limit of normal (2 or more nerves).^b
3. Evidence of unequivocal temporal dispersion or a proximal to distal amplitude ratio less than 0.7 (1 or more nerves).^{b,c}
4. F response latency exceeding 125% of upper limit of normal (1 or more nerves).^{a,b}

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^aExcluding isolated ulnar or peroneal nerve abnormalities at the elbow or knee, respectively.

^bExcluding isolated median nerve abnormality at the wrist.

^cExcluding the presence of anomalous innervation (e.g., median to ulnar nerve crossover).

Guillain-Barré syndrome. It may be due to particular susceptibilities at entrapment points²⁴ or may reflect a distal predilection of involvement.

Conduction abnormalities in classic Guillain-Barré syndrome are similar to those recorded from isolated guinea-pig sciatic nerve following induction of experimental allergic neuritis.¹¹ The *in vitro* abnormalities include nerve conduction block in some fibers with nerve conduction slowing in others, resulting in excessive temporal dispersion. The degree of slowing exceeds that expected with primary axonal degeneration. The findings suggesting distal predilection are consistent with the experimental observations of Sumner,³⁰ who found that distal nerve twigs and common compression sites were, along with nerve roots, potential areas of vulnerability to humorally induced demyelination because of an impaired blood-nerve barrier.

The role of needle EMG in evaluating patients with Guillain-Barré syndrome is secondary. Initial needle electromyographic findings consist only of abnormal MUAP recruitment, without configuration changes. Myokymic discharges occasionally are observed during the first few weeks of illness.¹² Abnormalities 3–4 weeks after onset vary from only reduced recruitment to evidence of extensive denervation.²⁰ Interestingly, abnormal spontaneous activity appears simultaneously in proximal and distal muscles,¹ consistent with either random axonal degeneration along the axon or predominant distal predilection.

Some patients having a clinical diagnosis of Guillain-Barré syndrome have no slowing of conduction velocity or temporal dispersion.²⁶ The diagnosis of acute intermittent porphyria should be considered in such patients. Electrodiagnostic studies suggest that porphyric neuropathy is predominantly an axonal neuropathy with major involvement at the root or cord level.² Conduction study abnormalities include low amplitude CMAPs without evidence of substantial temporal dispersion, conduction block, or slowed conduction velocity, although patients with variegate porphyria may have electrodiagnostic evidence suggestive of demyelination with partial conduction block.⁸

The majority of acute toxic neuropathies are of the axonal loss type, and electrodiagnostic studies easily distinguish them from Guillain-Barré syndrome. A notable exception involves high-dose arsenic poisoning, where early electrodiagnostic testing may reveal findings suggestive of an acquired segmental demyelinating polyradiculoneuropathy.¹³

Prediction of the course or eventual outcome of Guillain-Barré syndrome based upon clinical or CSF findings has been disappointing. No significant correlation exists between persistence of neurologic deficit and patient age, sensory loss, papilledema, or CSF pleocytosis.²⁵ Although ventilator dependency and rapid evolution of weakness are more common in patients with a poor prognosis, the best predictor of poor outcome is a reduced average CMAP amplitude less than 10% of the lower limit of normal.²³ This is consistent with observations of others^{10,22,26} who have identified 2 patterns of electrodiagnostic abnormality. One is characterized by gross abnormalities of conduction velocity with little evidence of fibrillation potentials on needle EMG, and the other is characterized by evidence of extensive denervation, with or without nerve conduction velocity abnormalities. Patients in the first group recovered rapidly and relatively completely. Patients in the second group demonstrated poor recovery with pronounced residual deficits.

The seeming paradox,²² that patients with the slowest conduction velocities improved rapidly, reflects the most readily reversible underlying pathophysiology, e.g., demyelination. This emphasizes that CMAP amplitude loss with distal stimulation usually reflects axonal degeneration. The main prognostic distinction then depends upon identification of the degree and extent of axonal degeneration. Although most patients have evidence of some axonal degeneration during the

course of their illness, substantial axonal degeneration is associated with a protracted clinical course and marked residual impairment.²¹

Although the hallmark of axonal degeneration is low-amplitude or absent evoked responses with distal stimulation, extensive conduction block could mimic these findings, although this only rarely causes confusion.^{10,15,28} The relative preservation of average CMAP amplitude in the patient reported (70% of the lower limit of normal) suggested a good prognosis. Although less sensitive, the paucity of fibrillation potentials at 5 weeks also suggested a good prognosis. The fluctuation in CMAP amplitude is demonstrated by sequential studies in another patient shown in Fig. 2.

In summary, complete electrodiagnostic evaluation of patients with suspected Guillain-Barré syndrome requires both motor and sensory conduction studies (performed on multiple nerves in upper and lower extremities), F response latency measurements, and needle electromyography. Because of the patchy or multifocal distribution of involvement, an isolated normal sensory or motor conduction study does not exclude substantial in-

volvement elsewhere. Conversely, focal entrapment with apparent slowing of conduction velocity and/or conduction block must be excluded before concluding the patient has a demyelinating polyneuropathy. The ulnar and peroneal nerves are particularly vulnerable at the elbow and knee, respectively.

To detect proximal involvement, F response latencies should be recorded even when distal extremity conduction velocities are normal. Evidence of conduction block and temporal dispersion are hallmarks of multifocal demyelination and may be present even when there is little abnormality of maximum conduction velocity.

Electrodiagnostic studies are useful in establishing prognosis, particularly when sequential studies are performed during the first 5 weeks of illness. Evidence of conduction block, abnormal temporal dispersion, and preservation of average CMAP amplitude with distal stimulation above 10% of the lower limit of normal are all associated with a good prognosis, while evidence of markedly reduced average CMAP amplitude with extensive, profuse fibrillation potentials are indicative of axonal degeneration and a poor prognosis.

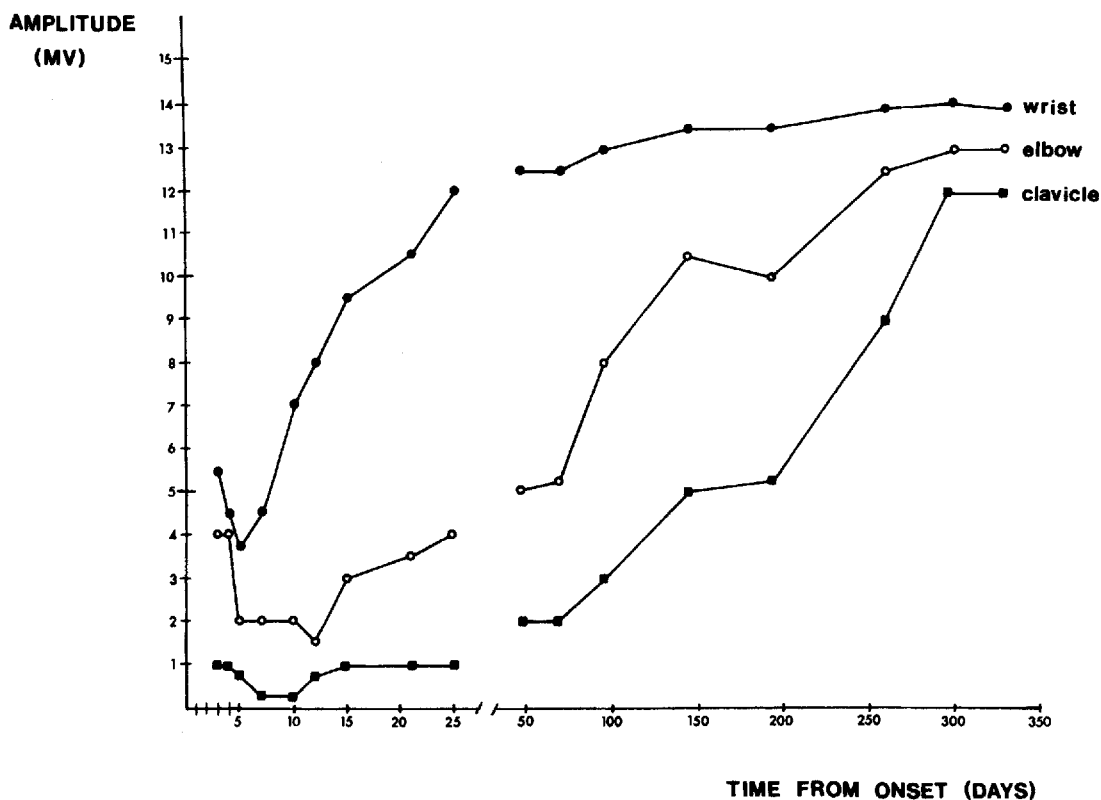


FIGURE 2. Serial ulnar compound muscle action potential amplitudes with proximal and distal stimulation, recording from hypothenar muscles. Same patient as Fig. 1. Modified from Albers JW: Electromyography in the prognosis of nerve injury. *American Academy of Neurology Special Course No. 22: Clinical Electromyography*, 1980.

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