

PERIPHERAL NEUROPATHY

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Quite frequently, podiatrists are faced with difficult patients without an obvious diagnosis. These are patients who may have seen several other specialists who were also stumped by the patient's disease. Often this type of patient involves laboratory testing which includes nerve conduction studies and electromyography. A case of such a patient will be reviewed here. An explanation of how to read and interpret the NCV/EMG report and an analysis of this patient's diagnosis is discussed.

Case Presentation

This 65-year-old retired professional athlete presented with progressive numbness in both feet which began about six years ago. In the last few months the numbness appeared to extend to the distal aspect of his fingers. In the last year he noticed complete collapse of the arch of his right foot (Figure 1). The patient most recently developed an ulcer on the distal aspect of the right hallux.

The patient's medical history included rheumatic fever as a child. There was no history of diabetes mellitus. He occasionally had pain in the left side of the low back but this was not a regular occurrence. Six years ago he noticed that he was unable to sleep because of a feeling of tightness in the feet. Sitting and standing helped to relieve the problem. He was told he was not getting enough blood to his feet. Two years later he had surgery for replacement of an aortic valve. His feet swelled after the operation, however the problem resolved. Vascular consultation at that time revealed normal arterial and venous circulation.

Medications included Coumadin(r), and triamterene and Maxide(r) for hypertension. His father died of a heart attack at age 70. His mother died at age 87 of "old age." He has a sister who died of diabetes mellitus and two other healthy siblings. He used to drink socially, but has stopped drinking entirely since 1989.

Objectives

After reading this article, the podiatric physician will be able:

1. To know the basic characteristics, clinical presentation, and etiology of peripheral neuropathy.
2. To understand the purpose of electrodiagnostic testing and be able to interpret basic electrodiagnostic findings.
3. To be familiar with demyelinating and axonal neuropathies including clinical characteristics and electrodiagnostic findings, and to be able to provide several examples of peripheral neuropathies in each of these two categories.
4. To be able use electrodiagnostic findings in distinguishing between the demyelinating and axonal neuropathy.
5. To use information in the history, physical examination, and laboratory testing to determine a diagnosis.
6. To be able to make distinctions between contributory findings in the history, physical examination, and laboratory testing which lead to the major diagnosis and isolated findings not relevant to the diagnosis.

Neurologic examination revealed absent deep tendon reflexes. There was loss of sensation in the feet especially the distal aspect, relative loss of joint position sense in the toes, decreased vibration sensation in both legs above the knee, and decreased pinprick in the hand relative to the upper leg. Vascular examination was normal with the foot warm with palpable pulses.

Collapsing rigid pes planovalgus of the right foot with marked heel valgus and forefoot abduction was present (Figures 1 & 2). The patient had full muscle power of the ankle dorsiflexors, plantarflexors, and evertors of the foot, but a decrease in muscle power for active inversion of the right foot. The right calf was 1/2 inch thinner than the left. Gait was asymmetric with right collapsing pesplanovalgus, severe hallux valgus and genu valgum. The patient could not stand on his toes on his right foot. At this point in time, what is your diagnosis for this patient?



Figure 1: Patient standing shows collapsing pes planovalgus of the right foot present for about a year.



Figure 2: Patient from the rear. Right foot in marked valgus. "Too many toes" sign is evident.

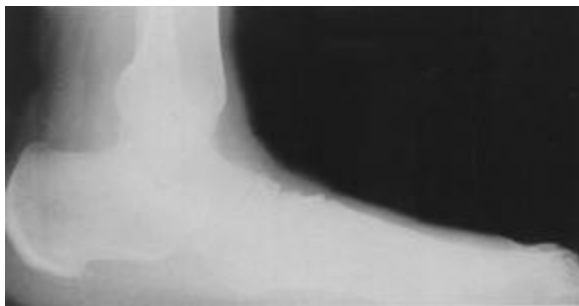


Figure 3 (Above): Lateral radiograph shows collapsing pes planovalgus with equinus of the calcaneus and collapse of the medial column.

Figure 4 (Right): Anteroposterior radiograph shows very early neuroarthropathy of the midfoot (Charcot changes).



Plain radiographs of the right foot revealed collapsing pes planus on the lateral view, hindfoot equinus with lowering of the calcaneal inclination angle, and collapse of the medial column (Figure 3). Anteroposterior views showed very early Charcot changes in

the midfoot (Figure 4). The patient was sent for electrodiagnostic testing to determine the cause of his peripheral neuropathy. Results showed moderate decrease in nerve conduction velocity of the right peroneal nerve with increased distal latency and diminished amplitude (Table 1). There was 1+ fibrillation of the right medial

Table 1: NERVE CONDUCTION VELOCITY/ ELECTROMYOGRAPHY		
PERONEAL NERVE	RIGHT	NORMAL
Distal Latency	17 msec	6 msec
Amplitude	17 mV	2.5 mV
NCV	26 m/sec	>40 m/sec
1+ Fibrillations Right medial gastrocnemius & Right Tibialis Anterior		
2+ PSW (Positive Sharp Wave) Right medial gastrocnemius		

gastrocnemius and right tibialis anterior muscle and 2+ positive sharp waves of the right gastrocnemius. The report was read as moderate to severe predominantly axonal, distal sensory-motor polyneuropathy.

Laboratory tests showed blood glucose performed on two occasions was 102 and 105 mg/DL (70-115 mg/DL); Hemoglobin-A 1C 5.3% (4.2-5.9%); uric acid levels of 9.5 mg/DL (2.5 to 8 mg/DL), cholesterol 249 mg/DL (120-240 mg/DL), Lactic

Dehydrogenase LDH 325 U/L (90-250 U/L), triglycerides 198 mg/DL (30/175 mg/DL). Antinuclear antibody testing, erythrocyte sedimentation rate, vitamin B-12, serum protein electrophoresis were all normal. Reactive plasma reagin (RPR) was nonreactive.

Electrodiagnostic Testing -- Nerve Conduction Velocity

Nerve conduction velocity studies consist of the stimulation of a peripheral sensory or motor nerve or a mixed nerve. A physiologic action potential (AP) is generated by electrical stimulation of the axon of the nerve. In a motor nerve the AP of the corresponding muscle is recorded. The action potential propagates down the axon where it is detected at a distant site by surface electrodes. The latency time is the time between the stimulus and the response and the nerve conduction velocity is the speed of impulse propagation. The amplitude tells how many muscle fibers are firing in a motor unit.

Nerve conduction studies evaluate the integrity of large myelinated sensory and motor neurons.¹ In the patient with peripheral neuropathy NCV testing is used to distinguish between the two major forms of neuropathy, axonal and demyelinating neuropathy. Although history and physical examination can detect the general clinical features of peripheral neuropathy such as loss of sensation, diminished reflexes, weakness and atrophy, clinical examination cannot distinguish between axonal and demyelinating neuropathy.

Demyelinating diseases are characterized by severely decreased nerve conduction velocities, markedly prolonged distal latencies, and normal (or only slightly decreased) AP amplitude (Table 2). In demyelinating neuropathy there is at least a 60% reduction in nerve conduction velocity. Demyelination is directly responsible for reduced speed of the nerve and may produce severe motor and sensory deficiencies. Clinically demyelinating polyneuropathies have greater motor impairment than sensory and little or no pain. Examples of demyelinating neuropathies include Charcot Marie Tooth Disease Type 1A, Guillain-Barre syndrome, and peripheral neuropathy associated with cancer (Table 2).

Axonal neuropathies are usually milder than demyelinating neuropathies and are characterized by spontaneous activity in distal muscles with low amplitude evoked responses, with relatively normal conduction velocity (Table 2). Conduction velocities are normal despite extensive axonal degeneration. This is because myelin is responsible for the speed of the nerve impulse and the myelin remains intact in axonal neuropathies.

Clinically axonal polyneuropathies are symmetrical, begin distally with subacute onset. Polyneuropathies due to axonal degeneration are common and include diabetic neuropathy, alcoholic neuropathy, Charcot Marie Tooth Disease Type 2, and neuropathies caused by nutritional deficiency, connective tissue disease and toxins (Table 2). Many neuropathies are due to mixed axonal-demyelinating processes. Diabetes mellitus may also be associated with a mixed peripheral neuropathy. Other mixed disorders include neuropathy associated with multiple myeloma or lymphoma, and several drug induced neuropathies.

The patient in this report was diagnosed by NCV/EMG studies with a moderate to severe predominantly axonal distal sensory-motor polyneuropathy which is consistent with many neuropathies including diabetic neuropathy and alcoholic neuropathy. The patient's nerve conduction velocity for the right peroneal nerve was 26 meters per second which was reduced to approximately half that of the normal value (Table 1). A typical motor nerve conducts at approximately sixty to seventy meters per second.² See Table 3. Upper extremity motor fibers should conduct at greater than 50 meters per second and lower

**Table 2:
CATEGORIES OF PERIPHERAL
NEUROPATHY**

	Demyelinating Neuropathy	Axonal Neuropath y
Conduction velocity	Markedly reduced	Normal
Distal latency	Prolonged	Normal
Amplitude	Normal	Reduced
Examples	Guillain-Barre Syndrome CMT Type I	Alcoholic CMT Type II

**Table 3:
NERVE CONDUCTION VELOCITY**

Motor Nerve	40-60 meters/second
Upper Extremity	60-70 meters/second
Lower Extremity	>40 meters/second
Highest possible value	80 meters/second
Unmyelinated nerve	7-10 meters/second
Newborns	1/2 adult value with normal adult value achieved by age 5
Age 60	NCV reduced 5%
Temperature	Reduces NCV 5% for each 1 degree temperature drop

extremity motor fibers generally conduct at greater than 40 meters per second,¹ but the fastest that any nerve can travel is 80 meters per second.³ See table Table 3. Unmyelinated nerve conducts at a much slower speed of seven to ten meters per second.²

Electromyography

Electromyography (EMG) is a direct recording of the inherent electrical activity of the muscle itself.⁴ Electromyography involves the measurement and observation of action potentials caused by the depolarization and repolarization of a muscle fiber or a group of muscle fibers as a result of the injury of a probing needle (electrode).⁵ In the normal muscle there is no spontaneous activity.⁶ Fibrillation is spontaneous muscle activity of a resting muscle and represents the denervation of a muscle fiber and is tested by EMG. Fibrillation action potentials develop in muscle fibers two to four weeks after motor axons have been cut or injured.⁶ The fibrillation potential in involved muscle fibers is the single most significant diagnostic finding of nerve damage.⁵ In nerve injuries, at least three weeks should pass before performing electrodiagnostic testing. When a muscle is denervated it becomes irritable and the membrane is unstable.⁷ Positive sharp waves represent unstable muscle membrane and occur about two weeks after denervation.

Electromyography permits localization of a nerve lesion by testing several muscles. For example, abnormal spontaneous potentials present in the tibialis anterior and extensor hallucis longus may be indicative of either a common peroneal nerve problem, a deep peroneal nerve problem, or an L-5 nerve root condition. However, a normal examination of the peroneus longus muscle would localize the abnormality to the deep peroneal nerve.⁴

The patient presented here had 1+ fibrillation potential of the gastrocnemius and anterior tibial muscle and 2+ positive sharp waves of the gastrocnemius all on the right side. Since the gastrocnemius muscle is innervated by the tibial nerve and the anterior tibial muscle is innervated by the peroneal nerve, denervation occurred in at least two different nerves.

Electrodiagnostic testing confirmed the presence of neuropathy for this patient and revealed the nature of the peripheral neuropathy to be a distal predominantly axonal polyneuropathy which involved both sensory and motor nerves. However, the pathology causing the peripheral neuropathy cannot be determined by electrodiagnostic testing. A biopsy of the peripheral nerve might have determined the etiology of the peripheral neuropathy. Nerve biopsy can help to determine types of nerve axon degeneration and patterns of demyelination as well as specific diagnoses responsible for peripheral neuropathy such as vasculitis or amyloidosis.⁸

Explanation of Patient's Finding

Clinically, severe acquired collapsing pes planovalgus foot deformity present unilaterally bears a superficial resemblance to posterior tibial tendon rupture.⁹ However, the incipient neuropathic osteoarthropathy in the midfoot displayed on radiographs are more

characteristic of a pes planovalgus deformity produced by neuropathic arthropathy than that of a traumatic or degenerative posterior tibial tendon dysfunction. More importantly the patient was numb in both legs and the tips of the fingers, therefore a diagnosis of posterior tibial tendon rupture would be too localized and ignores the entire clinical presentation.

Laboratory tests showed slightly elevated uric acid, cholesterol, lactic dehydrogenase, and triglycerides. All other results were normal. The elevated lactic dehydrogenase is a consequence of the patient's heart disease and was not a factor in the diagnosis.¹⁰ The patient's clinical description was more systemic than a localized gouty arthritis so the elevated uric acid was not considered to be a factor in the patient's main diagnosis.

Peripheral Neuropathy

Clinically the patient demonstrates peripheral neuropathy. The two most common causes of peripheral neuropathy in the United States are Diabetes mellitus and alcoholism¹¹ (Table 4). Trauma and entrapment are common, but considered in the differential diagnosis of mononeuropathies.¹² The most frequent pattern of polyneuropathy is seen with metabolic diseases such as diabetes mellitus, nutritional deficiency and renal failure.¹³ Neuropathy develops slowly, often over months or years and begins with sensory abnormalities in the lower extremities.

Sensory loss manifested as numbness, tingling, muscle weakness, atrophy, and diminished deep tendon reflexes are the hallmarks of peripheral neuropathy¹³ (Table 5). Pain when present tends to be worse at night. The patient presented here had a recurring ulcer on the right hallux. All patients with neuropathy are at risk for insensitive foot ulcerations.¹⁴ The plantar hallux ulcer does not help to explain the specific etiology of the peripheral neuropathy.

Most neuropathies give rise to a mixed sensorimotor disturbance, therefore the patient will experience weakness as well as loss of sensation usually described as numbness. The first manifestation in symmetrical polyneuropathies are usually in the legs¹⁵ and the legs are involved more than the arms (Table 5). Distal muscle involvement occurs more frequently than involvement of proximal muscles.¹⁴ Peripheral neuropathy involves muscles of extension and abduction such as the anterior groups leg muscles and the peroneal muscles. Therefore drop foot and weakness in eversion are much more common than weak posterior group muscles.^{16,17} Paresthesias are usually described as

COMMON ETIOLOGIES AND CLASSIFICATION OF PERIPHERAL NEUROPATHY

Metabolic

- Diabetes mellitus - in patients who have had diabetes for more than 10 years. Distal sensory polyneuropathy most common form.

Chemical/medicines/toxins

- Alcohol
- Heavy metal-lead, mercury
- Isoniazid
- Phenytoin

Vitamin Deficiency

- Vitamin B12, most common, also Vitamins A, B2, B6, folate, E

Inherited Sensorimotor Neuropathy

- Charcot Marie Tooth Disease Type I Demyelinating
- Charcot Marie Tooth Disease Type II Axonal

Infectious

- HIV
- Leprosy
- Lyme disease

Autoimmune

- Guillain-Barre syndrome

Connective Tissue Diseases

- Rheumatoid arthritis
- Systemic lupus erythematosus
- Sarcoidosis
- Polyarteritis nodosa

Paraneoplastic Syndromes

- Carcinoma
- Lymphoma
- Myeloma

Table 5: PERIPHERAL NEUROPATHY -- FEATURES

Hallmark Features of Peripheral Neuropathy

- Numbness
- Tingling
- Pain
- Weakness

numbness, tingling, buzzing, or a feeling of constriction.

Differential Diagnosis

Peripheral neuropathy may be confused initially with myopathic disease since both kinds of disorders may involve varying degrees of weakness with diminished or absent reflexes. Peripheral neuropathy is distinguished from myopathic disease by its predominantly distal muscle involvement whereas myopathies tend to affect the proximal muscles of the pelvic and pectoral girdle. Also myopathies, in contrast with peripheral neuropathies do not have a sensory component.

Diabetic Neuropathy

Distal symmetrical sensorimotor polyneuropathy is the most commonly encountered form of diabetic neuropathy⁸ and is a common complication of diabetes mellitus.¹⁸ It is estimated to be present in about one-quarter of diabetic patients.¹⁹ Diabetic peripheral neuropathy is a progressive neuropathy, initially involving the more distal parts of the lower extremities and then progressing centrally to involve the hands, thighs, and in severe cases the trunk.²⁰ The major symptoms consist of numbness in the legs and feet, hyperesthesia, hyperpathia, with aching and burning pain. Not all patients have pain. Sensory symptoms consist of reduced or absent sensation to pain, touch, cold, heat, and vibration. Slowing in motor and sensory conduction is a common finding in diabetics and is attributed to axonal degeneration with secondary demyelination.

Diabetic neuropathy was consistent with the patient's clinical and electrodiagnostic testing. In addition, the patient had a sister that died of diabetes. However, two normal blood glucose tests as well as a normal hemoglobin A1-C test adequately ruled out diabetes mellitus as the etiology of this patient's peripheral neuropathy. Since peripheral neuropathy caused by diabetes usually does not occur until the patient has had diabetes for at least ten years,²¹ a subclinical case of diabetes responsible for this patient's neuropathy was considered unlikely.

Alcoholic Neuropathy

Alcoholic neuropathy is slowly progressive distal sensorimotor polyneuropathy.²²⁻²⁴ The disorder begins distally and slowly spreads more proximally. Patients present with paresthesias, pain and weakness. Foot drop, wrist drop, proximal weakness with difficulty rising from a squatting position or chair may eventually be present. Plantar ulcers and Charcot foot type disorders may occur.^{25,26} Although alcoholic neuropathy does have a similar clinical presentation to this patient's neuropathy, and the patient admitted to social drinking which he had not done since 1989, it was most probably not sufficient to be a cause of peripheral neuropathy. Alcoholic neuropathy only develops in cases of long-term alcohol abuse that involves dietary deficiency.¹⁴

Genetic Neuropathy

The genetic neuropathies are characterized as slowly progressive over years with insidious onset. Charcot Marie Tooth Disease is the most common inherited peripheral neuropathy.²⁷ It is most frequently inherited by autosomal dominant inheritance, but can be inherited by autosomal recessive or sex-linked inheritance. It is characterized by progressive distal muscle wasting and weakness, areflexia and cavovarus foot deformities. Patients with CMT disease present most frequently with pes cavus (85% of cases) and drop foot.²⁸ There is severe slowing of nerve-conduction velocities and pathologic analysis of nerves reveals simultaneous remyelination and demyelination. This disorder usually begins in late childhood. This patient clearly did not have cavovarus foot type with drop foot characteristic of CMT Disease. There was also no family history of CMT disease.

Nutritional neuropathy (most frequently vitamin B12, but also other B vitamins, vitamin A, E, and folate) are common causes of peripheral neuropathy.¹⁴ The patient's B12 and folate level were normal ruling out pernicious anemia and nutritional deficiency as a cause of the patient's neuropathy.

Connective tissue disease is sometimes associated with peripheral neuropathy.²⁹ However, negative antinuclear antibody testing and normal erythrocyte sedimentation tests ruled out connective tissue disease as a cause of this patient's peripheral neuropathy. HIV neuropathy is a distal symmetric sensory polyneuropathy present in the late stages of HIV infection.³⁰⁻³⁴ The patient had a negative HIV test ruling out HIV neuropathy. Serum protein electrophoresis was negative ruling out cancer as a cause of peripheral neuropathy.

Any medication or drug potentially can cause peripheral neuropathy. The patient here was taking Coumadin(r), triamterine, and Maxide(r). However, none of these have previously been related to peripheral neuropathy. Possibly the patient could have had temporary ischemia prior to the aortic valve surgery in 1992 which eventually resulted in the sequela of peripheral neuropathy. Ischemic vascular disease may result in subacute or chronic neuropathy.³⁵

Finally, malignancy as a cause of peripheral neuropathy is suspected in an elderly patient with a subacute sensory neuropathy or polyradiculopathy of obscure cause. The course is usually progressive and usually there is weight loss.

Final Diagnosis

The patient had a slowly progressive peripheral neuropathy of undetermined etiology. This patient saw three neurologists, an internist and orthopedist. Although electrodiagnostic testing revealed the neuropathy to be a distal predominantly axonal polyneuropathy, the specific etiology remained unknown. Chronic neuropathies have been known to evolve over a half-century.³⁶ In a high proportion of peripheral neuropathy the explanation remains uncertain.^{37,38} Even with extensive testing for peripheral neuropathy, the etiology can be found in no more than 50 percent of patients admitted to the hospital for evaluation.³⁹

In a series of 205 cases of undiagnosed neuropathy referred to the Mayo Clinic, it was possible to find an etiology in 76%.⁴⁰ The majority of patients in this study obtained a diagnosis by family investigation and were found to have hereditary sensorimotor neuropathy. In evaluating the patient with peripheral neuropathy, a detailed family, social, and medical history; neurologic examination; and electrodiagnostic testing and nerve biopsy are often necessary to make a diagnosis. Even then the diagnosis may remain unknown in a certain percentage of cases.

QUESTIONS

1. The best clinical diagnosis for this patient is:
 - A. Posterior tibial tendon dysfunction
 - B. Peripheral neuropathy
 - C. Rigid flatfoot secondary to tarsal coalition
 - D. Rigid flatfoot secondary to vertical talus
2. What were the findings of the patient's x-rays?
 - A. Inconclusive
 - B. Showed pes planovalgus changes produced by posterior tibial tendon rupture
 - C. Showed incipient neuro-osteoarthopathy of the midfoot
 - D. Showed bony erosions and osteoporosis
3. The patient's electrodiagnostic testing revealed:
 - A. The type of peripheral neuropathy
 - B. Nerve entrapment at the level of the peroneal nerve
 - C. Peroneal mononeuropathy
 - D. The cause of the patient's peripheral neuropathy
4. As compared to myopathy, peripheral neuropathy:
 - A. affects proximal muscles and has a sensory component
 - B. affects proximal muscles and has no sensory component
 - C. affects distal muscles and has a sensory component
 - D. affects distal muscles and has no sensory component
5. Peripheral neuropathy can be characterized best by:

- A. Weakness, sensory loss, pain, diminished reflexes, atrophy over time
- B. Weakness, sensory loss, but painless, diminished reflexes, and atrophy over time
- C. Sensory loss, but does not involve motor loss
- D. Increased deep tendon reflexes and spasticity

6. Electrodiagnostic testing can confirm the presence as well as the nature of the peripheral neuropathy, however, the pathologic etiology of peripheral neuropathy cannot be determined by electrodiagnostic testing.

- A. True
- B. False

7. What etiology of peripheral neuropathy can be determined by serum protein electrophoresis testing?

- A. Connective tissue disease
- B. Vasculitis
- C. Cancer
- D. Infectious disease

8. What are the two main forms of neuropathy?

- A. peripheral and central
- B. voluntary and involuntary
- C. voluntary and autonomic
- D. demyelinating and axonal

9. An example of a demyelinating neuropathy would be:

- A. Alcoholic neuropathy
- B. Guillain-Barre Syndrome
- C. Charcot Marie Tooth Disease type 2
- D. Connective tissue neuropathy

10. A patient claimed to have peroneal nerve damage after undergoing knee surgery three days ago. Electromyography performed 3 days after the surgery revealed positive sharp waves and fibrillation potentials. The best conclusion is:

- A. Normal EMG
- B. Abnormal NCV
- C. The surgery was responsible for damage to the peroneal nerve.

D. The nerve damage was most probably present prior to the surgery

11. The cause of the patient's flatfoot was most probably:

- A. neuropathic osteoarthropathy
- B. posterior tibial tendon dysfunction
- C. Arthritis
- D. Connective Tissue Disease

12. The cause of this patient's peripheral neuropathy was:

- A. Diabetes mellitus
- B. Charcot Marie Tooth Disease
- C. Nutritional neuropathy
- D. Never determined

13. Which is INCORRECT about alcoholic neuropathy?

- A. It develops only with long-term alcohol abuse and dietary deficiency
- B. Symptoms can range from mild to severe
- C. The neuropathy can be asymptomatic
- D. Neuropathy is a sensory polyneuropathy

14. What is the greatest velocity of a peripheral nerve?

- A. 7-10 meters per second
- B. 40 meters per second
- C. 60 meters per second
- D. 80 meters per second

15. As compared to axonal neuropathy, demyelinating neuropathy has:

- A. reduced amplitude and increased latency
- B. Increased amplitude and increased latency
- C. reduced latency and reduced velocity
- D. Increased latency and reduced velocity

16. The lowest nerve conduction velocity would be present in:

- A. Neonate
- B. Person over sixty years old

- C. Nonmyelinated nerve
- D. Motor nerve

17. The results of the patients electrodiagnostic testing would be consistent with all of the following diseases EXCEPT:

- A. Diabetic neuropathy
- B. Alcoholic neuropathy
- C. Connective tissue neuropathy
- D. Guillan-Barre disease

18. As compared to an upper extremity motor neuron, a lower extremity motor neuron would NORMALLY travel:

- A. Faster
- B. Slower
- C. About the same speed
- D. Could be slower or faster

19. The two most common cause of peripheral neuropathy in the United States is:

- A. Diabetes mellitus and Charcot Marie Tooth Disease
- B. Diabetes mellitus and alcohol
- C. Diabetes mellitus and syphilis
- D. Nutritional deficiency and alcohol

20. On EMG examination abnormal fibrillation potentials are present in the extensor digitorum longus and the anterior tibialis muscle, but the peroneus brevis muscle is normal. What can be concluded from the results of the EMG examination?

- A. The problem is localized to the deep peroneal nerve
- B. The problem is localized to the common peroneal nerve
- C. The patient most probably has an L-5 radiculopathy
- D. The results may be indicative of a deep peroneal nerve lesion, a common peroneal nerve lesion, or an L-5 radiculopathy

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