AANEM Monograph #2
Important Anomalous Innervations of the Extremities

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EDUCATIONAL OBJECTIVES  This monograph directs the attention of the physician learning electrodiagnostic medicine to the important anomalous innervations of the extremities that affect the interpretation of electrodiagnostic studies. The monograph discusses (1) median-to-ulnar nerve communications, (2) ulnar-to-median nerve communications, (3) variations in the innervation of intrinsic muscles of the hand, (4) accessory deep peroneal nerve, and (5) tibial-to-peroneal nerve communication. Emphasis is placed on the anatomy of these anomalous innervations, their electrophysiologic recognition and the manner in which they affect the interpretation of electrodiagnostic studies. The practicing physician should be able to apply this information to improve diagnostic accuracy and thereby contribute to better patient care.

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Anomalies of peripheral nerves and variations in the innervation of muscles of the extremities and cutaneous areas occur commonly. The anatomy of most of these are reviewed in detail in *Nerves and Nerve Injuries*, by Sydney Sunderland. Knowledge of a few of these is of crucial importance to the electrodiagnostic (EDX) physician in the performance of nerve conduction and electromyographic (EMG) studies in normal patients and those with peripheral nerve lesions. A lack of awareness of these common anomalies and variations risks faulty interpretation of the electrophysiologic data obtained during nerve conduction and EMG examination. This monograph deals with anomalous median-to-ulnar nerve communication and ulnar-to-median nerve communication both in the forearm, variations in the innervations of the intrinsic muscles and sensory dermatomes of the hand, anomalous innervation of the extensor digitorum brevis (EDB) muscle by the accessory deep peroneal nerve, and an anomalous connection between tibial and peroneal nerves.

**MEDIAN-TO-ULNAR COMMUNICATIONS**

Communications between the median and ulnar nerves occurring in the forearm have been well-described in the literature but receive scant attention in most anatomy books (Figure 1). The anomaly was described as early as 1763 by the Swedish anatomist R. Martin and later by W. Gruber in 1870. Other reviews are by Rowntree, Mannerfelt, and Wilbourn and Lambert.

**Anatomic Aspects.** The median-to-ulnar nerve communications always involve axons descending in the median nerve, crossing through the forearm to join the ulnar nerve, ultimately innervating intrinsic hand muscles. Srinivasan and Rhodes noted the anomaly in 15% of normal fetuses and that it originated from the anterior interosseus nerve in most cases. Less commonly, the anastomosis did not join the ulnar nerve directly but innervated the flexor digitorum superficialis or flexor digitorum profundus. In addition, they noted the anomaly bilaterally in all their cases.

**FIGURE 1.** Compound muscle action potential (CMAP) from several muscles on median nerve stimulation at the elbow and wrist in a normal individual with median-to-ulnar nerve communications. Stimulation at the elbow evoked a large CMAP from the first dorsal interosseus (FDI). Wrist stimulation evoked a small CMAP from the FDI. The initial positive deflection suggested it arose from thenar muscles. The small hypothenar CMAP preceded by a positive deflection on both elbow and wrist stimulation suggested it was volume conducted from thenar muscles.
cases (8 of 8) of trisomy 21. Although a significant number of axons in a given patient may take this anomalous route, it does not involve all the axons of the median nerve. The median-to-ulnar nerve communications are sometimes referred to as Martin-Gruber anastomosis after its two early describers. Gruber\textsuperscript{10} and Mannerfelt\textsuperscript{20} described this anomaly to occur in 15% of extremities studied. Wilbourn and Lambert\textsuperscript{36} using electrophysiologic techniques similar to those described in this manuscript, found it to occur in 31% of subjects and 21% of limbs studied. These median nerve axons that cross to the ulnar nerve in the forearm innervate various intrinsic muscles of the hand. In a total of six cases, Mannerfelt\textsuperscript{20} found these axons to innervate the first dorsal interosseous (FDI) in five, adductor pollicis in four, and hypothenar muscle in two. Wilbourn and Lambert\textsuperscript{36} in 22 limbs showing the anomaly, found these median axons to innervate the FDI area 21 times, hypothenar 9 times, and thenar 3 times. Crutchfield and Gutmann\textsuperscript{5} noted the anomaly in 28% of 50 persons and 62% of family members of 5 propositi with this variant. This suggested a dominant mode of inheritance.

**Electrophysiology—General Aspects.** Anomalous median-to-ulnar-communication involves motor axons almost exclusively. The studies of Srinivasan and Rhodes\textsuperscript{31} would predict this to be the case, because the anastomosis in most cases occurs through the anterior interosseus nerve and this is a pure motor branch of the median nerve. Involvement of sensory axons is unusual but was reported in one case by Santoro and colleagues.\textsuperscript{28} Valls-Solé\textsuperscript{34} reports a case of carpal tunnel syndrome (CTS) and median-to-ulnar nerve communication in whom an additional sensory anomaly occurred. The ulnar side of the third finger and the radial side of the fourth were supplied by ulnar nerve axons that were confined to the ulnar nerve and did not traverse the median nerve at any point. Despite the presence of both a motor and sensory anomaly involving the hand, they were not linked together, again speaking to the expected purely motor component of the median-to-ulnar communication in most cases.

This anomaly may be suspected in otherwise normal individuals when carrying out routine motor conduction studies. In the median nerve, the compound muscle action potential (CMAP) tends to be mildly higher in amplitude (and mildly shorter in duration) when the nerve is stimulated supramaximally distally (wrist) as compared with more proximally (elbow). This is due to increased synchronization at distal stimulation. The amplitude of the CMAP on wrist stimulation is normally larger than that on elbow stimulation and this increase is up to 120%. In the presence of the anomaly, the CMAP on elbow stimulation may be significantly larger. This is so because there are more axons available for stimulation in the median nerve at the elbow than the wrist. Although the CMAP is, in each case, recorded from the thenar muscle group (with the recording electrode usually lying over the motor point of the abductor pollicis brevis [APB]), contributions from FDI, adductor pollicis, and thenar muscles innervated by the anomalous axons (located in the ulnar nerve at the wrist) contribute to the larger CMAP on median nerve stimulation at the elbow.

Determining whether this is in fact the case requires supramaximal median nerve stimulation while recording from FDI and hypothenar muscles. If the anomalous axons are present, stimulation of the median nerve at the elbow will evoke a CMAP from one or both muscles. Wrist stimulation of the median nerve will evoke a much smaller or no CMAP from the FDI and no CMAP from the hypothenar muscle.

In Figure 1, from a normal individual, stimulation of the median nerve at the elbow evoked a much larger CMAP from the FDI muscle than stimulation at the wrist. There is no evidence of median crossover innervation to the hypothenar muscle. Figure 2 shows the same occurrence in a patient with concomitant CTS with median to ulnar anastomoses involving both the FDI and hypothenar muscles.

**FIGURE 2.** Compound muscle action potential (CMAP) evoked from several muscles on median nerve stimulation at the elbow and wrist in a patient with both carpal tunnel syndrome and median-to-ulnar nerve communications. The thenar CMAP on elbow stimulation is preceded by a positive deflection not seen on wrist stimulation. Stimulation at the elbow evoked a CMAP from the first dorsal interosseus and hypothenar muscles. Wrist stimulation evoked a much smaller CMAP which, by virtue of the preceding positive deflection, probably arose from the thenar muscle. The motor conduction velocity (recording thenar muscle) was spuriously fast at 74.4 m/s (difference in latencies in 2.5 over a distance of 186 mm).
Conversely, conduction studies in the ulnar nerve may show hypothenar and/or FDI CMAPs that are much larger (greater than 120%) on wrist stimulation than elbow stimulation. This increase is more than that expected from just synchronization and, of course, reflects the increased number of axons present in the ulnar nerve at the wrist as a result of the anomaly (Figure 3). Such ulnar nerve changes might also indicate an ulnar neuropathy if the concomitant median nerve conduction changes described above are not present. It is rather important at this point to make certain that the increased size of the CMAP on ulnar stimulation at the wrist is not related to the spread of stimulus to the median nerve lying nearby at the wrist. These changes in the ulnar nerve (i.e., a much larger hypothenar or FDI muscle CMAP on wrist compared with elbow stimulation) may be easily misinterpreted by the unwary EDX physician as a physiologic block (neurapraxia) above the elbow in the ulnar nerve. That these findings are due to the median-ulnar nerve communications is, however, easily documented by demonstrating the smaller CMAP not only on stimulation of the ulnar nerve above the elbow (arguing against a physiologic block in the region), in addition to seeing the appropriate changes noted above on median nerve stimulation. This is an extremely important point. Last, Kimura has demonstrated that this anomaly can be easily documented using the collision technique.

**Electrophysiological Aspects—Median Nerve Lesions.** When anomalous communication occurs in association with median and ulnar nerve lesions, it may produce additional electrophysiological changes that make interpretation difficult unless the EDX physician has a clear awareness of the anomaly. In the presence of CTS, median nerve stimulation at the elbow produces a thenar CMAP that is preceded by an initial positive deflection despite the fact that recording electrode lies over the motor point. This initial positive deflection is not seen on median nerve stimulation at the wrist (Figures 2 and 4). If it is, this now reflects faulty recording electrode placement. Median and ulnar nerve stimulation studies while recording from thenar, hypothenar, and FDI muscles (as described above) will document that this characteristic change in the CTS is due to the anomaly.

Gutmann noted this change in 20% of median nerves in 63 consecutive patients with bilateral CTS. This is in keeping with the incidence of the anomaly. The thenar CMAP preceded by an initial positive deflection on elbow stimulation, but not on wrist stimulation, occurs for the following reason. Conduction in the median nerve axons going through the carpal tunnel and innervating thenar muscles is slower than those median nerve axons crossing to the ulnar nerve and supplying FDI, adductor pollicis, abductor digiti minimi (ADM), and flexor pollicis brevis (FPB). The CMAP from the latter muscles, on median nerve stimulation at the elbow, is generated prior to that from thenar muscles innervated by median axons going through the carpal tunnel. The earlier-appearing CMAP is incorporated into the overall thenar CMAP, but, because it originates at some distance from the recording electrode, produces an initial positive deflection. This initial positive deflection

![FIGURE 3](image-url)

**FIGURE 3.** Compound muscle action potential (CMAP) from several muscles on ulnar nerve stimulation at the elbow and the wrist in a patient with carpal tunnel syndrome and median-to-ulnar nerve communications. First dorsal interosseous CMAP is larger on stimulation at the wrist than elbow. Thenar and hypothenar CMAP are slightly larger on wrist stimulation.

![FIGURE 4](image-url)

**FIGURE 4.** Thenar compound muscle action potential (CMAP) following median nerve stimulation at the elbow and wrist in a patient with carpal tunnel syndrome and median-to-ulnar nerve communications. The CMAP evoked on elbow stimulation is slightly larger and preceded by an initial positive deflection. In contrast to Figure 2, median ulnar conduction velocity was normal.
is due to the volume conduction effect and is not present when stimulating the median nerve at the wrist, below the level of the anomalous interchange.

In six patients with characteristic symptoms of CTS studied by Gutmann and colleagues, electrophysiologic studies were normal except for the initial positive deflection of the thenar CMAP with median nerve stimulation at the elbow but not at the wrist (Figure 5). Specifically, distal motor latencies and sensory latencies were normal in median nerves. As normal electrophysiologic studies occur in CTS (up to 8%) and median-to-ulnar communications are common (15-31%), this observed finding can be of practical clinical importance in the diagnosis of mild CTS, despite otherwise normal electrophysiologic studies when the anomaly is present.

Another less-common finding noted in CTS by the presence of the anomaly, reported by Kimura and Iyer and Fenichel, is an erroneously normal proximal (elbow) motor latency in the median nerve with prolongation of the distal motor latency. The near normal proximal latency is, again, due to the median nerve communications (for the reasons noted above) and results in a spuriously fast-calculated conduction velocity. In the 63 consecutive patients studied by Gutmann, this finding occurred only once (Figure 2).

Additionally, Lambert described a thenar CMAP with two components on median nerve stimulation at the elbow, again related to the slower conduction in the median nerve at the wrist and faster conduction in those axons that have crossed to the ulnar nerve. The ulnar-nerve component is absent when the median nerve is stimulated at the wrist (Figure 6). This also appears to be an uncommon finding.

**Electrophysiologic Aspects—Ulnar Nerve Lesions.** Anomalies may also affect the interpretation of motor conduction studies, as well as the clinical examination, in the presence of an ulnar nerve lesion. This is best exemplified by a complete ulnar neuropathy due to a lesion at the level of the elbow. In this circumstance, stimulation of the ulnar nerve just above and below the elbow evokes no hypothenar or FDI CMAP while stimulation at the wrist does. The same changes could, of course, be produced by a physiologic block of the ulnar nerve in the forearm. That this change is due to the median-to-ulnar nerve communications is further demonstrated by stimulation of the median nerve. The hypothenar and FDI CMAP is evoked on elbow stimulation while wrist stimulation evokes little or no response (Figure 7). This clearly documents that the hypothenar and FDI CMAPs are derived from stimulated axons lying in the ulnar nerve at the wrist and median nerve at the elbow. A correct interpretation of these findings would avoid the incorrect conclusion that some ulnar nerve axons going through the lesion at the elbow are still anatomically intact.

In a case reported by Miller, the anomaly complicated the evaluation of an ulnar neuropathy localized to the cubital tunnel. This resulted in the ulnar conduction block appearing more severe by 2 mV (stimulating the median nerve at the elbow resulted in a 2 mV CMAP from the ADM). Nonetheless, even after subtracting this 2-mV contribution, the amplitude reduction in the ulnar nerve stimulating above the cubital tunnel was still approximately 50%.

This anomaly may complicate the clinical examination when an ulnar neuropathy with severe or complete axonal loss occurs. Because of median-to-ulnar communicating axons innervating hypothenar, FDI, and adductor pollicis muscles, the degree of clinical weakness may be considerably less than anticipated. When the median-to-ulnar nerve communications in the forearm are especially generous, there may be only mild weakness of intrinsic muscles of the hand despite complete degeneration of

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**Figure 5.** Thenar compound muscle action potential (CMAP) evoked by stimulating the median nerve at the wrist from 2 cases, (A) and (B). Both had symptoms of carpal tunnel syndrome but normal distal motor and palmar sensory latencies. The arrow denotes initial positive deflection seen on elbow stimulation only. The larger amplitude of the thenar CMAP on stimulation at the elbow compared with wrist is also a reflection of the median-to-ulnar communications.
the ulnar nerve at the elbow. It is under this circumstance that the “all median hand” occurs as described by Marinacci; however, the “all median hand” occurs only in the presence of an ulnar neuropathy in this author’s experience. At best, one can only see a “too much median hand” in the presence with median-to-ulnar nerve communications in persons having no nerve lesions.

ULNAR-TO-MEDIAN NERVE COMMUNICATIONS

In contrast to the common occurrence of median-to-ulnar nerve communications, that of ulnar-to-median nerve communications is rare. They have been reported on three occasions involving motor axons and once involving only sensory axons. The initial case of Marinacci involved a patient with median nerve trauma at the forearm. Denervation occurred in the forearm flexor muscles, usually supplied by the median nerve, but hand muscles remained unaffected. Motor axons supplying the thenar muscles could be stimulated in the median nerve at the wrist or ulnar nerve at the elbow. A case described by Komar and colleagues had only partial loss of thenar function and hand sensation after a complete median nerve CMAP was evoked on ulnar stimulation at the elbow but not the wrist. Streib described a woman with fatigue and generalized weakness showing the thenar CMAP 4.9 mV larger stimulating the median nerve at the wrist than elbow and 6.6 mV larger stimulating ulnar nerve at the elbow than the wrist. No evidence of a sensory axon abnormality was noted. The findings in these cases clearly document axons to thenar muscles in the ulnar nerve at the elbow that crossover to the median nerve in the forearm and the median nerve at the wrist.

Hopf noted ulnar-to-median nerve communication limited to sensory axons in 1 of 30 normal subjects in a study of ulnar sensory distribution. The sensory nerve action potentials evoked by stimulating the digital nerves of the ulnar side of the middle finger and radial side of the ring finger were recorded from the median nerve at the wrist and ulnar nerve at the elbow. Motor conduction studies showed no similar communication.

Although uncommon, an awareness of this anomaly is useful. It can affect the clinical and electrophysiological presentation of ulnar and median neuropathies.

VARIATIONS IN THE INNERVATIONS OF INTRINSIC MUSCLES OF THE HAND

All the intrinsic muscles of the hand are innervated by the ulnar and median nerves but may have variations. This is in part due to the median-to-ulnar nerve communications discussed in the previous section. These variations of muscle innervation, however, may
occur without actual anomalous communications between these two nerves in the forearm or the hand.

Anomalous communications in the hand were described by Riche and Cannieu and are sometimes referred to as the Riche-Cannieu anastomosis. They are not as well defined as the median-to-ulnar nerve communications in the forearm. They are thought to involve communications between the motor branch of the median nerve and the deep branch of the ulnar nerve in the radial part of the hand. The anatomic and clinical importance of these Riche-Cannieu anastomoses has not been clarified. It is not clear whether these communications are sensory, motor, or mixed, and if they pass from ulnar to median nerve or in the opposite direction. Mannerfelt noted these communications in three of nine hand dissections. Harness and Sekeles noted anastomoses between the deep branch of the ulnar nerve and various branches of the median nerve deep to the thenar muscles in 27 of 35 (77%) hand dissections.

Most commonly, the FDI, adductor pollicis, and ADM are innervated by the ulnar nerve and the APB and opponens pollicis by the median nerve. The FPB may have a dual nerve supply with the median nerve innervating the superficial head and the ulnar nerve of the deep head.

Rowntree’s study (also reviewed by Seddon) documented many variations in the pattern of innervation in 226 cases with ulnar and median nerve lesions: 33% of patients showed all thenar muscles (FPB, APB, and opponens pollicis) to be entirely innervated by the median nerve; 32% showed median nerve innervation of the APB and opponens pollicis and the entire FPB by the ulnar nerve; 15% showed innervation of the APB and opponens pollicis by the median nerve and dual innervation of the FPB; 2% of the cases were interpreted as having all intrinsic hand muscles supplied by the ulnar nerve; and in 1% of the cases the entire thenar musculature and adductor pollicis were interpreted as being exclusively supplied by the median nerve while the FDI and ADM were supplied by the ulnar nerve. In this author’s laboratory, however, examples of all intrinsic hand muscles innervated entirely by the ulnar nerve or the FDI and adductor pollicis innervated entirely by the median nerve have not been seen. Rowntree apparently recognized that the hypothenar muscles may at times have innervation from the median nerve but did not comment on this.

Kimura and Ayyar investigated the median-to-ulnar nerve communications in the hand stimulating the median and ulnar nerves at the wrist recording from FDI and APB muscles using surface electrodes. Ulnar nerve innervation to thenar muscles was 83%. Median nerve innervation to FDI was 43% and ADM 16%. The changes were more common in caucasians and hispanics than blacks. Although the numbers of communication may be inflated by virtue of the volume conduction effect, the findings again indicate the common occurrence of these communications.

Two publications highlight the clinical importance of these variations. Dumitru and colleagues documented a patients with an ulnar neuropathy at the elbow in whom denervation seen in the APB was due to an anastomosis from the deep ulnar nerve in the hand to partially innervate the APB. Seradge and Seradge documented two patients with CTS showing partial and complete innervation, respectively, of the ADM by a nerve branch leaving the median nerve at the midcarpal tunnel area. It then crossed the tunnel superficial to the flexor tendons and penetrated the transverse carpal ligament to innervate the muscle.

An awareness of these variations is essential for the correct interpretation of the findings on clinical examination and needle EMG. Especially important is that signs of denervation may occur in FBP with ulnar nerve lesions because of its frequent ulnar innervation. Further, one may see denervation in FDI with median lesions and APB with ulnar nerve lesions on rare occasions due to anomalies cited above.

**ACCESSORY DEEP PERONEAL NERVE**

The clinical importance of the EDB is that it extends the second to fifth toes and is commonly used to record CMAPs when carrying out peroneal nerve conduction studies. The EDB is usually innervated exclusively by the deep peroneal nerve, a major branch of the common peroneal nerve; however, in as many as 28% of patients, one or both of the EDB muscles are partially innervated by the accessory deep peroneal nerve, a branch of the superficial peroneal nerve. Anatomic studies by Bryce, Winckler, and Crutchfield have demonstrated that the accessory deep peroneal nerve may innervate the lateral portion of the EDB. The accessory deep peroneal nerve arises from the superficial peroneal nerve midway on the lateral aspect of the leg. It passes deep and posterior to the peroneus brevis tendon, behind the lateral malleolus, and subsequently enters the lateral portions of the EDB (Figure 8). Electrophysiologic studies have demonstrated this anomaly, involving one or both EDB muscles in 21-28% of individuals. This anomaly appears to be inherited in a dominant fashion. Crutchfield and Gutmann showed that 78% of relatives of 5 subjects also had the anomaly, compared with 22% of unrelated individuals.
When carrying out peroneal nerve conduction studies, the amplitude of the EDB CMAP, stimulating the deep peroneal nerve at the ankle, should be 90-120% of the amplitude compared with stimulating the common peroneal nerve at the knee. In the presence of an accessory deep peroneal nerve innervating the EDB, the EDB CMAP is considerably smaller when stimulating the deep peroneal nerve at the ankle because some of the axons of the common peroneal nerve to the EDB are in the accessory deep peroneal nerve. This can be proven by stimulating the accessory deep peroneal nerve behind the lateral malleolus, evoking an additional CMAP from the EDB (Figure 9). Although this anomaly does not mimic any pathologic lesion, the smaller CMAP on supramaximal stimulation of the deep peroneal nerve at the ankle may be difficult for the electrodiagnostic physician to explain unless he is aware of the anomaly.

This anomaly may cause minor confusion in the evaluation of lesions of the common peroneal nerve. In a partial lesion involving a physiologic block at the knee, the expected smaller CMAP, stimulating above the head of the fibula (compared with at the ankle), may be lost due to the concomitant smaller CMAP on ankle stimulation of the deep peroneal nerve resulting from the anomaly. However, in this circumstance one would expect a larger EDB CMAP on common peroneal nerve stimulation below the head of the fibula than that seen at the other two stimulation sites.

The accessory deep peroneal nerve may cause considerable difficulty in both the clinical and electrophysiologic evaluation of deep peroneal neuropathies. These occur much less commonly than neuropathies of the common peroneal nerve. In a neuropathy of the deep peroneal nerve, as due to a nerve compression by osteochondroma or ganglion cyst, one would expect weakness of the anterior tibialis and EDB muscles with complete sparing of muscles innervated by the superficial peroneal nerve (i.e., peroneus longus and brevis). In the presence of an accessory deep peroneal nerve, there would be unexpectedly good function of the EDB (especially in its lateral portion innervating the fourth and fifth digits). A complete or almost complete neuropathy of the deep peroneal nerve showing complete or almost complete loss of function in the anterior tibialis muscle and good function in the EDB might lead to confusing and erroneous conclusions.

Similarly, stimulation of the deep peroneal nerve at the ankle, in this circumstance, would evoke a very small or no EDB CMAP, while stimulation of the common peroneal nerve at the knee would evoke a much larger (and possibly normal) CMAP. The situation would, of course, be clarified once it was

**FIGURE 8.** Diagrammatic representation of (A) common peroneal nerve, (B) accessory deep peroneal nerve (branch of superficial peroneal nerve), and (C) deep peroneal nerve.

**FIGURE 9.** Compound muscle action potentials evoked from extensor digitorum brevis in the presence of an accessory deep peroneal nerve.
discovered that this same CMAP could be evoked on stimulation behind the lateral malleolus, i.e., stimulating the accessory deep peroneal nerve (Figure 10). From a clinical point of view, residual function in the face of an otherwise complete deep peroneal palsy, should suggest this anatomic variation.

**TIBIAL-TO-PERONEAL NERVE COMMUNICATION**

Phillips and Morgan\(^25\) described the findings in a case of tibial-to-peroneal nerve communication on the basis of intraoperative nerve conduction studies. Stimulation of the tibial nerve produced a contraction from the peroneus longus muscle and a nerve action potential in the distal peroneal nerve. This crossover resulted in confusing clinical and electrophysiologic findings including the presence of a few motor unit action potentials in the peroneus longus muscle with otherwise complete peroneal denervation. A prior tibial-to-peroneal communication was reported but no details were given.\(^35\)

**REFERENCES**


**SUGGESTED READING**

CME QUESTIONS
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Ludwig Gutmann, MD

FOR EACH OF THE FOLLOWING MULTIPLE-CHOICE QUESTIONS, SELECT THE ONE BEST ANSWER.

1. When stimulating the median nerve and recording from thenar muscle, the evoked muscle action potential is twice as large when stimulating the median nerve at the wrist as compared to the elbow. Which of the following is LEAST likely to explain this finding?
   A. Median-to-ulnar nerve communication in the forearm.
   B. Faulty stimulating techniques.
   C. A generalized peripheral neuropathy.
   D. A physiologic block in the median nerve in the forearm.
   E. Ulnar-to-median nerve communication in the forearm.

2. When stimulating the ulnar nerve and recording from hypothenar muscle, the muscle action potential evoked wrist stimulation of this nerve is twice as large as that when stimulating the nerve just above the elbow. Which of the following is LEAST likely to explain this finding?
   A. Median-to-ulnar nerve communication in the forearm.
   B. A generalized peripheral neuropathy.
   C. An ulnar neuropathy with a physiologic block at the level of the elbow.
   D. Ulnar-to-median nerve communication.
   E. Faulty stimulating techniques.

3. Which of the following is most likely to be partially innervated by ulnar nerve?
   A. Abductor pollicis brevis.
   B. Opponens pollicis.
   C. Flexor pollicis brevis.
   D. Flexor carpi radialis.

4. In the presence of clinical and electrophysiologic data implicating the presence of a carpal tunnel syndrome, the presence of a thenar muscle action potential preceded by an initial positive deflection, when stimulating the median nerve at the elbow but not when stimulating at the wrist, suggests the presence of:
   A. A superimposed ulnar neuropathy.
   B. Median- or ulnar nerve communications in the forearm.
   C. A generalized peripheral neuropathy.
   D. A superimposed radial neuropathy.
   E. Opponens pollicis.

5. Which of the following muscles is most likely to be innervated by axons coursing through the median nerve at the elbow in the presence of median-to-ulnar communications?
   A. First dorsal interosseus.
   B. Flexor pollicis brevis.
   C. Abductor digiti minimi.
   D. Abductor pollicis brevis.
   E. Opponens pollicis.

6. Stimulation of the deep peroneal nerve at the ankle evokes a muscle action potential from the extensor digitorum brevis muscle that is twice as large as that evoked on stimulating the common peroneal nerve at the knee. Which of the following is most likely?
   A. The presence of an accessory deep peroneal nerve.
   B. A neuropathy of the deep peroneal nerve.
   C. A neuropathy of the superficial peroneal nerve.
   D. A neuropathy of the accessory deep peroneal nerve.
   E. A neuropathy of the common peroneal nerve.

7. The accessory deep peroneal nerve is a branch of:
   A. The common peroneal nerve.
   B. The deep peroneal nerve.
   C. The superficial peroneal nerve.
   D. The tibial nerve.

8. The accessory deep peroneal nerve innervates which of the following?
   A. Extensor digitorum brevis.
   B. Posterior tibial muscle.
   C. Anterior tibial muscle.
   D. Extensor hallucis.
   E. Flexor hallucis.

9. Which of the following anomalous innervations is LEAST common?
   A. Median-to-ulnar communications.
   B. Ulnar-to-median communications.
   C. Accessory deep peroneal nerve.

10. An evoked muscle action potential would NOT be expected from the first dorsal interosseus in the presence of median-to-ulnar communication stimulating which of the following?
    A. Ulnar nerve at the elbow.
    B. Ulnar nerve at the wrist.
    C. Median nerve at the elbow.
    D. Median nerve at the wrist.
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Revised November 2006

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B. Sign the study certification below.

C. Print or type your name and address.

D. Tear out this entire page and mail to:
   Faxes or photocopies will not be accepted.
   AANEM
   2621 Superior Dr NW
   Rochester, MN 55901

E. Enclose a stamped, self-addressed envelope.

CERTIFICATION:

I claim ________ AMA PRA Category 1 credit(s)™ for participating as a learner in this activity (1 credit for each hour of participation, not to exceed 2 credits).

Signature ___________________________________________ Date __________________

Name ____________________________________________________________________________
Address __________________________________________________________________________

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Receipt of CME credit earned is contingent upon completion of both the certification statement above and the evaluation form on the reverse side. Correct answers will be mailed with the certificate. Monographs published by the AANEM are reviewed every 3 years by the AANEM Education Committee for their scientific relevance. CME credit is granted for 3 years from the date of publish, review, or revision date. Individuals requesting credit for monographs that have been discontinued will be notified that CME credit is no longer available.
1. Did this CME activity meet the educational objectives outlined on page ii?
   A. Yes.
   B. No. (Please explain below.)

2. Overall, did this CME activity meet your expectations?
   A. Yes, very well.
   B. Yes, at least on a par with other activities of its type.
   C. It generally did not meet my expectations. (Please explain below)
   D. My expectations were completely unmet. (Please explain below)

3. Did you have unanswered questions remaining after completion of the activity that should have been covered in the study material?
   A. No.
   B. Yes. (Please explain below)

4. Will completion of this study material enhance your professional effectiveness?
   A. Yes, substantially.
   B. Yes, somewhat.
   C. Not sure.
   D. Probably not. (Please explain below)
   E. The subject matter was not applicable to my patients.

5. Would you recommend this material to others?
   A. Yes.
   B. Under some circumstances. (Please explain below)
   C. No. (Please explain below)

6. Should this type of CME activity be continued?
   A. Yes.
   B. No. (Please explain below)

Explanation for above questions (please refer comments back to original question number) and/or recommendations for changes to this study material when it is next revised.

What changes, if any, should be made to the format or guidelines for this CME activity?

Please list any recommendations for topics to be considered for future monographs: