EMG Rotation Objectives (Answers)

I. Basic Concepts of Electricity and Electronics in Clinical EMG

1. **What is charge?** A property of certain subatomic particles (especially electrons and protons) that gives rise to and interacts with the electromagnetic force. Symbol is Q. Unit is coulomb (C) which is approximately equal to 6.24 x 10^18 electrons.

2. **What is voltage?** The electromotive force required to make electricity flow through a conductor. Symbol is E. Measured in volts (V).

3. **What is current?** The flow of electrically charged particles, usually electrons. Symbol is I. Unit is ampere (A), which is 1 coulomb passing a point in a conductor in 1 second.

4. **What is impedance?** The total opposition to current flow in an AC circuit, including resistance, capacitive reactance, and inductive reactance. Symbol is Z. Measured in Ohms (Ω).

5. **What are filters?** Electronic circuits that perform the function of processing a signal (i.e. remove unwanted electrical noise). Electrodiagnostic studies use low-frequency (high-pass) and high-frequency (low-pass) filters to exclude high- and low-frequency electrical noise to reproduce the signal of interest.

6. **What are amplifiers?** Devices that increases the amplitude (i.e. voltage or current) of a signal.

II. Nerve Conduction Studies

1. **What is the difference between the anode and the cathode?** An anode is the terminal on the stimulator where current flows in. The cathode is the terminal on the stimulator where current flows out. Depolarization of a nerve occurs under the cathode, and the depolarization proceeds both orthodromically and antidromically from the cathode. The cathode should be placed closer to the active recording electrode than the anode because the anode has the potential to hyperpolarize the nerve and block the depolarization caused by the cathode; this could cause the recorded potential to be reduced or absent.

2. **What are the filter settings and gain settings for sensory and motor NCS?** For a sensory NCS, the low-frequency filter is set at 2 Hz with the high-frequency filter set at 10 kHz. For a motor NCS, the low-frequency filter is set at 20 Hz with the high-frequency filter set at 2 or 3 kHz. For a motor NCS, the low-frequency filter is set at 2 Hz with the high-frequency filter set at 10 kHz. See EMG machine settings for other settings.

3. **Where should G1 and G2 be placed?** For a sensory NCS, the active recording electrode (G1) is placed over the nerve to be tested with the reference electrode (G2) placed over the nerve 3 – 4 cm distal to the G1 electrode. For a motor NCS, G1 is placed on the center of the muscle belly with G2 placed distally over the tendon of the muscle.

4. **What is the signal-to-noise-ratio and when is this important? What can be done to improve the response?** Signal-to-noise ratio is the ratio of the desired signal power to the background noise signal power. The most common background noise is 60-Hz noise from electrical devices in the surrounding environment. This is most important outside of the EMG lab such as in a ICU setting. Since the signals recorded during NCS and EMG are based on the differences between the active and reference electrodes, making sure that the two electrodes have the same impedance will decrease the background noise. This can be done by making sure the electrodes are the same type, have intact wires and good connections, the underlying skin is clean and intact, a conducting jelly is used between the skin and electrodes, the electrodes are secured to the skin with tape, a ground is in place between the stimulator and recording electrodes, and coaxial cables are used.

5. **Motor Amplitude**
   a. **What is the physiologic basis of the motor amplitude?** CMAP amplitude represents the number of muscle fibers that depolarize.
   b. **What are the units used to measure it?** Millivolts (mV).
   c. **Why do we record over the muscle motor point?** This is the location where muscle depolarization first occurs. If the recording electrode is not placed here, nerve conduction studies can be artificially abnormal because (a) the initial positive deflection makes the onset latency difficult to accurately measure, and (b) the CMAP amplitude may appear artificially reduced.
   d. **What types of disorders cause a reduction of the CMAP amplitude and how can these be distinguished electrodiagnostically?** Axonal loss (i.e. axonal neuropathy), conduction block from demyelination between the stimulation site and recording, some neuromuscular junction disorders, and some myopathies.

6. **Sensory amplitude**
   a. **What is the physiologic basis of the sensory amplitude?** It represents the summation of all the individual sensory fibers action potentials.
   b. **What are the units used to measure it?** Microvolts (µV).
   c. **What is the localization value of a normal versus abnormal sensory amplitude in the setting of both normal and abnormal motor amplitudes in the corresponding motor nerve?**

7. **Motor latency**
   a. **What is the significance of the motor latency?** It represents 3 things: (1) The conduction time from the stimulation site to the neuromuscular junctions, (2) the time delay across the neuromuscular junction, and (3) the depolarization time across the muscle.
   b. **Do we look at the onset or peak?** Onset peak, which represents the fastest fibers of the nerve.
   c. **What are the units?** Milliseconds (ms).

8. **Sensory latency**
   a. **What is the significance of the sensory latency?** The onset latency represents the time it takes for conduction through the largest (i.e. fastest) cutaneous sensory fibers. The peak latency represents a mix of large and small fibers.
   b. **Do we look at the onset or peak?** The onset latency represents the time it takes for conduction through the largest (i.e. fastest) cutaneous sensory fibers. The peak latency represents a mix of large and small fibers.
   c. **What are the units?** Milliseconds (ms).

9. **Conduction velocity**
a. What is the physiologic basis of a slow conduction velocity? Conduction velocity slows due to either loss of the fastest fibers, such as in an axonal neuropathy, or by loss of saltatory conduction, as with a demyelinating neuropathy.

b. How do you calculate conduction velocity in a motor nerve? To do this, you first need to stimulate at two different sites along the motor nerve. Then, measure the distance between the two stimulation sites. Now, divide the distance by the difference of the proximal latency from the onset latency between the two responses. More simply put, motor conduction velocity = distance / (proximal latency - distal latency). (Example: If a median nerve, measured at the abductor pollicis brevis, has an latency of 3.5 ms when stimulating at the wrist and a latency of 7.5 ms when stimulating at the elbow and the distance between the two stimulating sites is 200 mm, then the calculation would be 200 mm / (7.5 - 3.5) ms = 200 mm / 4 ms = 50 mm/ms = 50 m/s.)

c. What are the units? Meters per second (m/s).

d. Why do you need to stimulate at two different sites along the nerve for a motor conduction velocity but not for a sensory conduction velocity? Since a motor nerve study includes the time across the neuromuscular junction and the time for the muscle to depolarize, the conduction velocity from a single motor nerve study will not represent the true conduction velocity of the nerve alone. When two sites are used, the distance between the two sites divided by the difference in latency represents the conduction velocity of only that portion of the nerve. Since that portion of the nerve does not include the neuromuscular junction or the muscle, the calculation will represent the true conduction velocity of the nerve.

e. What is the difference between an orthodromic and an antidromic study? An orthodromic study uses a stimulus directed in the way the nerve normally depolarizes, while an antidromic study uses a stimulus directed in the opposite direction. For example, stimulating a sensory nerve away from the sensory receptor is an antidromic study, but stimulating towards a sensory receptor is an orthodromic study. Motor nerve studies are orthodromic since the stimulus is always directed towards the neuromuscular junction. Most sensory studies are performed antidromically because this results in a higher SNAP amplitude than orthodromic studies.

11. Pitfalls

a. How can you tell if you're not over the motor point of the muscle? (A) There will be an initial positive deflection of the CMAP, which makes the onset latency difficult to determine. (B) The CMAP amplitude may be artificially reduced.

b. What will happen to the NCS responses if the patient's skin is too cold (<32°C)? Lower temperatures causes sodium channels to depolarize more slowly. This causes a delay in polarization leading to a prolonged latency and consequently a slower conduction velocity. The slowed depolarization and thus longer time to inactivation allows for a greater influx of sodium which leads to an artificially increased amplitude.

c. What is the significance of supramaximal stimulation? It ensures that all intact nerve fibers are depolarized. If this is not achieved, amplitudes can be artificially low and latency prolonged.

d. What does 60 Hz interference look like and what can be done to eliminate it? 60 Hz interference appears as a regular sine wave tracing. Interference can be decreased by ensuring that the recording and reference electrodes appear electrically identical. This is done by cleaning the skin, applying conductive jelly to the electrodes, and making sure the electrodes are securely affixed to the skin using tape or pressure if necessary.

e. What disease states correlate with a prolonged F-response?

f. Do F-responses correlate with a prolonged F-response?

g. Do you apply a supramaximal or submaximal stimulus in the F-response?

e. What disease states correlate with a prolonged F-response?

13. H-Reflex

a. Are the afferent and efferent arms of the H-reflex sensory or motor?

b. Is there a synapse in the H-reflex?

c. What is the best nerve to study the H-reflex?

d. Do you apply a supramaximal or submaximal stimulus in the H-reflex?

e. What disease states correlate with a prolonged H-reflex?

f. Do H-reflexes detect radiculopathies not found on needle exam?

14. Physical Skills. How do you set up each of these nerve conduction studies?

a. Sensory nerves

i. Median at the wrist

ii. Ulnar at the wrist

iii. Median midpalmar

iv. Ulnar midpalmar

v. Radial at the forearm

vi. Sural at the calf

vii. Medial and lateral antebrachial cutaneous

viii. Lateral femoral cutaneous

ix. Superficial peroneal

x. Medial plantar

xi. Lateral plantar

b. Motor nerves

i. Ulnar

ii. Median

iii. Peroneal

iv. Tibial

v. Musculocutaneous

vi. Radial

vii. Facial

viii. Spinal accessory

ix. Femoral
III. Normal Values

1. Sensory nerves. What are the normal amplitudes and latencies for each of these nerves?
   a. Median
   b. Ulnar
   c. Median midpalmar
   d. Ulnar midpalmar
   e. Radial
   f. Sural

2. Motor nerves. What are the normal amplitudes and latencies for each of these nerves?
   a. Ulnar
   b. Median
   c. Peroneal
   d. Tibial
   e. Facial

3. What is a normal upper extremity motor conduction velocity?
4. What is a normal lower extremity conduction velocity?
5. F-responses
   a. What is a normal median/ulnar F-wave value?
   b. What is a normal tibial/peroneal F-wave value?
6. What is a normal tibial/soleus H-reflex value?
7. How are normal values affected by
   a. Height
   b. Age
   c. Lower extremities vs. upper extremities
   d. Proximal vs. distal segments of the same nerve

IV. Repetitive Stimulation

1. Which nerves are typically studied with repetitive simulation?
2. What is the rate of stimulation that is given?
3. Regarding the physiology of the neuromuscular junction how is Ach synthesized?
   a. What are quanta?
   b. What is a mini-endplate potential (MEPP)
   c. What is an end-plate potential (EPP)
   d. What is a muscle fiber action potential (MFAP)
4. Explain the significance of primary, secondary, and tertiary stores of acetylcholine in neuromuscular transmission, and the ramifications of these various stores on repetitive stimulation testing.
5. Describe the exercise protocol with repetitive stimulation
6. What is post-exercise facilitation and in which disorders is this a prominent finding?
7. What are the expected findings with repetitive stimulation in each of the following disorders:
   a. Myasthenia gravis
   b. Lambert Eaton Myasthenic Syndrome
   c. Botulinum intoxication

V. Needle EMG - Resting Muscle

1. How many locations should be evaluated in each muscle?
2. What sweep speed and sensitivity are best for observing spontaneous activity?
3. What sweep speed and sensitivity are best for observing voluntary motor units?
4. Insertional activity
   a. What causes increased insertional activity?
   b. What causes decreased insertional activity?
5. Endplate noise
   a. What is endplate noise?
   b. Are endplate spikes regular or irregular?
   c. Is the initial deflection positive or negative?
6. Fibrillation potentials and positive sharp waves
   a. What is the physiologic basis of fibrillations and positive sharp waves?
   b. How fast do fibrillations fire? How does this compare to normal motor units?
   c. Are fibrillations generally regular or irregular?
   d. Is the initial deflection positive or negative?
   e. What is the significance of tiny fibrillation potentials?
   f. How long after nerve injury do positive waves and fibrillations appear?
   g. What disease states other than nerve injury can produce positive waves and fibrillations?
   h. Fibrillations are graded on a scale of 0-4+. What is the difference between 1+, 2+, 3+, and 4+ fibrillations?
   i. Which myopathies cause fibrillations?
   j. Which myopathies generally do not cause fibrillations?
   k. After denervation, please note that fibrillations may continue until complete reinnervation has occurred.
7. Complex repetitive discharges
   a. What causes complex repetitive discharges?
   b. What is the appearance of a complex repetitive discharge on needle EMG?
8. Myotonia
   a. What is myotonia?
   b. What are the features of a myotonic discharge on EMG?
   c. What disease states are associated with myotonic discharges?
   d. How can complex repetitive discharges be distinguished from myotonic discharges?
9. Fasciculations
9. a. What is the physiologic difference between a fasciculation and a fibrillation?
b. Are fasciculations regular or irregular?
c. At what rate do fasciculations fire?
d. What conditions or disease states are associated with fasciculations?

10. Myokymia
   a. How can myokymia be distinguished from complex repetitive discharges?
   b. How can myokymia be distinguished from tremor?
   c. What disease states cause myokymia?

11. What is the difference between cramp and neuromyotonia?

VI. Needle EMG – Motor Unit Activity

1. How do you know you are recording directly over a motor unit action potential?
2. Duration
   a. What does the duration of a motor unit action potential (MUAP) reflect?
   b. What is the physiologic basis for prolonged duration in neurogenic disease?
   c. What does a long duration MUAP sound like?
   d. What is the importance of MUAP rise time?
3. Polyphasia
   a. How do you calculate the number of phases in a MUAP?
   b. In normals, what percent of MUAPs have 3 or 4 phases?
   c. What is the morphological difference between phases and serrations?
   d. What is the physiologic basis for increased polyphasia in neurogenic disease?
   e. What is the physiologic basis for increased polyphasia in myopathic disease?

4. Satellite potential
   a. What does a satellite potential look like?
   b. What is the physiologic basis for a satellite potential?

5. Amplitude
   a. What is the upper limit of normal for the amplitude of a MUAP?
   b. What does the amplitude of a MUAP reflect?
   c. What is the physiologic basis for high amplitude MUAPs in neurogenic disease?
   d. What is the physiologic basis for low amplitude MUAPs in myopathic disorders?
   e. What does a high-amplitude MUAP sound like?

6. Recruitment
   a. What is the lowest frequency at which a voluntary motor unit can fire?
   b. In a normal recruitment pattern, what is the ratio of firing frequency (in Hertz) of the fastest motor unit to number of units firing?
   c. What is the difference between reduced recruitment and reduced activation?

VII. Electrodiagnostic Findings in Common Clinical Scenarios

1. In each of the following conditions, describe what would be expected on nerve conduction studies (sensory, motor and F-responses) and needle EMG (including the pattern of abnormal spontaneous activity, MUAP duration, amplitude, polyphasia, and recruitment): (see [http://rehabmanual.com/book/print/443](http://rehabmanual.com/book/print/443))
   a. Axonal loss
      i. Hyperacute axonal loss (<3d)
      ii. Acute axonal loss (>1 week, <3-6 weeks)
      iii. Subacute axonal loss (>3-6 weeks, <2-3 months)
      iv. Subacute to chronic axonal loss (>2-3 months, >many months/years)
      v. Chronic axonal loss (>many months/years)
   b. Demyelinating disease
      i. Acquired demyelinating polyneuropathy
      ii. Hereditary demyelinating polyneuropathy
      iii. Focal demyelination (a single distal lesion)
   c. Myopathy producing muscle fiber necrosis
   d. Myopathy not producing muscle fiber necrosis (metabolic myopathy)
   e. Neuromuscular Junction Lesions (excluding abnormalities seen on repetitive stimulation and single fiber EMG)
   f. Central nervous system disease
2. What is the electrodiagnostic approach to the diagnosis of
   a. Painless weakness
   b. Unsteady gait
   c. Rhabdomyolysis
   d. Elevated serum CK levels
   e. Muscle cramps
   f. Foot drop
   g. Wrist drop/hand weakness

VIII. Pathophysiology of Peripheral Neuropathies

1. What is neurapraxia?
2. What is neurotmesis?
3. What is axonotmesis?
4. What is Wallerian degeneration and how does it occur?
5. Describe the histology of normal nerves and connective tissue
6. What are the effects on conduction of an axonopathy?

IX. Normal Anatomy

1. What are the nerve, nerve root, and trunk innervations of the following upper extremity muscles? (See the [Muscle and Nerve Cheat Sheet](#))
   a. Rhomboids
   b. Supraspinatus
   c. Infraspinatus
d. Deltoid
e. Biceps Brachii
f. Serratus Anterior
g. Pectoralis Major – clavicular
h. Pectoralis Major – sternocostal
i. Brachioradialis
j. Ext carpi radialis longus
k. Triceps
l. Extensor carpi Ulnaris
m. Extensor digitorum communis
n. Extensor indicis
o. Extensor pollicis longus
p. Supinator
q. Pronator teres
r. Flexor carpi radialis
s. Flexor pollicis longus
t. Flexor digitorum profundus 1&2
u. Flexor digitorum superficialis
v. Abductor pollicis brevis
w. Opponens pollicis
x. Flexor carpi ulnaris
y. Flexor digitorum profundus 4&5
z. First dorsal interosseus of the hand
aa. Abductor digiti quinti of the hand

2. What are the nerve and nerve root innervations of the following lower extremity muscles?
   a. Iliopsoas
   b. Sartorius
   c. Rectus femoris
d. Vastus lateralis
e. Vastus medialis
f. Adductor longus
g. Gluteus medius
h. Gluteus maximus
i. Quadratus femoris
j. Peroneus longus
k. Peroneus brevis
l. Anterior tibialis
m. Extensor digitorum longus
n. Extensor hallucis
o. Extensor digitorum brevis
p. Internal hamstrings
q. External hamstrings
r. Short head of the biceps femoris
s. Posterior tibialis
t. Medial gastrocnemius
u. Lateral gastrocnemius
v. Abductor hallucis
w. Abductor digitorum (Ped)
x. First dorsal interosseus pedis

X. Compressive Neuropathies 1: Median Nerve

1. What is the difference between compressive neuropathy and entrapment neuropathy?
2. What are the signs and symptoms of carpal tunnel syndrome?
3. What are the risk factors for carpal tunnel syndrome?
4. What are the AANEM electrodiagnostic criteria for carpal tunnel syndrome (sensory, midpalmar sensory, and motor)?
5. What are our criteria for carpal tunnel syndrome? Note that these relative differences are not considered absolute or diagnostic.
6. Proximal Median mononeuropathy## The pronator syndrome### What is it and what are the symptoms?
   a. i. What are the expected nerve conduction and EMG findings?
   b. Compression at the ligament of Struthers### What are the signs and symptoms?
   i. What are the expected nerve conduction study and EMG findings?
7. The anterior interosseus syndrome:
   a. Which muscles are innervated by the anterior interosseus nerve?
   b. What are the signs and symptoms?
   c. What are the expected nerve conduction study and EMG findings?

XI. Compressive Neuropathies 2: Ulnar Nerve

1. What muscles are supplied by the ulnar nerve?
2. Ulnar mononeuropathy at the elbow
   a. What are the clinical features of ulnar mononeuropathy at the elbow?
   b. What are risk factors for ulnar mononeuropathy at the elbow?
   c. What are the expected nerve conduction study and EMG findings in ulnar mononeuropathy at the elbow?
   d. How are the flexor carpi ulnaris and flexor digitorum profundus 4&5 useful localizing an ulnar mononeuropathy?
   e. How can an ulnar mononeuropathy at the elbow be distinguished from ulnar mononeuropathy proximal to the elbow?
3. Ulnar mononeuropathy at the wrist
   a. What are the various branches of the ulnar nerve that can be affected at the wrist?
   b. How do the clinical presentations vary?
   c. What are the nerve conduction study findings associated with these?
XII. Compressive Neuropathies 3: Peroneal Nerve (fibular nerve)
1. What muscle is supplied directly by the common peroneal branch of the sciatic nerve above the fibular head?
2. What muscles are supplied by the deep peroneal nerve?
3. What muscles are supplied by the superficial peroneal nerve?
4. Which branch or branches of the peroneal nerve are usually involved in an entrapment neuropathy at the fibular head?
5. What are the expected nerve conduction studies and EMG findings in peroneal mononeuropathy at the fibular head?

XIII. Compressive Neuropathies 4: Radial Nerve
1. What muscles are supplied by the radial nerve before the spiral groove? Which is the last one?
2. What muscles are supplied by the radial nerve after the spiral groove but before the branch to the posterior interosseous nerve? Which is the first one?
3. What muscles are supplied by the posterior interosseous nerve?
4. What is the first muscle innervated by the posterior interosseous nerve as it emerges from the supinator?
5. What are the expected nerve conduction study and EMG findings in a radial neuropathy at the spiral groove?
6. What are the expected nerve conduction study findings and needle EMG findings in a posterior interosseous syndrome?

XIV. Compressive Neuropathies 5: Uncommon Compressive Neuropathies
1. What is the difference between vascular and neurogenic thoracic outlet syndrome?
2. What are the expected nerve conduction study and EMG findings in neurogenic thoracic outlet syndrome?
3. What are the features of digital nerve entrapment of the hand?
4. Sciatic neuropathy: What muscles are supplied by the sciatic nerve?
   a. What etiologic factors may contribute to sciatic mononeuropathy?
   b. What are the expected nerve conduction studies and EMG findings?
5. Tarsal Tunnel Syndrome: What are the clinical features of tarsal tunnel syndrome?
   a. What etiologic factors may contribute to tarsal tunnel syndrome?
   b. What are the expected nerve conduction study and EMG findings?
6. Femoral Neuropathy: What are the sensory branches of the femoral nerve?
   a. What is the clinical presentation of lateral femoral cutaneous neuropathy?
   b. What muscles are innervated by the femoral nerve?
7. What are the findings in long thoracic neuropathy?
8. What are the findings in dorsal scapular neuropathy?
9. What are the findings in suprascapular neuropathy?
10. What are the findings in musculocutaneous neuropathy?
11. What are the findings in ilioinguinal, genitofemoral, and saphenous neuropathies?
12. What are the findings in obturator neuropathy?

XV. Anomalous Innervation
1. What are the three types of median-ulnar anastomoses?
2. Which is the most common type of median-ulnar anastomosis?
   a. What nerve conduction study finding suggests the presence of this anastomosis?
   b. How would this anomaly be confirmed?
3. What are the findings in a patient with a median-to-ulnar anastomosis and carpal tunnel syndrome?
4. What muscle may be innervated by an accessory peroneal nerve?
5. How is an accessory peroneal nerve identified on nerve conduction studies?

XVI. Axonal vs. Demyelinating Neuropathy
1. What types of nerve fibers are involved in conveying pain?
   a. Temperature
   b. Pressure
   c. Vibration
   d. Two-point discrimination
   e. Proprioception
2. What physical exam findings may be present in patients with peripheral neuropathy?
3. What electrodiagnostic findings differentiate peripheral neuropathy from polyradiculopathy?
4. What electrodiagnostic findings are suggestive of an axonal peripheral neuropathy?
5. Demyelinating peripheral neuropathy
   a. What electrodiagnostic findings are suggestive of an acquired demyelinating peripheral neuropathy?
   b. What conditions cause an acquired demyelinating peripheral neuropathy?
   c. What electrodiagnostic findings are suggestive of a hereditary demyelinating peripheral neuropathy?
   d. What conditions cause a hereditary demyelinating peripheral neuropathy?
6. How can cool skin temperature be misleading in determining whether a peripheral neuropathy is axonal or demyelinating?
7. How do you define peripheral neuropathy in the geriatric population?
8. Hereditary neuropathies: What are the most common hereditary neuropathies and how are they classified?
   a. What genetic tests are available for hereditary neuropathies?
   b. In approximately what fraction of hereditary demyelinating neuropathies is there no known family history?
9. What is multifocal motor neuropathy and what are the findings on nerve conduction studies?

XVII. Radiculopathies and Plexopathies
1. Radiculopathy
   a. What are the expected findings on motor nerve conduction studies in a radiculopathy?
b. What are the expected findings on sensory nerve conduction studies in a radiculopathy?
c. After an acute radiculopathy, which muscles (proximal or distal) will show abnormal spontaneous activity first? Which muscles will show changes of reinnervation first?
d. What are the most common cervical and lumbar radiculopathies?
e. What are the electrodiagnostic findings in lumbar spinal stenosis?
f. What is the minimum number of muscles that should be tested to screen for radiculopathy in an upper extremity?
g. What is the minimum number of muscles that should be tested to screen for radiculopathy in a lower extremity?
h. What abnormalities do you expect on needle exam in each of the following radiculopathies?
   i. L4 radiculopathy
   ii. L5 radiculopathy
   iii. S1 radiculopathy
   iv. C5 radiculopathy
   v. C6 radiculopathy
   vi. C7 radiculopathy
   vii. C8 radiculopathy

2. Plexopathy
   a. How can you differentiate plexopathy from radiculopathy on the basis of nerve conduction studies?
   b. What are the clinical features of an upper trunk brachial plexopathy?
   c. What are the clinical features of a lower trunk brachial plexopathy?
   d. Which nerves are supplied by the posterior cord?
   e. Which nerves are supplied by the lateral cord?
   f. Which nerves are supplied by the medial cord?
   g. Draw the brachial plexus. In an upper trunk plexopathy, which of the following sensory nerve action potentials will be affected?### Median at D1
      i. Median at D3
      ii. Ulnar
      iii. Radial
      iv. Medial antebrachial cutaneous
      v. Lateral antebrachial cutaneous

   h. In a lower trunk plexopathy, which of the following sensory nerve action potentials will be affected?### Median at D1
      i. Median at D3
      ii. Ulnar
      iii. Radial
      iv. Medial antebrachial cutaneous
      v. Lateral antebrachial cutaneous

   i. In a middle trunk plexopathy, which of the following sensory nerve action potentials will be affected?### Median at D1
      i. Median at D3
      ii. Ulnar
      iii. Radial
      iv. Medial antebrachial cutaneous
      v. Lateral antebrachial cutaneous

   j. In a lateral cord plexopathy, which of the following sensory nerve action potentials will be affected?### Median at D1
      i. Median at D3
      ii. Ulnar
      iii. Radial
      iv. Medial antebrachial cutaneous
      v. Lateral antebrachial cutaneous

   k. In a medial cord plexopathy, which of the following sensory nerve action potentials will be affected?### Median at D1
      i. Median at D3
      ii. Ulnar
      iii. Radial
      iv. Medial antebrachial cutaneous
      v. Lateral antebrachial cutaneous

   l. In a posterior cord plexopathy, which of the following sensory nerve action potentials will be affected?### Median at D1
      i. Median at D3
      ii. Ulnar
      iii. Radial
      iv. Medial antebrachial cutaneous
      v. Lateral antebrachial cutaneous

   m. What is diabetic amyotrophy?
   n. What is brachial plexitis (Parsonage-Turner Syndrome?)
   o. q. Are paraspinal muscles affected in plexopathy?

3. Paraspinal Mapping## How much paraspinal denervation can one expect in the low back muscles of asymptomatic persons?
   a. What is the specificity of needle EMG in differentiating clinically and radiographically apparent spinal stenosis from low back pain or no symptoms?
   b. How does one place an EMG needle in the L5-innervated paraspinal muscles?

XVIII. Cranial Nerve Studies

1. For what clinical situations is a facial motor response a useful study?
2. In the blink reflex, what pathway does the R1 represent?
3. In the blink reflex, what pathway does the ipsilateral R2 represent?
4. In the blink reflex, what pathway does the contralateral R2 represent?

XIX. Motor Neuron Disease

1. What are the different motor neuron diseases that affect adults and children and how are they can be distinguished clinically?
2. What is the differential diagnosis for weakness with normal sensation?
3. What are the clinical features of amyotrophic lateral sclerosis?
4. In motor neuron disease:
4. a. What are the expected sensory and motor nerve conductions study findings?
b. What findings may be noted on repetitive stimulation?
c. What findings are expected on needle examination and what is the distribution of involvement?
5. A cervical lesion may cause polyradiculopathy (wasting and weakness of the upper limbs) and myelopathy (spasticity in the lower limbs). How can this be distinguished from ALS?
6. What are the Escorial definitions of definite, probable, and possible motor neuron disease? What are the four body segments that can be tested to meet these criteria?
7. What does muscle biopsy show in motor neuron disease?
8. Why are thoracic paraspinous muscles useful in the EMG evaluation of ALS?

XX. Introduction to Muscle Pathology

1. Be able to identify each of the following in muscle tissue, and describe the clinical significance of the findings that are abnormal:
   a. Normal H&E
   b. Atrophic muscle fibers
   c. Fiber type grouping
   d. Increased glycogen storage
   e. Split fibers
   f. Central nuclei
   g. Vacuoles
   h. Z band, I band, and A band
2. What are the features of Type I muscle fibers?
3. What are the features of Type II muscle fibers?
4. How do the following relate to each other: Muscle fibers, myofibrils, sarcomeres?

XXI. The Muscular Dystrophies

1. What are the clinical features, mode of inheritance, and EMG findings in:
   a. Duchenne muscular dystrophy
   b. Becker muscular dystrophy
   c. Limb Girdle muscular dystrophy
   d. Fascioscapulohumeral muscular dystrophy
   e. Oculopharyngeal muscular dystrophy
   f. Myotonic dystrophy
2. How are muscular dystrophies classified?
3. For which muscular dystrophies is a gene test available?

XXII. Inflammatory/Metabolic Myopathies

1. What are the clinical features and EMG characteristics of:
   a. Dermatomyositis
   b. Polymyositis
   c. Inclusion body myositis
   d. Hypokalemic periodic paralysis
   e. Hyperkalemic periodic paralysis
   f. Statin myopathy
2. What are the limitations of EMG in the diagnosis of steroid myopathy?
3. What are the EMG findings in an incompletely treated inflammatory myopathy?

XXIII. Congenital/Storage Myopathies

1. What are the clinical features and muscle biopsy findings in:
   a. Central core myopathy
   b. Nemaline myopathy
   c. Myotubular (centronuclear) myopathy
   d. Mitochondrial myopathy
2. What are the clinical features and muscle biopsy findings in:
   a. Acid maltase deficiency (Pompe’s disease)
   b. Myophosphorylase deficiency (McArdle’s disease)
   c. Carnitine palmitoyltransferase deficiency

XXIV. Myotonia

1. What are the clinical features of myotonic dystrophy?
2. What are the clinical features of myotonia congenita?
3. What are the clinical features of paramyotonia congenita?
4. What ion channels are involved in each of the myotonic disorders?

XXV. Neuromuscular Complications of Cancer

1. What conditions can cause facial myokymia?
2. Describe the clinical picture in the various paraneoplastic syndromes of the peripheral nervous system?
3. How can radiation plexopathy be differentiated from plexopathy caused by metastatic tumor?

XXVI. An Approach to the Diagnosis of Neuromuscular Disease in the Floppy Infant

1. How do the nerve conduction study findings differ between normal infants and adults?
2. What is the most likely diagnosis in a floppy infant with neuropathic motor unit changes on EMG?
3. What is neonatal myasthenia gravis?

XXVII. SFEMG

1. What is jitter and how is it measured?
2. What is the significance of increased jitter?