EMG in Lumbosacral Radiculopathy

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Needle EMG abnormalities in radiculopathy are the result of an inflamed motor nerve root. Ultimately this inflammation may cause death of axons. However, during the dynamic phase of the radiculopathy the inflamed motor root may result in:

a) Positive waves
b) Fibrillation potentials
c) Increased proportion of polyhasic MUP's
d) Reduced number of MUP's as compared to strength of contradiction
e) If (d) is neurapraxic weakness, motor nerve stimulation will evoke a normal compound muscle action potential.
f) Fasciculation potentials

Sequence of Abnormalities
Days
After Onset

O+ Reduced numbers of MUP's; reduced recruitment interval (increased recruitment frequency); increased proportion of early polyphasic MUP's (see Appendix 1); fasciculation potentials. Prolonged "H" latency in S-1. Reduced number of F-waves in weak muscle.

4+ Evoked compound muscle AP will differentiate clinical weakness (and reduced # MUP's) as either neurapraxia or true weakness (i.e. denervation); CAMP will be small compared to contralateral (in true weakness).

7+ Positive sharp waves in posterior primary ramus distribution.

12+ Positive sharp waves in proximal limb muscles; fibrillation potentials in paraspinals.

15+ Positive sharp waves in distal limb muscles and fibrillationpotentials in proximal limb muscles.

18+ Fibrillation potentials in most affected muscles.

TECHNIQUE OF EXAM

Begin with cogent history and physical examination. Needle exam should begin with weakest muscles. Explore the proximal, middle and distal portions of the muscle.

Step I **Muscle at Rest**

Move needle electrode in at least 10 areas radially around the point of entry. Watch each general area for 1 minute to observe for fasciculation potentials. Note also for fibrillation potentials.

Step II **Insertional Activity**

As the needle is briskly thrust 10-20 times in a circular field around the skin penetration, this burst of insertional activity represents "injury potentials" as the needle electrode disrupts the muscle cell membranes. The first appearing membrane abnormal irritability is a few positive sharp waves just after the provoked injury potentials. Whether a prolongation or slurring of the cessation of the normal injury potentials (insertional activity) is detectable just prior to the few positive sharp waves is a subjective phenomenon apparent to only a few expert and experienced electromyographers.

Step III Minimal Muscular Contraction

Here it is theoretically possible to determine the recruitment interval (Petajan). This is the time in milliseconds between succeeding activations of the first recruited MU at the moment of the second MU is recruited. It would be shortened in a radiculopathy.

Also the increase proportion of polyphasic MUP's would be noted -- generally greater than 1/6.

Step IV Maximal Muscular Contraction

When the message to activate the muscle is partially blocked at the inflamed root, a maximal effort by the patient will result in a reduced number of MUP's firing rapidly. N.B. It is impossible to obtain a maximal effort in a 2 or 3 joint muscle with patient reclined!

Step V Distribution of EMG Abnormalities

Keep and anatomy book nearby.
Sharpen you knowledge of surface anatomy.
Do not stereotype your exam by always exploring the same muscles for each root.

EVOKED COMPOUND MUSCLE ACTION POTENTIAL

In order to identify the proportion of clinical weakness as neurapraxic, the motor nerve should be stimulated with a surface recording electrode over the weak muscle. For example: L-5, ext dig long; S-1, medial gastroc; L-4, vastus medialis. Compare with the compound muscle AP on contralateral (normal) side. This is useful, after 4 days from onset, in managing or advising the referring physician.

Positioning

I recommend the prone position for L-S radiculopathy EMG's (under abdomen). To get relaxation of paraspinal muscles, suggest contraction of antagonist, i.e. abdominal, or better, poke abdominal muscles with end of fingers to get a mild contraction.

Post Laminectomy EMG

Ideally, one should have a preoperative EMG to compare. It is, however, useful to examine the paraspinal muscles in spite of the laminectomy. Explore at least 3 cm. Lateral to the scar and 3-4 cm. deep. If localized EMG abnormalities are present only in the post pr. ramus distribution but correlate with the clinical dx and signs, these abnormalities should be considered in the diagnositic equation.

Remote Radiculopathies

Membrane abnormalities (fibrillation potentials and positive waves) may persist for a year or more. However, the amplitude is smaller in the long duration deneravation. (We have observed fibrillation potentials 5-6 years after laminectomy in patients who were asymptomatic.) Reinnervation (i.e. adopting of denervated muscle fibers by neighboring viable axons) does occur, resulting in polyphasic MUP's. Large amplitude MUP's of normal duration which many electromyographers ascribe to mature reinnervation can be rather the result of altered architecture. Single fiber EMG studies may shed more light on this.

Complex repetitive discharges (iterative) may be seen in severe radiculopathy as a consequence of ephaptic conduction, among several denervated muscle fibers. These are called high frequency if F exceeds 45 Hz.

Technique

Apply recording (active) electrode over the medial aspect of the soleus and reference over Achilles tendon. Needle stimulating electrode inserted over the tibial nerve in the popliteal fossa (junction of middle and lateral 1/3) just lateral to the popliteal artery.

Apply a low intensity stimulus at rate of .5 Hz. Duration should be .5-1 ms. Sweep speed setting should allow for 50 milliseconds across the osciloscope face. Gain should be 1 miliovolt per vertical division (cm.).

"H" wave is recognized as a muscle action potential appearing after a latency from 28-35 ms. As stimulus intensity is increased the "M" wave will appear at 5-8 ms. and the "H" wave will get smaller. At maximal stimulating intensity the "H" wave will disappear and "F" wave (variable and low amplitude) will appear after approximately the same latency. The latency of the H-wave is constant.

Formula for predicting "H" latency in tibial nerve is .46 times length in cm. for medial malleolus to point of stimulation (popliteal area) plus .1 of age in years plus a constant of 9.13 2SD equals 4.75 ms. Difference between involved and contralateral univolved latency is less than 1 ms. (S.D. = .4 ms.).

1. If observed latency is greater than predicted mean, do the study on contralateral limb. Greater than 1.0 ms. difference is diagnostically significant. (3 S.D. = 1.2 ms. side-to-side).

2. If both tibial nerve "H" latencies are greater than the predicted mean latency, determine the tibial nerve conduction velocity. The patient could have a peripheral neuropathy. Sural nerve latency is also a sensitive indicator of certain neuropathies, so should be determined.

3. If the history and physical exam and needle EMG are highly suggestive of radiculopathy, yet the observed latency is less than the predicted latency (mean), determine the contralateral latency. More than 1 ms. difference side-to-side is diagnostically significant.

Value of "H" Latency

1. In the first 7-10 days of a suspected S-1 radiculopathy, the "H" latency may confirm.

2. If needle EMG findings are limited to the paraspinal muscles (posterior primary rami), prolonged or absent "H" latency is diagnostic of S-1 radiculopathy.

3. If minimal or inconclusive needle EMG findings, prolonged or absent "H" latency will support S-1 radiculopathy.

4. If needle EMG findings do not distinguish between L-5 and S-1 radiculopathy, "H" latency determination will be helpful. Its normality suggests L-5, and its absence or prolongation, S-1. This is probable since fewer than 1/20 radiculopathies are L-4.

5. In recurrent pain after previous lumbar laminectomy with questionable needle EMG findings, prolonged or absent "H" (unilateral) suggest S-1 radiculopathy.

EMG IN RADICULOPATHY

Guidelines for Muscle Innervation by Root Levels

I. Only L-4 below knee is anterior tibial muscle.
II. Only L-5 below ankle is Ext. Dig. Br.
III. From medial to lateral in foot more S-1 to more S-2.

N.B. In all muscles as one explores proximally to distally; medially to laterally; anteriorly to posteriorly, one goes cephalad to caudad with roots.

EMG IN L-S RADICULOPATHY

Selected Muscles with Root Levels

Lower Extremity

Gluteus Max	L5,S1,2
Gluteus Med.	L4,5,S1
Tensor Fasc. Lata	L4,5,S-1
Quadriceps	
V. Med.	L2,3,4
V. Lat.	
V. Int.	
Rect. Fem	
Sartorius	L2,3,4
Adductor Longus	L2,3,4
Gracilis	L2,3,4
Biceps Fem.	L5,S1
Semimembran	L5,S1
Semitend	L4,5
Ant Tibial	L4,5
Ext. Dig Long	L5,S1
Ext. Hall Long	L5,S1
Per Long & Brev	L5,S1
Gastrocnemius	L5,S1,2
Medial more S-1	
Lateral more S-2	

Soleus	S1,2
Flex Dig Long	L5,S1
Flex Hall Long L5, S1	
Ext Dig Br	L5,S1
Abd. Hallucis	S1,2
1st Dorsal Int	S1,2
Abd Dig V ped	S1,2

INTERPRETING AND REPORTING THE EMG OF RADICULOPATHY

Have an anatomy book nearby -- reports are replete with anatomic errors.

A few caveats --

1. Fibrillation potentials and positive sharp waves are NOT synonymous with denervation.
2. Most radiculopathies represent a SINGLE root, so explore sufficiently to be sure. Do not report multiple roots, e.g. L4,5,S1, when only one root is involved.
3. Don not stereotype the EMG -- the most common error in the exam. This is exploring the same small list of muscles irrespective of the clinical exam.
4. Use terminology recommended by AAEE, i.e. no disease or pathologic labels for electrical activity. Describe wave forms by parameters, e.g. amplitude, duration, number of phases, rate of firing, etc.
5. Do not over interpret abnormalities.
6. Specific roots are identified by distribution in anterior primary distribution. Posterior primary ramus distribution places the compromise at or proximal to the root. "H" reflex assists in L-S root identification.
7. "Prolonged" insertional activity is NOT an objective EMG abnormality.
8. Root distribution of EMG abnormalities may occur in "vulnerable" nerve syndrome, e.g. diabetic, toxic, idiopathic neuropathies, presumable the result of dural sleeve entrapment of the root.
9. Use the patient with classic specific radiculopathy to confirm the root distribution in the various muscles.
10. Simplify your reporting sheets to give:
    MUSCLE (incl innervation)
    INSERTIONAL ACTIVITY (positive waves)
    SPONTANEOUS ACTIVITY (fasciculation and fibrillation potentials)
    MOTOR UNIT POTENTIALS (describe and note recruitment)

N.B. Never be reluctant to say, 'I don't know." Repeat the EMG: suggest other diagnostic tests.

EMG IN RADICULOPATHY

Historical Perspectives

1929	Adrian & Bronk devised coaxial needle electrode.
1938	Denny-Brown & Penny Backer described differences between fibrillation and fascuculation.
1943	Weddell et al described insertional activity and course of denervation.
1945	Jasper described portable electromyography.
1950	Golseth-EMG in office.
1951	Woods & Shea, Value of EMG. Mack, E., EMG in post disk pts. Bonner, F., EMG in disc dis Shields, C., EMG
1954	AAEE is founded
1955	Marinacci authored first book exclusively devoted to EMG.
1957	Lambert describes origin of positive wave.
1959	Johnson notes 25% of proved radiculopathies (by surgery) demonstrated EMG abnormalities only in posterior primary rami.
1973	"H" reflex is prolonged in S-1 radiculopathy.
1980's	Dermatol stimulation in SEP. F-waves in radiculopathy. Spinal nerve stimulation.

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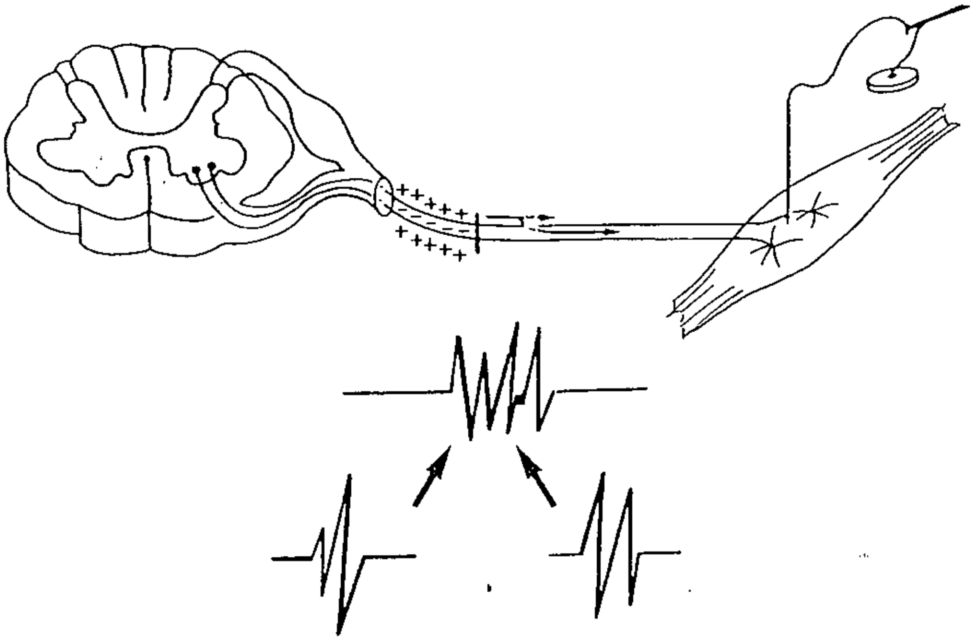
THIS REPRESENTS THE HYPOTHESIS TO EXPLAIN THE POLYPHASIC-"APPEARING" MUP SEEN EARLY IN RADICULOPATHY (W/IN FIRST 2 WEEKS)

Early Polyphasic M.U.P



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Cardiovascular Response of Paraplegics on Arm Cranking Exercise

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Kulsomboon W, Khunphasee A, Teeranet K, Ing-Aram R, Khunadorn F. Carciovascular response of paraplegics on arm cranking exercise. J Thai Rehabil 1993;3(2):12-17

Abstract Paraplegics cannot exercise simple methods such as running, walking or bicycling. Arm cranking on a modified bicycle ergometer was used as a means of exercise stress test and endurance training for paraplegics. The purpose of this study was to assess the cardiovascular response on arm cranking stress test between 10 normal persons and 10 paraplegics and the response after arm cranking training for three weeks in 5 paraplegics. Heart rate (H.R.), systolic blood pressure (S.B.P.), diastolic blood pressure (D.B.P.) and rate pressure product (R.P.P.) were evaluated at rest and at the peak point. Power(W) and rate of perceived exertion (R.P.E.) were evaluated at peak point. Normal persons' mean resting heart rate and mean resting rate pressure product were significantly lower than those of paraplegics, confirmed the hypothesis of deconditioning in paraplegics. After three weeks period of endurance exercise, post-training mean resting diastolic blood pressure was significantly higher than pre-training value and the other parameters were nonsignificant. Post-training mean peak systolic blood pressure was significantly lower than pre-training value which indicated paraplegics' ability to improve the cardiovascular response with arm cranking exercise.

บทคัดย่อ ผู้ป่วยอัมพาตครึ่งท่อนไม่สามารถที่จะออกกำลังโดยใช้วิธีธรรมดา เช่น การวิ่ง, การเดิน หรือการปั่นจักรยานได้ การออกกำลังกายโดยวิธี Arm Cranking โดยดัดแปลงจาก bicycle ergometer จึงเป็นวิธีหนึ่งที่ผู้ป่วยเหล่านี้สามารถออกกำลังกายได้ ในการศึกษานี้ได้เปรียบเทียบการตอบสนองของระบบหลอดเลือดและหัวใจของคนปกติ และของผู้ป่วยอัมพาตครึ่งท่อนล่าง เมื่อทำการทดสอบ stress test โดยใช้วิธี arm cranking และเปรียบเทียบในผู้ป่วยก่อนและหลังการออกกำลังโดยใช้วิธี arm cranking ครบ 3 สัปดาห์ พบว่าชีพจรเฉลี่ยและค่าเฉลี่ยของ rate pressure product ขณะพักในคนปกติมีค่าต่ำกว่าของผู้ป่วยอย่างมีนัยสำคัญ ยืนยันถึงสมมติฐานของ deconditioning ในผู้ป่วยเหล่านี้ และเมื่อผู้ป่วยออกกำลังครบ 3 สัปดาห์แล้วพบว่า ค่าความดัน diastolic เฉลี่ยขณะพักในช่วงหลังฝึกมีค่าสูงกว่าช่วงก่อนฝึกอย่างมีนัยสำคัญ และค่าความดัน systolic เฉลี่ยในช่วงหลังฝึกมีค่าน้อยกว่าช่วงก่อนฝึก อย่างมีนัยสำคัญทั้งในขณะพักและขณะสูงสุด ซึ่งชี้ให้เห็นถึงการตอบสนองของระบบหลอดเลือดและหัวใจของผู้ป่วยที่ดีขึ้นจากการออกกำลังโดยวิธี arm cranking