Neuromuscular Junction Disorders

Thomas Willis (1621-1675), English physician, published a book, De anima brutorum in 1672 in which he wrote about "a woman who temporarily lost her power of speech and became 'mute as a fish'." This has been interpreted as being the first written description of myasthenia gravis.

Learning objectives:

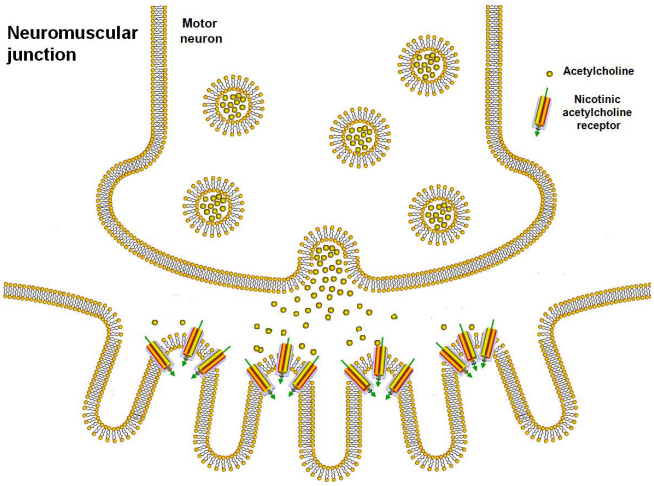
Disorders affecting the neuromuscular junction (NMJ) are among the most interesting and rewarding seen in the EMG laboratory. These disorders are generally pure motor syndromes that usually preferentially affect proximal, bulbar, or extraocular muscles. They are confused occasionally with myopathies. With knowledge of normal NMJ physiology, most of the abnormalities affecting the NMJ can be differentiated using a combination of nerve conduction studies, repetitive stimulation, exercise testing, and needle EMG.

NMJ disorders can be classified into immune-mediated, toxic or metabolic, and congenital syndromes. They usually are distinguished by their clinical and electrophysiologic findings. All are uncommon, but among them, myasthenia gravis (MG) and Lambert-Eaton myasthenic syndrome (LEMS) are the disorders most often encountered in the EMG lab. Both are immune-mediated disorders.

This paper will review the different classifications of NMJ, pathophysiology, clinical findings, nerve conduction recordings and repetitive nerve stimulation (RNS) parameters and technique, and a case study. The reader will gain insight on the function of the NMJ and be able to identify abnormalities with RNS to confirm the diagnosis of NMJ disorder.

Myasthenia Gravis:

Myasthenia gravis (MG) is a chronic autoimmune neuromuscular disease characterized by varying degrees of weakness of the skeletal (voluntary) muscles of the body. The name myasthenia gravis, which is Latin and Greek in origin, literally means "grave muscle weakness". With current therapies, however, most cases of MG are not as "grave" as the name implies. In fact, for the majority of individuals with MG, life expectancy is normal.



The hallmark of MG is muscle weakness that increases during periods of activity and improves after periods of rest. Certain muscles such as those that control eye and eyelid movement, facial expression, chewing, talking, and swallowing are often, but not always, involved in the disorder. The muscles that control breathing and neck and limb movements may also be affected.

Because MG is limited to the NMJ, there is no abnormality of the mental state or sensory or autonomic function.

Pathophysiology:

Myasthenia gravis, the best understood of all autoimmune diseases, is caused by an immunoglobulin G (IgG) directed attack on the NMJ, aimed specifically at the nicotinic acetylcholine (ACH) receptor.

The mechanism of antibody damage to the ACH receptor and *postsynaptic* membrane involves several steps. First, binding of the antibody to the receptor can directly block the binding of ACH. Second, there is a complement-directed attack, with destruction of the ACH receptor and post junctional folds. Last antibody binding can result in an increase in the normal removal of ACH receptors from the postsynaptic membrane. Thus, although the amount of ACH released is normal, there is reduced binding of ACH to the ACH receptor, resulting in a smaller endplate potential and a reduced safety factor of NMJ transmission.

The thymus gland, which lies in the chest area beneath the breastbone, plays an important role in the development of the immune system in early life. Its cells form a part of the body's normal immune system. The gland is somewhat large in infants, grows gradually until puberty, and then gets smaller and is replaced by fat with age. In adults with MG, the thymus gland is abnormal. It contains certain clusters of immune cells indicative of lymphoid hyperplasia, a condition usually found only in spleen and lymph nodes during an active immune response. Some individuals with MG develop thymomas or tumors of the thymus gland. Generally thymomas are benign, but they can become malignant. The relationship between the thymus gland and MG is not yet fully understood.

Clinical findings:

Although MG may affect any voluntary muscle, muscles that control eye and eyelid movement, facial expression, and swallowing are most frequently affected. The onset of the disorder may be sudden. Symptoms often are not immediately recognized as myasthenia gravis.

In most cases, the first noticeable symptom is weakness of the eye muscles. In others, difficulty in swallowing and slurred speech may be the first signs. The degree of muscle weakness involved in MG varies greatly among patients, ranging from a localized form, limited to eye muscles (ocular myasthenia), to a severe or generalized form in which many muscles, sometimes including those that control breathing, are affected. Symptoms,

which vary in type and severity, may include a drooping of one or both eyelids (ptosis), blurred or double vision (diplopia) due to weakness of the muscles that control eye movements, unstable or waddling gait, weakness in arms, hands, fingers, legs, and neck, a change in facial expression, difficulty in swallowing and shortness of breath, and impaired speech (dysarthria).

Age and Gender:

Myasthenia gravis presents at any age. Female incidence peaks in the third decade of life, whereas male incidence peaks in the sixth or seventh decade.

Transient (temporary) neonatal myasthenia gravis occurs in infants of myasthenic mothers. This occurs when maternal autoantibodies pass through the placenta, resulting in the same clinical syndrome in newborn infants. The illness usually is mild and self-limited and disappears after 2-3 months of age.

MG also may be seen in patients treated with penicillamine. The clinical syndrome is similar to idiopathic MG, including the presence of antiacetylcholine receptor antibodies, except that most patients slowly improve once the penicillamine has been discontinued.

The female-to-male ratio is said classically to be 6:4, but as the population has aged, the incidence is now equal in males and females.

Nerve Conduction Studies:

In any patient suspected of having MG, at least one motor and one sensory nerve should be tested in an upper and lower extremity just to be sure there is not an underlying problem such as peripheral neuropathy or motor neuron disease. Pay special attention to the compound muscle action potential (CMAP), which should be normal in patients with MG. Only a small number of patients have a baseline CMAP that falls below the normal range. With a small CMAP, LEMS should be considered.

Routine nerve conduction studies also must be performed to ensure the integrity of any nerve that subsequently will be used for RNS. A decrement on RNS can be seen in various denervating conditions (e.g., neuropathies, motor neuron disorders, inflammatory myopathies) and myotonic disorders, in addition to primary disorders of the NMJ. For instance, a decrement on RNS of the median nerve may be seen in a severe carpal tunnel syndrome.

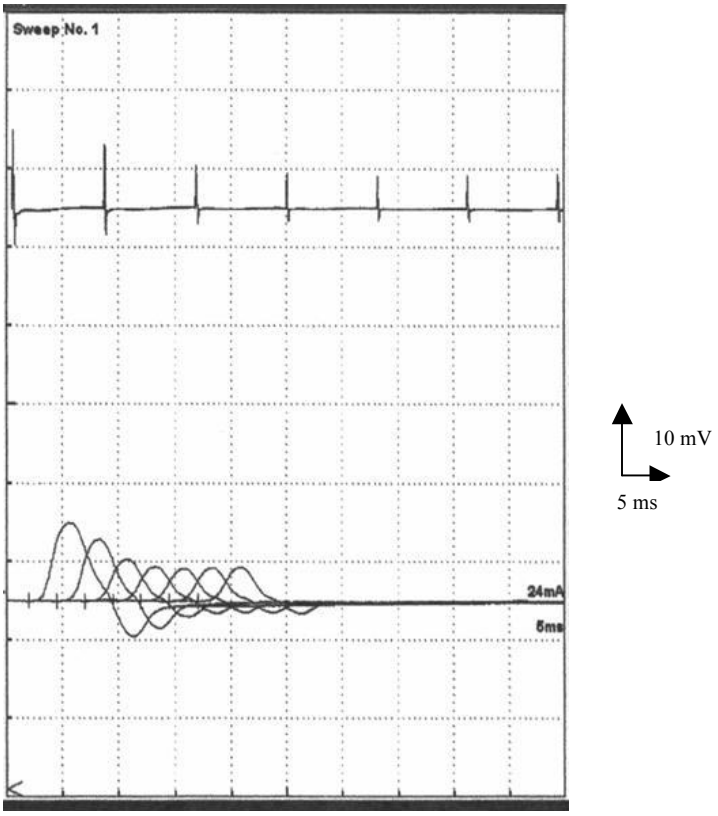
Repetitive Nerve Stimulation:

A decremental response on RNS is the electrical correlate of clinical muscle fatigue and weakness. RNS is abnormal in more than 50% to 70% of patients with generalized MG but often is normal in patients with the restricted ocular form of MG. A decrement of 10% on slow RNS (2 or 3 Hz) is characteristically seen in patients with MG. Distal nerves and proximal nerves should be tested. Though the distal nerves are easier to

obtain, the diagnostic yield of proximal nerves is greater due to these being more involved. Checking the spinal accessory or facial nerves may have better outcome for diagnosis. A baseline RNS tracing followed by one minute of exercise with four consecutive tracings one minute apart, looking for a CMAP decrement secondary to post-exercise exhaustion should be done. If a significant decrement develops, the patient should perform a brief 10 second isometric contraction, immediately followed by a single stimulation, looking for an increment in the CMAP and repair of the decrement secondary to post-exercise facilitation.

Temperature and Repetitive Nerve Stimulation:

Much as a patient tends to experience less intense symptoms in cold weather, so will cool extremities attenuate a decrement. These false negative results with a cool extremity may occur because of reduced release of Ach release with the first stimuli, leaving more quanta for subsequent stimuli or possibly because of the reduced rate of removal of calcium ions from the nerve terminal after stimulation. For this reason it is important to maintain a suitable skin temperature (i.e. 32ºC – 34ºC) before beginning RNS.



Ulnar nerve stimulation at 3 Hz

Further Correlation and differential diagnosis:

Electromyography is useful to rule out severe denervating disorders (e.g., motor neuron disease, polyneuropathy, inflammatory myopathy) and myotonic disorders. Also, the needle examination may demonstrate motor unit action potential (MUAP) abnormalities

suggestive of a NMJ disorder; unstable MUAP's; small, short-duration MUAP's similar to myopathies units; or both.

Single-fiber electromyography is used to measure the relative firing of adjacent single muscle fibers from the same motor unit and can detect both prolonged jitter (the measurement of the mean value of consecutive differences of successive interpotential intervals or the MCD) as well as blocking of muscle fibers (when impulses intermittently fail to occur).

Lambert-Eaton Myasthenic Syndrome

Lambert-Eaton myasthenic syndrome (LEMS) is a disorder of the NMJ transmission characterized by reduced release of ACH from the *presynaptic* terminal. There is now clear evidence that this disorder, like MG, is an immune-mediated disorder.

Pathophysiology:

The pathogenesis of LEMS is fairly well understood and involves the production of IgG antibodies directed at the presynaptic voltage-gated calcium channel. These antibodies interfere with the calcium-dependent release of ACH quanta from the presynaptic membrane and subsequently cause a reduced endplate potential on the postsynaptic membrane, resulting in NMJ transmission failure.

Clinical findings:

In striking contrast to the fatigue phenomena in MG, weakness peaks after rest or immediately upon awakening in the morning. Strength tends to transiently improve with brief exercise, although it is not sustained during a prolonged effort. Weakness and fatigability primarily affect the lower limbs, particularly the pelvic girdle and thigh muscles. Thus patients have difficulty in climbing stairs and, to a lesser degree, arising from a chair. The abnormality also involves the shoulders and upper limbs, usually but not always sparing the neck, bulbar, and extraocular musculature. This distribution of weakness stands in sharp contrast to the typical patterns seen in MG with conspicuous bulbar symptoms such as ptosis, diplopia, dysphagia, and dysarthria.

Rare patients have been diagnosed with the disorder after they have been prescribed calcium channel blockers or have failed to wean after anesthesia. Small cell carcinoma is eventually found in 60% of patients with LEMS.

Age and Gender:

Patients older than 40 years, usually males and smokers, are at greatest risk. The remaining patients, usually younger women, have a primary autoimmune disease without any evidence of carcinoma.

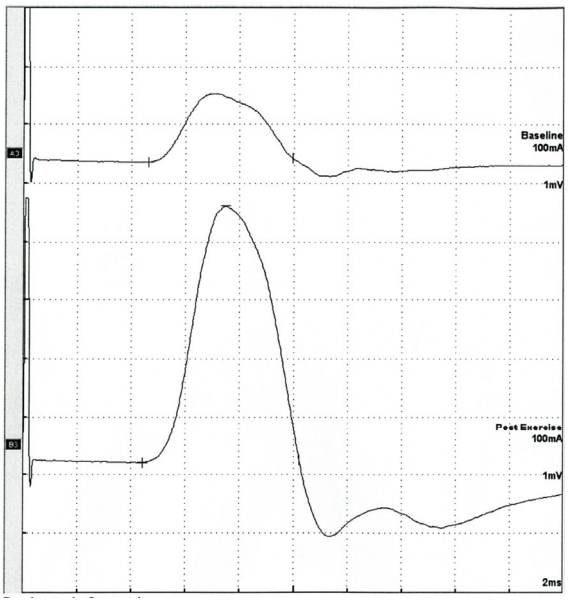
Nerve Conduction Study:

Similar to patients suspected of having MG, patients suspected of having LEMS should have at least one motor and one sensory nerve in an upper and lower extremity tested. If a patient with LEMS also has a superimposed neuropathy, either from an unrelated cause or as a paraneoplastic process from underlying carcinoma, the diagnosis of LEMS is frequently missed. *In any patient* with low or borderline low CMAP amplitudes at rest during routine motor and sensory studies, the distal motor stimulation should be repeated after 10 seconds of brief exercise to look for LEMS. As the electrical hallmark of the syndrome, nerve stimulation typically elicits very small CMAP amplitudes and in striking contrast, entirely normal sensory responses.

Repetitive Nerve Stimulation:

Repetitive stimulation at low rates (2 or 3Hz) further diminishes muscle action potential similar to the decrement seen in MG. Stimulation at high rates (30-50Hz) will cause substantial increments, usually exceeding 50-200% of the baseline value in amplitude and area. A brief voluntary contraction for up to 10 seconds will produce a marked increase in the CMAP amplitude (postexercise facilitation) due to calcium accumulation in the presynaptic nerve terminal with subsequent enhancement of the release of ACH quanta. This technique of brief, intense isometric exercise is preferable to rapid RNS, which can be quite painful. Slow RNS should always include a single stimulation after brief exercise looking for LEMS.

The marked postexercise facilitation of the CMAP is the electrical correlate of the clinical facilitation of muscle strength and reflexes seen after brief exercise.



Median nerve stimulation over the APB at baseline and after 10 seconds of exercise

Further correlation:

Needle EMG results in LEMS are similar to those in MG. Insertional activity is normal, and abnormal spontaneous activity is not seen. MUAPs usually are normal. Occasionally they are unstable: rarely they are short, small and polyphasic, similar to myopathic MUAPs. SF-EMG shows increased jitter or blocking, similar to MG, and cannot routinely differentiate between these two disorders.

Botulism

Botulism is caused by the potent exotoxin of Clostridium botulinum, which blocks presynaptic release of ACH at both somatic and autonomic synapses. The result is NMJ and parasympathetic blockade. Although there are eight strains of botulism, three are most commonly associated with clinical diseased: types A, E and F. These are most commonly found in improperly prepared food, especially canned vegetables or fish. Botulism also can occur as the result of a wound infection. Infantile botulism is the most common clinical presentation.

Clinical:

In adults, symptoms usually will occur within 1 to 2 days after consumption of an exotoxin or 1 to 2 weeks after a deep wound has been inoculated with toxin. Nausea, vomiting, and abdominal pain are common initially. These symptoms are followed by blurred vision, diplopia, and dysarthria. Rapidly progressive descending weakness follows. The illness progresses for 1 to 2 weeks, with recovery occurring slowly over several months.

Infantile botulism presents with decreased muscle tone and movement, a weak cry, and constipation.

Nerve Conduction Study and RNS

The pathophysiology of botulism is presynaptic blocking of ACH, similar to LEMS. Likewise, the electrophysiologic evaluation and findings in Botulism and LEMS are similar. Sensory conduction studies are normal. CMAP amplitudes are decreased with normal latencies and conduction velocities. A decremental response may be seen with slow RNS. An incremental response characteristically occurs after brief exercise or fast RNS. This finding usually is present in mild or early cases. Note, however, that in severe botulism, if the amount of ACH release has dropped severely below threshold, even facilitation with rapid RNS or brief exercise may not result in a threshold response, and no increment occurs in the CMAP amplitude. Thus, the lack of an incremental response to rapid RNS or brief exercise cannot completely exclude the diagnosis of botulism.

Differential Diagnosis:

Clinically, MG is not usually associated with such a rapid progression, nor is there any autonomic dysfunction. Guillain-Barre' syndrome is included, but sensory complaints usually are prominent.

Further Correlation

Botulinum toxin is such a potent NMJ blocker that the muscle fibers are effectively chemo-denervated. Thus, in needle EMG, fibrillation potentials and positive sharp waves are common. Similar to other NMJ disorders, MUAPs may be normal or small, short and polyphasic, similar to myopathic MUAPs. Depending on the severity, recruitment may be normal, early, or reduced. The latter may occur if every muscle fiber of a motor unit is blocked by the botulinum toxin, effectively reducing the number of motor units. Likewise, SF-EMG shows increased jitter and blocking, signifying the underlying NMJ dysfunction.

Usually, differentiating between botulism and MG is straightforward, both by clinical and electrodiagnostic findings. In contrast, the electrodiagnostic findings in botulism and LEMS may be indistinguishable (depending on the degree of denervation present in botulism), yet their clinical presentations are markedly different.

Other NMJ Disorders

A variety of natural toxins of animal, plant, tick, and bacterial origin can cause disorders of the NMJ. Animal toxins include those from venomous snakes and arthropods, certain marine creatures, skin secretions of dart-poison frogs, and poisonous fish, shellfish, and crabs. These toxins act at single or multiple sites of the neuromuscular apparatus, interfering with voltage-gated ion channels, ACH release, depolarization of postsynaptic membrane, or generation and spread of the muscle action potential.

The administration of some drugs, notably kanamycin and neomycin and all other polypeptide aminoglycoside antibiotics, may cause abnormalities of neuromuscular transmission.

The congenital myasthenic syndromes are a group of exceptionally rare disorders caused by an inherited defect in NMJ transmission. These disorders are not immune mediated and thus are not associated with autoantibodies in the blood and do not respond to prednisone, other immunosuppressants, or plasma exchange. They are different from transient neonatal MG, which is caused by the transfer of antibodies via the placenta from a mother with MG to her baby.

Repetitive Nerve Stimulation Parameters and Techniques:

Repetitive nerve stimulation (RNS) techniques can be used in diagnosing diseases of the NMJ; however, technical errors can give erroneous results, and careful attention to technique is essential.

1. For 24 hours before testing the patient must discontinue any medication that affects the NMJ (i.e. Mestinon).
2. Immobilize the muscle as best as possible.
3. The skin temperature is maintained at 32ºC – 34ºC to avoid false-negative results.
4. All stimuli must be supramaximal.
5. Perform routine motor nerve conduction first to ensure that the nerve is within normal limits.
6. If the CMAP amplitude is low at baseline, have the patient perform 10 seconds of maximal voluntary exercise, then stimulate the nerve supramaximally immediately postexercise, looking for an abnormal increment (>40% above baseline).
7. Perform RNS on one distal and one proximal motor nerve. Always try to study weak muscles. If no decrement is found with a proximal limb muscle, a facial muscle can be tested, keeping in mind technical considerations.
8. Two or 3 Hz is appropriate rate of RNS because rates of 5 Hz (=200ms between stimuli) or less, there is significant time for the intracellular calcium concentration to diminish, resulting in the depletion of the stores of ACH readily available for release, thereby increasing the likelihood of detecting MG.
9. In LEMS, the deficit is present in any muscles tested.
10. If the patient is suspected of having botulism, any clinically weak muscle is tested.
11. In MG, muscle weakness increases with exertion but improves with rest and with anticholinesterase drugs (i.e. Tensilon, Mestinon). Proximal muscle testing is much more sensitive than distal muscle testing; however, testing proximal muscles is technically more difficult.
12. If >10% decrement occurs and is consistently reproducible:
    a) Have the patient perform maximal voluntary exercise for 10 seconds.
    b) Immediately repeat the 2 or 3-Hz RNS postexercise to demonstrate postexercise facilitation and repair of the decrement.
13. If <10% decrement or no decrement occurs:
    a) Have the patient perform maximal voluntary exercise for 1 minute, then perform 2 or 3-Hz RNS immediately and 1, 2, 3, and 4 minutes after exercise to demonstrate postexercise exhaustion.
    b) If a significant decrement occurs, have the patient perform maximal voluntary exercise again for 10 seconds and immediately repeat 2 or 3-Hz RNS to demonstrate repair of the decrement.

Proximal Stimulation:

a. Facial nerve: Stimulate behind the earlobe at the stylomastoid foramen with the active recording electrode over the nasalis or orbicularis oculi muscle.
b. Spinal accessory nerve: Stimulate the nerve as it descends along the posterior border of the sternocleidomastoid muscle with the active electrode over the upper trapezius at the angle of the neck and shoulder. The patient is upright in a chair; the arms are adducted and extended with the hand holding the bottom of the chair. Exercise is obtained by having the patient shrug the shoulders against his/her own resistance.
c. Brachial plexus/axillary nerve: Stimulate at Erb's point with the active electrode over the deltoid muscle. This technique is more painful, and movement artifact can be a problem.
d. Musculocutaneous nerve: Stimulate in the axilla with the active electrode over the biceps muscle. With this technique, the stimulus can be unstable.
e. Femoral nerve: Stimulate in the inguinal region with the active electrode over the vastus medialis muscle. This technique can be painful.

Distal Stimulation:

a. Ulnar nerve: Stimulate the ulnar nerve at the wrist with the active electrode placed over the abductor digiti minimi muscle.
b. Median nerve: Stimulate the median nerve at the wrist with the active electrode over the abductor pollicis brevis muscle. The disadvantage of this technique is that the thumb is difficult to immobilize.

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Case Study

Date of Birth:	3/21/1958	Temperatures:
Age:	50	Right wrist: 32°C after warming
Gender:	Female	Right leg: 31°C after warming

REASON FOR STUDY: History of generalized myasthenia gravis. Patient recently discontinued all her immunomodulating medications with fairly minimal clinical symptoms since. She continues to use Mestinon and pseudoephedrine as means to treat her symptoms on a daily basis. Her last dose of Mestinon was more than 12 hours ago.

NERVE CONDUCTION STUDIES:

1. Motor conduction studies of the right ulnar and right peroneal nerves were normal.

2. Sensory nerve conduction studies of the right ulnar and right sural nerves were normal.

3. 3-Hertz repetitive stimulation of the right ulnar nerve (recording from the abductor digiti minimi) revealed a decrement at baseline which corrected after 10 seconds of maximal voluntary contraction (post-exercise facilitation). A decrement was seen after one minute of exercise when recording every minute for five minutes (post-exercise exhaustion).

NEEDLE ELECTROMYOGRAPHY:

Needle examination of selected proximal and distal muscles in the right arm was carried out. There was no evidence of abnormal insertional or spontaneous activity in any muscles sampled. Unstable motor unit action potentials were recorded in the right biceps muscle. The remainder of the motor unit action potentials appeared normal as tabulated below. There were no myopathic units.

SINGLE FIBER EMG

A single fiber EMG study of the frontalis muscle was carried out. A total of 15 fiber pairs were analyzed. The MCD for each individual fiber pair varied from 39 to 187 microseconds; with only three fiber pairs under 51 microseconds (upper limit of normal for patient's age is 57.5). The mean jitter for 15 fiber pairs was 96 (upper limit of normal is 37.3). Blocking was seen in all fiber pairs and ranged from 1% to 44.5%. The clearly abnormal single fiber EMG study supports the diagnosis of a neuromuscular transmission disorder.

CLINICAL CORRELATION:

This is an abnormal study. The clearly abnormal repetitive stimulation and single fiber EMG study supports the diagnosis of a neuromuscular transmission disorder. There is no electrical evidence to suggest the presence of an underlying neuropathy or myopathy.

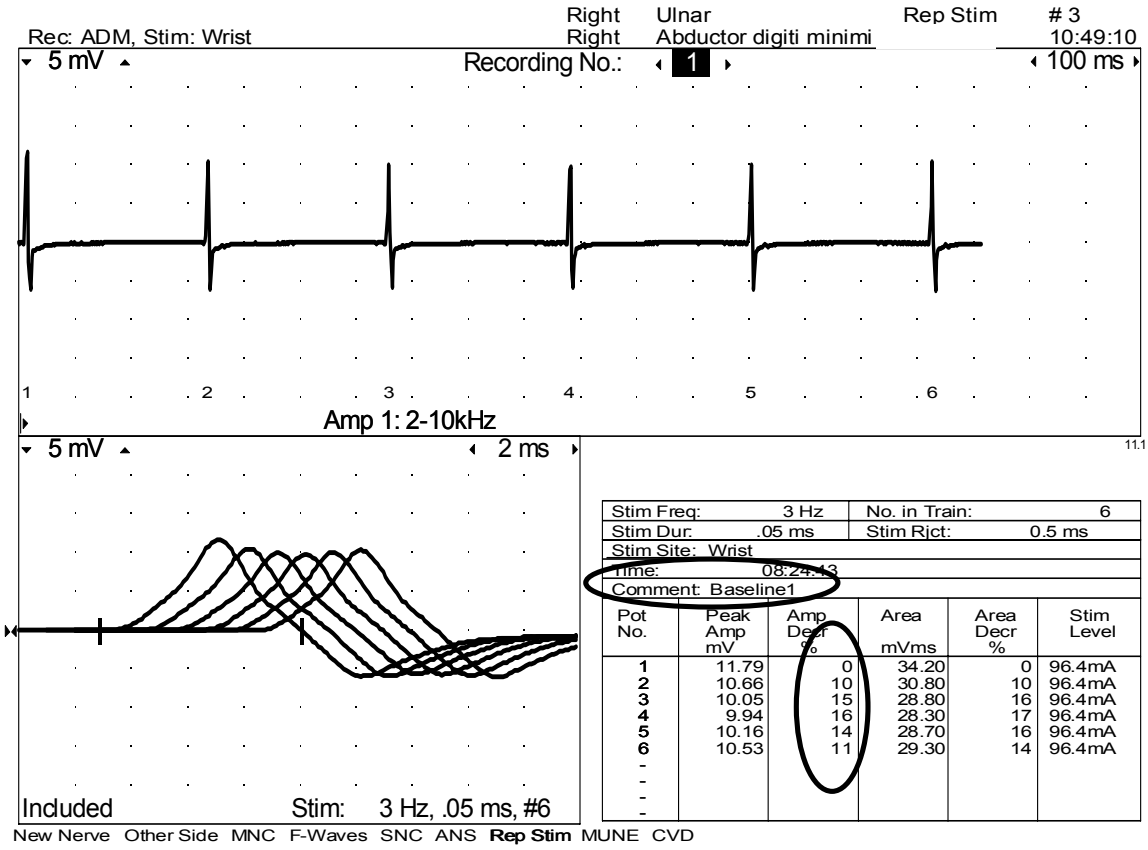
Motor Nerve Conduction:

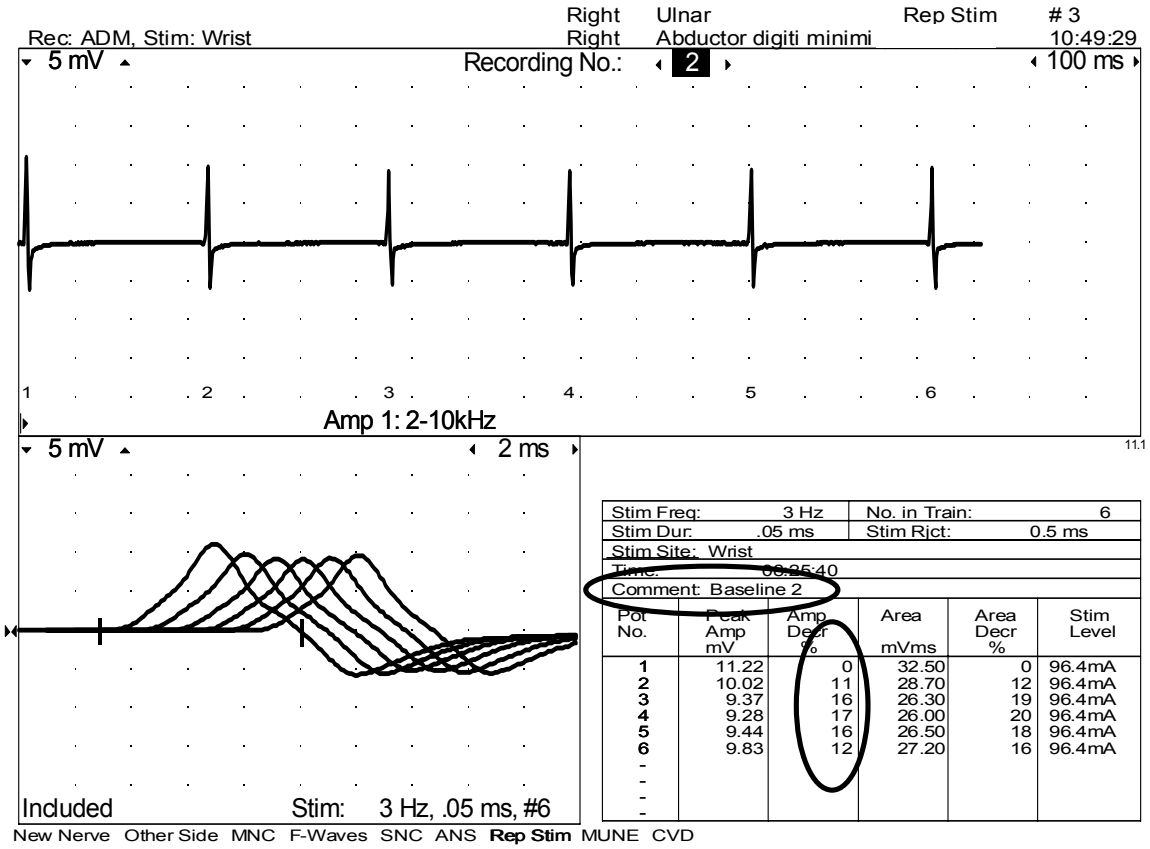
Nerve and Site	Segment	Distance	Latency	Amplitude	Conduction Velocity
Right Ulnar					
Wrist	Abductor digiti minimi-Wrist	60 mm	4.2 ms	9.61 mV	
Elbow	Wrist-Elbow	225 mm	8.4 ms	8.33 mV	53.5 m/s
Right Peroneal					
Ankle	Extensor digitorum brevis-Ankle	90 mm	4.4 ms	5.61 mV	
Fibula (head)	Ankle-Fibula (head)	280 mm	10.0 ms	5.30 mV	50.0 m/s
Popliteal fossa	Fibula (head)-Popliteal fossa	90 mm	12.0 ms	4.94 mV	45.0 m/s

Sensory Nerve Conduction:

Nerve and Site	Segment	Distance	Amplitude	Peak Latency
Right Ulnar				
Digit V (little finger)	Wrist-Digit V	130 mm	23.8 µV	3.4 ms
Right Sural				
Lower leg	Lateral malleolus-Lower leg	140 mm	11.1 µV	3.3 ms

Repetitive Stimulation:





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Stim Dur:	.05 ms	Stim Rjct:	0.5 ms
Stim Site: Wrist			
Time:	08:25:40		
Comment: Baseline 2			

Pot No.	Peak Amp mV	Amp Decr %	Area mVms	Area Decr %	Stim Level
1	11.22	0	32.50	0	96.4mA
2	10.02	11	28.70	12	96.4mA
3	9.37	16	26.30	19	96.4mA
4	9.28	17	26.00	20	96.4mA
5	9.44	16	26.50	18	96.4mA
6	9.83	12	27.20	16	96.4mA
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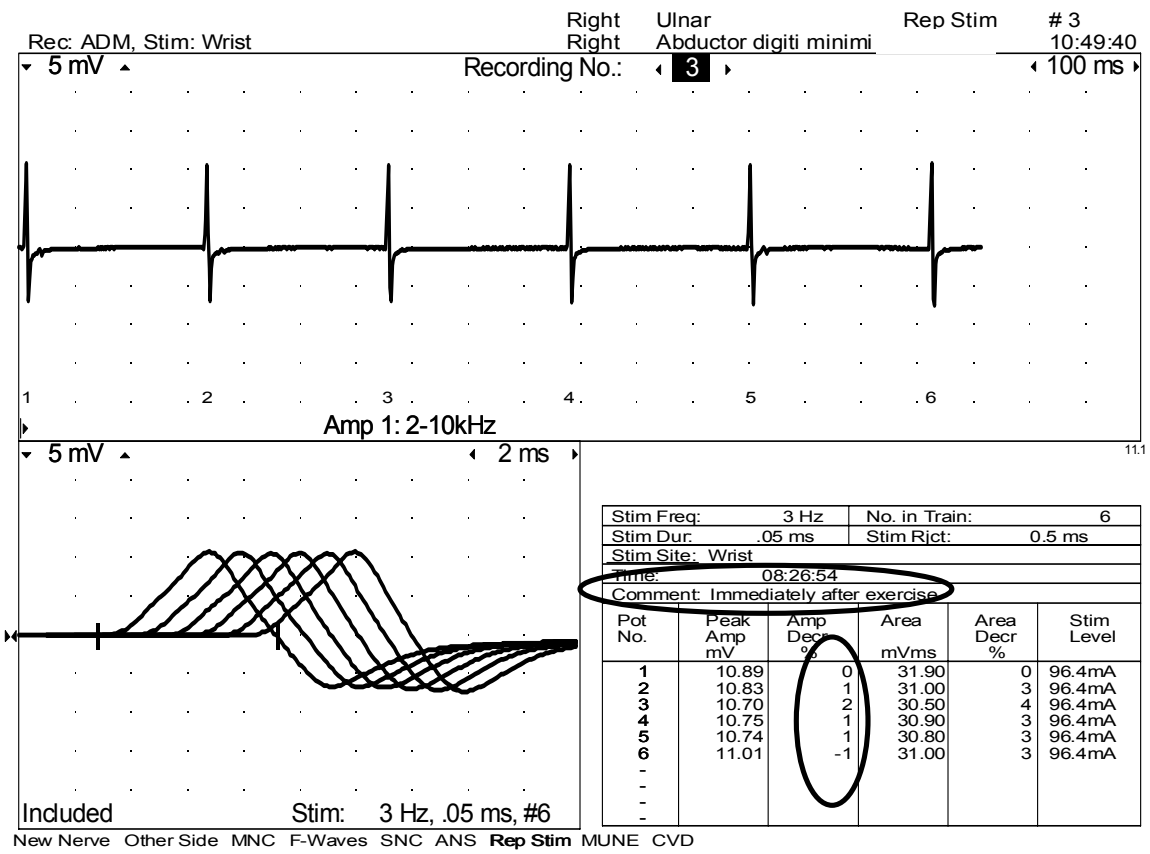
-					
-					
-					



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Stim Dur:	.05 ms	Stim Rjct:	0.5 ms
Stim Site: Wrist			
Time:	08:26:54		
Comment: Immediately after exercise			

Pot No.	Peak Amp mV	Amp Decr %	Area mVms	Area Decr %	Stim Level
1	10.89	0	31.90	0	96.4mA
2	10.83	1	31.00	3	96.4mA
3	10.70	2	30.50	4	96.4mA
4	10.75	1	30.90	3	96.4mA
5	10.74	1	30.80	3	96.4mA
6	11.01	-1	31.00	3	96.4mA
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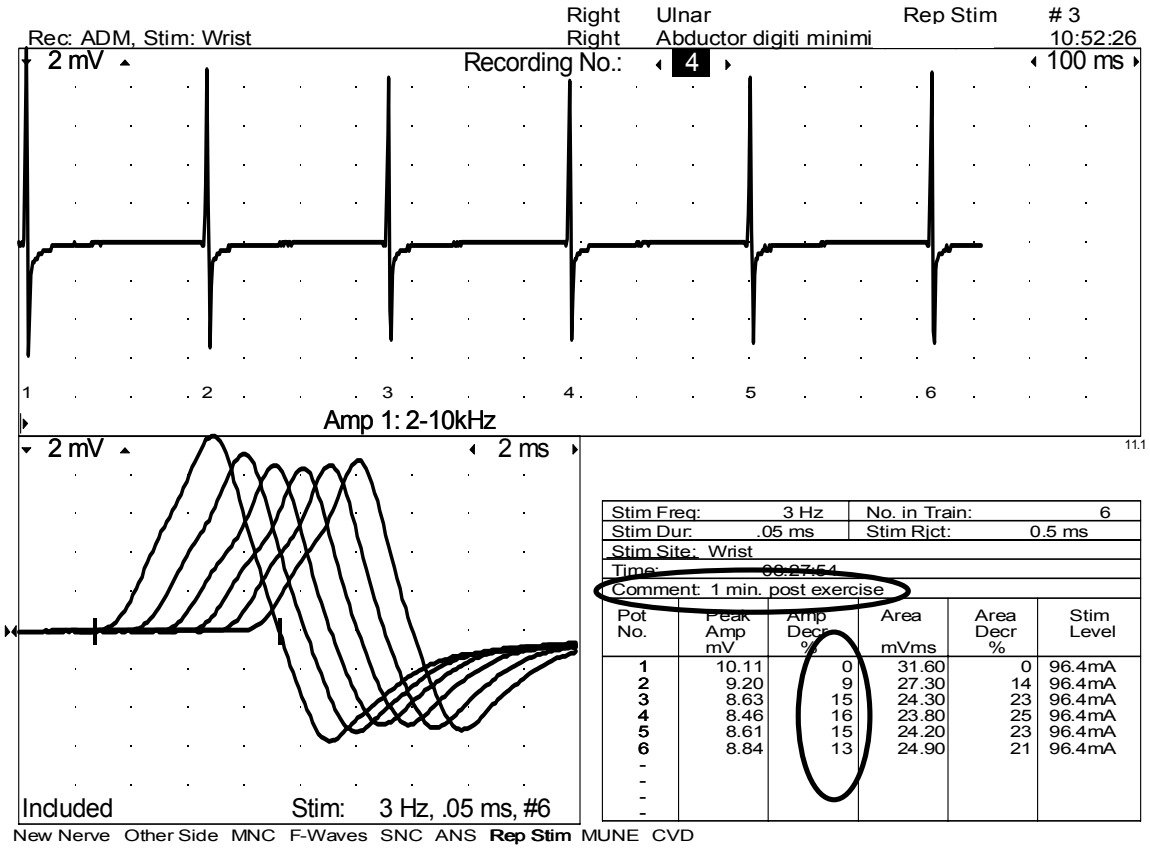

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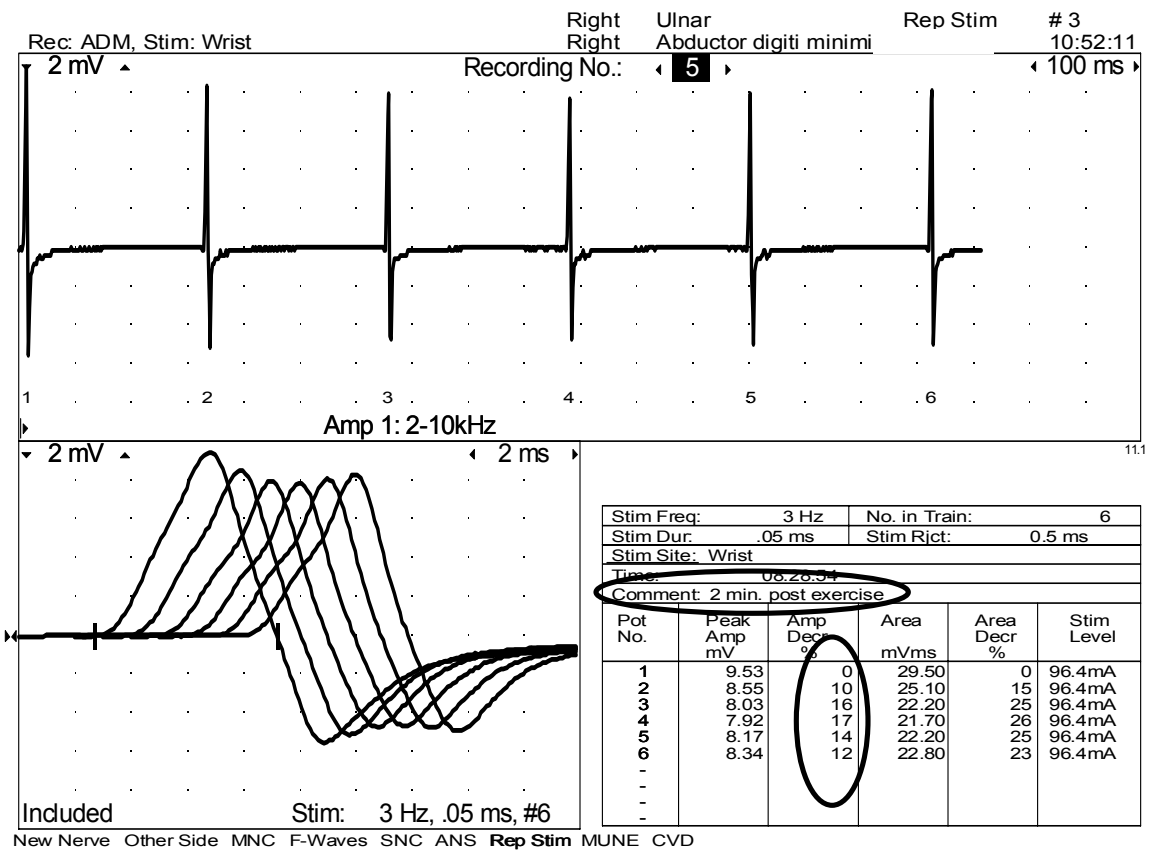
Stim Dur:	.05 ms	Stim Rjct:	0.5 ms
Stim Site: Wrist			
Time:	08:27:54		
Comment: 1 min. post exercise			

Pot No.	Peak Amp mV	Amp Decr %	Area mVms	Area Decr %	Stim Level
1	10.11	0	31.60	0	96.4mA
2	9.20	9	27.30	14	96.4mA
3	8.63	15	24.30	23	96.4mA
4	8.46	16	23.80	25	96.4mA
5	8.61	15	24.20	23	96.4mA
6	8.84	13	24.90	21	96.4mA

New Nerve Other Side MNC F-Waves SNC ANS **Rep Stim** MUNE CVD



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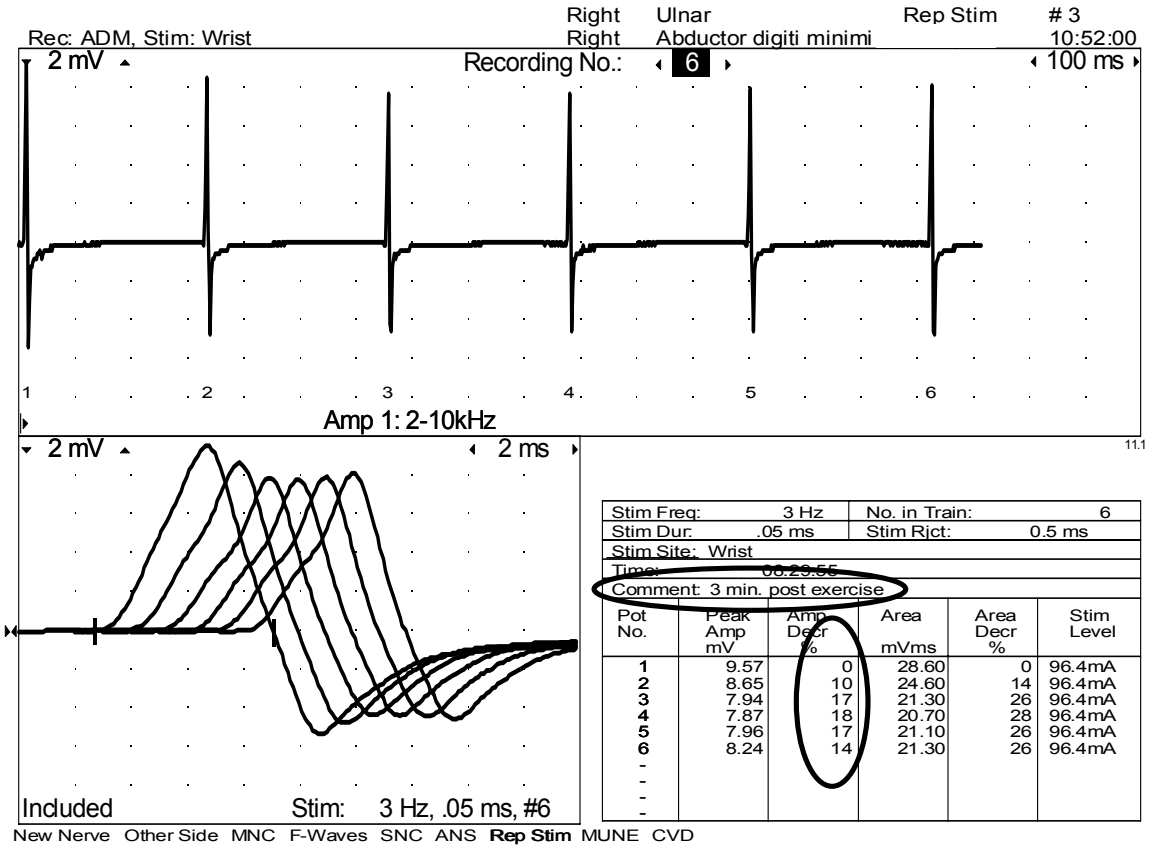
Stim Site: Wrist			
Time:	08:28:54		
Comment: 2 min. post exercise			

Pot No.	Peak Amp mV	Amp Decr %	Area mVms	Area Decr %	Stim Level
1	9.53	0	29.50	0	96.4mA
2	8.55	10	25.10	15	96.4mA
3	8.03	16	22.20	25	96.4mA
4	7.92	17	21.70	26	96.4mA
5	8.17	14	22.20	25	96.4mA
6	8.34	12	22.80	23	96.4mA

New Nerve Other Side MNC F-Waves SNC ANS **Rep Stim** MUNE CVD



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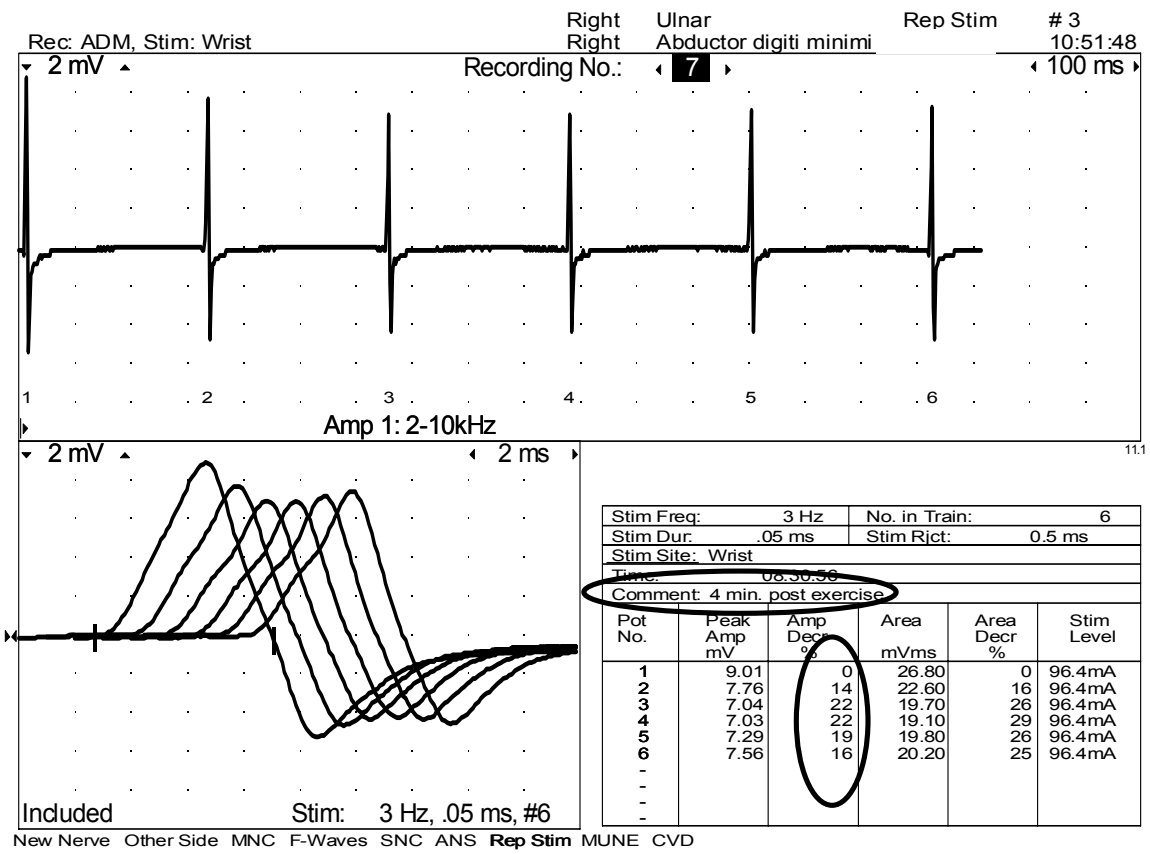
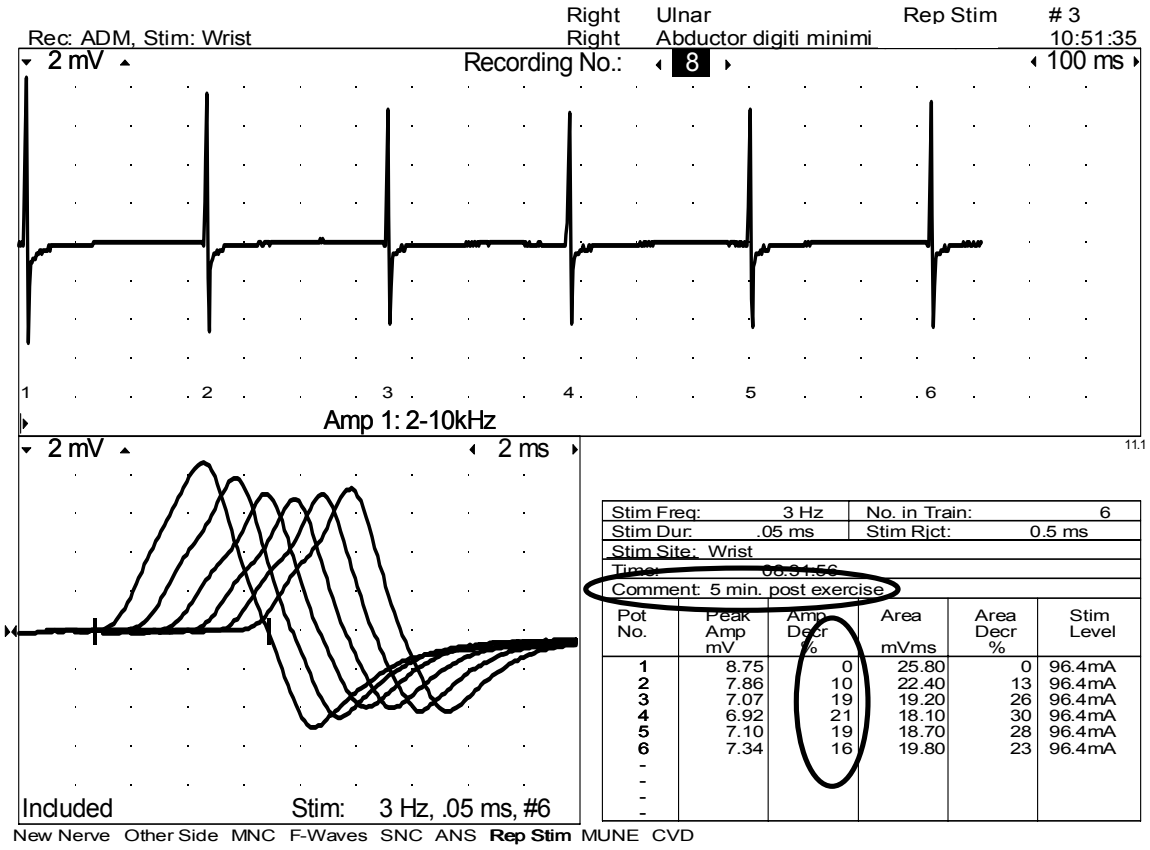
Stim Site: Wrist			
Time: 08:29:55			
Comment: 3 min. post exercise			

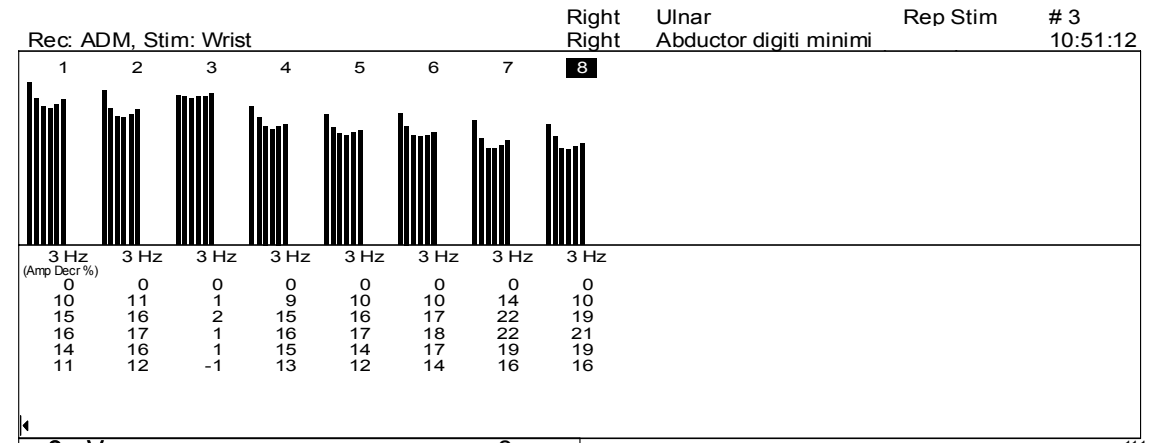
Pot No.	Peak Amp mV	Amp Decr %	Area mVms	Area Decr %	Stim Level
1	9.57	0	28.60	0	96.4mA
2	8.65	10	24.60	14	96.4mA
3	7.94	17	21.30	26	96.4mA
4	7.87	18	20.70	28	96.4mA
5	7.96	17	21.10	26	96.4mA
6	8.24	14	21.30	26	96.4mA
-					
-					
-					
-					



Stim Freq:	3 Hz	No. in Train:	6
Stim Dur:	.05 ms	Stim Rjct:	0.5 ms
Stim Site: Wrist			
Time: 08:30:56			
Comment: 4 min. post exercise			

Pot No.	Peak Amp mV	Amp Decr %	Area mVms	Area Decr %	Stim Level
1	9.01	0	26.80	0	96.4mA
2	7.76	14	22.60	16	96.4mA
3	7.04	22	19.70	26	96.4mA
4	7.03	22	19.10	29	96.4mA
5	7.29	19	19.80	26	96.4mA
6	7.56	16	20.20	25	96.4mA
-					
-					
-					
-					


Rec: ADM, Stim: Wrist
Right Ulnar
Right Abductor digiti minimi
Rep Stim # 3
10:51:35
2 mV
Recording No.: 8
100 ms
1 2 3 4 5 6
Amp 1: 2-10kHz
11.1
2 mV
2 ms
Included
Stim: 3 Hz, .05 ms, #6
Stim Freq: 3 Hz
No. in Train: 6
Stim Dur: .05 ms
Stim Rjct: 0.5 ms
Stim Site: Wrist
Time: 08:31:56
Comment: 5 min. post exercise
Pot No. | Peak Amp mV | Amp Decr % | Area mVms | Area Decr % | Stim Level
1 8.75 0 25.80 0 96.4mA
2 7.86 10 22.40 13 96.4mA
3 7.07 19 19.20 26 96.4mA
4 6.92 21 18.10 30 96.4mA
5 7.10 19 18.70 28 96.4mA
6 7.34 16 19.80 23 96.4mA
New Nerve Other Side MNC F-Waves SNC ANS Rep Stim MUNE CVD


Rec: ADM, Stim: Wrist
Right Ulnar
Right Abductor digiti minimi
Rep Stim # 3
10:51:12
1 2 3 4 5 6 7 8
3 Hz 3 Hz 3 Hz 3 Hz 3 Hz 3 Hz 3 Hz 3 Hz
(Amp Decr %)
0 0 0 0 0 0 0 0
10 11 1 9 10 10 14 10
15 16 2 15 16 17 22 19
16 17 1 16 17 18 22 21
14 16 1 15 14 17 19 19
11 12 -1 13 12 14 16 16

Needle EMG Examination:

	Spontaneous Activity			Volitional MUAP's			
Muscle	Insertional	Fibs/+Waves	Fasciculations	Duration	Amplitude	Polyphasic	Recruitment
Right Deltoid	Normal	None	None	Normal	Normal	None	Normal
Right Biceps brachii	Normal	None	None	Unstable	Unstable	None	Normal
Right 1st dorsal interosseous	Normal	None	None	Normal	Normal	None	Normal

Single Fiber EMG:

Right Frontalis

Recording	MCD	Blocking	Firing Rate	Sweeps
2	88 µs	17.9 %	12 Hz	78
3	82 µs	4.0 %	8 Hz	100
4	102 µs	3.0 %	11 Hz	100
5	89 µs	2.0 %	14 Hz	100
6	62 µs	40.2 %	18 Hz	97
7	107 µs	14.0 %	11 Hz	100
8	130 µs	35.6 %	8 Hz	87
12	129 µs	37.8 %	8 Hz	82
13	187 µs	44.3 %	11 Hz	79
14.1	154 µs	1.4 %	7 Hz	68
14.2	83 µs	14.5 %	7 Hz	55
16	51 µs	2.5 %	7 Hz	77
17	86 µs	8.9 %	16 Hz	78
18	50 µs	1.0 %	19 Hz	100
19	39 µs	2.0 %	19 Hz	100

Mean:	96 µs	15.3 %	12 Hz
SD:	40 µs	16.0 %	4 Hz
Max:	187 µs	44.3 %	19 Hz
Min:	39 µs	1.0 %	7 Hz