Electromyography in Disorders of Muscle Tone

Andrew Eisen

ABSTRACT: No single clinical electrophysiological test can evaluate disorders of muscle tone. These disorders, symptomatic of a variety of diseases have a multifactorial physiological basis. The several tests used are complimentary each aiming to study different aspects of spinal and supraspinal reflexes which become deranged. The H reflex and F wave (H max/M max and F max/M max ratios) measure motoneuron pool excitability in general. The tendon reflex includes spindle mechanisms bypassed by the H reflex and, with limitations, comparison of H max/M max and T max/M max yields information about the γ system. Tonic vibration of a tendon inhibits the H reflex from the same muscle. The TVR measures autogenous presynaptic inhibition exerted by the Ia afferents of the muscle. Recurrent inhibition via Renshaw cells is evaluated by studying the effect of collision on the H reflex. Reciprocal inhibition of the Ia afferents can be assessed by measuring H reflex change induced by stimulating Ia afferents from antagonists. Changes in the H reflex recovery cycle measure polysynaptic influences on spinal motoneuron excitability. Cutaneomuscular (flexor) reflexes measure poly- and oligosynaptic excitatory drive to spinal motoneurons and the blink reflex evaluates the excitatory drive to brainstem motoneurons. Long loop (segmental) responses can be evaluated by limb pertubation using a torque motor or electrical stimulation applied during voluntary muscle contraction. Finally needle electromyography is a more relevant test in several disorders of muscle tone such as the stiff-man syndrome and Isaacs' syndrome.

RÉSUMÉ: L'électromyographie dans les perturbations du tonus musculaire. Les perturbations du tonus musculaire ne peuvent être évaluées au moyen d'une épreuve électrophysiologique clinique unique. Ces perturbations, symptomatiques de différentes maladies, ont une base physiologique multifactorielle. Les multiples tests utilisés sont complémentaires, chaque test évaluant différents aspects des réflexes spinaux et supraspinaux qui peuvent être perturbés. Le réflexe H et l'onde F (les rapports H max/M max et F max/M max) mesurent l'excitabilité du pool de motoneurones en général. Le réflexe ostéotendineux inclut des mécanismes en fuseau contournés par le réflexe H et, dans une certaine mesure, la comparaison de H max/M max et de T max/M max fournit de l'information sur le système. Les vibrations toniques d'un tendon inhibent le réflexe H du même muscle. Le RTV mesure l'inhibition présynaptique autogène exercée par les afférents Ia de ce muscle. L'inhibition récurrente via les cellules de Renshaw est mesurée par l'étude de l'effet de collision sur le réflexe H. L'inhibition réciproque des afférents Ia peut être évaluée en mesurant le changement du réflexe H induit par la stimulation des afférents Ia qui proviennent des antagonistes. Les changements du cycle de récupération du réflexe H mesurent les influences polysynaptiques sur l'excitabilité du motoneurone spinal. Les réflexes cutanéomusculaires (flexion) mesurent la transmission excitative poly- et oligo-synaptique vers les motoneurones spinaux et le réflexe de clignotement évalue la transmission excitative vers les motoneurones du tronc cérébral. Les réponses des boucles longues (segmentaires) peuvent être mesurées par la perturbation d'un membre au moyen d'un moteur qui imprime un moment de torsion ou de stimulation électrique pendant la contraction musculaire volontaire. Finalement, l'électromographie à l'aiguille est le test le plus approprié dans plusieurs perturbations du tonus musculaire telles le syndrome de l'homme raide et le syndrome d'Isaac.

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Altered muscle tone is symptomatic of a variety of syndromes manifested, for example by spasticity, rigidity, abnormal posturing (dystonia) and hypotonia. These are usually identifiable without difficulty but are not readily defined in physiological terms.[1-5] The physiological mechanisms underlying abnormal tone are complex and multifactorial. One or more of the following are involved in many syndromes characterized by altered tone: peripheral sensory receptors, primary afferent fibers and their presynaptic terminals, excitatory and inhibitory interneurons, alpha motorneurons and gamma (fusimotor) motorneurons. The commonly held view that disorders of muscle tone are due primarily to defective fusimotor control of muscle spindle endings is probably naive.[6] Nevertheless, common to many of these disorders are altered supraspinal influences. As a result, changes in spinal cord function and segmental reflexes are reflected clinically by paresis or loss of dexterity.

Over the last 2 decades numerous electrophysiological methods have been developed for assessing altered muscle tone. Many complement each other, evaluating different aspects of neuronal circuitry, and it is unlikely that a single technique will

From the Neuromuscular Diseases Unit (EMG), The Vancouver General Hospital, Vancouver
Reprint requests to: Dr. A. Eisen, The Neuromuscular Diseases Unit (EMG), The Vancouver General Hospital, 855 West 12th Ave., Vancouver, B.C., Canada V5Z 1M9

evolve which will be applicable to all situations. In evaluating altered muscle tone, clinical electrophysiology attempts to answer at least three questions:[7] 1) Is tone abnormal and if so to what extent? 2) What physiological factor(s) underly the abnormality? 3) Is a particular therapeutic regimen efficacious and what is its mode of action?

This paper overviews many of the presently available electrophysiological tests used clinically to assess altered muscle tone (Table 1). It does not consider how these tests can be used to evaluate myorelaxant agents, (but see references 1, 4, 8). Other contributors to this symposium will concentrate on specific aspects of clinical electrophysiology in altered muscle tone.

The H Max/M Max Ratio

This measures alpha motoneuron excitability reflecting the percentage of motorneurons activated via the monosynaptic reflex.[9] It assumes that presynaptic inhibition, acting upon the terminal arborizations of the Ia afferents, remains constant.[10] Long duration (1 ms) shocks, preferentially exciting Ia afferents, are commonly applied to the sciatic nerve in the popliteal fossa. The H reflex, with the maximum amplitude, is recorded from the soleus muscle (H Max). The same stimulation site is used to evoke a maximal motor compound action potential employing a supramaximal shock of 1 ms duration. The response is recorded over the soleus muscle (M Max). The normal range of H Max/M Max is 5-35%. Although, the soleus H reflex is most commonly employed, monosynaptic responses with the same properties as the soleus H reflex can be elicited from many other muscles. They become more easily elicited in upper motor neuron lesions.[11]

The H Max/M Max ratio is increased in spasticity.[9,12] In parkinsonian rigidity it is normal or reduced.[9,13,14]

The T Max/M Max Ratio

This additionally measures excitability of the muscle spindles and the rheological properties of the stretched muscle bypassed by the H reflex.[15] The ankle jerk is elicited by a single tap applied to the achilles tendon using an electromechanical hammer. The direction and percussive force of the hammer is varied to obtain a maximal amplitude response recorded over the gastrocnemius-soleus complex (T Max). This site is also used to record the maximal M wave elicited by supramaximal stimulation to the sciatic nerve at the popliteal fossa (M Max). In parkinsonian patients with rigidity the ratio is within the normal range (5-40%). It is increased in spasticity.

Table 1: Electrophysiological tests used in assessment of muscle tone

Test	Evaluate/Measures
H Max/M Max Ratio	Alpha Motoneuron Excitability
T Max/M Max Ratio	Alpha Motoneuron Excitability Incorporates Spindle Mechanism
Tonic Vibration of Muscle	Presynaptic Inhibition of Monosynaptic Reflex
H Reflex Recovery Cycle	Polysynaptic Changes in Motor Neuron Excitability
Modification of H Reflex by Maximal Conditioning Shock	Recurrent Renshaw Inhibition
Modification of H Reflex by Simultaneous Sensory Stimulation	Reciprocal Ia Inhibition
F Wave Responses F Max, F/M Ratio	Motoneuron Excitability
Limb Perturbation During Intended Motor Acts	Short and Long-Loop Responses
Nerve Stimulation During Muscle Contraction	Short and Long-Loop Responses
Blink Reflex	Poly and Oligosynaptic Excitatory Drive to the Brainstem Interneurons
Cutaneomuscular Flexor Reflexes	Poly and Oligosynaptic Excitability Drive to Spinal Interneurons
Needle Electromyography	Recruitment Pattern, Firing Frequency of Motor Units, Motor Unit Morphology

Vibratory Inhibition of the H Reflex (Tonic Vibration Reflex)

In normal subjects, vibration applied to a limb muscle produces an illusion of movement, a slowly augmenting contraction (the tonic vibration reflex) and relatively non-specific inhibition of the monosynaptic reflex.[16] This last effect predominantly results from autogenic presynaptic inhibition of the muscle Ia afferents responsible for the monosynaptic reflex.[17-20]

A 100 Hz vibrator is attached to the achilles tendon and the maximum amplitude H reflex is measured under control conditions and during vibration. Inhibition is expressed as [H Max during vibration/control H Max × 100]. In normal (young) subjects there is about a 40% inhibition of H Max in the relaxed muscle. Presynaptic inhibition declines in older subjects and inhibition is reduced during muscle contraction in normal subjects.[10]

In spasticity, inhibition is significantly reduced and may be absent.[10,20,21] This is also true during contraction, a modification of the technique that ensures that the inhibition being measured is truly presynaptic.[10] Presynaptic inhibition is not reduced in parkinsonism[12] and returns to normal within a year of complete spinal lesion.[16]

H Reflex Recovery Cycle

This monitors polysynaptic changes in motoneuron excitability secondary to segmental and suprasegmental mechanisms.[18,22] Paired stimuli of equal strength, each of sufficient intensity to elicit a maximum H reflex, are applied at various interstimulus intervals. Results are expressed as a (percentage) ratio of the second (test) response, H2, to the first (conditioning) response, H1. In certain individuals results are stable over many hours.[23]

The normal H reflex recovery cycle follows a characteristic pattern: complete inexcitability for 2 msec, reappearance of the reflex for about 10 msec, marked inhibition for about 100 msec, facilitation for about 200 msec and moderate inhibition for several seconds. Several aspects of the recovery cycle are measureable: T_0, time of onset of recovery, T_1, time of peak facilitation, T_2, duration of facilitation, T_3, recovery time constant, R_0, amplitude at onset of recovery and R_1, degree of facilitation.[24]

In spastic subjects there is a trend toward increased time of peak facilitation, increased duration of facilitation and increased recovery time constant. However, only the degree of facilitation, which approximately doubles, differs significantly from normals.[24] The parkinsonian recovery curve exhibits a longer and higher period of facilitation.[25]

Recurrent Renshaw Inhibition

Inhibition by Renshaw cells reduces the excitability of motoneurons and when less effective results in motoneuron hyperactivity. Renshaw inhibition can be assessed using a double collision technique.[26,27] This prevents excitatory postsynaptic

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potentials, created by Ia conditioning volleys, negating recurrent inhibition in the tested motoneurons.[26] In essence the H reflex amplitude is compared when elicited by a stimulus adjusted to produce a maximum H response (conditioning shock) with that obtained by collision of this volley with a shock adjusted for a maximum M wave (test shock). Conditioning-test interval is about 10 msec, being varied according to the subjects height.[26] The intensity of recurrent inhibition is normal in parkinsonian rigidity[12] but increased in spasticity.[26]

Reciprocal Inhibition of Ia Afferents

Muscle Ia afferents normally exert a measurable inhibitory influence upon the motoneurons of antagonists.[28] This peripheral Ia reciprocal inhibition is different from central, supraspinal inhibition, but both types converge onto the same spinal Ia inhibitory interneurones.[29,30] In the upper limbs, peripheral inhibition, is assessed by comparing the H reflex recorded from relaxed forearm flexor muscles evoked by median (flexor) nerve stimulation at the elbow with the effect of applying a simultaneous, conditioning, low intensity, stimulation of the radial (extensor) nerve at the spiral groove. Lower limb inhibition is assessed by measuring the decline in the soleus H reflex amplitude induced by simultaneous, threshold stimulation of the peroneal nerve. Unlike the upper limb, inhibition in the lower limb is weak in normal subjects and usually requires voluntary dorsiflexion of the foot before it is seen.[28]

In parkinsonism inhibition of the soleus H reflex is obvious, even at rest, and is more marked with voluntary contraction than in normals.[12,29,30]

The F Wave Response

The F wave reflects recurrent discharge of antidromically activated motoneurons.[31,32] Its shortest latency is a useful measure of conduction through the proximal peripheral nervous system,[32] but other characteristics such as F wave persistence[33-35] and amplitude[36,37] are of particular relevance for evaluating central disorders. For example, increased F wave persistence[34] and amplitude[36-38] has been demonstrated in spasticity. These studies suggest the F wave is a measure of central motoneuron excitability.[31,39] In acute stroke the amplitude of the F wave is decreased.[39,40] Recently, an increased amplitude of the F wave has also been described in parkinsonian rigidity.[12,41] However, before concluding that the F wave amplitude is increased it is essential to be sure that baseline muscle activity was absent or the same as on the other side.

Long-Loop Responses to Limb Perturbations

Long-loop responses are synchronized muscular contractions associated with phasic aspects of movement; acceleration and velocity. The term response rather than reflex is preferred since volitional movement is an essential element when eliciting them.[5] They are recognized by their latencies, which are considerably longer than a spinal reflex, but shorter than a pure voluntary contraction in response to a command.

The subject is instructed to maintain a limb posture to oppose a sudden perturbation (push or pull) delivered via a torque motor. The raw EMG is amplified, averaged and rectified. The stretched muscle reacts with a short latency spinal reflex and medium-latency and long-latency responses followed by voluntary movement. The responses, named M1, M2 and M3,[42] elicited by wrist perturbation, have latencies of about 30 msec (spinal reflex), 50 msec (medium-latency) and 80 msec (long-latency) respectively. M3 is inconsistent and M2 and M3 may not be distinguishable. Long-loop responses elicited by leg perturbation have appropriately longer latencies since the conduction pathway includes much of the spinal cord.[43]

Long-loop responses are unrecordable when a perturbation is applied distal to a spinal lesion indicating the conduction pathway is supraspinal. In hemiplegia M1, the spinal reflex component, is usually enhanced. M2 may be normal, enhanced or absent in the affected arm and leg. In many hemiplegics, late activity is seen occurring at about 100 msec. This is not voluntary since it is seen in a paretic limb, and not clonus because its frequency would be too fast.[44] The dorsal columns, medial lemniscus, sensorimotor cortex and capsular corticomotoneurone pathways are essential for long-loop responses which are attenuated or lost when these are involved selectively.[5,43] In rigid parkinsonians, long-loop responses are enhanced,[42,44] an enhancement that correlates positively with the degree of rigidity.[45]

Short and Long Latency Responses Elicited by Electrical Stimuli

Electrical stimulation of the median nerve at the wrist induces three potentials of increasing latency, recordable from the contracting thenar muscle. The one with the shortest latency is a direct motor response (M-wave). It is followed by two responses, named R1 and R2 by Eisen et al.[46] Their latencies are about 28 msec and 46 msec respectively. R1 has many of the properties of an H reflex.[47,48] Evidence derived from patient studies suggests that R2 is a long-loop response synapsing within the sensorimotor cortex.[49,50] It is likely, but unconfirmed, that R2 is the same response as M2 evoked by perturbation of a limb.[51,52] Short and long latency responses can also be elicited by electrical stimulation of a leg nerve.[46,50] R2 has a longer latency than that obtained with arm stimulation, the difference being in keeping with the time it takes to transit along the spinal cord.

The Blink Reflex

The blink reflex can be elicited by mechanical or electrical stimulation. An ipsilateral early latency response, R1, (less than 12.9 msec) and longer latency bilaterally recorded responses, R2, (less than 43.7 msec) are evoked by supraorbital nerve stimulation.[53] R2 is equivalent to the clinically visible glabellar reflex. Both normally habituate rapidly and failure to do so is characteristic, but not specific of early Parkinson's disease.[54,55] In stroke, especially early on, both early and late components are often absent or reduced in amplitude on the involved side.[56]

Cutaneous Polysynaptic Reflexes

When an electrical stimulus, of intensity sufficient to produce a tactile sensation is applied to the sural nerve, a reflex response of short latency (40-60 msec) is recordable from the biceps femoris muscle. A stronger, painful, stimulus elicits a longer latency response (85-120 msec). If the stimulus is increased further both early and late components are recordable from other flexor muscles of the lower limb, for example the tibialis anterior.[14,57,58] Inhibitory and subliminal excitatory effects may preclude recording cutaneomuscular reflexes from an inactive muscle. This may be overcome by introducing a test activity such as a monosynaptic (H) reflex or tonic voluntary EMG activity.[59]

In spasticity, stimuli that are threshold for the late response, recordable in normal subjects only from the biceps femoris, elicit widespread flexor reflexes.[59] In parkinsonian patients with rigidity the short latency response is enhanced and may become more widespread. However, the longer latency response is lost.[58]

Needle Electromyography

Abnormal muscle tone, although most commonly due to upper motoneuron disease and associated spasticity or extrapyramidal disease associated with rigidity, may also be seen as a manifestation of a variety of conditions having characteristic findings on needle electromyography.

Neuromyotonia (Isaacs' syndrome), is a sporadic disease usually starting with muscle twitching in the legs. As the disease progresses intermittent and then permanent muscle stiffness develops. If untreated, the arms become involved and laryngeal stridor may develop. Tendon reflexes are depressed or absent.[60] Abnormalities are seen on nerve-muscle biopsy affecting predominantly the motor nerve terminal which is considered to be the site of the abnormality responsible for the syndrome.[61] Electromyography shows sustained or repetitive firing of motor units (continuous muscle fiber activity). In addition there are characteristic very high frequency (300 Hz) spontaneous discharges. Decrement in successive potential amplitude produces a unique and pathognomonic sound on the speaker. Many patients respond to phenytoin and carbamazepine.[62] Similar EMG findings are seen in the much rarer congenital Schwartz-Jampel syndrome.[63]

Stiff-man syndrome is another disease characterized by continuous firing of otherwise normal motor units. Clinically there is rigidity initially involving axial musculature and later spreading to the limb girdles. Voluntary movement is difficult or impossible.[64,65]

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