The Use of Manual Muscle Testing to Assess Functional Integration of High-Threshold Versus Low-Threshold Alpha Motor Neurons

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Abstract

- Preloaded muscle tests are sensitive to the integration of low-threshold motor neurons, and by extension the areas of the nervous system that preferentially activate those neurons.
- Post-movement muscle tests are sensitive to the integration of high-threshold motor neurons, and by extension the areas of the nervous system that preferentially activate those neurons.
- Input-output relation of alpha motor neurons, selective activation of low-threshold and high-threshold alpha motor neurons, and functional implications of gamma motor neuron-muscle afferent feedback loops are discussed.

Introduction

Slow-twitch non-fatiguing muscle fibers, and the small, low-threshold alpha motor neurons (AMN) that control them, maintain postural muscle tone. (Powers and Binder, 1999) Small, slowly-firing, low-threshold dynamic gamma motor neurons (GMN) innervate dynamic bag muscle spindle fibers that signal small degrees of stretch through 1A afferents. (Taylor, Ellaway, and Durbaba, 1999) The 1A afferents send their greatest synaptic input back to the low-threshold motor neurons that control postural tone. (Powers and Binder, 1999) Postural muscle tone is modulated primarily from the ipsilateral brainstem, (Reference) which in turn is influenced by projections from the ipsilateral neocortex. Preloaded muscle tests evaluate the function of slow twitch, low-threshold motor units, and by extension the areas of the nervous system that preferentially activate those motor units.

Fast-twitch fatigable muscle fibers and the high-threshold AMNs that control them supply the power for fast, powerful movements. Large, quickly-firing static GMNs innervate static bag and chain muscle spindle fibers (Taylor, Ellaway, and Durbaba, 1999) Static bag and chain fibers contract to maintain spindle sensitivity during movement, and they signal muscle length through group 2 afferents (Taylor, Ellaway, and Durbaba, 1999) Secondary afferents send their greatest synaptic input back to the large AMNs that are primarily involved with fast powerful movements. (Powers and Binder, 1999) Voluntary movements are controlled from the contralateral side of the brain. Post-movement muscle tests evaluate the function of larger, fast-twitch fatigable motor units and by extension the segmental, suprasegmental and interneuronal inputs that preferentially activate those motor units.
Alpha motor neuron physiology

Every part of the nervous system involved in the control of muscle function must do so by acting directly or indirectly on AMNs. Motor neurons are the "final common pathway" in the control of movement and are among the most highly studied neurons in the mammalian central nervous system. (Powers and Binder, 1999) They have large cell bodies that are easily identifiable, their output targets are easy to identify and their output can be measured in a number of ways.

An alpha motor neuron and the motor fibers it innervates are a motor unit. Motor units within each muscle have a wide range of properties. Motor units can be classified as type S (slow twitch, low force, non-fatigable), type FR (fast-twitch, intermediate force, intermediate fatigue time) and type FF (fast twitch, high force and quickly fatigable). (Powers and Binder, 1999) Force output of these motor units varies by 100-fold, fatigue resistance varies by 10-fold, and contraction speed varies by 5-fold. (Binder et al. 1996, 2002)
Postural muscles, non postural, expand motor unit descriptions

Small amns innervate small muscle fibers. Small diameter axons, slow conduction velocity.

The intrinsic properties of the AMNs vary as well. Low-threshold AMNs innervate a relatively fewer number of the smaller, slow twitch muscle fibers. They have smaller cell bodies, thinner axons, and slower conduction times. They have a higher input resistance, and need less synaptic current to initiate an action potential. (The higher the input resistance, the greater the change of voltage elicited by any current that reaches the soma, and the less current is

needed to bring the neuron to threshold.) They have a longer afterhyperpolarization and therefor a slower frequency of firing. (Powers and Binder, 1999)

High-threshold AMNs innervate a relatively larger number of the larger, fast-twitch fatigable muscle fibers. They have larger cell bodies, thicker axons, and faster conduction times. They have a lower input resistance, and need more synaptic current to initiate an action potential. They have a shorter afterhyperpolarization and therefor a higher frequency of firing. (Powers and Binder, 1999)

Alpha Motor Neuron Recruitment Pattern, Early Studies

When the membrane potential of the motor neuron moves from its resting value to a threshold value, the motor neuron fires an action potential and is said to be recruited.

The numbers of motor units that are firing and the rate at which they fire determine the strength of a muscle contraction. The order in which AMNs begin to fire during any given task is called the recruitment pattern. In the 1960s researchers developed the principle of orderly recruitment with a series of studies with anesthetized cats. (Binder et al. 1996, Gandevia, S.C., 2001, Powers and Binder, 1999) AMNs were isolated and currents were injected into the cell body or the dendritic tree, or presynaptic neurons were stimulated. The amount of current reaching the cell body was compared to the rate of action potentials elicited.

Each cell has a unique (and later discovered to be changeable) relationship between the amount of injected or synaptic current and its firing rate. (Binder et al, 1996) It usually takes approximately 1.5 times as much current to elicit a steady repetitive discharge as to elicit one action potential. (Binder et al, 1996) In the absence of modulation described later there is usually a linear relationship between synaptic current and the firing rate of the motor neuron. (Binder et al, 1996)

Early researchers concluded that AMNs are always recruited in order of increasing size, and described this as a process of orderly recruitment. . (Binder et al. 1996, Gandevia, S.C., 2001) For a given amount of current reaching the cell body, smaller AMNs began firing sooner than larger AMNs. Two functional advantages were originally postulated. With orderly recruitment, motor units are recruited in order of increasing fatigability, so that fatigable units are reserved for brief forceful contractions. A second postulated advantage was that the relative precision of force control is the same at all force levels. Each newly recruited unit contributes approximately the same percentage increment to the total force of the muscle at that point in the contraction. . (Binder et al. 1996)

Later Recruitment Studies; Selective Recruitment

Later work with decerebrate, or moving cats demonstrated that the principle of orderly recruitment is a useful, but overly simple model. It does not take into account distribution patterns of synaptic input within the motor neuron pool or modulation of alpha motor neuron function by reticulospinal projections. (Hultborn, 1999, 2002) Motor neurons are not simply recruited in order of increasing size. (Delgado-Lezama and Hounsgaard, 1999, Burke, 2002) AMN recruitment is controlled by pre-synaptic input from both segmental and descending systems, each with a different magnitude and distribution of input to low- and high-threshold motor neurons. Binder, Heckman and Powers (Binder et al. 2002) report that researchers measured the effective synaptic current of six different input systems to the anesthetized cat. Effective synaptic current is defined as the amount of current reaching the soma of the motorneuron during high frequency repetitive stimulation of presynaptic cells. Researchers (Powers and Binder,

1999, Binder 2000) found that:

- Rubrospinal and corticospinal input facilitates high-threshold motor neurons and inhibit low-threshold motor neurons.
- Group 1-a afferent input is larger to low-threshold motor neurons.
- Group 2 afferent input is larger to high-threshold motor neurons.
- Group1-b afferent input inhibits low-threshold motor neurons, but facilitates high-threshold motor neurons.
- Recurrent inhibition from Renshaw cells and reciprocal 1A inhibition is distributed uniformly between low- and high-threshold motor neurons.
- Input from the lateral vestibular nuclei preferentially stimulates high-threshold motor neurons.

Synaptic Integration; Drive and Modulation

There are two major types of synaptic influence on motor neuron discharge, drive and modulation (Kernell, 2002, Powers and Binder, 1999). Driving currents are provided by input from fast-acting ionotropic synapses that summate spatially and temporally in the cell soma. Driving current does not change the neuron's intrinsic properties of input resistance and duration of afterhyperpolarization, and driving currents from different sources summate in a close to linear fashion. (If distributed evenly within the motor neuron pool, driving current produces an orderly recruitment)

Modulating synapses alter the neuron's intrinsic properties. Neuromodulators acting through metabotropic receptors can increase membrane resistance by 50% and shorten the duration of afterhyperpolarization allowing an increased frequency of firing. They alter both sub-threshold and supra-threshold behaviors of the AMNs. Modulating synapses alter the neuron response to driving currents. For example, 1-a afferent input elicits very different responses in the cord depending on the presence of monoamines. The same amplitude of 1-a input generates 2-3 nA of effective synaptic current in anesthetized animals and 12-17 nA of synaptic current in decerebrate animals. (Heckman And Lee, 2001)

One of the primary ways that modulators create changes in the target neurons is by triggering persistent inward currents (PICs) (Heckman And Lee, 2001, Hochman et al. 2001, Alaburda et al., 2002, Kernell, 2002). PICs depolarize the cell and create a long-lasting change in membrane potential known as a plateau potential. The plateau potential makes the cell easier to activate, and it sometimes generates self-sustaining firing in the absence of continued synaptic input. The PIC and its associated plateau potential last from a second or so to more than a minute. Low-threshold AMNs generate stronger, longer-lasting PICs than high-threshold AMNs. Some plateau potentials decay spontaneously after a period of time and some are stopped by a burst of inhibitory input.

Motor neurons often exhibit bistable behavior in which brief periods of excitation and inhibition can toggle self-sustained firing on and off (Heckman and Lee, 1999, Collins et al, 2002). Low-threshold AMNs are fully bistable. They can produce self-sustaining firing for many seconds up to a minute. High-threshold AMNs are partially bistable. They can generate at most 1-2 seconds of self-sustaining firing.

Preloaded Muscle Tests

Preloaded muscle tests are performed with 2 seconds of light pressure to elicit an isometric contraction prior to applying the test. This test procedure frequently demonstrates a weak muscle when non-preloaded tests do not. The test is more sensitive if the examiner uses light pressure rather than moderate or heavy pre-loading pressure. Both preloaded and non-preloaded muscle tests require the muscle being tested to resist a quick increase of force attempting to lengthen the muscle. The test causes quick stretch, a postural perturbation that is signaled to the nervous system by primary afferents that activate primarily low-threshold AMNs.

What might cause a muscle to weaken in a preloaded context and be strong without the preload? Isometric contractions cause greater 1-B inhibitory impact on low-threshold than on high-threshold motor neurons. In the case of reduced descending facilitation from the brainstem, the preload inhibitory input is enough to cause failure of the low-threshold activation that is necessary to resist the test pressure. The lower the preload pressure the lower the percentage of high-threshold AMNs recruited to resist the preload pressure, and the lower the percentage of high-threshold AMNs available to resist the final test pressure. (Increased preload pressure activates a larger percentage of high-threshold AMNs which are then available to resist the final test pressure)

To summarize, descending monoaminergic projections activate persistent inward currents in the dendrites of motor neurons, which in turn contribute tonic activation to low-threshold AMNs and GMNs and regulate postural muscle tone. Preloaded muscle tests are sensitive to the integration of low-threshold motor neurons, and by extension the areas of the nervous system that preferentially activate those neurons.

Decreased output of brainstem monoaminergic projections (often secondary to decreased neocortical output) results in characteristic postural changes. Decreased neocortical output typically causes decreased tone of posterior compartment muscles above T6 (arm extensors and external rotators) and anterior compartment muscles below T6 (leg flexors, leg adductors, foot dorsiflexors, and internal rotators). This pattern is sometimes called a soft pyramidal pattern of weakness because it is associated with normal or decreased tendon reflexes and is caused by loss of modulation from the ipsilateral brain stem. Soft pyramidal muscle weakness is more frequently observed with preloaded muscle tests than with other types of muscle tests.

Post-Movement Muscle Tests

Sometimes a muscle is strong in a test situation that does not involve an immediate prior movement, but fails to resist test pressure after it has been lengthened or shortened. Post-movement muscle tests are sensitive to the condition of high-threshold AMNs and GMNs and by extension, the segmental and suprasegmental inputs acting on them.

Fast-twitch fatigable muscle fibers and the high-threshold AMNs that control them supply the power for fast, powerful movements. Large, quickly-firing static GMNs innervate static bag and chain muscle spindle fibers (Taylor, Ellaway, and Durbaba, 1999) Static bag and chain fibers contract to maintain spindle sensitivity during movement, and they signal muscle length through group 2 afferents (Taylor, Ellaway, and Durbaba, 1999) Secondary afferents send their greatest synaptic input back to the large AMNs that are primarily involved with fast powerful movements. (Powers and Binder, 1999) Voluntary movements are controlled from the contralateral side of the brain. Post-movement muscle tests evaluate the function of larger, fast-twitch fatigable motor units and by extension the segmental, suprasegmental and interneuronal inputs that preferentially activate those motor units.

Muscle Spindle Cells

Muscle spindle cells provide information to the central nervous system about muscle length and changes of muscle length. There are three important functional parts of the muscle spindle cell; the intrafusal fibers, the GMNs that innervate the contractile polar sections of the intrafusal fibers, and the sensory receptors that originate in the central non-contractile parts of the intrafusal fibers. (Pearson and Gordon, 2000)

Muscle spindle cells contain three types of intrafusal muscle fibers (dynamic bag, static bag, and chain) enclosed within a spindle-like (or fusiform) fluid-filled connective tissue capsule situated in parallel with the longer, stronger extrafusal muscle fibers that do the work of the muscle. The length of the spindle and the length of the intrafusal fibers within the spindle changes in parallel with the length of the extrafusal fibers. Each intrafusal fiber has a contractile portion and a non-contractile sensory portion. The sensory portion increases its firing rate when it is stretched. Tension in the contractile portion of the intrafusal fiber effects the degree of stretch in the sensory portion of the fiber. The sensory portion of the spindle is stretched when the entire spindle lengthens and when the contractile portion of the intrafusal fiber contracts causing stretch of the non-contractile sensory portion. (Pearson and Gordon, 2000)

Gamma Motor Neurons

The motor innervation of the intrafusal fibers comes from small motor neurons called gamma motor neurons to distinguish them from larger AMNs that innervate extrafusal fibers. Like AMNs, GMNs show variation in size, frequency of firing, presynaptic pools, and output targets. Traditionally two types of GMNs have been recognized, static and dynamic. Recently it is clear that there are two types of static GMNs. (Taylor et al. 1999)

Dynamic GMNs are smaller, and have a lower frequency of firing than static GMNs. They innervate the slow, weakly contracting dynamic bag fibers. The contractile portion of the dynamic bag does not contract. It simply stiffens. This increases the sensitivity of the dynamic bag to stretch. The sensory portion of the dynamic bag activates primary afferents that have an increased firing rate during the first few degrees of muscle stretch. (Taylor et al. 1999)
Static GMNs are larger and have a faster frequency of firing. They innervate static bag and chain fibers. Static GMNs can activate static bag or chain fibers independently, or both together. Different populations of static GMNs are active at different parts of a muscle movement cycle. (Taylor et al. 1999)
Intrafusal Fibers

Each intrafusal muscle fiber has a characteristic type of contraction. (Taylor et al. 1999)

Dynamic bag fibers have weak, slow contractions. They have a slow frequency of firing, and a slow tetanic fusion frequency of less than 30 Hz. They do not actually shorten, but they stiffen, making the contractile polar portion of the dynamic bag more resistant to stretch so that the middle sensory portion of the dynamic bag stretches more with each degree of spindle stretch. This causes a higher rate of firing for the primary afferent with its annulospiral ending on the dynamic bag. (Taylor et al. 1999)

Static bag fibers have a higher frequency of firing than dynamic bag fibers, and less than chain fibers. Static bag tetanic fusion frequency is about 30 Hz. They do contract and can change the bias (tonic firing rate) of the primary

and secondary afferents that have sensory endings on the static bag. They do not contract quickly enough to prevent unloading of the sensory portion of the bag while the muscle is shortening. Static bag fibers do shorten enough to cause resumed activation of the primary and secondary afferents that have sensory endings on the static bag once the muscle is resting in the new shortened position. (Taylor et al. 1999)

Chain fibers contract quickly and powerfully. Their tetanic fusion frequency is greater than 100 Hz. They contract quickly enough to prevent unloading of the sensory portion during muscle shortening, but as they fire at unfused tetanic frequencies they drive the primary afferents with annulospiral endings on the chain fibers causing a distorted stretch response in the primary afferents. (Taylor et al. 1999)

To summarize, dynamic bag fibers (innervated by dynamic GMNs) control the stretch sensitivity of primary afferents. The primary afferent stretch response is phasic and does not last more than a few milliseconds. Because the dynamic bag fibers contract slowly they do not maintain the stretch sensitivity once the muscle has moved. The static bag sets the bias or tonic-firing rate for both primary and secondary afferents. It does not contract quickly enough to prevent unloading during shortening. Those static GMNs that innervate static bag fibers alone are best suited to set the bias of both primary and secondary afferents. Static bags and chains together (and the static GMNs that innervate them) are best suited to maintain spindle sensitivity during muscle shortening.

Muscle Afferents

The central portion of each intrafusal fiber contains a non-contractile sensory section that activates primary or secondary afferents. There are two types of sensory endings in the muscle spindle. Primary endings (annulospiral) are located in the central portion of dynamic bag, static bag, and chain fibers. They activate large fast primary (group 1a) afferents. They signal rapid changes of length, and to a lesser extent resting length of the muscle. (Pearson and Gordon, 2000)

Primary afferents have two sensory branches; one to the annulospiral ending on the dynamic bag and the other to annulospiral endings on static bag and chain fibers. The frequency of the primary afferent matches the rate of the sensory branch with the highest rate of firing. This changes at different stages of a movement cycle. Primary afferents excite low-threshold AMNs more than high-threshold AMNs and they have relatively little input to GMNs (Taylor, 2002, Banks et al, 1997)

Secondary endings (flower spray) are located in the central portion of static bag and chain fibers. They activate group 2 (secondary) afferents. Secondary afferents have one or more sensory branches with endings on static bag, chain fibers or both. They signal muscle length and the degree of intrafusal fiber contraction (spindle bias). The sensory branch (from the static bag or the chain) with the highest firing rate determines the firing rate of the secondary afferent. This changes in different phases of a movement cycle. (Taylor, 2002, Taylor et al. 1999)

Group 2 afferents have disynaptic, trisynaptic and polysynaptic input to AMNs and GMNs, and as recently suggested, some monosynaptic input to AMNs and GMNs as well. (*****) Secondary afferents have greater input to high-threshold AMNs than low-threshold AMNs (Powers and Binder, 1999). The effect of secondary afferents on GMNs is strongly influenced by monoaminergic modulation from the brainstem as described below.

Alpha-gamma co-activation

When a muscle shortens the muscle spindle cells and the intrafusal fibers within the spindle shorten as well. This tends to unload the central sensory part of the spindle and reduce afferent input to the cord. To counteract this tendency, static GMNs cause contraction of static bag and chain fibers to maintain tension in the central sensory portions of the intrafusal fibers. (Taylor et al, 1999)

Voluntary movement initiated through corticospinal and rubrospinal pathways activates AMNs and GMNs. The pathways controlling voluntary movement are distributed preferentially to high-threshold AMNs (Powers and Binder, 1999, Binder 2000) and to static GMNs. Static GMNs are activated slightly out of phase to high-threshold AMNs. (Ellaway et al, 2002)

Cord Integration of Group 2 Afferent Input

Secondary afferents send collaterals to a large number of segmental and suprasegmental destinations. Signal transmission to each destination is independently modulated by presynaptic inhibition, and by excitatory and inhibitory input to postsynaptic neurons. (Stuart, 2002). Information is routed to one destination or another, a process that is strongly influenced by monoaminergic modulation from the brainstem. (Stuart, 2002)
Spinal interneurons are modulated and interneuronal pathways are reorganized during voluntary movements. Spinal reflexes are adjusted to assist rather than counteract the execution of planned movements (Jankowska and Hammar, 2002). The same stimulus evokes different responses in different situations, including different phases of a movement cycle such as locomotion. (Stuart, 2002)

Group 2 afferents project to the dorsal horn lamina 4, the intermediate zone lamina 5 and 6 and the ventral horn lamina 7 and 8 (Jankowska et al. 2002) (Jankowska and Hammar, 2002). Ventral horn targets include AMNs and GMNs. Intermediate zone targets include interneurons that are immediately premotor to AMNs and GMNs. Secondary afferent projections to both intermediate and ventral horn targets are subject to presynaptic inhibition from GABAergic dorsal horn interneurons (Jankowska et al. 2002).

Serotonin and Norepinephrine Modulate Afferent Input

Serotonin and norepinephrine have several opposing actions in the cord. Serotonin facilitates mono and di-synaptic pathways from secondary afferents through the intermediate and ventral horn to motor neurons. Norepinephrine facilitates polysynaptic pathways with integration in the dorsal horn en route to intermediate and ventral horn targets (Jankowska et al. 2002).

Both serotonin and norepinephrine have direct excitatory action on AMNs but they have opposite effects on secondary afferent input to GMNs. (Jankowska et al. 2002).

Serotonin enhances secondary afferent input to GMNs. It does this with a combination of direct excitatory action on GMNs, facilitation of intermediate zone interneurons that are active in di-and tri-synaptic pathways, and inhibition of dorsal horn GABAergic interneurons that presynaptically inhibit secondary afferent terminal projections to the intermediate and ventral horn sites (Jankowska et al. 2002).

Norepinephrine depresses secondary afferent input to GMNs. It does this with a combination of direct inhibitory action on GMNs and inhibitory action on intermediate zone interneurons that are active in di-synaptic pathways.
Positive Feedback Loop between Secondary Afferents and Gamma Motor Neurons

Activity of static GMNs and secondary afferents forms a positive feedback loop that can be modulated at the step of afferent activation of static GMNs (Jankowska and Gladden, 1999). Increased gamma motor neuron activity causes more afferent activity. This is a potent effect. Increased afferent activity causes gamma motor neuron activation. This is a weaker effect and it is modulated by the ratio of norepinephrine and serotonin in the cord (Jankowska and Gladden, 1999) Serotonin enhances the positive feedback loop and norepinephrine inhibits the loop.

Serotonin and Muscle Tone

Serotonin and its receptors are distributed throughout the CNS. There are billions of cells in the central nervous system but only 10-20,000 that produce serotonin. Cells that produce serotonin are all located in the brainstem from the medulla to the mesencephalon (Jacobs et al, 2002).

A rostral group of serotonergic nuclei project to the cortex, and a caudal medullary group project to the spinal cord. Jacobs and his research group (Jacobs et al, 2002) implanted micrometers in order to study the function of serotonin in behaving cats. He reports that the primary role of the medullary serotonergic system appears to be increasing motor tone and facilitating repetitive motor activity. Serotonin inhibits nociception and increases sympathetic nervous system activity in order to support its primary function of increased motor tone. Serotonin levels decrease during sleep, and fall to zero during periods of REM sleep, when muscle tone is profoundly reduced Serotonergic neurons in the medulla are sensitive to carbon dioxide and/or pH. (Jacobs et al, 2002) Increased carbon dioxide (3%, which is fairly sensitive) causes increased serotonergic activity and increased breathing. Decreased levels of carbon dioxide cause decreased serotonergic activity in medullary neurons that project to the spinal cord. There does not appear to be phasic activity of serotonergic neurons related to the respiratory cycle.

Respiration and Muscle Tone

If the patient takes six or seven deep rapid breaths, they temporarily decrease the levels of carbon dioxide in the blood stream, and often show widespread preloaded muscle test weakness (described previously) and post-shortening muscle test weakness (described later). If the patient holds their breath for ten seconds they temporarily increase the levels of carbon dioxide in their blood stream, and increase the levels of serotonergic activity in the cord, and they often show widespread weakness after lengthening the muscle to be tested.
Low CO_2 tends to cause decreased activation of AMNs as evidenced with preloaded muscle test weakness and post-shortening muscle test weakness, but it has the opposite effect on other neurons. Hyperventilation tends to cause increased neuronal excitability. Low CO_2 causes an increase in pH, which causes decreased free calcium because of increased association with binding protein. Low calcium causes voltage gated sodium channels to open with lower levels of activation and nerves become more excitable.

Post-Movement Muscle Tests

Post-movement strength tests are performed after the muscle has been lengthened or shortened through at least a quarter of its range of motion. The patient is asked to exert a strong force. The examiner resists and then adds additional test pressure in the direction of lengthening the muscle. The examiner assesses the patient's ability to resist the added pressure.

The post-movement test conditions cause increased corticospinal and rubrospinal drive to the ventral horn. Corticospinal and rubrospinal pathways excite high-threshold AMNs and static GMNs and inhibit low-threshold AMNs and dynamic GMNs.

If high-threshold AMNs do not have adequate stimulation from corticospinal and rubrospinal projections the patient is unable to resist the added test pressure and the muscle lengthens. (This is true whether the patient is initiating a high force test post-movement or not)

If high-threshold neurons do not have adequate stimulation from secondary afferents, the patient is unable to resist the added test pressure and the muscle lengthens. (The larger the movement, the more important the effect of secondary afferent integration on muscle function) Two conditions can cause decreased stimulation of high-threshold AMNs from secondary afferents.

- Decreased drive to the GMNs from corticospinal and rubrospinal projections causes decreased secondary afferent activation.
- An increased ratio of norepinephrine to serotonin in the cord directly inhibits GMNs and facilitates presynaptic inhibition of secondary afferent input to AMNs and GMNs.

Shortening Versus Lengthening Pre-Test Movements

As a muscle is shortened gamma drive is initiated to maintain an appropriate bias in the muscle spindle. Inadequate gamma drive results in off-loading of the spindle and reduced afferent input to the cord. The cord reflexively acts as if the muscle is shorter than it actually is and stops trying to shorten it. Inadequate gamma drive results in post-movement weakening after the muscle being tested is shortened.

Excessive gamma drive causes excessive bias in static bag fibers and chain fibers of the muscle which is shortening (the antagonist of the muscle which is lengthening), and increased secondary afferent input to the cord. The cord reflexively acts as if the muscle being shortened is longer than it actually is and reflexively tries to get it to contract more. This causes reciprocal inhibition of the muscle that is lengthening, the muscle that will be tested. Excessive gamma drive results in post-movement weakening after the muscle being tested is lengthened.

Clinical correlations

The preloaded muscle test is an effective way to evaluate postural influences on muscle tone. This test is sensitive to the integration of low-threshold motor neurons, and by extension the areas of the nervous system that preferentially activate those neurons.

The post-movement test is an effective way to evaluate the body's ability to effectively generate and control movement, and to evaluate corticospinal and rubrospinal influences on muscle tone.

Many patients have pre-loaded weakness, often in a soft pyramidal distribution, on one side of the body, and post-shortening muscle test weakness on the other side of the body. The side of pre-loaded muscle test weakness correlates with signs of decreased hemispheric function such as sluggish pupil constriction, palatal paresis, increased ratio of retinal vein-to-artery size, and increased blood pressure.

Patients with abnormally increased muscle tone driven by the contralateral side of the brain typically have post-lengthening muscle test weakness, especially in flexors. Post-lengthening weakness correlates with signs of increased contralateral mesencephalic activity such as brisk, slowly fatiguing pupil contractions.

Conditions of transneural degeneration complicate the picture. Refer to the article by Walter Schmitt for a review of transneural degeneration (Schmitt, 1999). When an area of the nervous system is in the initial stages of degeneration it frequently shifts closer to its threshold of activation and it fires spontaneously or in response to trivial stimulation. It activates easily, but it fatigues quickly.

Many patients have transneural degeneration of the Purkinje cell system of the cerebellum. When the purkinje system is in the early stages of degeneration vestibular stimulation such as a few bounces on a mini-trampoline, or closing the eyes and moving the head back and forth often causes a temporary right to left reversal of one of the patterns described above. Treatment should be appropriate for the condition.

Summary

Muscles serve both postural and movement-generating functions. They have different fiber types and different controlling systems to accomplish those functions. Pre-loaded muscle tests and post-movement muscle tests are used to assess these functions and the areas of the nervous system that control them.

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