The Brainstem and Manual Muscle testing

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Abstract

This article offers a brief review muscle physiology, spinal cord function, and the modulating effects of norepinephrine (NE) and serotonin (5HT) on muscle function, all with an emphasis on factors that effect muscle test outcome. It describes five manual muscle test procedures that are used to evaluate brainstem function, the neuro-physiological relevance of each procedure, and expected muscle test outcomes in response to physiological brainstem stimulation. It proposes that in the context of a full neurological exam, specific muscle test procedures can be used as a sensitive, easily administered diagnostic tool for the evaluation of brainstem function.

Key words: reticular formation, reticular activating system, norepinephrine, serotonin, manual muscle testing

Introduction

Each muscle participates in a number of functions. Shoulder muscles participate in posture and locomotion, reaching, throwing, and lifting. Each function requires the activation of specific combinations of motor neurons in the spinal cord and each function is controlled from different areas of the brain. Muscle tests can be designed to evaluate specific motor functions and by extension the areas of the brain that control those functions. This article describes five manual muscle test procedures that are used to evaluate brainstem function, the neuro-physiological relevance of each procedure, and expected muscle test outcomes in response to physiological brainstem stimulation.

Section 1: Motor Control

Overview

The spinal cord and brain coordinate all of our movement, voluntary and automatic. The cerebral cortex initiates and sets the direction for voluntary movements. Cortical motor areas connect with a variety of neurons in the brainstem that in turn project to the spinal cord. Cortical motor areas also project to the spinal cord where they connect with interneurons, which in turn project to the alpha motor neurons (AMNs) that activate muscle fibers. Cortical motor areas have very few direct connections to the AMNs that activate muscle cells, and the direct connections that do occur are mainly with AMNs that innervate hand muscles.

The cerebral cortex delegates the detailed work of coordinating muscle movement to groups of cells in the brainstem and spinal cord. Commands from the cerebral cortex and other areas of the brain activate specific areas of the brainstem, which in turn activate pattern-generating neurons in the spinal cord. . The brainstem coordinates automatic and repetitive movements, and muscle tone. (1) Mammals that have had their forebrains removed can be made to walk, trot and gallop by stimulating different locomotor regions in the brainstem. The movements are well coordinated and the animal is able to maintain largely adequate balance. (2)

Muscle Physiology

Movement and posture is accomplished by muscle activity. Each muscle contains hundreds of thousands to millions of independent, contractile units called muscle fibers. There are three types of muscle fibers, slow-twitch fibers (S), fast-twitch fatigable fibers (FF), and fast-twitch fatigue-resistant fibers (FR). Type-S fibers are smaller, and type-FF are larger. Fibers of the same type are grouped together as motor units. Each motor unit is controlled by a single motor neuron in the spinal cord or brainstem.

Motor units within each muscle have a wide range of properties. Type-FF motor units can generate 100 times the force of type-S motor units and they can contract five times as fast. On the other hand type-S motor units have ten times the endurance of type-FF motor units. (3)

Each muscle contains a mixture of fiber types. Proximal, postural muscles have a higher percentage of slow-twitch fibers, and distal hand muscles have a higher percentage of fast-twitch fibers. Routine posture and locomotion is accomplished almost entirely by slow-twitch fibers. Fast, explosive forceful movements of proximal muscles engage a higher percentage of fast-twitch fibers as do movements of the eyes and fingers.

Alpha motor neurons (AMNs)

Every part of the nervous system involved in the control of muscle function must do so by acting directly or indirectly on AMNs. AMNs innervate the extrafusal muscle fibers that do the work of the motor system. AMNs vary in size, physiological properties, and function. (3,4,5,6,7) Small AMNs and their associated type S motor units are most active in tonic postural activities. Large AMNs and the associated type FF motor units are most active in fast forceful motions. A higher percentage of small AMNs innervate postural muscles, and a higher percentage of large AMNs innervate finger muscles.

Small AMNs are more excitable; they have a lower threshold of activation. They have a lower frequency of firing, thinner axons and slower conduction speeds. They innervate as few as 100 small, slow-twitch muscle fibers. Large AMNs are less excitable; they have a higher threshold of activation. They have a higher frequency of firing, large diameter axons and faster conduction speeds. They innervate as many as 1000 large, fast-twitch muscle fibers. Large and small AMNs have different properties, different functions, and they are activated from different parts of the nervous system. Many parts of the nervous system preferentially activate one group or the other, often facilitating one and inhibiting the other. (3,4,5,6,7)

- Rubrospinal and corticospinal input facilitates small AMNs and inhibits small AMNs.
- Group 1-a afferent input projects primarily to small AMNs.
- Group 2 afferent input projects primarily to large AMNs (as well as gamma motor neurons GMNs as discussed later)
- Group1-b afferent input inhibits small AMNs and facilitates large AMNs.
- Recurrent inhibition from Renshaw cells and reciprocal 1A inhibition is distributed uniformly between small and large AMNs.
- Input from the lateral vestibular nuclei preferentially stimulates large AMNs.

Plateau Potentials (PP)

Until the 1960's it was thought that the membranes of motor neurons were passive and the amount of synaptic input to the cell correlated directly with the output from the cell. In the 1970's researchers discovered that motor neurons could exhibit periods of sustained firing that were relatively independent of the amount of synaptic input. It was discovered that this sustained firing is due to persistent inward currents that were large enough to create a sustained shift in the membrane potential, called a plateau potential (PP) (6,7,8)

In the 1980's it was discovered that these phenomena are dependent upon projections of NE and 5HT from the brainstem. 5HT and NE facilitate AMNs by creating persistent inward currents in the cell membrane that create a long lasting (up to a minute) increase in the membrane potential called a plateau potential. This increases the cell's excitability so that the same amount of synaptic input creates up to six times greater output from the cell. (6) PPs are most likely to occur in small AMNs and least likely to occur in large AMNs. PPs last for up to a minute for small AMNs, but only 1-2 seconds for large AMNs (3,5,6) This is discussed more in a later section of this article about pre-loaded muscle tests.

Gamma Motor Neurons (GMN) and Muscle Spindle Cells (MSC)

GMNs innervate the intrafusal muscle fibers in muscle spindle cells that keep stretch receptors at a relatively constant length as the muscle changes length. MSCs contain stretch receptors that signal how fast the muscle is changing position (phasic signal) and how much it has changed position (tonic signal). MSCs are embedded in muscles. The ends of the MSC are firmly attached to the connective tissue matrix of the muscle and the length of MSCs changes in tandem with the length of the muscle. The sensory stretch receptors are suspended between the ends of the muscle spindle cell by intrafusal muscle fibers. As the muscle changes length, intrafusal fibers contract and relax in order to keep the stretch receptors at a relatively constant length. If the stretch receptors are overly slack or overly tensed they will send inappropriate signals to the central nervous system. GMNs regulate the

strength of the stretch reflex by effecting the sensitivity of stretch receptors, which in turn determine the strength of the stretch signal that travels back to the spinal cord. (7,9,10)

GMNs are activated independently of AMNs. GMNs receive input about muscle length from group-2 afferents, propriospinal networks and a variety of suprasegmental sources. (11,12)

5HT and NE provide strong (and opposite) modulation to the amount of group-2 feedback to GMNs. (13,14) This is discussed more in a later section of this article about post-movement muscle tests.

Brainstem Reticular Formation and Muscle Tone

The brainstem consists of the medulla, pons, and mesencephalon. It controls many important functions in the body including respiration, cardiovascular function, gastrointestinal function, equilibrium, posture, and automatic, stereotyped movements of the body.

The reticular formation and the vestibular nuclei located in the brainstem play an especially large role in the control of posture. Reticulospinal pathways project primarily to small AMNs, and vestibulospinal pathways have a larger effect on large AMNs. This article discusses reticulospinal control of posture. A future article will discuss vestibulospinal influence on posture and muscle function.

The reticular nuclei are divided into two major groups; the pontine reticular nuclei that extend throughout the pons and into the mesencephalon, and the medullary reticular nuclei that extend the length of the medulla. These two sets of nuclei function largely antagonistically to each other. The pontine reticular formation excites extensor antigravity muscles and the medullary reticular formation inhibits them. They transmit signals to the spinal cord through medial and lateral reticulospinal tracts respectively. (15)

The pontine reticular nuclei receive especially strong input from vestibular nuclei. The medullary reticular nuclei receive especially strong input from rubrospinal, corticospinal and other higher brain centers. When the brain stem is severed between the pons and mesencephalon (leaving the pontine and medullary reticular systems as well as the vestibular system intact) the animal develops decerebrate, or extensor rigidity. This occurs because the medullary reticular formation fails to inhibit extensor muscles when it is deprived of its normal excitatory input from the cerebral cortex, red nucleus, and basal ganglia. Rigidity occurs primarily in antigravity muscles of the neck, trunk and the extensor muscles of the legs. (15)

In simple terms the pons facilitates extension and the medulla facilitates flexion, and these effects are strongest in muscles of the neck, back and legs. When a person is standing, tonic activation of the pons facilitates extensor antigravity muscles. When the person begins to walk, signals from higher brain centers activate specific parts of the medulla to inhibit extensor muscles and allow the swing phase of gait. (1)

Reticular Activating System and Muscle Tone

The reticular-activating system (RAS) acts through reticulospinal projections to modulate posture, muscle tone, and locomotion. It is comprised of three primary cell groups in the mesopontine tegmentum; the pedunculopontine nucleus that secretes acetylcholine (ACH), the locus coeruleus that secretes NE, and the raphe nuclei that secrete 5HT. These three groups of neurons each project to widespread areas of the nervous system where they have modulatory effects. (16,17,18)

The three groups of neurons which make up the RAS interact with each other, facilitating or inhibiting each other as they project to their respective targets to control the sleep-wake cycle and arousal, modulate the fight or flight response, and regulate posture and locomotion. It is not surprising that the RAS is linked to the motor system in order to optimize attack or escape. During deep sleep and REM sleep the RAS causes a loss of muscle tone so that we don't act out our dreams. During waking the RAS modulates muscle tone and locomotion via the reticulospinal tracts. (16,17,18,19)

This article will focus on NE and 5HT projections from the RAS, and their effect on muscle test outcomes.

NE and Muscle Tone

NE modulates the function of many diverse parts of the nervous system. Neurons containing NE have the widest divergence and project to more parts of the nervous system than any other neurons in the brain. (20) All of the NE in the central nervous system is produced in the brainstem, most of it in the locus coeruleus (LC).
The LC neurons lie in a cluster in the periventricular gray area in the dorsolateral corner of the fourth ventricle in the rostral pons. It is named for the bluish color that is cast from its melanin content onto the floor of the fourth ventricle. The LC is located immediately medial to the medial vestibular nucleus and to the caudal end of the mesencephalic trigeminal nucleus.

The pattern of distribution and the types of contact that projections from the LC make with other neurons is similar to the pattern of distribution and the types of contacts that are made by peripheral sympathetic neurons. The proximity and close association of the LC with the mesencephalic trigeminal nucleus suggests that it, like the mesencephalic trigeminal nucleus, developed from neural crest cells and migrated into the central nervous system to form the equivalent of a widely divergent peripheral sympathetic ganglion. (20)

The close association of the LC and mesencephalic trigeminal nucleus is the basis of a physiological brainstem challenge discussed in a later section of this article.

The LC projects ipsilaterally to effect multiple areas in the spinal cord where NE has the following actions.

- Facilitates small AMNs (3,5,6)
- Inhibits renshaw cells (disinhibits AMNs) (21,22)
- Dampens stretch reflexes (23)
- Strongly depresses synaptic actions of group-2 muscle afferents (which monitor muscle position) on GMNs and on intermediate zone neurons that provide input to AMNs and GMNs. (13)

The LC exerts prominent facilitory influence on posture by directly exciting AMNs for extensor (and flexor) muscles and by releasing those AMNs from recurrent renshaw cell inhibition. (21,22)

NE has its greatest effects on proximal extensor, abductor, and external rotator muscles, especially in the legs. (See the discussion in Brainstem Reticular Formation and Muscle tone above.)

5HT and Muscle Tone

Cells that produce 5HT are all located in raphe nuclei in the midline of the brainstem from the medulla to the mesencephalon. They project extensively through the nervous system. Raphe nuclei in the pons and mesencephalon project rostrally, and those in the medulla project to the spinal cord. Jacobs and his research group (24) implanted micrometers in order to study the function of 5HT 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. 5HT inhibits nociception and increases sympathetic nervous system activity in order to support its primary function of increased motor tone. 5HT levels decrease during sleep, and fall to zero during periods of REM sleep, when muscle tone is profoundly reduced. (24,25)

5HT has its greatest effects on proximal flexor, adductor, and internal rotator muscles, especially in the legs. (See the discussion in Brainstem Reticular Formation and Muscle tone above.) Medullary raphe nuclei project bilaterally to the spinal cord where 5HT has the following effects.

- Facilitates small low threshold AMNs (3,5,6)
- Facilitates gamma motor neurons, GMNs, and thereby enhances stretch reflexes.
- Strongly facilitates synaptic actions of group 2 afferents on GMNs and on intermediate zone neurons that provide input to AMNs and GMNs. (12,13)

Serotonergic neurons in the medulla are sensitive to carbon dioxide and/or pH. 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. (24)

Summary: Contrasting effects of 5HT and NE on Muscle Function

- 5HT and NE each increase muscle tone of both flexor and extensor muscles. As a pontine nucleus the LC and its adrenergic projections (NE) have a greater effect on extensors than on flexors. Serotonergic projections from the raphe nuclei in the medulla have a greater effect on flexors than extensors.

• 5HT enhances feedback from group-2 muscle afferents to GMNs and to the intermediate zone neurons that provide input to GMNs. NE depresses group-2 muscle afferent feedback to those same targets.

• 5HT and NE have opposite effects on GMNs and the stretch reflex. 5HT weakly facilitates the muscle stretch reflex and NE strongly dampens the stretch reflex.

Spinal cord functions that effect muscle tests

Stretch reflex

Abrupt non-volitional lengthening of a muscle elicits a stretch reflex that causes the stretched muscle to contract and resist lengthening. The reflex is a postural mechanism to prevent unintended perturbations of a desired posture. It is accomplished by the activation of dynamic bag stretch receptors in MSCs. The receptors send signals to the cord through 1A afferents that have direct monosynaptic input to small AMNs that maintain posture. (Group 2 afferents also contribute to the stretch reflex in a lesser extent.)

In response to the stretch signal from 1A afferents, AMNs in the spinal cord increase their rate of firing to activate the muscle and resist lengthening. This process is not under voluntary control. An inadequate stretch reflex causes muscle tests that involve an abrupt increase of force to fail.

The effectiveness of the stretch reflex depends upon

1. The sensitivity of the dynamic bag stretch receptors which is in turn dependent upon activation from dynamic GMNs in the ventral horn of the spinal cord.
2. The ability of the AMNs to respond to excitatory input.

NE and 5HT modulate both factors determining the efficacy of the stretch reflex.

1. NE facilitates AMNs and inhibits GMNs. It has a strong net damping effect on the stretch reflex. (23) (NE agonist medications are helpful in reducing exaggerated stretch reflexes and spasticity after spinal cord injuries.)
2. 5HT provides weak facilitation for stretch reflexes.

Group-2 afferent input to the cord

As a muscle changes length the nervous system is challenged to maintain adequate feedback from the stretch receptors that are embedded in the muscle. Muscle spindle cells are embedded in the muscle, and stretch receptors are embedded in the muscle spindle. MSCs change length when the muscle changes length. Muscle fibers within the MSC contract and relax to maintain a relatively constant length in the receptor portion of the spindle as the muscle and the muscle spindle gets shorter or longer. This is coordinated by GMNs in the spinal cord. (7,9,10)

GMNs activate intrafusal muscle fibers (inside the muscle spindle cells) to maintain optimal length of the receptor as the muscle changes length. GMNs are activated (independently of AMNs that innervate the extrafusal muscle fibers) by descending commands from the brainstem and cerebral cortex, from spinal propriospinal networks, and by feedback from group-2 muscle afferents.

Group-2 afferents convey signals from receptors (static bag and chain fibers in muscle spindle cells) that register muscle length. Group-2 afferents project to numerous targets in the spinal cord including neurons in the dorsal horn, intermediate zone and GMNs in the ventral horn. GMNs use information from group-2 afferents to calculate appropriate drive to intrafusal fibers and thereby maintain optimal spindle sensitivity.

5HT and NE modulate the input from group-2 afferents to spinal cord targets. 5HT facilitates the transmission of information from group-2 afferents to GMNs and NE depresses that transmission. (13,14)

Modulation from NE and 5HT allows the brainstem to reconfigure spinal circuits and spinal reflexes on a second by second basis to meet the needs of different movements, and appropriate modulation is necessary in order to maintain spindle sensitivity during and after movement. The post-movement muscle tests described below evaluate the nervous system's ability to maintain spindle sensitivity and an appropriate stretch reflex during and after movement.

Interactions of Homologous columns in the spinal cord

The AMNs and GMNs that innervate a muscle reside in a continuous column of cells that span three or four spinal segments in the ventral horn of the spinal cord. There is a somatotopic organization of motor neurons (MNs) such that MNs innervating proximal muscles are in the medial portion of the ventral horn and MNs innervating distal muscles are in the lateral portion of the ventral horn. MNs to flexor muscles are more dorsal and MNs to extensors are more ventral.

Neurons that go to muscles with similar functions are grouped in close proximity in homologous columns. MNs in homologous columns interact in predictable ways. Excitation and inhibition spread from one group to another. When a muscle is activated, antagonist muscles are inhibited through reciprocal inhibition, and muscles with synergistic actions are facilitated. (10)

For example, when a flexor muscle is activated,

- Other flexors in that limb are activated to some degree.
- The antagonist extensor is inhibited, and
- Other extensors in that limb are inhibited to some degree as well.

Recurrent Inhibition

Recurrent inhibition is a negative feedback system that is used in many areas of the nervous system. Renshaw cells in the ventral horn of the spinal cord function as part of a recurrent inhibition loop that inhibits AMNs. AMNs send collateral axons that excite Renshaw cells that in turn inhibit the AMNs.

Renshaw cells are inhibitory cells in the ventral horn with a variety of targets and a variety of segmental and suprasegmental input sources. They are part of the recurrent AMN inhibitory loop described above. They serve as a regulator that can vary the gain of AMN recurrent inhibition.

NE depresses the activity of Renshaw cells, thus reducing the degree of recurrent inhibition to AMNs. NE facilitates the action of AMNs directly and through disinhibition (inhibiting the Renshaw cells that inhibit the AMNs) (21,22)

Section 2: Manual Muscle testing to Evaluate Integration of Small AMNs

Introduction

AMNs have a range of sizes, physiological properties and functions. Small AMNs innervate slow-twitch muscle fibers that predominate in proximal muscles and perform most of the work of posture and locomotion. Large AMNs innervate fast-twitch muscle fibers that predominate in distal muscles (especially hand) that perform fine, delicate, fractionated movement. Fast-twitch muscles also supply fast explosive power for proximal muscles.

Small and large AMNs perform different functions and they are activated from different areas of the nervous system. Muscle tests can be modified so that they challenge one group of AMNs more than the other. This article will focus on tests that challenge small AMNs. (A future article will discuss tests that challenge large AMNs)

5HT and NE are projected to the spinal cord from different parts of the brainstem and in the spinal cord they have opposite effects on GMNs and on group-2 afferent input to GMNs. GMNs regulate stretch reflexes during and immediately following movement. Muscle tests can be designed so that they challenge the ability of GMNs and the brainstem systems that regulate them to maintain appropriate stretch reflexes during and immediately following movement.

This article describes manual muscle tests that are used to evaluate the integration of small AMNs. Within the context of a complete neurological examination, manual muscle testing provides a sensitive, easily administered method of evaluating brainstem function. Each test involves examiner application of a fast final force that elicits a stretch reflex. The muscle is evaluated to determine whether it can in can resist lengthening.

Muscle test variables

In addition to the final eccentrically applied test pressure, tests for small AMNs have the following variable factors.

1. Contraction of muscles distal to the test muscle. The test force can be applied while the patient maintains simultaneous isometric contraction of muscles distal to the test muscle.

1. Amount and duration of pre-load force: The test force can be applied after the muscle is already contracting strongly to resist a pre-loading force, after it is contracting lightly to resist a pre-loading force, or when the muscle has not been subjected to a pre-loading force.

1. History of the antagonist muscle. The test force can be applied after the antagonist muscle is contracted or stretched

1. Pre-test movement. The test force can be applied immediately after the muscle is lengthened, immediately after it is shortened, or while the muscle is at a constant length.

Muscle test procedure: Isometric contraction of muscles distal to the test muscle

Isometric contraction of muscles distal to the test muscle: Mechanics

• The patient maintains isometric contraction of muscles distal to the test muscle while the test is performed.

Isometric contraction of muscles distal to the test muscle: Physiology

• Isometric contraction activates Golgi tendon organs that send 1b afferent input to the spinal cord. 1b input inhibits small AMNs and facilitates large AMNs (4,26,27,28) Due to the interactions of homologous columns of neurons in the spinal cord, excitation or inhibition of one group of muscles spreads to other muscles in that limb which have a similar function. For example inhibition of small AMNs that innervate distal flexor muscles causes inhibition of the small AMNs that innervate proximal flexor muscles and vice versa.

Isometric contraction of muscles distal to the test muscle: Significance

• Isometric contractions of distal muscles can be used to enhance the sensitivity of proximal muscle tests. For example holding a fist (isometric contraction of finger flexors) will cause a higher percentage of pre-load tests (described below) for shoulder flexor muscles to fail, and holding the fingers and wrist in extension (isometric contractions) causes a higher percentage of rebound tests (described below) for shoulder abductors to fail.

Muscle test procedure: Pre-loaded muscle test

Pre-loaded muscle test: Mechanics

• Have the patient resist light isometric pressure for two seconds prior to applying the test pressure.

• Pre-load weakness is most often observed in proximal rather than distal, leg more than arm, flexor rather than extensor, and adductor, internal rotator muscles rather than abductor external rotator muscles (muscles that are most strongly effected by medullary, serotonergic projections)

• The sensitivity of pre-load tests is enhanced when homologous muscles distal to the test muscle are isometrically contracted for the duration of the test. For example, a pre-load test of arm flexors is enhanced when the fingers are held in flexion ("make a fist"), and a pre-load test of hamstring muscles is enhanced if the test is performed while the patient maintains foot dorsiflexion.

Pre-loaded muscle test: Physiology

• The final test force causes a stretch that reflexively causes the muscle to resist lengthening. The stretch reflex occurs through activation of small AMNs. If the stretch signal is dampened, or if the small AMNs fail to respond adequately, the stretch response is inadequate and the muscle test fails.

• Light pre-load pressure creates an isometric contraction that inhibits the small AMNs that respond to the stretch signal. Heavy pre-load pressure activates large AMNs that are then available to resist the test pressure. (Large AMNs are not activated by the stretch reflex) For this reason, light pre-load pressure often causes a test that fails, while heavy pre-load pressure does not.

• PPs of small AMNs persist for up to a minute. PPs of large AMNs decay after 1-2 seconds. The two-second pre-load activation period is longer than the PPs produced for large AMNs, and the length of time reduces the force generated by large AMNs and fast-twitch fibers. (3,5,6)

• 5HT and NE have the following effects on pre-load muscle tests.

• Low 5HT causes decreased integration of small AMNs. It has little effect on the stretch reflex. It tends to cause pre-load muscle weakness.

• Low NE causes decreased integration of small AMNs and it also causes an increased stretch reflex. Because of the increased stretch reflex it does not tend to cause pre-load muscle weakness.

Pre-loaded muscle test: Significance

• Pre-load weakness is frequently indicative of decreased serotonergic drive from the ipsilateral medulla.

Muscle test procedure: Rebound muscle test

Rebound muscle test: Mechanics

• Tap the arm or leg in a direction that stretches the antagonist of the muscle that is to be tested. Do this immediately prior to applying the test pressure.

• Rebound tests are best seen with extensor, abductor and external rotator muscles (that receive relatively large pontine/NE drive)

• Rebound tests are enhanced if the wrist or ankle is held in extension. (Plantar flex the foot while performing a rebound test with the piriformis muscle and hold the wrist and fingers in extension when performing a rebound test with the middle deltoid)

Rebound muscle test: Physiology

• Tapping elicits a stretch response. If the stretch response is exaggerated the muscle that is stretched inhibits the test muscle (through reciprocal inhibition) to an extent that the test muscle fails to resist test pressure.

Rebound muscle test: Significance

• Rebound test weakness is frequently due to an increased stretch reflex secondary to decreased NE.

Muscle test procedure: Antagonist-activation muscle test

Antagonist-activation muscle test: Mechanics

• Apply light isometric pressure to cause contraction of the antagonist muscle for two seconds immediately prior to applying the test force.

• The test is enhanced if distal muscles homologous to the antagonist are held in isometric contraction for the duration of the test. (Hold a fist while performing an antagonist activation test for the middle deltoid muscle)

Antagonist-activation muscle test: Physiology

• Under normal conditions, light isometric contraction inhibits small AMNs. This author believes that in the case of excessive 5HT facilitation of AMNs, type 1b afferent feedback fails to inhibit the AMNs. Instead the antagonist AMN are facilitated and cause reciprocal inhibition of the test muscle.

Antagonist-activation muscle test: Significance

• This author believes that weakness following antagonist activation frequently indicates exaggerated reciprocal inhibition due to increased 5HT drive to the AMNs. (The weakness pattern is frequently temporarily abolished by physiological challenges that decrease CO2 as described below)

Muscle test procedure: Post-movement muscle test

Post-movement muscle test: Mechanics

• Post-shortening test: Have the patient move the muscle to be tested through (at least) 30 degrees of its range of motion in a direction which shortens the muscle. Have them stop at a predetermined position. Immediately apply the test pressure.

• Post-lengthening test: Have the patient move the muscle to be tested through (at least) 30 degrees of its range of motion in a direction which lengthens the muscle. Have them stop at a predetermined position. Immediately apply the test pressure.

Post-movement muscle test: Physiology

• Post-shortening and post-lengthening tests evaluate the ability of GMNs to maintain spindle sensitivity during (and immediately after) movements. Two primary factors influence the GMNs ability to maintain post-movement receptor sensitivity.

1. The efficiency of synaptic input from group-2 afferents to the GMN, as discussed earlier.
2. A variety of descending commands from supersegmental sources to the GMN (subject of a future paper)

• The efficiency of group-2 afferent input is strongly modulated by the ratio of NE and 5HT in the spinal cord. The higher the 5HT / NE ratio, the more group-2 feedback to GMNs. (13)

• A low 5HT/NE ratio (low 5HT and /or high NE) causes decreased feedback from group-2 afferents to GMNs. GMNs think that the muscle has moved less than it has. GMNs fail to maintain adequate drive to intrafusal fibers and the receptor portion of MSCs is offloaded. It goes slack and fails to generate an adequate stretch signal. The muscle fails to resist test pressure after it is shortened.

• A high 5HT/NE ratio (high 5HT and /or low NE) causes increased feedback from group-2 afferents to GMNs. GMNs think that the muscle has moved more than it has. GMNs generate excessive drive to intrafusal fibers and the receptor portion of MSCs is loaded excessively. This generates excessive tone in the muscle that in turn weakens the antagonist muscle through reciprocal inhibition. A muscle fails to resist test pressure after it is lengthened (after its' antagonist is shortened)

Post-movement muscle test: Significance

• Post shortening weakness might indicate:
1. Inadequate drive to GMNs from suprasegmental command signals.
1. Decreased feedback from group-2 afferents to GMNs due to a decreased ratio of 5HT/NE.

• Post-lengthening weakness might indicate
1. Excessive drive to GMNs from suprasegmental command signals.
1. Excessive feedback from group-2 afferents to GMNs due to an increased ratio of 5HT/NE.

Muscle test procedure: Physiological brainstem challenges

Carbon dioxide (CO_2) and trigeminal challenges can be used to evoke predictable changes in muscle function. Challenges are helpful in determining whether positive muscle test results are due to physiological problems in the brainstem or to lesions elsewhere in the nervous system.

Muscle test procedure: CO_2-evoked changes in muscle function

Increased CO_2: mechanics of the challenge

• Have the patient wear a mask or breathe into a paper bag for 5 or 6 respirations immediately prior to performing the tests.

Increased CO_2: physiology of the challenge

• Breathing into a mask causes increased CO_2 in the serum that triggers an increase of 5HT.
• This increases the 5HT / NE ratio and muscles fail to resist test pressure after they are lengthened (or no longer weaken after they are shortened)
• Increased 5HT causes antagonist activated weakness (or abolishes pre-load muscle test weakness)

Decreased CO_2: mechanics of the challenge

• Have the patient take 5 or 6 respirations deep rapid respirations immediately prior to performing the tests.

Decreased CO_2: physiology of the challenge

• Hyperventilation causes decreased CO_2 in the serum that triggers a decrease of 5HT.
• This decreases the 5HT / NE ratio and muscles to fail resist test pressure after they are shortened (or no longer weaken after they are lengthened)
• Decreased 5HT causes pre-load muscle test weakness (or abolishes antagonist activated muscle test weakness).

Significance of the challenges:

• If these CO2 challenges abolish a positive muscle test finding, it is likely that the weakness is due to a reversible, physiological medullary raphe nuclei lesion on that side. If CO2 challenges fail to abolish a positive muscle test outcome, there is increased likelihood that the causative lesion is elsewhere in the nervous system.
Muscle test procedure: Trigeminal-evoked changes in muscle function
Bite challenge: mechanism

• Have the patient lightly bite a tongue depressor between the molar teeth on one side of the mouth, and test extensor/external rotator muscles on the same side of the body.
Bite challenge: physiology

• This author believes that because of their proximity and (presumed) shared lateral crest origins (Jones, 1991), activation of the mesencephalic trigeminal nucleus causes activation of the locus coeruleus, which results in increased noradrenergic projections to spinal cord neurons, especially those innervating extensors.

• Increased NE causes decreased stretch reflex for extensor muscles and they often fail to resist examiner initiated test pressure.
• Increased NE causes decreased stretch reflexes and abolishes rebound muscle test weakness.
• Increased NE causes a decreased 5HT / NE ratio, and muscles weaken after they or shortened (or no longer weaken after they are lengthened)
Bite challenge: significance

• If the trigeminal bite challenge does abolish a rebound or post-lengthening muscle test weakness, there is increased likelihood that the weakness is due to a reversible metabolic lesion of the pons on that side. If it fails to abolish the weakness, there is increased likelihood that the weakness is due to a lesion elsewhere in the nervous system.

Mouth-open trigeminal challenge

• Holding the mouth open often causes muscle test outcomes indicative of decreased activation of the locus coeruleus and decreased NE projection to the spinal cord. This is not as consistent as the biting challenge
Discussion

A muscle test evaluates whether a muscle can perform a certain function or group of functions; resist a perturbation, move and then resist a perturbation, generate a large force, etc. Each muscle function requires the activation of specific combinations of large and small AMNs, and large and small GMNs (7) and each function is most effectively directed from a particular area of the brain.

The brainstem coordinates posture and locomotion. Higher brain centers initiate and direct posture and locomotion, but delegate the details to the brainstem and spinal cord. Higher centers of the brain are more directly involved with the generation of fast powerful forces and with the control of hand and finger movement. Corticospinal and rubrospinal pathways carry signals from higher centers in the brain to the large AMNs that activate fast-twitch muscle fibers that are necessary for those actions.

Muscle tests evaluate the performance of particular muscle functions, and by extension the areas of the brain that control those functions. Muscle testing is a sensitive, easily administered diagnostic tool for the evaluation of brainstem (and other supraspinal) function.

This article has focussed on the evaluation of motor function controlled or modulated by the pontine and medullary reticular formation, the LC and the medullary raphe nuclei. It has hopefully extended the theoretical understanding of how to use muscle testing to evaluate brainstem function.

The tests described in this paper can be (and in the population of patients seen by this author, usually are) positive (weak) due to physiological, reversible brainstem dysfunction. They can also be positive due to dysfunction in other areas of the nervous system including the ipsilateral cerebellum, contralateral cerebral cortex or basal ganglia, and the spinal cord or peripheral nerve.

If CO2 and trigeminal challenges reverse positive findings, it is likely that those findings are due to physiological brainstem dysfunction. Physiological brainstem dysfunction can in turn be due problems in other areas of the nervous system. The brainstem does not function well if it receives aberrant input from any area of the brain. For this reason, positive muscle test findings must be considered in the context of a complete neurological examination. If CO2 and trigeminal challenges do not reverse positive muscle test findings, it is more likely that the lesions are due to lesions other than physiological brainstem lesions.

Abbreviations

Abbreviations used in this article

- 5HT serotonin
- AMN alpha motor neuron
- GMN gamma motor neuron
- LC locus coeruleus
- MN motor neuron
- MSC muscle spindle cell
- NE norepinephrine
- PP plateau potential
- RAS reticular activating system

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