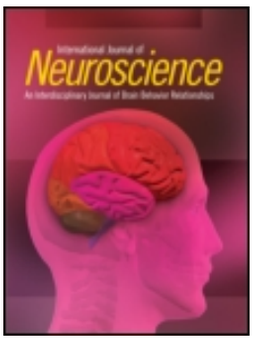

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EXPANDING THE NEUROLOGICAL EXAMINATION USING FUNCTIONAL NEUROLOGIC ASSESSMENT: PART II NEUROLOGIC BASIS OF APPLIED KINESIOLOGY

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Functional Neurologic Assessment and treatment methods common to the practice of applied kinesiology are presented. These methods are proposed to enhance neurological examination and treatment procedures toward more effective assessment and care of functional impairment. A neurologic model for these procedures is proposed. Manual assessment of muscular function is used to identify changes associated with facilitation and inhibition, in response to the introduction of sensory receptor-based stimuli. Muscle testing responses to sensory stimulation of known value are compared with usually predictable patterns based on known neuroanatomy and neurophysiology, guiding the clinician to an understanding of the functional status of the patient's nervous system. These assessment procedures are used in addition to other standard diagnostic measures to augment rather than replace the existing diagnostic armamentarium. The proper understanding of the neurophysiologic basis of muscle testing procedures will assist in the design of further investigations into applied kinesiology. Accordingly, the neurophysiologic basis and proposed mechanisms of these methods are reviewed.

Keywords: Functional neurology; functional neurologic assessment; neurologic examination; manual muscle testing; physiology; functional medicine; functional illness; neurologic assessment; kinesiology; muscle testing; muscular strength; muscle contraction; skeletal muscle; diagnosis; physical examination

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INTRODUCTION

Goodheart (1964) introduced manual muscle testing for functional neurological assessment (Walther, 1988). He called his observations applied kinesiology (AK). AK is a functional neurologic assessment and treatment process that extends the neurological examination taught in medical and chiropractic colleges to include the identification of subtle shifts away from optimal neurologic status. These shifts are associated with declines in function that may contribute significantly to patient morbidity (Fries, 1992, 1996). Changes in patterns of motor function that occur in response to the introduction of sensory stimuli of known value can be used to evaluate the functional status of central and peripheral neurologic pathways and guide the clinician to therapeutic measures to restore optimal neurologic function.

Much of the data gathering process unique to applied kinesiology relies on the manual assessment of muscular function as a method to evaluate changes in functional neurologic status reflected as changes in motor function. These observed changes in muscular function are assumed to be associated with changes in the central integrative state (CIS) of anterior horn motor neurons. The CIS is defined as the summation of all excitatory inputs (EPSPs) and inhibitory inputs (IPSPs) at a neuron. It is possible, therefore, to have a wide variation of central facilitated states and central inhibited states of neurons summating from many sources. The functional strength of a skeletal muscle is affected by the CIS (Goodheart, 1964; Walther, 1988; Guyton, 1991; Denslow, 1942) of the anterior horn motor neuron cells, (Feinstein, 1954) which in turn reflects changes elsewhere in the neuraxis.

The CIS may also be affected by the health of the neuron itself. The neuron's ability to maintain metabolic pathways, membrane receptors, and membrane pumps so that it is capable of responding to the demands placed on it by EPSPs and IPSPs is a critical factor, and one which may be identified by the AK assessment process as well. However, consideration of transneural degeneration and various axonopathies is beyond the scope of this paper. The clinical factors presented here will assume normal health of neurons. Although this may or may not be the case in clinical practice and must be considered by the clinician, the addition of this variable in this presentation would complicate and obscure the principles which are being presented. Similarly, although characteristics of individual neurological receptors and pathways are emphasized herein, the clinician must consider

all of the interconnectedness of the nervous system, which is also within the realm of AK assessment but beyond the scope of this presentation.

Functional neurological assessment is performed by introducing sensory receptor-based stimuli, monitoring changes in the CIS through manual muscle testing, and interpreting the outcomes of manual assessment according to the knowledge of the relevant neuroanatomy. The introduction of sensory receptor-based stimuli of known value usually creates predictable changes in patterns of motor output. These motor changes are observed through muscle testing responses, and compared with the predicted responses, allowing the clinician to derive data about the state of the patient's neuraxis. Each step in the process of diagnosing and treating a patient using AK consists of creating a specific neurologic context, which is thought to be the sum of all sensory receptor-based afferent stimulation and all centrally generated effects at that moment, and observing changes in the patient's motor responses to that context.

AK clinical diagnostic procedures are focused on identifying functional neurological changes before they become end stage tissue disorders. Since the health of the nervous system is dependent on its ability to receive and respond to sensory information, treatment procedures are primarily sensory receptor based therapies designed to normalize afferentation.

For example, the activation of touch, pressure, vibration, and other types of mechanoreceptors (MRs) is known to block afferent signals from nociceptors (Sherrington, 1948; Feinstein, 1954). In the presence of adequate nociceptor activation, as when touching a hot stove with the hand, there is flexor reflex afferent (FRA) activity that creates muscle facilitation and inhibition patterns associated with the flexor withdrawal reflexes. There will typically be facilitation of limb flexors and inhibition of limb extensors with contralateral stabilization, creating withdrawal of the affected limb away from the painful stimulus. There will be patterns of facilitation and inhibition associated with these activated reflex pathways which can be identified using manual muscle testing. Introducing MR inputs (mechanically rubbing an area of tissue whose nociceptors are firing, for example) to block the pain will also result in a facilitation effect of muscles whose inhibition was caused by the FRA response. The effect of such an introduced stimulus may also be assessed through manual muscle testing. Treatment procedures are aimed at restoring a balanced level of neurologic function, with appropriate levels of facilitation, which are observed clinically to be associated with restoration of other normal functions such as autonomic and neuroendocrine balance, proper neuro-immune function, and reduction of pain.

MUSCULAR FACILITATION AND INHIBITION

Clinicians using AK commonly refer to the result of a manual muscle test as a "strong" response or a "weak" response (Leisman, Zenhausern, Ferentz, Tesfera and Zemcov, 1995). A muscle that cannot meet the demands of testing pressure is termed "weak". The weak testing outcome is hypothesized to be associated with an inhibitory CIS of the muscle's alpha motor neuron (AMN) pool. If the motor neurons in the pool are inhibited (further away from depolarization threshold, or hyperpolarized), then the subject cannot adequately depolarize the pool on demand and adequate muscle contraction to meet the demands of the manual muscle test cannot take place. The result is a weakness in the muscle test outcome. In this paper, the terms "strong" and "weak" are used interchangeably with the terms "conditionally facilitated" and "conditionally inhibited". These latter terms are intended to refer to the hypothesized conditional facilitation or inhibition of alpha motor neurons, reflecting changes in their CIS.

The functional status or CIS of the anterior horn motor neurons is maintained by convergence of multiple segmental and suprasegmental pathways. The segmental pathways are sensory pathways that are either of somatic or visceral origin and arise from a variety of sensory receptors in skin, joints, fasciae, viscera, and from various chemoreceptors. The supra-segmental pathways are descending pathways that can be of a conscious origin (cortical) or of a reflexogenic origin (brainstem, cerebellum) including postural and gait patterns. A conditionally inhibited muscle is thought to be associated with an inhibitory CIS summation of the converging pathways to the alpha motor neuron controlling that muscle (Leisman, 1989).

Leisman *et al.* (1995) have provided the first electrophysiologically based definition of what AK practitioners observe as conditionally facilitated and inhibited muscle responses to manual muscle testing procedures: The ability or inability of a muscle to lengthen but to generate enough force to overcome resistance is what is qualified by the examiner and termed "Strong" or "Weak".

Leisman and his colleagues found that there were significant differences in force/integrated EMG ratios in what is described as muscle testing strengths and weaknesses and that these differences could not be attributed to fatigue of the tested muscles (Leisman *et al.*, 1995).

In an earlier paper, Leisman *et al.* (1989) also identified differences in somatosensory evoked potentials (SSEPs) recorded during the testing of conditionally facilitated versus conditionally inhibited muscles. SSEPs were recorded from the contralateral median nerves of fifteen subjects while three

different, previously identified muscles were tested. In all subjects the baseline (no muscle test) and control "strong" muscle test recordings were comparable while recording from the "weak" muscle showed increased amplitudes in the contralateral layer components.

Subjective reports differentiating between pathological and functional weaknesses have been supported by objective evidence (Lawson, 1997; Leisman *et al.*, 1989, 1995). If the peripheral nerve and the neuromuscular junction are intact, then a weak muscle test response is hypothesized to represent inadequate anterior horn motor neuron activity due either to excessive inhibition or to inadequate facilitation of these neurons. If a muscle is conditionally facilitated, it is presumed that the summation of inputs to the AMNs results in net excitation. With manual muscle testing, it is usually possible to subjectively localize which particular well-defined neurological pathways are overly excited or inhibited and to identify the source(s) of these deviations from normal function. Though one might typically assume that the normal state of a muscle involves adequate facilitation to meet the demands of manual testing, this is not always the case neurophysiologically. Some normal states are characterized by inhibition. For example, when a patient assumes a stance which resembles a gait pattern (standing with opposite arm and leg forward), neurophysiology would predict that the alpha motor neurons controlling arm flexors ipsilateral to the forward leg would be in a state of inhibition and alpha motor neurons controlling ipsilateral arm extensors would be in a state of facilitation, so that the ipsilateral arm could move into extension, in keeping with the normal contralateral motion of walking. If one were to test the arm flexors in this gait position and find them adequately facilitated, the clinician would consider this result abnormal.

Accordingly, the interpretation of findings of conditional facilitation and inhibition are only relevant in the context of predictions based on known neurologic normals. When a variable in the form of sensory receptor stimulation is introduced, the clinician looks for the presence or the absence of a predictable change. If the stimulus creates a change in central integration that affects motor outputs, a change in muscle testing response will be observed, toward either facilitation or inhibition, according to the circumstances.

RECEPTORS AND THEIR EFFECTS ON CENTRAL MODULATION

In addition to MR and nociceptors, receptor-based afferent stimulation can be introduced using retinal, olfactory, and taste receptors. These receptors

introduce afferent stimuli that can impact on suprasegmental targets, affecting their central integrative states, and therefore affecting all motor pathways either directly or indirectly.

In addition to general sensory and special sensory receptor-based afferents, impact on neurological function may arise from alterations in biochemical status affecting neuronal membrane potentials, neurotransmitter levels, and hypothalamic monitoring of the blood milieu. Neuronal activity of cortical origin, whether from cognitive, emotional, or other processes, also impacts the neuraxis significantly.

Receptor activity is transmitted throughout the entire nervous system and interacts with other factors to affect the CIS of virtually all neuron pools. When a group of sensory receptors fire, the function of other non-adjacent portions of the nervous system may be affected. This may lead to changes in modulation of other neurological systems downstream from the functional changes. Any neural system which has motor connections, either locally or by ascending or descending pathways, may have its functional status evaluated using the AK system of functional neurologic assessment. This includes not only somatic sensory systems, but also central systems for visual motor activity (*e.g.*, accommodation reflexes), vestibular reflexes (*e.g.*, tonic labyrinthine reflexes), reticulospinal (autonomic) pathways, emotional (psychosomatic) pathways, and so on. By evaluating the effects of olfactory and gustatory stimuli, neurochemical evaluation may be performed as well.

AFFERENT STIMULUS AS RECEPTOR-BASED CHALLENGE

In normal individuals, muscles function in a relatively predictable manner. Patterns of excitatory or inhibitory stimuli, introduced by the clinician through sensory receptor stimulation, are observed clinically to create predictable changes in the outcomes of manual muscle tests. These changes are hypothesized to be associated with changes in patterns of facilitation and inhibition of motor neurons. Applied kinesiologists commonly refer to the introduction of a known sensory stimulus as a "challenge". Such sensory challenges constitute the first half of the process basic to applied kinesiology, in which afferent stimuli of known value are introduced. The second half of the process is the observation of changes in motor function through manual muscle testing. The clinician must then interpret the observed change according to the known neuroanatomy and neurophysiology

associated with the stimulus and the resulting motor response, in much the same way that a clinician would interpret the outcome of a deep tendon reflex or the constriction of a pupil in response to a light stimulus.

NORMAL GAIT PATTERNS OBSERVED WITH MANUAL MUSCLE TESTING

Normal gait involves alternate shoulder flexor and extensor facilitation and inhibition. In the normal individual testing the general shoulder flexors and extensors in the neutral standing position should reveal normal muscle strength testing outcomes. Placing a subject in a simulated gait position (for example, standing with the left leg and right arm forward and the right leg and left arm back) should result in a conditional inhibition (muscle testing weakness) of the shoulder flexors on the left side as well as a weakness of the shoulder extensors on the right side. This is what would be predicted based on the normal arm swing of gait.

It is hypothesized that alterations from this normal pattern would be observed if there was improper afferent activity arising from sensory receptors that are a part of gait activity. This deviation from normal is thought to consist of alterations in patterns of facilitation and inhibition of muscles. This effect has been observed as muscle testing responses which vary from the predicted normal patterns (Leisman *et al.*, 1989). Therapeutic interventions are aimed at normalizing afferent activity to restore normal neuromuscular function.

FUNCTIONAL EVALUATION OF SPINAL CORD INTERNEURON FUNCTION

The following tests are used to evaluate spinal cord interneuron function. Muscle testing responses that diverge from predicted neurologic normals guide the AK practitioner to various therapeutic measures, aimed at restoring normal receptor afferentation. The therapeutic measures vary according to the specific clinical circumstance, and are beyond the scope of this paper.

Spinal interneurons affecting AMNs are facilitated by both segmental and suprasegmental pathways. Segmental influences include: (1) autogenic facilitation (such as by the la fiber mediated deep tendon reflex), (2) reflexogenic inhibition from a contracted muscle, or (3) flexor reflex

afferent (FRA) activity. Excitation of each of the three pathways is predicted to be associated with strengthening of a primary muscle and inhibition of its antagonist. One may examine these pathways to identify possible sources of aberrant influence on the alpha motor neuron of the muscle being tested.

For example, tapping the right patellar tendon to elicit a deep tendon reflex should result in facilitation of the right quadriceps, inhibition of the right hamstring, inhibition of the left quadriceps, facilitation of the left pectoralis major, and inhibition of the right pectoralis major. A strong voluntary contraction of the right quadriceps is expected to create the same pattern just described.

AK practitioners could examine the functional status of these pathways by observing patterns of conditional facilitation and inhibition of muscles subserved by the pathways in question. Failure of the predicted response immediately following a patellar tendon tap or muscle contraction may indicate a functional neurological problem.

Pinching or squeezing a tendon to fire an inhibitory afferent from the Golgi tendon organ is predicted to be associated with conditional inhibition of the muscle of origin followed by an immediate return to normal facilitation on a subsequent muscle test. Failure of this response should lead the clinician to investigate the source of neurological interference resulting in the dysfunction.

Axial traction of a joint for eight to ten seconds activates Type III joint receptors, whose afferents are inhibitory to muscles which cross the joint being stretched. This traction is followed by testing a muscle that crosses the joint. The muscle should be inhibited and the muscle testing response would be predicted to reflect this conditional inhibition (Leisman *et al.*, 1996). Failure of the inhibited response leads to further investigation.

In a similar fashion, the facilitatory effects of Type II joint receptors may be evaluated. A conditionally inhibited muscle is predicted to respond with a shift toward facilitation when its related joint is stretched quickly. This should occur even when the muscle spindles of the muscle are prevented from stretching by the nature of the joint stretch. Failure of this strengthening response is grounds for further investigation.

All of the above mentioned tests evaluate well known, well defined interneuron and reflex pathways. These pathways are also dependent on other segmental and suprasegmental influences for normal function. AK muscle testing combined with traditional neurological examination procedures greatly enhances the data gained from the neurological exam.

USING MECHANORECEPTOR AND NOCICEPTOR STIMULATION TO GUIDE THERAPY TO INJURED AREAS

The effects of pain are dependent on the interaction of two types of sensory receptors and their afferent pathways: nociceptors (NOCs) and MRs. Skin NOCs can be manually stimulated by pinching. Skin MRs can be manually stimulated by rubbing or stroking. These receptor-based stimuli constitute a mechanism by which inputs can be strategically introduced, as a challenge to the system's central modulatory mechanisms. The intent of such a "challenge" is to assess the extent to which changes in sensory input create alterations of motor function.

By observing muscle testing outcomes resulting from such sensory receptor diagnostic challenges, there are five possible outcomes: rubbing facilitates, rubbing inhibits, pinching facilitates, pinching inhibits, or no change in muscle status results. Each of the first four possibilities would direct the clinician to a different treatment procedure to normalize the patient's functional neurological status and reduce pain if present.

If an alteration in motor function is a consequence of nociceptive afferent stimulus, one would expect to observe a return to normal motor function if the nociceptive stimulus was blocked or terminated. Since the activation of mechanoreceptors blocks the input from adjacent nociceptors, one may selectively stimulate mechanoreceptors in tissues (such as rubbing or stroking the skin over the effected area) and observe motor control patterns in muscles to identify those areas of the body whose stimulation results in a normalization of motor function, presumably by achieving a blocking effect of nociception. Observed patterns are interpreted in light of the known neuroanatomy.

Nociception has extremely important effects on muscular and autonomic activity *via* reflex pathways both segmentally at the spinal cord level and suprasegmentally at the hypothalamus and brainstem levels, regardless of whether or not a person experiences conscious pain.

SUPRASEGMENTAL MODULATION OF PAIN

In the assessment of functional alterations related to pain, MR and NOC receptor stimuli are the basis for sensory receptor diagnostic challenges that may guide therapeutic choices. If rubbing or stroking the skin over an area

of trauma results in neuromuscular facilitation, there will also be an abnormal response to autogenic facilitation of the muscle spindle as described above (Lynn, 1985).

If pinching over an area of recent or old injury results in neuromuscular facilitation, the result suggests that the additional nociception causes the facilitation response *via* the descending medullary pathways through interneurons to the MNs in the anterior horn (Fitzgerald, 1985). This suggests that the weakness is present due to a net MN inhibition (due to a lack of excitation) which may be overcome by activating the CRN descending pathway.

If pinching (or adding any increased nociception from any stimulus) over an area of injury results in a general neuromuscular inhibition response, the result suggests excessive NOC activity, affecting all MNs (Guilbaud, Peschanski and Besson, 1985).

By these sensory receptor challenges, the neurological pathways related to a specific patient's pain may be assessed and the most appropriate pain control techniques may be employed.

TONIC LABYRINTHINE REFLEXES

The tonic labyrinthine reflexes (TLR) are present at birth and create patterns of excitation and inhibition of the limbs helping to maintain upright posture or to catch the body in the case of a fall (Guilbaud *et al.*, 1985). There are four distinct TLR response patterns: face up, face down, right ear down, and left ear down. In the face down position of the head, the flexors of all four limbs are facilitated. In the face up head position, the extensors of all limbs are facilitated.

The right ear down position is associated with excitation of the right limb extensors and an inhibition of the right limb flexors. Likewise, this head position is associated with excitation of the left sided limb flexors and an inhibition of the left limb extensors.

The effects of TLRs on muscle testing are profound. The TLRs are not strong enough to create muscle testing weakness by themselves, but they are powerful enough in their excitatory effects to overcome many other sources of inhibition hence hiding otherwise weak muscle patterns. Clinical application of TLR principles suggest that the supine (face up) would not be expected to demonstrate conditional inhibition of extensor muscles tested manually, due to the power of these lateral vestibulospinal tract mediated

reflex pathways (Guilbaud *et al.*, 1985). Likewise, a prone (face down) patient would not be expected to demonstrate conditional inhibition of a flexor tested manually. In the supine position a weak flexor muscle should be strengthened while the head is in the opposite ear down position. These patterns of head position in relation to gravity must be considered when performing muscle testing for any purpose.

For example, when an orthopedist tests the muscles of the rotator cuff in a supine patient, the generalized facilitation of extensors by the TLR creates an artificially increased facilitation of supraspinatus, infraspinatus, and teres minor muscles, all of which are extensors. These muscles should either be tested in a prone position, or other measures should be taken to momentarily disable the facilitating effect of the TLR. Such measures are beyond the scope of this discussion.

TLR head position patterns may also be examined to ascertain the status of the TLR and the lateral vestibulospinal tracts. Variations from predicted facilitation and inhibition patterns guide the clinician's attempt restore the adequate function of this postural control mechanism. For example, if a right side extensor is identified as conditionally inhibited in a supine patient, turning the head to the right ear down position should increase the extent of facilitation of the extensor. Failure of right ear down position to yield this effect is suggestive of an inability of the TLR to create the expected efferent pattern of facilitation to right sided extensors. Further assessment and therapeutic measures are pursued to normalize this effect.

FUNCTIONAL DEAFFERENTATION

Patterns of conditional muscular inhibition have been often ascribed to "functional deafferentation" of the various afferent pathways affecting a muscle's motor neurons. Functional deafferentation may be simply defined as the loss of normal and expected sensory activity. A common source of functional deafferentation is the loss of normal joint range of motion resulting in decreased mechanoreceptor (MR) activation. AK therapeutic procedures are often directed at normalization of the sources of facilitation and inhibition, with the intent of restoring normal efferent motor control mechanisms for muscular activity. This effect restores normal muscular control of joint movement. Changes in the status of joint and muscle function can be both a cause and effect of changes in the extent of afferentation.

STROKE ANTALGIA-LIKE PATTERNS OF INHIBITION

Functional deafferentation of the cerebral cortex can affect not only its somesthetic sense but also its motor responses. If, due to a lack of normal afferentation, a motor area does not receive adequate stimuli, its inability to fire its usual motor pathways can result in a dysfunction in which the muscle imbalances, and sometimes the symptoms, are reminiscent of stroke antalgia, but without the typical tissue pathology. Autonomic concomitants will accompany this pattern *via* the impact of the reticulospinal pathway at the intermediolateral cell column (IML) (Lynn, 1985). The effects of altered reticulospinal activity include changes in autonomic modulation that influence among other things, dilation of the pupils, respiration, heart rate and rhythm, circulation, sweating, shivering, digestion, *etc.*

The hypothalamoreticulospinal tract inhibits ipsilateral anterior muscles above T6, and ipsilateral posterior muscles below T6. Decreased frequency of firing of the neurons in this pathway, as is the case in functional deafferentation, has been observed to move the CIS of involved alpha motor neurons toward facilitation, resulting in an increased tone of the anterior muscles above T6, and an increased tone in the posterior muscles below T6 on the ipsilateral side (Willis, 1985). This increased facilitation yields increased inhibition of the antagonistic muscles. As a result, some or all of the ipsilateral upper limb extensors and abductors and ipsilateral lower limb flexors and adductors will be conditionally inhibited, and unable to meet the demands of manual testing. The observed pattern is very similar to stroke antalgia, and also resembles the normal gait pattern. It has been called a "pyramidal distribution of weakness". It is actually an inhibition of the extrapyramidal tract that has lost its ability to perform its requisite inhibitory functions that modulate pyramidal influences.

This stroke antalgia-like distribution of inhibition is primarily due to MR deafferentation from joints and muscle spindles *via* the thalamus and cerebellum. These muscle imbalances create symptoms that can lead to joint pathology and increased injury potential. Since joint MR afferents provide a significant portion of the total afferent stimulation to the contralateral thalamus, any reduction of MR stimulation would yield a reduced frequency of firing of thalamic interneurons, and therefore a reduced frequency of firing of descending efferents from the thalamus through the hypothalamoreticulospinal pathway.

Another area where the effects of asymmetrical thalamic firing can be observed is in vision. Primary visual afferents terminate in the lateral geniculate body of the thalamus and are conveyed from the thalamus to the

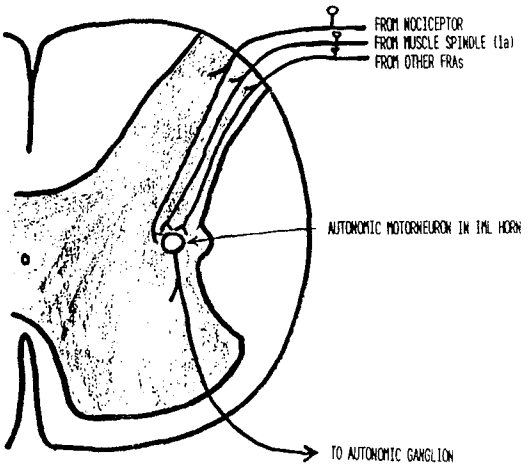

FIGURE 1 Nociceptive sensory fibers.

cord *via* reticulospinal tracts which affect both IML MNs and AMNs. Therefore, certain systemic and local changes in sympathetic (SYM) and parasympathetic (PS) function affect, in a predictable, specific fashion, the CIS of AMNs and hence, muscle strength and weakness patterns.

Nociceptors cause predictable FRA muscular activity previously discussed. An additional effect of many FRA afferents is stimulation of the SYM IML (Burt, 1993).

Since changes in autonomic functions affect somatic motor pathways through established mechanisms, changes in muscle function will accompany changes in autonomic (SYM and PS) status in predictable ways. AK evaluation of autonomic functions employs observations of conditional muscular facilitation and inhibition associated with observed changes in autonomic function. Such patterns of facilitation and inhibition may be present in a static fashion when alterations of autonomic tone are also present in a static fashion, as can be observed in routine neurological examination. Subtle changes in autonomic tone may be identified through the observation of patterns of change in conditional facilitation or inhibition of muscles in response to sensory receptor challenges directed at altering autonomic tone. Altered autonomic tone in a visceral organ will be clinically associated with either increased or decreased SYM activity and/or increased or decreased PS activity to that organ (Willis, 1985). Each of these four states can be identified by evaluating muscle testing patterns and responses to sensory receptor stimuli.

occipital cortex. Should the CIS of the thalamus (including the lateral geniculate body) be shifted toward inhibition, then the ability to convey primary afferents through the thalamus to the visual cortex where the visual inputs are perceived will be decreased. As a result, the physiologic blind spot will usually be larger in the visual field contralateral to the side of the thalamus receiving inadequate afferentation. The long literature on stabilized retinal image addresses this well (Riggs, 1953; Leisman, 1974, 1976). Blind spots may be mapped in the office setting and correlate with the muscle testing findings of a stroke antalgia-like distribution of neuromuscular inhibition. Significant changes in blind spot size have been demonstrated after manual manipulation of joints on the side ipsilateral to the enlarged blind spot. Such manipulation is presumed to have its effect *via* restoration of adequate afferent firing from joint MRs to the contralateral thalamus.

VISCEROSOMATIC AND SOMATOVISCERAL INTERACTIONS

The autonomic nervous system motor neuron cells in the IML column receive significant input from somatic factors (Lynn, 1985; Willis, 1985). Nociceptive sensory fibers are flexor reflex afferents (FRAs) which synapse in the IML column (see Fig. 1).

The significance of this fact neurologically is that clinicians cannot even touch their patients, much less manipulate them, without creating substantial effects on the IML column and the autonomic nervous system motor neurons. It is impossible to treat patients for neuromuscular or musculoskeletal problems without having meaningful effects on the motor neurons of the autonomic nervous system.

The body is constituted in such a way that somatic inputs into the nervous system cannot be made without affecting visceral function. Nor can visceral function be activated by any means (manipulative, nutritional, allopathic, homeopathic, *etc.*) without having significant effects on somatic motor function as well. Those who profess to treat musculoskeletal complaints without creating visceral effects are misinformed.

AUTONOMIC COMPONENTS OF AK EVALUATION

Autonomic signals which originate at the hypothalamus are transmitted to the brainstem reticular formation. This information descends to the spinal

Specific sensory receptor based diagnostic challenges may be used to identify segmental (local) or suprasegmental influences on the function of the primary autonomic motor neurons in the IML, hence on the AMNs and muscle function. Systemic influences affect the entire autonomic nervous system, whereas local influences may be used to evaluate the level of function of one organ at a time. An example of each of these is discussed below.

LOCAL AUTONOMIC STIMULATION OF VISCERAL REFERRED PAIN AREAS

Due to viscerosomatic convergence, (Swedlow, 1986) the body cannot tell the difference between nociception arising in the skin and that arising from an organ. Activation of the skin NOCs over the classical visceral referred pain (VRP) area for a given organ (see Fig. 2) will often result in a change in patterns of neuromuscular facilitation and inhibition, yielding changes in muscle testing outcomes. Similarly, activation of MRs over the same referred pain area may also bring about a change in muscle testing response by blocking visceral NOC activity.

Clinically, if increasing NOC activity by pinching an area results in a conditional facilitation of muscle, this can usually be interpreted as an indication that an increase in SYM stimulation in the area of the pinch has yielded a favorable effect. One might infer from this that the ambient SYM

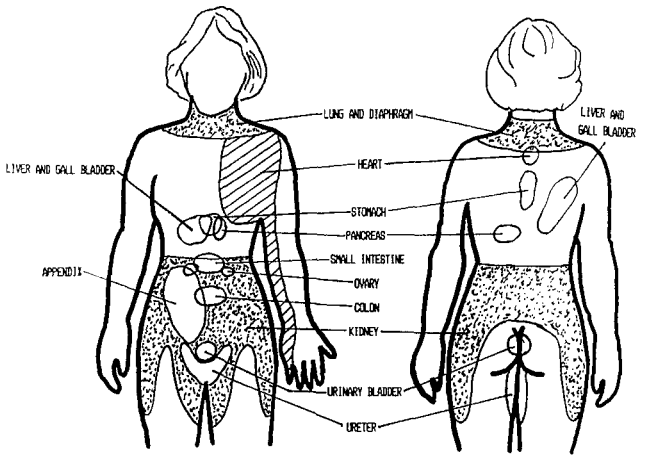

FIGURE 2 Visceral Referred Pain Areas.

activity in the area is inadequate. This decreased SYM activity is due to an inhibited CIS of the IML arising from lack of facilitation or lack of inhibition of inhibition by the afferents to the IML. This interpretation is especially applicable when a pinching or other NOC stimulus is applied to the VRP areas (see Fig. 2). A conditional facilitation of muscles following pinching a VRP area would suggest that the related organ was relatively deficient in SYM activity, as reflected in the CIS of its IML MNs, whose state is identified by observing the state of the AMNs for the muscles tested. This could be due to either deficient SYM activity or increased PS activity or both.

The converse of this, pinching over a VRP causing a conditional inhibition of muscles, suggests excessive SYM activity of that organ.

Nociceptive reflexes, including those affecting the IML and SYM function, are blocked by mechanoreceptor (MR) activity. Hence, if rubbing over a VRP area to increase MR afferents creates a conditional facilitation of muscle, it suggests a need for less SYM activity, more PS activity, or both for that organ.

If rubbing over a VRP creates a conditional inhibition of muscle, this suggests that the associated organ has excess PS activity.

When many or all VRPs respond to the same pinching (SYM) or rubbing (PS) challenges, a systemic challenge is often positive, as we will discuss below.

SYSTEMIC AUTONOMIC STIMULATION – VISUAL

There are a number or systemic autonomic stimulation patterns associated with changes in patterns of neuromuscular facilitation or inhibition. Only one of these is presented here.

It is established that the act of focusing from near to far and focusing on a distant object are events that involve activation of the SYM system. When conditionally inhibited muscles become facilitated with visual near-to-far activity or with focusing on a distant object, this is often evidence of a need for more systemic SYM function. A conditional inhibition response on near-to-far activity or distant focusing usually implies an excess of SYM activity. In either case, changes in patterns of facilitation or inhibition of muscle are interpreted in light of the impact of the shift of the system toward increased SYM function.

It is likewise established that the act of focusing from far to near is an event that involves activation of the PS system. If a conditionally inhibited muscle becomes facilitated with focusing on a near object such as the tip of

the nose, it suggests a need for more PS function. A conditional inhibition response on far-to-near activity or near object focusing usually implies an excess of systemic PS activity. Normalization of these systemic autonomic imbalances may involve many procedures including dietary changes, manipulation, sensory receptor manipulation to normalize afferent activity (see next section), nutritional supplementation, stress reduction techniques, and so on.

A MODEL FOR SO-CALLED NEUROLYMPHATIC REFLEX ACTIVITY INCREASING PARASYMPATHETIC ACTIVITY

The so-called neurolymphatic reflexes (NLs) are somatovisceral reflexes first described by Chapman. Most are located in the intercostal spaces. Chapman identified palpatory findings of nodular, indurated areas localized segmentally in intercostal and paraspinal areas, and associated them with disease in visceral organs neurologically associated with each segmental level. Chapman recommended manipulation of the tender areas until the tenderness or induration decreased.

Goodheart (1965) first described the use of the NL reflexes in relation to manual muscle testing. He found that specific patterns of conditional neuromuscular inhibition would respond with facilitation after manipulating one of the areas as described by Chapman. In this way, specific reflex areas described by Chapman became associated with specific observed patterns of conditional muscular inhibition. Since Chapman had identified relationships between these reflexes and specific organs, the associated patterns of conditional muscular inhibition associated with specific reflexes began to be likewise associated with the same specific organs. This was the beginning of clinical observations of a specific muscle-organ relationship. Areas of increased tissue irritability would be expected to send increased afferent signals to the spinal cord. This effect would be increased during weight bearing and during movement (including respiration), when the tissues are irritated by movement or pressure.

Increased afferentation in the intercostal spaces would be expected to reflexogenically increase SYM activity. This has been shown in laboratory animals by increasing both NOC and MR sensory input (Coote, Dowman, and Webber, 1969).

Clinical and anatomical evidence suggests that the response achieved by manipulating the NL reflexes is due to a relative increase of PS activity due

to a resolution of the pattern of ischemia and muscular spasm associated with the irritable NL area and a subsequent reduction of over stimulation of SYM activity at the IML. Although manipulation of the NLs often causes an increase of stimulation of local nociceptors during the manipulation, the net result following NL treatment is decreased irritability. This decreases the excessive afferent stimulation that is driving the local IML neurons to increased SYM activity. If PS outflow to those organs remains the same, the net result of treating an NL will be an increased relative PS activity of those organs that are affected. This is consistent with clinical observation. The need for the use of NL to increase PS activity is indicated clinically when the stimulation of MRs in a related organ's VRP yields a conditional facilitation of tested muscles.

The changes in muscular facilitation from treating a NL reflex are likely due to the collateral connections from the IML axons that reach AMNs. It is reasonable to expect increased muscular facilitation of conditionally inhibited muscles and a restoration of normal inhibition of "tight" or "spasmed" antagonists as a result of normalizing feedback from an active NL reflex.

GENERAL MUSCLE WEAKNESS PATTERNS FOLLOWING CHALLENGE PROCEDURES

A common observation by those practicing manual muscle testing procedures is a pattern in which all muscles exhibit a pattern of conditional inhibition following a sensory receptor challenge. This pattern suggests that anterior horn motor neurons must be inhibited throughout the neuraxis.

An area known as the inhibitory center of Magoun and Rhines (or the medial reticular extensor inhibitory area) in the medulla gives rise to descending inhibitory pathways which are generally inhibitory to extensor muscle reflex activity throughout the body (Kandel and Schwartz, 1985). Given the general inhibitory effect of certain sensory challenges on both flexor and extensor muscles, it is reasonable to speculate that there is a central site which creates a general inhibition simultaneously to both extensors and flexors. The exact location of the neurological center for general muscle weakness patterns remains a mystery, but one might speculate that it is related to the reticular formation and the descending reticulospinal tracts (Leisman, 1989).

DURAL CONCEPTS

AK techniques include cranial bone manipulation, performed with a light pressure. These techniques are based on a model of dura mater tension, particularly cranial dural tension. A cornerstone of the cranial dural concept is that cranial sutures have motion, and that this motion has an effect on nervous system function. This has been a much debated issue, but there is support for the concept that human sutures do have motion based on anatomical and developmental analysis, (Pritchard, 1956) squirrel monkey studies demonstrating lack of sutural ossification, (Retzlaff, 1976) squirrel monkey studies demonstrating nerve endings in sutural articulations, (Retzlaff, 1978) and clinical results (Weiner, 1990).

Alteration of normal cranial motion results in what are called "cranial faults" or "cranial lesions". The cranial bone motion is thought to affect and be affected by changes in dural tension, in both the cranium and the spinal canal. Since the dura mater has strong anatomical attachments to the sacrum and the coccyx, techniques involving cranial manipulation are often accompanied by sacral or other spinal manipulations. This has given rise to the terms like "craniosacral mechanism" and "craniosacral techniques".

Cranial faults are treated by manipulative procedures directed to the cranial bone structures and often accompanied by sacral or other spinal manipulation. These are most often very specific, gentle manipulations, occasionally performed coincident with a phase of respiratory activity. They will affect MR activity in the tissues manipulated as well as in the MRs in the cranial sutures and intracranial dural receptors.

Observations of the clinical outcomes from cranial manipulation demonstrate a wide diversity of physiological and therapeutic response including resolution of everything from low back pain to tachycardia. Considering the vast neuronal network affected by the trigeminal nerve, the vagus nerve, and the vestibular mechanism which may be impacted by cranial manipulative techniques, these responses seem plausible.

As discussed by Walther (1988), Upledger (1983), and others, the cranium is believed to function such that manipulation of one cranial bone will affect the entire craniosacral mechanism. It is thought that craniosacral techniques generally restore normal juxtapositional relationships and motion to the craniosacral mechanism, hence they are associated with a change in dural relationships or dural tension.

The innervation of both spinal and cranial dura arises almost totally from nociceptors (Willis, 1985). The cranial dural innervation and its areas of referred pain are reviewed in a paper by Schmitt (1992). The dura is

supratentorially innervated by the trigeminal nerve and infratentorially by sensory branches of the vagus nerve (Broadal, 1981). Many cranial faults are observed clinically to be accompanied by a pattern of bilateral conditional inhibition of the neck flexors, particularly the sternocleidomastoid (SCM) muscles. It is proposed that this bilateral neck flexor pattern should be interpreted as a representation of an FRA withdrawal reflex pattern associated with neck extension. This pattern is most likely initiated by nociceptive activity somewhere in the head and neck mediated through the various cranial afferents and their respective pathways. As with other pain patterns, MR stimulation over an area of NOC activity is expected to block the NOC afferents, briefly restoring facilitation to muscles whose inhibition is a consequence of the NOC activity. In this way, the clinician can introduce MR activity selectively by rubbing over each of the cranial bones or sutures, and observing changes in the pattern of inhibition of neck flexors. For example, if introduction of MR stimulation over one temporal bone yields a restoration of normal facilitation of neck flexors, therapeutic measures would be directed at the restoration of proper position and motion of that temporal bone.

It is also reasonable to describe the observed clinical therapeutic responses of cranial manipulation as taking place *via* the afferent effects of these nerves.

Upledger (1983) describes a great deal of movement in the craniosacral respiratory mechanism. The constant motion of the craniosacral mechanism may be enough to maintain a base line level of mechanoreceptor barrage from the associated structures. Accentuation of this movement by cranial manipulation may be adequate to bring hyperpolarized cranial receptors to threshold, firing the involved pathways, and reestablishing a frequency of firing that is maintained beyond the time of treatment. A normal amount of craniosacral motion will maintain a normal amount of afferent input to vital centers. An abnormal amount of afferent activity will create abnormal afferentation to these centers. This is thought to be normalized by mechanical manipulation of cranial bones to restore normal relationships and motions.

Examining extracranial MRs which are stimulated by craniosacral manipulative techniques sheds some light on the clinical responses. For example, one technique designed to correct mechanical torquing lesions of the sacroiliac joints involves placing a prone patient on orthopedic wedges (DeJarnette blocks) and repeatedly pressing on the sacrum coincident with respiration. This, of course, bombards the system with MR input from the

SI joints, the skin, muscles, and other tissues being contacted, intercostal and other respiratory activity.

THREE TYPES OF MANUAL MUSCLE TESTING

Manual muscle testing texts vary widely according to the type of contraction (*e.g.*, eccentric or concentric) to be measured, the timing and force of the tester, the timing and force of the patient's resistance to the movement, and other factors. Most describe the testing of muscles chiefly in order to establish gross muscular strength levels.

Our clinical observations have led to the discernment of three distinct methods of performing a manual muscle test. Clinically speaking, these are: (1) clinician started testing, (2) patient started testing with maximal contraction, and (3) patient started testing with submaximum contraction. Each of the three types of muscle testing responses is measured by the patient's ability to resist an incremental increase in force applied by the tester in the direction of eccentric contraction. The tester attempts to lengthen the patient's muscle out of a shortened pre-test position. Each type of test differs in the amount of pre-loading the patient is allowed prior to the addition of the eccentric testing force. The most significant factor in extracting clinical information from a muscle test response is who (tester or patient) starts the test, how much force is applied, and in which direction (*i.e.*, eccentric, concentric, or isometric).

1. Type I – Clinician Started Testing

Clinician started testing is an eccentric test of the muscle with no pre-loading. The tester positions the patient for the test, makes contact with the patient, and tells the patient to resist his force in the appropriate direction. Then the tester pushes against the line of pull of the muscle while the patient attempts to eccentrically contract the muscle, resisting the tester's attempt to further lengthen the muscle out of its shortened state.

2. Type II – Patient Started Testing to Maximum

In patient started testing with maximum contraction, the pre-loading allows a full isometric contraction of the muscle. The patient is instructed to push against the tester's hand while the tester holds the position as firmly as

possible. When the tester observes that the patient has reached a maximum isometric force, the tester then pushes with increased force in the direction of eccentric contraction. This type of testing is what Leisman *et al.* (1989, 1996), employed in their research efforts.

3. Type III – Patient Started Submaximum Testing

Patient started testing with submaximal contraction is a pre-loading of submaximal concentric contraction followed by eccentric test. The patient is instructed to push against the tester's hand in the direction of the muscle's normal pull, creating concentric shortening of the muscle to be tested. Immediately upon sensing the initial few degrees of concentric contraction, the tester then pushes against the patient's force in the direction of eccentric lengthening. The patient then resists the tester's attempt to further lengthen the muscle out of its shortened state.

It is obvious that training and skill are necessary to perform these tests and discriminate between the outcomes. Muscle testing for functional neurological assessment testing is far more sophisticated that simply having the patient shrug the shoulders to ascertain if cranial nerve XI is intact. The procedures, however, have reported significant reliability (Lawson and Calderon, 1997), as well as significant criterion validity (Leisman *et al.*, 1995).

A conditional inhibition response to an AK muscle test suggests that the CIS of those AMNs reflects either excessive inhibition or inadequate facilitation, in spite of the conscious descending excitatory inputs created by the patient attempting to perform the test. These conscious effects are considered to be a constant from one test to another. Measuring the eccentric portion of the test initiates a stretching of muscle spindles that should excite the AMNs and tend to reinforce the descending conscious pre-loading inputs to the AMNs for that the muscle.

NEUROPHYSIOLOGIC BASIS OF DIFFERENCES IN PATIENT RESPONSE TO THE THREE TYPES OF TESTS

Descending conscious effort by the patient in response to the eccentrically placed force creates inputs to the spinal cord that can be considered a constant in the manual muscle test. Any changes in muscle strength of the nature observed in AK are related to the eccentric portion of the test. Therefore, it is the effect primarily on the alpha motor neurons (AMNs) of

that muscle that is reflected in the outcome of the muscle test. The gamma motor neurons (GMNs) by themselves are not able to produce enough force to affect the type of testing changes that are perceived.

When a Type I test reveals a conditional inhibition of a muscle, it suggests that the central integrative state (CIS) of those AMNs is inhibited in spite of the conscious descending excitatory inputs present which are considered a constant from one muscle test to the next. Performing a Type I test causes a stretching of muscle spindles which should excite the AMNs and tend to reinforce the descending conscious inputs to the AMNs for that the muscle. With the exception of the primary muscle spindle la monosynaptic pathway, we know that (MR) activity at the spinal cord level affects muscle function through interneurons (INs) which affect the gamma loop. MR reflexes do not affect the AMNs directly, but affect muscle function through the GMNs and the gamma loop. (This is in direct contrast to nociceptors which synapse on INs which affect the AMNs directly and bypass the gamma loop.)

When a muscle shows a Type I weakness only, we can assume that the muscle spindle (which is being stretched from the onset of the test) is not adequately firing through the monosynaptic la pathway to the AMNs to reinforce the constant conscious descending facilitation of the AMNs adequately to overcome the tester's force. In a Type I only conditional inhibition, this is because the local CIS of the GMNs is being inhibited from the segmental MR inputs (*e.g.*, joint fixation with inadequate joint mechanoreceptor firing, active intercostal trigger points, *etc.*). This GMN inhibition results in a laxity (lengthened position) of the intrafusal fibers and slack in the muscle spindle, hence a lack of responsiveness to the stretching of the muscle as the test is initiated. There is a lack of reinforcement *via* the la reflex pathway to the AMNs which are thereby unable to keep the muscle firing up to the demands of the test. Thus, a Type I only pattern of conditional inhibition is present.

A Type II inhibition, then, is thought to be associated with inhibition of the AMNs even when the gamma loop is allowed to be fully facilitated by conscious descending pathways (both the constant pathways which always fire during any type of muscle test AND the additional pathways which are facilitated when the patient is allowed to recruit them during the isometric phase of the test). In other words, many descending pathways (reticulospinal, rubrospinal, cortico-cerebello-vestibulospinal, *etc.*) are thought to be recruited and activated when the patient is requested to perform a Type II test. These pathways are all thought to work through the gamma loop (and some also affect AMNs directly). Sources of inhibition (*e.g.*, autonomic, endocrine, cranial faults, TMJ, *etc.*) to one or more of these pathways to the

gamma loop are thought to result in an inhibition of the the gamma loop, hence inadequate reinforcement of the la pathway during the Type II test and an inhibition response to the testers added eccentric force at the end of the test. This is thought to be similar to the mechanism of a Type I test. But in a Type I test, the GMNs are being inhibited by local MR reflexes whereas in the Type II test, the GMNs are being inhibited by a descending suprasegmental pathway.

Descending suprasegmental pathways mentioned above which make up part of the corticofugal muscle activation system and operate through the gamma loop are affected by interferring inputs from the hypothalamus, vestibular equilibrium system, *etc.* This interference prevents maximum cortical activation of these corticofugal pathways, yielding inadequate activation of the GMNs and the gamma loop. Hence the la pathway will not be able to respond adequately to the stretching demands at the eccentric part of the muscle test and the Type II test will demonstrate inhibition.

The Type III test is also different in its mechanism. It is observed clinically that the Type III pattern of conditional inhibition is associated with nociceptive activity. It is known that nociceptors affect the AMNs through INs directly and bypass the GMNs and the gamma loop. This pathway is known as the flexor reflex afferent (FRA) pathway and the FRA IN group is sensitive to other segmental and suprasegmental inputs besides nociceptors (in fact to A-beta as well as A-delta and C afferents). The present discussion will focus on the nociceptors.

If a Type III pattern of inhibition is present, the FRA INs, (presumably in lamina VI and the intermediomedial group of lamina VII) are being activated for the FRA response by the nociceptors. These FRA INs both excite and inhibit both flexors and extensors by the flexor withdrawal reflex, the reciprocal extensor inhibition, and the crossed extensor reflexes which affect both flexors and extensors. This FRA IN pool is a major way station for various descending pathways on their route to effect affect AMNs. It is these FRA INs which are presumably impacted by immune and visual (autonomic) reflex imbalances transmitted *via* the medullary reticulospinal tracts (MRST).

Type III inhibition patterns are a result of activation of inhibitory INs which are part of the FRA reflex pathway from nociceptors. These INs pass directly to AMNs and bypass the GMNs and the gamma loop. Nociception is also transmitted to the caudal reticular formation (CRN) where it reflexively, *via* the MRST, descends in the dorsolateral fasciculus to inhibit the effects of incoming nociception at several spinal cord areas throughout the length of the spinal cord.

One might show a Type I and Type II but not a Type III pattern of inhibition if there are no local FRAs active, and if the MRST is functioning normally (from normal brainstem, hypothalamic, and cortical inputs), so that this pathway will be recruited in the muscle test to fire the FRA IN pool for the muscle in question which fires directly to the AMNs bypassing the gamma loop. So this powerful input to the AMNs is thought to be adequate to override the lack of stimulation from the Ia reflex, at least at the level of force required to overcome the tester's pressure in the Type III test.

When a maximum amount of force is necessary as in the Type II test, all AMN activity must be recruited which requires the recruitment of the gamma loop. That would explain why the Type II test, in which the patient exerts more force, could demonstrate a pattern of inhibition while the Type III test, in which the patient exerts less force, would demonstrate a pattern of facilitation.

RESPONSES OF THREE MUSCLE TEST TYPES TO RECEPTOR-BASED CHALLENGE STIMULI

Rubbing or stroking the skin over an area of injury is thought to activate MRs that block nociceptive afferents and facilitate previously inhibited muscles, if the inhibition pattern is due to the nociception. In the presence of excessive nociceptive stimulation, the muscle will demonstrate all three types of weakness to testing, with clinical significance placed on the Type III pattern. The clinician can confirm the nociceptive source of neuromuscular inhibition by activating skin MRs (*i.e.*, by gently rubbing the skin over the injured area) and observing a facilitation of the previously inhibited muscle.

In contrast, pinching over an area of injury, recent or ancient, may also create a facilitation of previously inhibited muscles observed with all three types of testing (or at least Type III if it was the only inhibition pattern present) due to excitation of medullary CRN reticular formation neurons that were previously being inhibited from some other source. This is the key to understanding the nature of Type III inhibition pattern.

Pinching activates the nociceptor – spinoreticular – MRST reflex pathway to descend and inhibit incoming nociception, in a similar but slightly different manner than the local MR effect. The MRST, affecting the same INs as the MRs, is also responsive to hypothalamic – reticulospinal influences which (presumably) include the effects of immune system related effects in the hypothalamus. Type III inhibition patterns have been noted clinically in conjunction with immune system problems. These immune

system problems presumably can be separate from other hypothalamic – reticulospinal influences mediated through the mesencephalic parabrachial nucleus pattern generator neurons that affect pontine reticular formation neurons. These neurons are the origin of the pontine reticulospinal tract (PRST). The PSRT neurons synapse on GMNs (and AMNs) but primarily rely on the gamma loop, hence the effect of inhibition in this pathway results only in Type I and Type II inhibition patterns.

Therefore, it is proposed that an immune system problem registers in the hypothalamus that affects descending hypothalamic – MRST pathways that in turn affect INs that directly affect AMNs (no GMNs) that will be associated with Type III inhibition patterns.

CLINICAL CORRELATIONS OF THE THREE TYPES OF TESTING

Any combination of the three types of weakness may be present in the same muscle. Patterns of neuromuscular inhibition limited to only one of these three test types are often missed in traditionally performed neurological testing. Clinical observations on the significances of the three different types of tests are as follows.

Type I testing is the most commonly employed type of testing by applied kinesiologists. Procedures that normalize weaknesses of this type have been found to include segmental inputs such as restoring afferentation by correction of a fixated vertebral motion segment, manipulation of trigger areas in muscles and other tissues, or treatment of acupuncture meridian therapy points associated with muscle in question. This type of testing is used for general screening. If weakness is found on clinician started (eccentric) testing, then the muscle is subsequently checked with the other two test procedures.

Type II inhibition patterns are clinically associated with a variety of problems that are presumably suprasegmental. These include chemical imbalances such as nutritional needs, hypothalamic monitored activities such as electrolyte imbalances, general autonomic imbalances, vestibular irregularities, temporomandibular dysfunctions, and other mechanical lesions which are suprasegmental in their origin including lesions of the craniosacral respiratory mechanism. (See previous discussion.) The functional neurological effects of these problems modulate GMNs and AMNs function *via* various descending suprasegmental pathways such as the reticulospinal tracts, the vestibulospinal tracts, or the rubrospinal tracts.

Type III inhibition patterns are related to withdrawal reflexes following injury (flexor reflex afferents), allergy and hypersensitivity type reactions, systemic functional endocrine imbalances, and visual motor problems such as functional problems with accommodation reflexes.

ORAL NUTRIENT TESTING

In 1968, Goodheart introduced testing nutritional substances by monitoring muscle testing responses associated with gustatory stimulation by nutritional substances. Placing substances on the tongue, such as nutrients in which the patient is lacking, were associated with a conditional facilitation of otherwise inhibited muscles. Placing offensive substances on the tongue, such as toxic substances, overdosed medications, and food allergens, were found to be associated with a conditional inhibition of otherwise facilitated muscles.

Afferents from the taste bud receptors of cranial nerves VII, IX, and X synapse in the nucleus of the tractus solitarius with ongoing projections to the thalamus, hypothalamus and cortex. Changes in muscle testing outcomes following taste bud receptor stimulation is hypothesized to be associated with changes in the CIS in the hypothalamus, cortex, or both.

This effect is commonly observed, for example, with gustatory receptor stimulation using syrup of ipecac, which induces an immediate and violent motor response which induces the patient to vomit.

Oral nutrient testing is widely used in AK practice to aid the clinician in making the best choice of nutritional substances, medications, herbs, and other substances when there are numerous possibilities from which to chose. It is also widely employed as a screening test to identify which laboratory evaluation may be best suited to a patient. For example, a patient who shows a strengthening response to insalivation of an anti-histamine would be considered a candidate for allergy testing, regardless of what symptoms are displayed. In this manner, the clinician may efficiently identify dysfunctional physiological processes at the root of patients' symptoms, rather than merely give the symptoms a named diagnosis.

A single blinded, uncontrolled pilot study of AK and allergy testing was performed by Schmitt and Leisman (1998). In this study, 19 of 21 foods associated with muscle weakness on oral challenge showed a subsequent elevation of serum IgE, IgG, and/or IgG immune complexes.

Later anecdotal observations indicate two emerging patterns: (1) strengthening on oral antihistamine is a useful screening test for allergies,

and (2) muscle testing with oral food challenges yields false negatives often enough so as to not be reliable to rule out a food allergy. AK identification of any food allergy reaction is a useful indication to perform allergy laboratory testing in a patient who is not responding adequately to other treatment.

THERAPY LOCALIZATION AS A FORM OF SENSORY CHALLENGE

Goodheart (1974) observed that a change in the outcomes of muscle tests could be elicited by having the patient apply their own persistent touching contact to an area of the body in need of treatment. This may be the first recorded recognition that the neurologically mediated effects of touch are diagnostic, as well as therapeutic. The effect became known as "therapy localization" (TL).

Changes observed to occur with TL are hypothesized to be a consequence of alterations in MR afferents from the tissues being stimulated by patient contact. Touching an area of the body increases afferent stimulation from the area, which increases the extent to which that area is represented in brain stem, cerebellum, and cortex. These changes in central representation are reflected as changes in the CIS of neurons in descending motor pathways, affecting the CIS of AMNs.

Therapy localization is extremely valuable in the AK assessment process. Therapy localization allows the clinician to stimulate areas of afferent input to identify those which impact muscle testing outcomes. The appropriate therapy, designed for the receptors whose stimulation alters motor function in a clinically relevant manner, has been found clinically to return the patient's motor system to a predictable pattern. Following treatment, touching the previously corrected area will have no effect on muscle testing outcomes. This tool helps to make AK assessment quick and precise.

ROLE OF THE PARABRACHIAL NUCLEUS IN DISSEMINATED TREATMENT EFFECTS

The observation of therapeutic effects far afield from a locally applied treatment is common to all complementary and alternative therapies. Structural treatment is often associated with improvements in patient

chemistry or emotional states. Biochemical treatment is often associated with resolution of structural or emotional problems. Treatment of emotional problems is often associated with improvement in structural or biochemical symptoms. The treatment of problems in all of these areas is observed clinically to be associated with changes in motor function observable through manual muscle testing. The following discussion proposes the suprasegmental pathways *via* which these changes in motor function may take place.

In the parabrachial nucleus (PB) in the mesencephalic reticular formation are located groups of neurons which are central pattern generators (CPGs). These CPGs, when stimulated, send efferent signals to groups of cells caudally which cause specific patterns of movement to take place. These stereotyped movements include turning about the midline, flexion-extension, and gait type patterns. These whole body patterned movements are activated when the CPG cells fire (Willis, 1985).

The PB CPGs are associated with these patterned movements by sending descending efferent signals to neurons in the pontine and the medullary reticular formations which give rise to the descending reticulospinal tracts (RSTs). The RSTs descend to all levels of the brainstem and the spinal cord and, through interneurons, affect AMNs and GMNs which affect muscle function. The RSTs also synapse on IML neurons which are the primary autonomic efferent neurons. Accordingly, activation of PB CPG neurons have facilitation and inhibition effects on muscles throughout the body as well as autonomic function throughout the body.

The PB CPGs receive direct afferent input from several areas which represent structural, chemical, and mental influences on the CIS of the CPGs, including the cerebellum, hypothalamus, and basal ganglia. These changes in CIS yield changes in the impact of CPG efferents on neuromuscular patterns, yielding changes in patterns of conditional facilitation and inhibition observed in the manual testing of muscles.

The cerebellum receives inputs from MRs throughout the body as well as from the cerebral cortex. Inputs from MRs in somatic tissue will change with changes in the status of structural problems. Inputs from the cerebral cortex will change with changes in mental and emotional status. The basal ganglia also impact the CPG in the PB area, giving rise to the synthesis of inputs this area receives, both mental and structural. The hypothalamus, the major neurochemical transducer of the body, is the basis for chemical inputs affecting muscular and autonomic function. The hypothalamus is also impacted by emotional activity which it transmits onward as muscular and autonomic outflow.

CONCLUSIONS

Muscle testing has been observed clinically to be related to many phenomena. But under all circumstances, muscles will only change strength when the CIS of AMNs is either inhibited (weakness) or excited (strength) during the performance of the test.

Muscle testing, applied with the precision used by AK practitioners, serves as a method of functional neurological assessment. Muscle testing has always been an integral part of the neurological exam. With AK techniques, the ability to assess predictable neurological responses to sensory receptor-based challenges of known value has been amplified. The use of AK can assist the clinician in identifying and treating sources of improper neurologic function in an efficient and accurate manner.

Muscle testing responses to sensory receptor challenges are compared to the predicted normals. These responses guide the clinician in the diagnostic and therapeutic process. Specific types of therapeutic receptor stimulation have two parallel affects in the nervous system. Properly designed receptor activation will normalize afferentation. An additional benefit of normalizing receptor afferentation is the restoration of normal muscle function which increases movement and hence increases joint and other MR activity further increasing afferentation.

Functional disorders can be expressed as many different complaints, ranging from pain to visceral disorders to mental depression. Visceral disorders such as hypochlorhydria, irritable bowel syndrome, and asthma, may be an expression of altered autonomic tone.

Sensory receptor based diagnostic challenges, including nociceptors, mechanoreceptors, visual, and gustatory receptors, may be used to evaluate the functional status of autonomic activity. With these testing procedures, it is no longer necessary to merely describe an organ as dysfunctioning or to guess at its status. Interpretations may be made regarding the SYM or PS imbalance.

The gradual loss of functional integrity of the organism yields a progressive loss of the ability to maintain homeostasis, which may progress to degenerative tissue changes and pathology. This makes the early detection and treatment afforded by AK potentially important. In addition, the diagnostic advantages provided by data derived from the functional neurologic assessment could help the clinician guide therapeutic measures and improve the potential for favorable outcomes. The significant benefit which these methods appear to provide, along with the favorable outcomes of well designed initial studies, warrants further exploration. The validity of

future studies of these methods rests with a proper understanding of their neurophysiologic basis.

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