

# The Neurological Rationale for a Comprehensive Clinical Protocol Using Applied Kinesiology Techniques

Walter H. Schmitt, DC, DIBAK, DABCN

*Presented at the International College of Applied Kinesiology – U.S.A. Meeting  
Marina del Rey, CA. June 2005*

Reference: Schmitt, Walter H., The Neurological Rationale for a Comprehensive Clinical Protocol Using Applied Kinesiology Techniques. Proceedings of the I.C.A.K. - U.S.A. Annual Meeting, Volume 1, 2005-2006. p. 157-191.

**Abstract:** A procedural protocol for the application of applied kinesiology techniques is presented. It is based on neurological and biochemical principles and thirty years of clinical observations of comparative applications of techniques. Short summaries of each section are included prior to the section to enable a brief review of the information.

**Key indexing terms:** applied kinesiology, manual muscle testing,

## INTRODUCTION

Applied kinesiology (AK) techniques can be interpreted in light of their effects on neurological function.<sup>1</sup> In this model a change in manual muscle testing response is ultimately due to changes in facilitation/excitation and inhibition at the anterior horn motorneurons (AHMNs) of the muscle in question.

Fundamentally, AK is all about excitation and inhibition of neural pathways. AK is a series of sensory receptor based diagnostic challenges followed by monitoring of manual muscle testing outcomes. All AK techniques are about creating sensory receptor stimulation that results in a net effect of excitation and inhibition leading to more optimal neurological function. These positive changes can be observed through somatic windows by changes toward normal in muscle facilitation and inhibition (muscle balance, range of motion, deep tendon reflexes) and through various autonomic windows that can also be monitored (pupil light response, blood pressure, heart rate, etc.) The changes in muscle testing responses are termed conditional facilitation and conditional inhibition dependent on the conditions present at the instant of the muscle test. In this paper, we will simply use the terms facilitation (or excitation) and inhibition. We will also use the terms strong and weak to denote muscle testing responses which represent AHMN facilitation/excitation and inhibition, respectively.

Many neurological pathways lead to AHMNs and impact the ability of the associated muscle(s) to respond to the demands of the manual muscle test. Through thirty years of observing both somatic and autonomic windows as well as symptom responses of patients, a step-by step clinical protocol for AK emerged. This protocol presents the most optimal application of AK techniques considering the neurological hierarchy of pathways that influence the AHMNs. The clinical application of the protocol is presented in a new reference manual that is coauthored by Kerry McCord, DC, DIBAK and this author.<sup>2</sup> McCord has also authored a paper presenting four diverse case histories each treated by following the same protocol.<sup>3</sup>

One of AK's most important and attractive features is that it is an open system. That is, AK allows for evaluation of the impact on a patient's nervous system of virtually any modality or therapy. This protocol is no exception to the openness of the AK approach. The hierarchy presented here is comprehensive, but the therapies listed are not all-inclusive. For example, evaluation and treatment of food allergies is presented at a certain point in the protocol as identification of the existence of a food allergy response is imperative for optimal progression through patient assessment and treatment. Yet any additional allergy diagnostic activity may be added and, although one particular allergy treatment procedure is included, any effective allergy treatment procedures may be used. The key is when to employ various diagnostic and

therapeutic steps. This protocol is open to the clinician's choice of techniques as it is based on fundamentally sound principles of the basic sciences of neurology and biochemistry.

Similarly, at the point in the protocol for emotional recall assessment, there are many emotional treatment procedures available to the practitioner. Choose the emotional technique that you have found to be the most effective, but employ it at the designated time in the protocol for optimal effects.

Richard Belli introduced the terms autogenic facilitation (AF) <sup>4</sup> and autogenic inhibition (AI) <sup>5</sup> to represent manual manipulation of muscle spindle cells or Golgi tendon organs to cause a net facilitating or inhibiting effect, respectively, at the anterior horn. Manipulating muscle spindle cells in the belly of the muscle to create a net facilitation can be used to help determine the status of a muscle's AHMNs.

The resulting net effect of facilitation and inhibition on a neuron at any given moment has been called the central integrated state of that neuron. Clinically speaking, we see three possible states for a particular muscle's AHMNs that can be reflected by muscle testing:

- 1) inhibited AHMNs (muscle tests weak)
- 2) normally facilitated AHMNs (muscle tests strong and AI weakens)
- 3) over facilitated AHMNs (muscle tests strong and AI doesn't weaken)

Each of these three states represents the central integrated state of the AHMNs and is a sum of the net effects of all pathways to those neurons. Hence, there are various combinations of excitation and inhibition arriving at the anterior horn that are derived from either direct sensory input (more or less segmental in nature) or from a distant pool of neurons, hence ascending and descending pathways including suprasegmental pathways. Suboptimal muscle function as seen in AK can be interpreted as a neuron pool somewhere that is:

- 1) inhibited and in need of facilitation (or increased afferentation)
- 2) over facilitated and in need of inhibiting

All of the above assumes neuron metabolism within normal ranges of function. Deranged neuronal metabolic function results in transneuronal degeneration (TND) <sup>6,7</sup>. The presence of TND would suggest an approach designed to address the metabolic function of the neurons as the primary goal rather than the typical AK approach that primarily addresses facilitation and inhibition. Although the clinical approach to TND (which is at the core of the chiropractic neurology approach) is the stuff of another paper, the concepts may be summarized here for contrast with the AK approach that is focus of this paper.

- 1) TND neurons may be too close to firing threshold (cells in danger of over stimulation and death by apoptosis) and these cells need to be stimulated with inhibitory activity to drive them away from firing threshold.
- 2) TND neurons may be in need of stimulation by excitation to increase their metabolic rate and make more intracellular protein (for its negative charge) to drive the cell away from threshold, but the level of stimulation must not be so close to threshold that stimulation will kill them.

AK procedures may be combined with the awareness derived from observing of autonomic windows on neurological function to address these issues, but as mentioned, that discussion is for another paper. However, in either approach, AK (the facilitation – inhibition approach) or chiropractic neurology (the neuron metabolic state approach), muscle testing can be seen as a somatic window on neurological function and is a valuable tool in assessing the patient

and directing therapy to the most optimal outcome. In fact, all of our therapies, and each and every therapy of any discipline can be seen as having their clinical influence by affecting one or more of the above states of neural activity.

## BEGINNING PROCEDURES

1. Postural Analysis
2. TS Line Analysis
3. Test Muscles - Find Weak Muscle(s)
  - a. Measure, Measure, Measure (ROM, Pain, etc.)
4. Does Autogenic Facilitation Strengthen?
  - a. No: Use IRT - Rubbing over Area(s) of Injury will Strengthen Weak Muscle
  - b. Yes: Use NSB and/or Set Point Technique for Recent Injury  
Use Origin/Insertion Technique for Muscle or Ligament Injury

Summary: Identify a weak / inhibited muscle first, then fix injuries to restore normal function of the ipsilateral cerebellum, the contralateral cortex, and the descending muscle spindle cell control mechanisms necessary for muscular and postural control. These corrections normalize autonomic, endocrine, and immune responses that are secondary to nociception.

Many AK procedures can be performed using either a strong or weak muscle to determine what procedures are optimal. It is most useful, however, to begin with an inhibited muscle if possible, and then work to identify the source of inhibition. Sometimes, this is difficult or impossible, so we may use a strong indicator muscle and observe for changes due to the effects of sensory challenges.

If a muscle is over facilitated, AI will not weaken the muscle, and this muscle should not be used as an indicator muscle. There are times when all muscles are over facilitated (referred to as “all muscles strong”) and then we must address some systemic source of over facilitation such as an over firing sympathetic nervous system in a fight or flee response. Further discussion of this concept will be left for another time.

The point is that our initial procedures are best served if we can identify an inhibited muscle(s). Postural analysis and temporosphenoidal line (T.S. Line) analysis are great time savers in this effort. Further, reassessment of postural analysis during and at the conclusion of the treatment provides objective feedback for the clinician as to the progress and effectiveness of the treatment session. The first goal, then, is to identify a weak (inhibited) muscle or muscles and then work from there.

Neurological patterns related to injuries creating a need for Injury Recall Technique (IRT) must first be considered first when encountering an inhibited muscle. The theory of IRT was discussed in a previous paper.<sup>8</sup> Simply put, IRT is thought to be an adaptation to an injury that is maintained by an ipsilateral cerebellar and a contralateral cortical response to the muscle (flexor reflex afferent) pattern created by the injury. Some injuries will result in an IRT pattern (an IRT injury) and others will not. An IRT injury on the right side, for example, will cause muscle response that will create a right cerebellar adaptation that will carry this message (via the dentatorubrothalamocortical tract) to the opposite cortex. The cortex will send messages back through the corticopontocerebellar pathway and this cerebellar-cortical-cerebellar loop then becomes active and maintains the muscular and postural adaptation to the injury. The net effect alters (probably by cerebellar effects of descending brainstem pathways – vestibulospinal and/or reticulospinal) muscle spindle control maintaining the adaptation.

The essentiality of optimal muscle spindle cell control cannot be over emphasized. This may be determined by using manual manipulation of the muscle spindle feedback loop to create

a facilitating effect to the inhibited muscle, or AF. If AF does not create a temporary strengthening of the weak muscle, this means that the most powerful reflex – and arguably the most important reflex, the only monosynaptic reflex in the body – is being over shadowed by some other source of inhibition, presumably from descending pathways in adaptation to the injury. IRT injuries must be corrected to restore this muscle spindle control system. Correction of an IRT injury will restore normal AF response to the weak muscle, or oftentimes, the muscle will return to a normal testing response following IRT correction.

Mechanoreceptor (MR) stimulation blocks the effects of nociceptors (NOC) at the spinal cord level, and at higher levels also. Rubbing the skin over the area of any IRT injury that needs treatment will strengthen the weak muscle due to MR stimulation. Rubbing will temporarily negate the effects of inhibition somewhere along the associated pathway (just as we instinctively rub an area we have just injured) even if the injury is ancient. Rubbing the skin over the suspected IRT injury is a quick and efficient way to identify the location that requires IRT.

There are two common IRT injuries that should be addressed at this point, but that may elude the AF screening process. Iliolumbar ligament IRT patterns will often not be seen unless the patient is weight-bearing. It is possible to directly challenge for the IL ligament at this point with the patient recumbent, and this is often a good idea. The other pattern is the hiatal hernia / GERD challenge. Once again, this is often an IRT problem that will not show up unless directly challenged. Although both of these problems will be identified at a later point in the protocol, since they are IRT injuries, the appropriate time to check for and correct them is at this juncture. The history should be your guide here. In the presence of hiatal hernia / GERD, the initial correction (inferiorly directed manipulation of the stomach) and the IRT should be performed at this time. Other associated traditional hiatal hernia / GERD corrections (e.g., dorsolumbar fixation, psoas imbalance) should be deferred until later in the protocol and only be corrected if they are not resolved by other procedures.

The importance of correcting IRT injuries first must be considered in the light of the cerebellar-cortical-cerebellar adaptation loop as well as their muscle spindle cell control consequences. Adaptations to injuries maintained by this loop impact both the cerebellum and the cortex in a significant way. Cerebellar tests such as finger-to-finger, one foot standing, Romberg, hypermetria, etc. will often be seen to move towards normal following IRT correction. Similar tests of cortical activity as well as those using muscle testing and right brain – left brain activity will often be normalized by IRT correction.

This implies (and in fact it is the recommended clinical procedure) that any cortical or cerebellar testing should be performed after (as well as before) IRT corrections in order to obtain a clear view of neurological function. Many neurological signs throughout the body are significantly changed towards normal by IRT corrections; so many in fact, that an initial neurological examination prior to IRT correction can often create misleading information as to the location of the primary area of lesion. Correction of IRT will allow the primary area of neurological involvement to be identified more clearly.

Asymmetrical cortical over stimulation from an injury becomes ingrained in the patient's nervous system and is often mistaken as the commonly found chiropractic neurology pattern referred to as hemisphericity. It might better be labeled as a decreased hemisphericity. Hemisphericity (or decreased hemisphericity) denotes an imbalance between the right and left halves of the cerebral cortex. For example, a right hemisphericity (or right decreased hemisphericity) means involvement of the right cerebral hemisphere.

When one cerebral hemisphere fires, there are three important patterns of muscle activity:

- 1) Ten percent of cerebral neurons fire via the corticospinal tract, corticoreticulospinal tract, and corticorubrospinal tract to muscles on the contralateral side of the body for meaningful, purposeful movement. The other ninety percent of cerebral neurons fire into the ipsilateral brainstem creating two additional patterns: 2) a general increase in muscle tone on that side of

the body, and 3) an ipsilateral increase in muscle tone of posterior (extensor) muscles above T-6 and an increase in muscle tone of anterior muscles below T-6. (Recall that in the embryological development of humans, anterior lower limb muscles are extensors as they have migrated from the posterior location where they would be found in lower animals.)

This third pattern originates in the pontomedullary reticular formation (PMRF) which is excited from above by the ipsilateral cortex. The reticulospinal tract that originates in the PMRF inhibits ipsilateral anterior muscles above T-6 (allowing for the increase in posterior muscle tone above T-6), and it inhibits ipsilateral posterior muscles below T-6 (allowing for the increase in anterior muscle tone below T-6.) For example, when the right cortex fires into the right PMRF, one might find weakness of the right biceps brachii along with increased triceps tone. Similarly, in the lower extremity, one might find a weakness of the foot plantar flexors tone with an increase in the dorsiflexors tone.

Decreased frequency of firing of the neurons in this pathway (as would occur in a decreased hemisphericity) has been observed to result in an increased tone of the anterior muscles above T-6, and an increased tone in the posterior muscles below T-6 on the ipsilateral side. This increased facilitation yields increased inhibition of the antagonistic muscles.

The observed pattern is very similar to stroke antalgia, and also resembles the normal gait pattern. As a result, some or all of the ipsilateral upper limb extensors (and abductors) and ipsilateral lower limb flexors (and adductors) may be inhibited, and unable to meet the demands of manual testing. Hence, with a decreased right cortex and decreased right PMRF, one might find increased biceps brachii tone and increased finger flexor tone accompanied by weakness of the right triceps and right finger extensors. In the lower extremity, this might be seen as increased tone of the right plantar flexors with weakness of the right dorsiflexors of the foot. (This has been called a “pyramidal distribution of weakness”, but it is actually an inhibition of the other descending motor pathways that have lost the ability to perform inhibitory functions that modulate pyramidal influences.)<sup>1</sup>

In summary, when one cortex fires, it causes 1) meaningful, purposeful movement on the opposite side of the body, 2) general increased muscle tone on the same side of the body, and 3) increased ipsilateral muscle tone of posterior (extensor) muscles above T-6 and increased ipsilateral muscle tone of anterior muscles below T-6. The third phenomenon creates a gait-like distribution of muscle facilitation and inhibition.

The first pattern of meaningful movement (on the opposite side of the body from cortical firing) requires the second and third patterns to create stability (on the same side of the body as cortical firing) and allow the movement to happen. For example, consider lifting a heavy ball with the left hand. The right cortex fires to initiate the left-sided activity, but without the right cortex also firing down to create stabilizing muscle activity on the right side of the body, the person would fall over toward the left side rather than be able to lift the ball.

Correcting IRT injuries will often resolve imbalanced afferentation to the opposite hemisphere and signs of a hemisphericity lesion disappear. Often, there is a history of multiple IRT injuries on both sides of the body. This can cause confusing clinical presentations and complicates the diagnosis of hemisphericity lesions. Although there are cases where correcting a hemisphericity pattern will correct an IRT, the IRT should be corrected first and then the patient should be evaluated for hemisphericity patterns. As will be discussed under gait patterns below, there are common mechanical and chemical presentations that also create a gait pattern of muscle imbalance. These, too, may be mistakenly diagnosed as a hemisphericity lesion.

Applications of therapies and rehabilitation procedures aimed at a hemisphericity pattern are often employed by the chiropractic neurologist with remarkable success. However, when the apparent hemisphericity lesion is actually secondary to an IRT injury (or other pattern mentioned later), such treatment is less effective, and even inappropriate in some cases.

Ignoring the IRT pattern will cause the clinician to “paint over rust” with further corrections. Adaptations will be treated rather than primary causes. AK techniques like reactive muscles and strain-counterstrain, that depend on manipulation of muscle spindle cells, become obsolete in light of the IRT correction. Restoring central muscle spindle control mechanisms eliminates the source of the problem identified and treated with these techniques.

In a similar fashion, acute injuries must be dealt with as a priority. (Acute injuries as used here means within approximately one week of the injury, sometimes longer for major fractures or surgeries.) Similar, but different neurological patterns will be maintained in adaptation to acute injuries. Nociceptor Stimulation-Blocking technique (NSB) is the preferred approach when dealing with acute injuries. (Other approaches such as cold laser treatment have been reported to be effective, but we have not seen any neurological comparison between these treatments and NSB. Whether both approaches fully normalize the same pathways is not known.)

When there is a need for IRT or NSB techniques, the interference with normal neurological function is of utmost significance. The presence of either pattern disrupts normal neuromuscular control mechanisms (flexor reflex afferent patterns), autonomic patterns (both locally at the IML and via ascending spinothalamohypothalamic connections to systemic autonomic control areas of the hypothalamus), endocrine patterns (via the same spinothalamohypothalamic connections), and cognitive activity. Any attempt to treat the patient with other therapies in the presence of these underlying disturbances will, at best, be treating adaptations, and is often useless in making any significant changes.

Nociceptor driven spinothalamohypothalamic pathways can create or obscure patterns of hypothalamic-pituitary-endocrine effects and their parallel neuromuscular effects via hypothalamoreticulospinal pathways. The hypothalamus (HPT) interacts with the immune system such that each affects the function of the other: immune system problems alter HPT function, and changes in HPT activity impact the immune response. In a similar fashion to nociception impacting endocrine effects, nociception changes immune system responses. Evaluation of the immune system and endocrine system must be deferred until correction of IRT injuries and other sources of nociception (such as those addressed by NSB or SP techniques) is completed.

There is a nociceptive driven reflex loop from the spinal cord synapse of nociceptors via the spinoreticular tract up to the caudal reticular formation. The large nuclei located in this lower brainstem area (nucleus gigantocellularis, nucleus raphe magnus, etc.) send descending axons back to the spinal cord area of nociceptor synapse and counteract the three effects of nociceptors in the spinal cord. That is, the reticulospinal tracts from these caudal reticular nuclei inhibit incoming nociception, restore muscle balance at the anterior horn motoneurons, and inhibit local IML sympathetic activity. The theoretical basis for NSB technique involves enhancing this pathway by tapping acupuncture head points (mechanoreceptors) in the presence of a nociceptive stimulation in order to restore spinal cord nociceptor transmission, neuromuscular balance, and IML autonomic activity to pre-injury status.

It is important to correct IRT and/or NSB prior to any nutritional testing (with one exception.) In the presence of the need for IRT or NSB, nutritional test results will be obscured by the effects of nociception on various areas. This includes effects in the brain stem (where oral nutrient testing stimuli impact gustatory centers in the nucleus of the tractus solitarius), the hypothalamus (where gustatory messages also synapse and can have autonomic and likely, endocrine effects), the cerebral cortex (where perception is distorted by nociception), and even the spinal cord’s neuromuscular response.

The one exception that allows nutrient testing prior to performing IRT or NSB is to do nutrient testing to see if any nutrient negates an IRT or NSB challenge. This can be a valuable tool in identifying which nutrient(s) will be most effective at reducing the pain and inflammation

related to the injury and to speed the healing of the injured area. Other systemic nutritional testing should be postponed until later in the protocol as indicated.

Set point technique may also be used in either acute or chronic injury patterns, but its application, when indicated in acute injuries, is best done near the beginning of the treatment procedures. The theoretical framework for set point technique is less clear. Clinically, SP technique restores muscle balance, reduces pain, and normalizes the autonomic consequences of an injured area. Hence, it would appear to affect the same three mechanisms as NSB. Whether the primary effects are in the spinal cord, brain stem, or higher levels is, at this juncture, undetermined.

An interesting note is that SP technique directed at a body area has been shown to have the same impact as using percussion over the area (whether applied manually or with a percussor machine.) SP technique will correct the same assessment parameters and create the same clinical outcome as percussion in many cases. (The effects of percussion will also be mentioned below relative to another therapy that may be used to duplicate or replace percussion effects.) This should shed some light on the neurological effects of both techniques.

One of the compelling characteristics of this protocol is that it presents AK as an open system. Following the order of the protocol is the important thing. The technique that is applied to address each step can be the doctor's choice. At this stage of the protocol, following IRT and/or NSB, the use of either SP technique or percussion over the injured area will be successful.

A clinical observation by Gerald Polino, DC<sup>9</sup> related eye position and acupuncture head points (B & E points.) Polino observed that responses to therapy localization (TL) to acupuncture head points would be paralleled by the patient looking in the direction of the same point. This procedure has been shown to be a valuable clinical tool and also sheds light on the various techniques using these acupuncture head points including NSB, SP, and LQM techniques.

Eye movements, directed by extraocular muscle motor nerves arising from cranial nerves III, IV, and VI, are coordinated in the brain stem gaze centers with strong cortical and cerebellar influences. Correction of techniques employing the acupuncture head points can result in normalization of eyes into distortion (EID) patterns. Recall that one of the effects of nociception is to drive the eyes toward the area of injury. NSB, SP, and LQM corrections can normalize the impact of EID eye position changes by neutralizing their source. When the eyes are relieved from responding to an area of injury, many postural adaptations return to normal.

These three cranial nerve nuclei (III, IV, and VI) are all midline nuclei and phylogenetically old. They have strong connections to other midline, phylogenetically old areas throughout the central nervous system. This includes the limbic areas of the brain (emotional brain) where centrally directed nociception is interpreted as pain. It also includes the AHMNs to the intrinsic spinal musculature which have no conscious control, but whose reflexogenic control, part of which is related to eye position, is critical to spinal position and function. There are obvious implications here regarding the spine and responses such as body into distortion (BID) technique that will be discussed later in relationship to "centering the spine" concepts.

## SYSTEMIC NUTRITIONAL FACTORS

5. Test Aspirin, Acetaminophen, Ibuprofen Mix - If Strengthens:
  - a. Check Essential Fatty Acids (BCSO, FSO, EPA, etc.)
  - b. Check EFA Cofactors (B-6, Mg, Zn, Niacin)
6. Test Antihistamine Mix – If Strengthens:
  - a. Challenge Orally for Offender(s) (Allergen)
  - b. IRT Chapman's Reflexes with Offender(s)
7. Sniff Tests – Aldehydes, Bleach, Ammonia

8. Test Nutrients Based on Patient History:
  - a. Vitamin E (Low Back Muscles), Vitamin C (Shoulder Muscles)
  - b. Iron, Folic Acid, Vitamin B-12
  - c. Cholesterol Lowering Nutrients (If Indicated)
  - d. Chondroitin Sulfate – If Strengthens:
    - i. Check Sulfur (Cysteine) & Associated Nutrients
    - ii. Check Blood Sugar Handling (Insulin, Magnesium)

Summary: Identifying and correcting these biochemical imbalances with nutritional supplementation and/or diet are essential for cellular, neuromuscular, and neurological support to encourage proper healing.

Some nutritionally treatable biochemical conditions are so important that they should be addressed very early on in the treatment process, regardless of the presenting symptoms. Each of the systemic nutritional factors in the protocol will have global impact at the cellular level. Each of these factors will also have global impact on muscle testing responses (assuming that injuries are corrected first.) In addition, most of these factors will have a direct impact on nerve function – either by directly supporting neurological metabolism and/or impacting neurotransmitter activity (synthesis or breakdown.)

Biochemically speaking, there are three factors affecting all nutrients that are applicable to both macronutrients or micronutrients. These are: 1) adequate dietary intake; 2) adequate delivery to cells; and 3) adequate metabolism of the nutrient in the cells.

It is difficult to prioritize within the systemic nutritional factors group. Each may be the most important biochemical issue in a given patient. There is, of course, a patient history driven priority. The history gives the best clues as to which nutritional factors to address first. In difficult cases, just work through the list.

It should be noted here, however, that assessment of the oxidative phosphorylation pathways for the synthesis for ATP [citric acid cycle (CAC) and respiratory chain or electron transport chain (ETC)], that could be arguably at the top of the list, are delayed in the protocol until later. The main reasons for this are as follows. Allergies (and chemical hypersensitivities) produce cytokines that are inhibitory to the CAC. Certain interleukins and tumor necrosis factor-alpha will cause the intracellular production of nitric oxide that inhibits the CAC aconitase enzyme thereby truncating the CAC at this step. The patient will test positive for carbon dioxide and CAC nutrients, but the need for supplementation of these nutrients at this juncture will be reduced or negated by the elimination of the cytokine blockade of CAC enzymes. \*\*\*

The ETC is dependent on coenzyme Q<sub>10</sub> (CoQ<sub>10</sub>), iron, and copper. Iron and copper will be addressed in this section when looking at the oxygen carrying capacity of the blood. In fact, it could be appropriate to check for CoQ<sub>10</sub> at this juncture, especially in heart patients, or others with classical CoQ<sub>10</sub> indications (gingivitis, myalgia, history of cholesterol lowering statin drugs.) But little is lost by checking it in conjunction with the CAC slightly later in the protocol.

5. Test Aspirin, Acetaminophen, Ibuprofen Mix - If Strengthens:
  - a. Check Essential Fatty Acids (BCSO, FSO, EPA, etc.)
  - b. Check EFA Cofactors (B-6, Mg, Zn, Niacin)

Prostanoids, which are derived from essential fatty acids (EFA) include prostaglandins (PGs), leukotrienes (LTs), and thromboxanes (TXs). Prostanoids influence inflammation, immune function, platelet aggregation, cholesterol synthesis, and modulate the responses of intracellular chemistry to membrane receptor driven functions. This includes neurotransmitter

(NT) activity. In addition, EFA imbalances are reflected in a variety of muscle imbalances that must be normalized for proper joint support and afferentation that is necessary for healing.

Further, prostanoids produced in the presence of improper EFA balance are highly inflammatory. These inflammatory substances (prostaglandin-2 family and leukotriene-B4 family) depolarize NOCs and cause an increase of potentially pain-causing sensory activity. We must control the prostanoids and negate their peripheral inflammatory effects in order to normalize afferentation from NOCs.

Most EFA imbalances are due to excessive intake of bad fats – rancid fats, saturated fats or most importantly, trans fats. Patients with EFA imbalances are prone to inflammation and tend to be users of aspirin and non-steroidal anti-inflammatory drugs (NSAIDs.) The small intestine takes the primary abuse from both bad fats and NSAIDs. This results in interference with proper absorption of other nutrients as well as inhibition of the quadriceps and/or abdominals, essential muscles for postural support. Although the presence of excess bad fats may be found when investigating cholesterol metabolism just below or digestive function later on in the protocol, the importance of proper EFA metabolism, both for clinical symptoms and cellular function, needs to be addressed at this earlier point.

Any problem will be made worse by EFA – prostanoid imbalance.

6. Test Antihistamine Mix – If Strengthens:
  - a. Challenge Orally for Offender(s) (Allergen)
  - b. IRT Chapman's Reflexes with Offender(s)
7. Sniff Tests – Aldehydes

Allergic reactions and chemical hypersensitivities result in mast cell release of histamine and other inflammatory agents. As mentioned above, cytokines from allergies (or other immune responses) will inhibit the CAC and consequently energy production. Histamine is a NT with potential global impact on cortical function as a part of the extrathalamic cortical modulatory systems (formerly called the reticular activating system.) Histamine neurons arise from the ventral posterior area of the hypothalamus (HPT) and project to nearly every region of the central nervous system as well as extensive projections to the cortex.<sup>10</sup> Clinically, we have observed that histamine reactions induce right brain – left brain activity in muscle testing responses.<sup>11</sup> These hemispheric imbalances must be addressed in order to obtain a clear picture of hemispheric dominance, so important in the chiropractic neurology approach.

Histamine is one of the most (some say, “the most”) powerful stimulator of the adrenal glands. Histamine produced by allergic reactions will disrupt adrenal activity, causing (resulting in) a hyperadrenia response initially, and possibly causing or aggravating a hypoadrenia further along the General Adaptation Syndrome progression. Adrenal problems, or any endocrine dysfunction, are best evaluated after correcting allergic responses and normalizing histamine levels.

Histamine is also one of the substances that depolarizes NOC resulting in an increase of potentially pain-inducing sensory activity. Therefore, we must neutralize the effects of histamine in order to normalize afferentation from NOC in the same manner that we must control the prostanoids for both their effects on both intracellular modulating and peripheral inflammation.

Food allergies and chemical sensitivities stress the immune system (thymus and spleen) which is why we address them prior to immune function. Detoxification of excess histamine stresses the spleen and liver, uses methyl groups related to sulfur amino acid metabolism that is essential in joint repair (as discussed below), and can deplete reserves of vitamin C. Degradation of histamine uses up the nutrients B-6 and folic acid. Both are essential for neurotransmitter (NT) synthesis. B-6 is necessary for ammonia metabolism, in particular as a transaminase enzyme (transports ammonia groups) activator. Ammonia groups are necessary for synthesis

and degradation of amino acids as well as urea cycle function. Folic acid is important for many vital functions, not the least of which is cell replication. Its deficiency can also cause right brain – left brain imbalances and switching related to the thyroid. All of the B-6 and folic acid related functions can be compromised in the presence of histamine mediated allergic reactions.

As with improper fat intake, food allergies create distress in the GI tract. Although any GI tract organ (or any body tissue for that matter) may be adversely affected by food or other histamine mediated allergies, the small intestine is the primary receiver of the abuse. As with bad fat intake, problems with nutrient absorption and weakness of the quadriceps and abdominals are once again implicated.

Chemical hypersensitivities deplete antioxidants, particularly selenium. In the presence of *Candida Albicans* or in other mycoses, the acetaldehyde produced by the fungus depletes molybdenum, an essential trace mineral necessary for aldehyde oxidase, xanthine oxidase, and sulfite oxidase enzymes. Impaired xanthine oxidase function interferes with ammonia elimination via uric acid and the build up of ammonia will shift NT functions, most notably, driving glutamate to glutamine instead of GABA and aspartate to asparagine. Impaired sulfite oxidase function interferes with the conversion of sulfite into sulfate. Sulfate is essential for liver detoxification as well as cartilage synthesis.

We check for and correct the need for Mo early on, helping many other functions further down the list. Also, in the presence of digestive fungal overgrowth, supplying Mo will help metabolize local acetaldehyde, a significant irritant of small intestine function. It is prudent to supply Mo in these patients prior to attempting to correct the digestive dysfunction.

Allergies and hypersensitivities can cause or aggravate any problem, any problem at all, regardless of whether or not the patient's symptoms seem to be related to typical allergic reactions.

## 7. Sniff Tests – Bleach, Ammonia

The need for Mo will usually be associated with a positive aldehyde sniff test and ammonia sniff test. The bleach sniff test may also be positive. The ammonia sniff test opens the door to assessing amino acid metabolism. The bleach sniff test is an excellent screening test for free radical pathology and the need for additional antioxidants beyond those already tested.

A positive ammonia sniff test causes alterations in amino acid synthesis and degradation and is usually accompanied by improper neurotransmitter synthesis of such important NTs as GABA, glycine, glutamate, and aspartate. Protein synthesis, essential for tissue repair and healing as well as intracellular (especially neuronal) metabolic function, depends on proper ammonia metabolism.

A positive bleach sniff test suggests free radical pathology which is a state of over oxidation of the tissues. This test is placed here prudently to assure the protection of tissues from oxidative free radical activity prior to instituting therapies which are designed to increase oxygen supply to tissues (red blood cell synthesis nutrients) and oxidative metabolic pathways (CAC and ETC.)

A positive bleach sniff test leads to consideration of both sulfur amino acid metabolism and antioxidant needs. Sulfur metabolism is discussed more thoroughly below under chondroitin sulfate and joint problems. In the absence of joint symptoms, a positive bleach sniff test will guide the clinician into this critical area of body chemistry mentioned below including homocysteinemia.

Free radical pathology is often driven by allergies and poor EFA metabolism, two issues that have already been addressed. So the bleach sniff test may be already negative by this juncture. If not, assessment of free radical chemistry and the antioxidants required to negate it are necessary at this point. This may include vitamins E and C, but we include these two

important nutrients in a separate step immediately below since their need may be present even in the absence of a positive bleach sniff test.

Neurological problems, both named and unnamed, are associated with a positive bleach sniff test. This may be due to the effects of homocysteinemia (homocysteic acid) on neuron metabolic function and/or the damaging effects of free radical pathology.

The long term devastating effects of homocysteinemia as a risk factor for neurological disease, heart disease, and cancer are well known. It is to the benefit of all homocysteinemia patients to have this problem identified as soon as possible in their lives so that counter measures may be put in place to negate the consequences. A positive bleach sniff test (and/or assessment of cartilage synthesis soon to follow) will guide the clinician to identify homocysteinemia in many patients who would otherwise continue to suffer the silent, degenerative effects of this condition leading to pathology. Screening for elevated homocysteine will also give us an early identification of important nutrient needs for other metabolic and NT activities including folic acid, vitamin B-12, magnesium, and vitamin B-6.

Any inflammatory problem, painful or not, visible or microscopic (such as autoimmune and neurological diseases) will likely show a positive bleach sniff test. Tissue healing response (of any unhealthy tissue) is dependent on removing the free radical destruction of the tissues by providing appropriate antioxidants. Free radicals will also interfere with the citric acid cycle and energy production as mentioned previously which is another reason that we perform the bleach sniff test prior to CAC assessment.

Correcting a positive ammonia sniff test and/or bleach sniff test will clear the way for proper neurochemical function that is essential for full neurological impact of subsequent structural therapies.

#### 8. Test Nutrients Based on Patient History:

##### a. Vitamin E (Low Back Muscles); Vitamin C (Shoulder Muscles)

Vitamin E and C are essential anti-oxidants. To supply them without first addressing the sources of oxidative (inflammatory) activity is to place the cart before the horse. So we place the testing of these nutrients after first addressing EFA imbalances and allergies / hypersensitivities. In the same hierarchy, we check for E and C (and the bleach sniff test) prior to testing for oxygen supplying nutrients to insure that increasing oxidation does not increase oxidative stress. The effects of normalizing antioxidants will also be seen by decreasing NOC activity in the presence of tissue inflammation.

Dr. Goodheart learned in the late 1970s that these two antioxidants were associated with right brain (vitamin E) and left brain (vitamin C) activity.<sup>12</sup> Once again, hemispheric imbalances may be manifested by the requirement of these two nutrients. Therefore, attempting to determine hemispheric activity without considering these vitamins may create an incomplete or even false picture.

Vitamin E is important for so many low back and pelvic muscles that to leave it out of the patient's program is to perpetuate musculoskeletal problems. The same is true for vitamin C and shoulder problems. So early on in the care of these symptoms, we test for these important antioxidants. Supplementation will help the associated muscle(s) maintain correction as well as provide the obvious biochemical effects.

#### 8. Test Nutrients Based on Patient History:

##### b. Iron, Folic Acid, Vitamin B-12

Oxygen supply to the tissues is of course, essential. But we first want to make sure that we have reduced any sources of excess oxidation (EFA, allergies/hypersensitivities, antioxidant

depletion.) So we place the assessment of the oxygen carrying capacity of the blood here. But we also place the oxygen supplying nutrients ahead of CAC and ETC assessment since adequate oxygen is necessary for these oxidative phosphorylation pathways.

Iron will first be depleted from reserves, then from muscles, and finally there will be a decrease in red blood cell (RBC) production. Aerobic muscle weaknesses and lack of muscle stamina, hence lack of treatment holding capacity, are present in iron deficiency.

Folic acid and B-12 are now evaluated. (We may have already identified the need for these two nutrients if a patient demonstrates excessive histamine and/or homocysteinemia.) It must be recalled that these two nutrients are not only necessary for RBC production, but for WBC production as well. Therefore, we check for these nutrients prior to immune assessment below. As mentioned, folic acid deficiency can also cause right brain – left brain imbalances and switching related to the hyoid. Folic acid is essential in the synthesis of a number of NTs including the catecholamines (dopamine, norepinephrine, and epinephrine) and the indoleamines, serotonin and melatonin. Iron plays a role in the synthesis of indoleamines as well. It is interesting to note that iron is also necessary for the CAC, and metabolites from the CAC are necessary for several other important NTs including glutamate, GABA, and aspartate.

#### 8. Test Nutrients Based on Patient History:

##### c. Cholesterol Lowering Nutrients (If Indicated)

Cholesterol problems may not seem as important in the hierarchy, yet recent awareness of the clinical findings of elevated cholesterol makes it important to evaluate fairly early. When cholesterol is elevated, there is a secondary muscle weakness present that will be recurrent until the cholesterol is addressed. In some patients, screening for cholesterol farther down the protocol (along with other liver detoxification procedures) might be appropriate, but if elevated cholesterol is known, it should be addressed earlier to encourage a more effective patient neuromuscular response.

Additionally, some of the nutrients (e.g., vitamin A, niacin, betaine) necessary for cholesterol metabolism will be important for other body functions, and if not checked here, they might not otherwise be checked until the associated muscle weakness recurs. Cholesterol metabolizing efforts are aided by proper EFA balance, already addressed.

When treating elevated cholesterol, a very common finding is large intestine toxicity. In light of elevated cholesterol, we might jump ahead and evaluate digestive activity, or at least large intestine function at this time, and then return to this point in the protocol.

#### 8. Test Nutrients Based on Patient History:

##### d. Chondroitin Sulfate – If Strengthens:

- i. Check Sulfur (Cysteine) & Associated Nutrients
- ii. Check Blood Sugar Handling (Insulin, Magnesium)

If the patient displays cartilage or connective tissue disorders, assessment of nutrients for joint repair must be done at this juncture if any progress is to be made. A need for these nutrients will often result in muscle imbalances that may perpetuate joint instability and altered ranges of motion interfering with sensory activity arising from affected joints and slowing the healing process.

Normalizing sulfur metabolism will lead the clinician into looking at sulfur amino acid metabolism and the methylation process that, when impaired, is usually related to elevated homocysteine and increased risk for heart disease, cancer, and neurological degenerative disorders such as Alzheimer's disease and Parkinson's disease.

Homocysteine converts to homocysteic acid, a potent excitatory neurotoxin. In the presence of homocysteic acid, metabolically challenged neurons will be placed at increased risk of metabolic exhaustion from over stimulation; even normal excitatory activity may place some neuron pools at risk for cell death by apoptosis. All manipulative therapies have excitatory and inhibitory effects. If the homocysteine patient is experiencing excitatory neuronal stress, some therapies will likely over stimulate neuron pools with short-lived success at best, and irreversible neuron damage at worst. Correction of sulfur metabolism is critical in the clinical course of these patients. Overlooking it can be neurologically disastrous, especially in patients who receive the powerful neurological effects of AK therapies. This is the second step included early on in this protocol to identify and correct the disastrous effects of elevated homocysteine.

When finding a need for addressing blood sugar handling problems as part of a joint problem, it is a good idea to address them and sulfur at the same time. So it may be appropriate to jump ahead in the protocol to the assessment of hyperinsulinism and then return to this point in the protocol.

Sulfur is also necessary for liver detoxification, especially for detoxification of steroid hormones, NSAIDs, and many other drugs. Finding a need for sulfur at this point in the protocol will lay a good foundation for addressing liver and/or hormone problems further downstream.

## SYSTEMIC STRUCTURAL FACTORS

9. Is TL to K-27 Positive?
  - a. Straight TL – Cranial – Pre-Test Imaging (Go to 11) – Immune or Mechanical?
  - b. Crossed TL – TMJ – IRT to TMJ (Go to 10) – Treat Immune Circuit
  - c. Dorsal Crossed TL – Use Tooth Techniques
10. Does TL to TMJ Strengthen Weak Muscle? (and/or) Is TMJ Present with IRT?
  - a. Right TMJ – Thymus (or Lower Sternum)
  - b. Left TMJ – Spleen (or Lower Sternum)
  - c. Check Nasosphenoid Cranial Fault
  - d. Check Temporoparietal Jam
  - e. Check Sphenoid Compression Fault
  - f. Correct TMJ / TMJ Muscles – Correct with IRT and/or Mechanically
11. Does Pre-Test Imaging Strengthen? If Yes – Check Cranial Bones
  - a. If IRT Positive:
    - i. Right Side – Check Thymus (or Lower Sternum)
    - ii. Left Side – Check Spleen (or Lower Sternum)
  - b. If No IRT – Make Mechanical Correction

Summary: The three K-27 switching patterns are related to cranial faults or TMJ faults. Cranial faults may be mechanical or secondary to immune system involvement. TMJ faults may be mechanical (related to cranial faults, TMJ muscle injuries, or tooth problems) or secondary to immune system involvement. About 80% of cranial and TMJ faults are immune related. The immune system affects hypothalamus activity that then becomes manifested as a cranial or TMJ fault with the consequent muscle testing patterns.

Switching is related to uncoupled cervical motion. In the presence of a positive K-27 TL, there will be a cervical subluxation (often C-2) that displays an uncoupled pattern. The uncoupling is secondary to a segment in the cervical spine serving two masters – one being its relationship to its neighboring vertebrae and the other being the aberrant muscular effects caused by a cranial fault or a TMJ fault. Therefore, no cervical adjustment should be made prior to the correction of switching and/or cranial or TMJ faults.

In the presence of cranial faults, pre-test imaging (PTI) is positive. To understand the theory behind PTI, one must understand how the cortex, cerebellum and muscles interact with each other. Communication between the cortex and the contralateral cerebellum and muscles include feedforward (FF), feedback (FB) and efferent copy (EC) mechanisms. Oftentimes, all three pathways are firing during a single motor event.

For simplicity's sake, it might be said that FB is the message received from the peripheral muscle feeding back into the cerebellum. This would occur whether the message originated from the brain or outside the body. FF is a message initiated in the cerebellum and going to the cortex. EC activity starts in the cortex and sends messages to the muscles and the cerebellum at the same time, that is, it sends copies to each. This utilizes the "error comparer" duty of the cerebellum that gets the message from the cortex and then waits for feedback from the movement to compare it with what was intended. If there is a discrepancy between what was intended and what actually occurred, then it will be noted in the cerebellum. When you imagine doing a movement you can actually do, like a muscle test, there is an efferent copy activity. (This is in contrast to imagining a movement that you could not actually do – such as flying - which is called body space imaging.)

It is proposed that cranial faults are neurologically mediated via dural nociceptors that fire powerfully into the cervical spinal cord, and then from cervical afferents into the cerebellum<sup>13</sup>. In the presence of a cranial fault, the associated disruptive input to the cerebellum results in aberrant cerebellar activity that presumably interferes with muscle controlling pathways (i.e., inhibited muscles) and imagining doing the test invokes enough of an EC response to override the interference with the muscle test resulting in a positive PTI. So it is proposed that PTI may be a test of interference to the cerebellum from cranial fault sensory activity integrity during a muscle test that can be overridden by imaging (EC to the cerebellum and the muscle) the movement.

Clinical observations of this author have shown that about 80% of cranial faults and TMJ faults are related to immune system problems. These adaptive (i.e., not actual primary mechanical lesions) cranial and TMJ faults will show up as IRT patterns – as if the cranial bone or the TMJ had been injured. That is, they will TL with the neck in extension to create an inhibition of a strong indicator muscle. The other 20% are mechanical in nature and require mechanical corrections. This includes mechanical cranial fault corrections or mechanical TMJ muscle corrections (usually involving IRT correction to one or more TMJ muscles attachments.)

The immune system problems spoken of here are related to weaknesses of the infraspinatus (thymus), lower trapezius (spleen), or pectoralis minor (parotid gland<sup>14</sup> and/or chemical sensitivities, including heavy metal toxicity.) The treatment that is required in most of these patients is rubbing the related Chapman's reflex.

Later in the protocol, endocrine problems are addressed. Balancing endocrine function will include normalizing adrenal glucocorticoid activity, in particular, cortisol. High cortisol levels will suppress or inhibit immune system function, including the thymus, the spleen, and gut-associated lymphoid tissues (GALT). Cortisol levels will increase as a result of our treatments in many patients and decrease as a result of our treatments in others. We must improve thymus and spleen function, prior to treating the endocrine system, so that our immune tissues can withstand any increased glucocorticoid activity we create. If we do not address the immune system prior to affecting adrenal activity, we run the risk of further suppressing immune system function by our endocrine-directed treatment causing both lowered immunity and increased symptomatology including emotional effects discussed below.

When the immune system is dysfunctional, there will be an impact on the hypothalamus (HPT.) This is the area that psychoneuroimmunology and neuroimmunomodulation research addresses. When the immune system is dysfunctional and the HPT responds, HPT connections to the mesencephalic reticular formation (RF) cause the manifestation of the immune system

problem to be seen in several ways. One factor observed is related to changes in TMJ muscle function and consequent patterns of body activity that are referred to as centering the spine patterns.<sup>15</sup> HPT adaptations to endocrine dysfunction may be manifested in the same manner (i.e., TMJ disturbance and/or muscle imbalance.) Another observation will be seeing changes in autonomic function. These include neurological signs such as alterations in pupillary reflexes and their fatigability that is mediated by cranial nerve III in the mesencephalon. They also include digestive problems such as ileocecal valve syndromes which can be monitored via muscle testing.

In the presence of immune system problems, the HPT, via descending pathways, synapses into the mesencephalic RF of the brainstem, and the effect appears to be inhibitory to the cells of the mesencephalon. (This becomes important when evaluating and treating aberrant metabolic states of the mesencephalon, the topic of a later paper as mentioned above.) The mesencephalic RF contains cell bodies that are affected by the HPT and includes the mesencephalic nucleus of cranial nerve V, the primary sensory cell bodies that arise from MRs of the TM joints and muscles. Due to interconnections within this area of the nervous system, the entire mesencephalon is affected by the presence of these sensory TMJ cell bodies and vice versa. This may very well be the mechanism whereby we see TMJ faults appear as secondary to immune system problems.

Among many other important structures in the mesencephalic RF are pattern generator cells located in the parabrachial nucleus area. That is, these nuclei (interstitial nucleus, prestitial nucleus, nucleus precomissuralis) fire down to the lower RF and the spinal cord resulting in groups of muscles firing in preprogrammed movements such as flexion and extension or rotation around the midline.<sup>16</sup> Mesencephalic involvement, via these pattern generator cells, creates muscle weakness patterns that can occur anywhere in the body. These include bilateral muscle weaknesses,<sup>17</sup> gait patterns, spinal flexion and extension patterns, etc.

The neurological awareness of cranial faults is also via the trigeminal nerve (V). The dura is innervated exclusively by nociceptors and these directly fire into the descending (cervical) nucleus of the trigeminal nerve affecting cervical muscle imbalance, hence creating uncoupled cervical motion. The altered cervical afferentation is carried directly to the cerebellum and the cerebellum carries these messages to the contralateral mesencephalon via the dentatorubral tract.

Hence, cranial and TMJ faults that are secondary in nature are associated with the mesencephalon. The mesencephalon, secondary to inputs from the HPT, creates muscle patterns that appear the same as if they originate in either a cranial or a TMJ fault. The cranial or TMJ will show up as IRT faults rather than by other indicators. That is, TL to the cranial bone or the TMJ with the neck in extension will cause an inhibition of a strong indicator muscle.

When the immune system is dysfunctional and the HPT reacts, the effects on the mesencephalic RF create muscle imbalances and IRT cranial faults and TMJ faults. Oral HPT tissue negates any muscle weaknesses as well as indicators of cranial and TMJ faults. The oral response to HPT tissue suggests that these findings are secondary to immune (or endocrine) dysfunction.

There also appears to be a sidedness to immune system problems. Thymus dysfunction is manifested in right-sided cranial and/or TMJ problems. If TL to a right-sided cranial or TMJ with the neck in extension is positive, there will be a weak infraspinatus on the right, possibly bilateral. Spleen problems are manifested in left-sided cranial and/or TMJ problems. If TL with the neck in extension to a left-sided cranial or TMJ is positive, there will be a weak lower trapezius on the left, possibly bilateral. Chemical hypersensitivities (and parotid problems) are manifested by either right-sided or left-sided cranial / TMJ faults and are accompanied by TL to the lower sternum and a bilateral pectoralis minor weakness. It appears that there are no specific patterns of muscle inhibition associated with any of the three immune patterns other than the

infraspinatus, lower trapezius, and pectoralis minor involvements. That is, any muscle weaknesses may be secondary to immune involvement. So important is this that no local problem (other than the systemic effects of injuries) should be addressed without first checking for cranial or TMJ patterns secondary to an immune problem.

The metabolic state of the mesencephalon may be observed by looking at two simple parameters: TMJ opening range of motion and pupillary light reflex responses. Measure the opening width of the TMJ (three knuckles of the less dominant hand should fit between the upper and lower central incisors.) Also measure the time it takes for the right and left pupillary light responses to fatigue. In most people, one side will fatigue more rapidly. After treating an immune system Chapman's reflex, recheck the TMJ opening width and the pupils. If the mesencephalon was over firing, and the TMJ reflexes were enhanced, then inhibiting the mesencephalon should allow the TMJ to open wider. If the mesencephalon was over firing to the point of fatigue, then inhibiting the mesencephalon by treating the immune system should cause a decrease in the fatigability of the pupillary light responses. You will note that the TMJ range of motion is a bilateral effect. Since the mandible is like a bucket handle, you cannot affect one side without affecting the other.

In the case of the pupillary light responses, treating the spleen will have an affect on the left pupil reflex and treating the thymus will impact the right pupil response. Treating the lower sternum area could affect either or both pupil reflexes.

Our clinical observations on the net effect of treating immune system Chapman's reflexes appears to be consistent with a shift toward inhibition in neurons in the mesencephalon: treating the thymus reflex results a shift toward inhibition in the right mesencephalon, treating the spleen reflex results a shift toward inhibition in the left mesencephalon, and treating lower sternum / parotid problems may result in a shift toward inhibition in either right or left mesencephalon.

The mesencephalon relates to emotional events via the mesolimbic system connections. Emotional states may be driven by primary mesencephalic imbalances. It is interesting to note clinical correlations of practitioners with totally different view points. In a number of systems that relate organ function to emotion, thymus (and/or triple warmer meridian) dysfunctions have been tied to hopelessness and despair. This author has observed on many occasions that treating the thymus can change a patient's emotional outlook from hopelessness / despair to a more reasonable state within just a minute or two of rubbing Chapman's reflex for the thymus. It has also been observed, prior to understanding this protocol, that sometimes a treatment procedure would cause a an emotionally fragile patient to abruptly decline into tears, and that treating the thymus would be the only thing that would pull the patient out of the emotional tailspin. An over firing right mesencephalon will drive the right mesolimbic pathway and activate the emotional areas for suffering in the right limbic system. Treating the thymus will affect the hypothalamus which inhibits the mesencephalon. This leads us to a neurological understanding of how and why treating the thymus is therapeutic for hopelessness and despair. Similar models may be advanced toward an understanding of other organ – emotion relationships.

#### 9. Is TL to K-27 Positive?

- c. Dorsal Crossed TL – Use Tooth Techniques

#### 10. Does TL to TMJ Strengthen Weak Muscle? (and/or) Is TMJ Present with IRT?

- c. Check Nasosphenoid Cranial Fault
- d. Check Temporoparietal Jam
- e. Check Sphenoid Compression Fault
- f. Correct TMJ / TMJ Muscles – Correct with IRT and/or Mechanically

11. Does Pre-Test Imaging Strengthen? If Yes – Check Cranial Bones
  - b. If No IRT – Make Mechanical Correction

About 20% of cranial faults or TMJ faults are related to problems that must be mechanically corrected. This is, of course, when there is a mechanical basis for the lesion. The clue that a cranial fault needs mechanical correction is that no IRT pattern (TL with the neck in extension) will be present. TMJ mechanical corrections include cranial fault based TMJ problems, IRT to TMJ related muscles, and reactions from tooth involvement. The three TMJ related cranial faults (nasosphenoid, temporoparietal jam, sphenoid compression), TMJ muscle imbalances, and tooth problems all create aberrant mechanical feedback into the TMJ and require correction to restore normal TMJ mechanics.

All of these mechanical faults create abnormal sensory receptor stimulation, either MR and/or NOC. The NOC sensory fibers synapse in the descending (spinal) nucleus of V. The MR sensory fibers (from TMJ joint and muscles), with their cell bodies in the mesencephalic nucleus of V, project to their primary synapse in the pontine motor nucleus of V.

The second order NOC neurons affect the anterior horn motoneurons of the cervical spine which changes MR feedback from the cervical spine. The cervical MR sensory activity powerfully feeds into the cerebellum, both directly (no interneuron) through cuneocerebellar fibers and indirectly. Hence, NOCs from the cranial dura, the TMJ, and teeth (as well as the sinuses, eyes, and tongue) have a very strong impact on cerebellar function through this sensory-motor-sensory loop. Problems with any of these structures will have similar powerful cerebellar effects. Theoretically, it is interference from cranial faults (arising from any of these structures) that disrupts the normal cortico-cerebellar (efferent copy) pathway and creates the phenomenon of positive pre-test imaging.

Disruption of cerebellar activity (as suggested by PTI) will often manifest in inadequate neurological expression of intentional movement. This will result in inefficient, uncoordinated, and partially inhibited motoneuron activity for any movement in the body. Simple cerebellar tests such as the finger-to-finger test will be seen as sub-optimal to downright abnormal. Correction of cranial faults, or those factors that influence or create cranial faults, will be seen to have a positive, often normalizing impact on the cerebellar finger-to-finger test or other indicators of cerebellar dysfunction.

#### Summary to this point, neurologically:

At this point in the protocol, we have accomplished the following (neurologically speaking): By correcting IRT, we have corrected aberrant input into the ipsilateral cerebellum and contralateral cortex and normalized the descending muscle spindle regulating systems that were adapting to injury.

We have addressed chemical imbalances (EFA, histamine) that impact peripheral nociceptors (so as to normalize NOC sensory activity) and neurotransmitter activity. We have investigated the need for folic acid and vitamin B-6, essential substances for the synthesis of some important NTs. Iron is also assessed and can affect NTs as well as being necessary for muscular function. When iron is needed, decreased muscle function, as seen by aerobic muscle testing weaknesses, will create an inefficient response to repeated motor inputs. So we have done much toward normalizing cerebellar and cortical afferentation and NT activity.

This has brought us to switching and the relationship to cranial faults, pre-test imaging and the immune system. Normalizing immune function stimulates hypothalamus pathways to inhibit the mesencephalon and favorably influence pattern generator cells that operate via descending reticulospinal (RS) pathways. Cerebellar responses to injuries that have been

corrected and the pathways for efferent copy, feedback and feed forward mechanisms have been addressed. The impact from mechanical cranial activity, so prominently affecting cerebellar firing, probably into the same pontomedullary descending pathways normalized by treating immune system function via hypothalamic-mesencephalic-pontomedullary descending RS pathways, is redressed. The effects of cranial correction to normalize the cerebellum via the dentatorubral tract directly into the opposite mesencephalon help to normalize potential mesencephalon imbalances from a direction other than the hypothalamus inputs.

These corrective effects negate aberrant cervical motor activity and allow normal coupling of the cervical spine and hence, easier adjustments that are more effective at bombarding the CNS with appropriate afferentation.

## SYSTEMIC NUTRITIONAL FACTOR

### 12. Does Rebreathing in a Paper Bag Strengthen?

#### a. If Yes: Check Citric Acid Cycle & Electron Transport Chain Nutritional Factors

Summary: CAC activity is necessary for carbon dioxide production for eventual synthesis of bicarbonate ion, which is necessary for CSF production and normal cranial bone function. CAC and ETC create ATP production for cellular function including maintenance of membrane polarization via membrane ion pumps. Several NTs are dependent on CAC function including glutamate, GABA, and aspartate.

As previously mentioned, we check for CAC activity following other systemic nutritional and structural factors rather than before. Inflammatory activity of cytokines and the production of nitric oxide cause a truncation of the CAC, and any attempt to correct CAC function is like painting over rust until the underlying source of inflammation (from EFA, allergies and hypersensitivities, and/or antioxidant deficiency) is negated.

Goodheart wrote many years ago about using zinc for recurrent cranial faults.<sup>18</sup> Zinc catalyzes the carbonic anhydrase enzyme for the reaction between  $H_2O$  and  $CO_2$  to produce carbonic acid ( $H_2CO_3$ ) which then dissociates into hydrogen ions and bicarbonate ions. The bicarbonate ions are necessary for the production of cerebrospinal fluid (CSF) as well as many other functions in the body (e.g., kidney, lung.) Goodheart hypothesized that the lack of bicarbonate interfered with adequate CSF synthesis that then led to cranial faults as adaptations to the change in CSF pressure and flow.

To continue this train of thought, even more fundamental than zinc in this process is the availability of  $CO_2$ .  $CO_2$  is a major byproduct of CAC activity. Inadequate CAC function will have an even more fundamental effect on CSF production than a need for zinc. Of course, there could be a need for both improved CAC activity and zinc, but zinc without the source of  $CO_2$  will be ineffective. The other source of  $CO_2$  is from vitamin B-6 dependent decarboxylation reactions throughout the body. B-6 is essential for the production of most NTs and its need will be found when  $CO_2$  strengthens and the CAC assessment finds no other CAC need.

So checking for CAC function at this juncture is also related to the possible recurrence of cranial faults that could indicate a problem with CAC activity (and/or zinc.) It is important to note that the excitatory NT glutamate and the inhibitory NT GABA are synthesized from alpha-ketoglutaric acid which is generated by the CAC. Sluggish CAC activity will interfere with the availability of these two important NTs and alter both sensory and motor responses to our other treatment procedures. Hence, it is a good time to make sure that these pathways are functioning adequately.

It is important to note that  $CO_2$  combines with ammonium ion to generate the urea cycle, the major source of ammonia waste for the body. Inability to rid the body of ammonia results in

hyperammonemia, resulting in changes in NT synthesis as ammonia groups are added to certain amino acid based NTs converting them into different substances altogether. For example, glutamate will become GABA, but in the presence of excess ammonia, it will be converted to glutamine and GABA will become deficient. Similarly, aspartate can be converted to asparagines and its NT function negated.

Speaking strictly about energy production rather than the other ramifications of CAC problems, we see that this is also the proper placement of CAC and ETC assessment. As stated previously, the need for CoQ<sub>10</sub> could be performed earlier. But the CAC and the ETC are a continuum in the oxidative phosphorylation process for the production of ATP. ATP provides energy for every cellular energy-using function: muscles, organs, and neurons. Once we have eliminated sources of CAC blockade (inflammation and/or allergy causing cytokines and nitric oxide) we can look to the possible need for B vitamins and manganese (and sometimes iron) necessary for efficient CAC activity.

It is important to recognize, from a neuron metabolism point of view, that all membrane receptor pumps (e.g., sodium-potassium pumps) that maintain neuron membrane polarization at a negative resting potential are energy (ATP) using mechanisms. If we want to have optimal depolarization (neuron firing) and repolarization, we must have adequate ATP formation, and this depends on CAC and ETC efficiency.

As mentioned previously, several neurotransmitters are dependent on the CAC including glutamate, aspartate, and GABA. Glutamate and Aspartate are excitatory NTs and GABA is the most important inhibitory NT in the CNS. Drugs that function as GABAergic agents include anti-anxiety drugs such as the benzodiazepines. In patients with low GABA, emotional treatments aimed at reducing anxiety will be short-lived, if successful at all. So, we address the production of GABA prior to treating emotional recall related disorders. In similar fashion, we have already dealt with the histamine produced from allergic responses above. Excess histamine, related to depression and other emotional responses, should be addressed prior to focusing on emotional recall techniques.

Similarly, in addition to the CAC nutrients, other nutrients necessary for NT formation (e.g., folic acid, B-6, iron) have also been covered to this point so that the patient will be able to maintain emotional as well as other NT based corrections. If the patient is treated for an emotional stress recall problem (or other NT dependent problem) in the same treatment session that a NT related nutrient need was identified, it is important to have patient insalivate and ingest the nutrient during the treatment session to reinforce the effects of the NT dependent treatment.

#### Summary to this point Biochemically:

In a general sense, excess oxidation is more dangerous than impaired oxidation since excessive oxidative activity produces free radicals that damage tissues (lipids, proteins, DNA, etc.) Impaired oxidation can cause a myriad of tissue dysfunctions, but it will only cause tissue damage secondarily. So our effort is directed first at correcting sources of inflammation / free radicals (allergies, EFA imbalances, antioxidant vitamins E & C) prior to addressing oxygen supply to tissues (improving RBC production.) In a similar fashion, we want to correct the immune system-related oxidative stressors since cytokines disrupt the CAC. Finally, we address the oxidative phosphorylation pathways (CAC and ETC) that may still show a need for support. When we correct immune dysfunction (thymus, spleen, chemical sensitivities) by rubbing the Chapman's reflexes, it appears to improve sluggish immune activity. Prior to addressing endocrine problems, which may involve increasing cortisol levels, we want to improve immune system function (thymus and spleen in particular) to insure that the immune system is capable of tolerating the immune suppression caused by cortisol.

Cholesterol production and cartilage repair become important factors only in those patients who present with related problems. But, in those patients, failure to address cholesterol

production and cartilage repair will allow consequent structural/neurological ramifications to block progress on other fronts, so we address these early on in the appropriate patients. Identification of homocysteine problems when assessing sulfur amino acid metabolism during the bleach sniff test and/or cartilage synthesis has huge ramifications on the patient's long term health.

## HEART-FOCUSED ACTIVITY

13. Does Specific Thought of Appreciation Felt in the Heart Strengthen?
  - a. Yes: Use Heart-Focused Technique(s)

Summary: Heart-focused (HF) activity is a self-induced therapy with autonomic, endocrine, immune, and emotional effects. The procedure is corrective of these important functions, but should be performed after the above procedures have set the framework for its effective implementation.

HF activity is based on research at the Institute of HeartMath in Boulder Creek, California. HF techniques not only result in profound physiological changes in heart-rate variability and cardiorespiratory function, but also lower elevated cortisol levels, raise low DHEA levels, and improve gut immune function (SIgA). HF is a powerful tool, and one that should be routinely taught to patients. It is a major technique for reducing emotional stress, yet, one HeartMath study showed 93% of participants improved doing these techniques. The question that begs to be answered is, "Why not 100%?"

We place HF activity at this point in the protocol because we have now cleared some of the potential obstacles to its optimal outcome. This includes treating all of the higher neurological centers associated with HF activity (cortex and cerebellum for imagery effects, hypothalamus, mesencephalon and pontomedullary RF areas for autonomic and immune function) as well as optimizing deficient NT activity that might alter the HF response. One might ask, "Why not use HF activity before this point to favorably influence immune problems including allergies?" In fact, HF activity has a profound effect on the immune system. But we must first make sure that imaging (PTI) is functioning properly to achieve adequate HF activity, and this means treating the hypothalamus and some immune circuits first. It is a bit like the chicken and the egg here, but considering the imagery aspects of HF, it is best assessed after normalizing PTI.

The only times we have seen poor results or adverse effects from HF activity is when the patient is switched. This includes small intestine related psychological reversal. Most small intestine problems are related to the dietary intake of food allergens and bad fats, so addressing these issues earlier on in the protocol allows us to clear this hurdle. However, if no small intestine dysfunction had been identified to this point, it may be prudent to quickly screen for small intestine involvement by rubbing and pinching the small intestine VRPs prior to initiating HF activity and correcting these circuits if still dysfunctional.

HF activity, once performed, should be taught to the patient and used daily at home. HF activity is placed before other endocrine and GI tract techniques due to its powerful and positive effects on these systems. Likewise, it is placed prior to other emotional stress related techniques. HF activity helps to correct and/or maintain endocrine (especially stress-related) and GI tract symptoms. Once the patient has been placed on daily HF home activity, check the endocrine system and GI tract prior to performing HF activity in the office. HF activity may have positive impact on these systems, but they still may require further treatment. Performing HF activity in the office may obscure your endocrine and GI tract findings – similar to jump starting a car with a dead battery, the next time you start the car, the battery is dead again - unless you have

addressed the underlying problem. Therefore, after initially giving the patient HF activity as homework, make sure that the patient is following your home care instructions, but do not do it in the office again, at least not until you have assessed and addressed any endocrine or GI tract dysfunctions.

## SYSTEMIC ENDOCRINE EFFECTS

14. Does TLR Strengthen as expected?
  - a. No: Identify and Treat Appropriate Endocrine Chapman's Reflex
  - b. Yes: Check for Endocrine Related Muscle Weakness – Treat Appropriately
15. Does Rubbing Adrenal Chapman's Reflexes cause Pituitary Chapman's Reflex to TL?
  - a. Yes: IRT to Adrenal Chapman's Reflexes with Offender.
16. Does Adrenal Challenge (Pinching) Induce Adrenal Muscle Weakness? If Yes:
  - a. TL to Adrenal Chapman's Reflexes – If Strengthens: Rub Reflexes
  - b. TL to Pituitary Chapman's Reflex – If Strengthens: Go to 15a
17. Does Ligament Stretch Cause Muscle Weakness?
  - a. Yes: Rub Adrenal Chapman's Reflexes
18. Test Endocrine Related Muscles – Identify and Treat Primary Chapman's Reflex
  - a. Test PMS (Liver) and TFL (Colon) – Treat Primary Chapman's Reflex
19. Test PMS – Rub and Pinch Liver VRP area – If Positive:
  - a. Test Liver Detoxification Nutrients
  - b. Challenge Liver Chapman's Reflex with Offenders – If Positive:
    - i. IRT Liver Chapman's Reflex with Offenders
  - c. Challenge PMS with Cholesterol – If Weakens: Go to 8c
    - i. Rub Liver Chapman's Reflex with Cholesterol in Mouth.
20. Pinch Pancreas VRP & Test Biceps Brachii (or Other Upper Limb Flexor) – If Weakens:
  - a. Test Chromium, Vanadium, Zinc, Pancreas Tissue, Sesame Seed Oil
  - b. Challenge Pancreas Chapman's Reflex with Offender – Offenders include:
    - i. Milk, Cortisol, Bad Fats, NE, Other Allergens

Summary: Identify the need for increasing or decreasing endocrine function. If indications are for both – correct toward increasing the sluggish organ function first. Any excessive hormone may be related to either over production or faulty liver detoxification. Liver assessment must include GI tract (especially large intestine) evaluation. Many endocrine problems include hyperinsulinism that must be addressed in conjunction with other endocrine dysfunction.

Injuries (especially those causing need for IRT or NSB) are stressful to the body and many endocrine (especially adrenal) indicators will be present until the sources of nociception are corrected. Nociception drives the ascending spinothalamic tract (part of the anterolateral spinal tracts) that impacts the hypothalamus causing the HPT-pituitary-adrenal axis to be stimulated to produce increased cortisol levels. In a similar fashion, nociception that reaches the HPT will create a sympathetic fight or flee reaction. This is why we never assess endocrine (or autonomic) function until injury patterns are corrected. Since inflammatory mediators depolarize nociceptors and drive the nociceptive pathways, corrections to the basic chemistry of inflammation (EFA, allergies, antioxidants) must also be addressed. Histamine is a powerful adrenal stimulant that must be moderated prior to endocrine evaluation.

As mentioned previously, Goodheart observed that muscles would respond differently to right brain activity (e.g., humming a tune) and to left brain activity (e.g., counting.)<sup>12</sup> He taught that right brain activity would often respond to therapies that increased steroid activity. Similarly, he noted that muscles that responded to left brain activity (e.g., counting) would often

respond the therapies that increased thyroid activity. Right-left brain imbalances of this nature can contribute to emotional stress (and cognitive) disturbances and the correction of endocrine imbalances at this point will set the stage for effective assessment of emotional recall issues later in the protocol.

14. Does TLR Strengthen as expected?

- a. No: Identify and Treat Appropriate Endocrine Chapman's Reflex
- b. Yes: Check for Endocrine Related Muscle Weakness – Treat Appropriately

Correction of sluggish immune system activity is necessary prior to endocrine correction to prevent any further inhibition of already suppressed immune tissues by increased adrenal glucocorticoid activity resulting from endocrine balancing efforts. On a neurological level, correcting injuries, TMJ and cranial problems (whether primary mechanical problems or secondary to immune system imbalances) is necessary prior to addressing endocrine dysfunction. These corrections will restore cerebellar afferent and efferent pathways enough to allow a clear assessment of tonic labyrinthine reflex (TLR) activity.

Hypofunction of an endocrine organ (adrenal, thyroid, reproductive) will be seen to cause an inappropriate muscular response to changes in head position relative to gravity. The first step in endocrine assessment is testing to see if the TLR are operating properly by testing inhibited muscles with the head in a position for TLR to facilitate those muscles. These reflexes operate primarily via the inferior vestibular nuclei, which receive direct input from the cerebellum. Failure of the TLR tells us that there is a under functioning endocrine organ, as long as other pathways to the vestibular nuclei are not interfering with its descending output.

Although not often mentioned, when expected muscles do not strengthen with the left ear down TLR pattern, it is always indicative of a low steroid function, either adrenal or reproductive, although most often adrenal. When the right ear down TLR pattern does not cause the expected muscle response, it is indicative of a relative hypothyroid state. However, these right and left correlations can be relied upon only if the patient has first had patterns of switching corrected (which is another reason that we place switching prior to the assessment of endocrine function in our protocol.)

15. Does Rubbing Adrenal Chapman's Reflexes cause Pituitary Chapman's Reflex to TL?

- a. Yes: IRT to Adrenal Chapman's Reflexes with Offender.

16. Does Adrenal Challenge (Pinching) Induce Adrenal Muscle Weakness? If Yes:

- a. TL to Adrenal Chapman's Reflexes – If Strengthens: Rub Reflexes
- b. TL to Pituitary Chapman's Reflex – If Strengthens: Go to 15a

17. Does Ligament Stretch Cause Muscle Weakness?

- a. Yes: Rub Adrenal Chapman's Reflexes

In addition to hypoadrenia, the other adrenal patterns assessed (hyperadrenia and ligament stretch adrenal stress syndrome - LSASS) should be tested only after the immune system's effects on the hypothalamus are resolved. The HPT drives the mesencephalon wherein the pattern generator cells of the parabrachial nucleus create "centering the spine" patterns including left convex lateral flexion (i.e., head and feet to right) associated with hyperadrenia (and increased reproductive steroids) and the right convex lateral flexion (i.e., head and feet to left) associated with increased thyroid activity. With the HPT-mesencephalon pathway free from immune interference (and other body chemistry problems previously addressed) we can accurately assess lateral flexion patterns.

Correction of endocrine dysfunction will center the spine and make adjustments easier (on both the doctor and the patient) and longer lasting. LSASS must be ruled out prior to any spinal adjusting, especially in patients with a history of poor response to manipulation.

18. Test Endocrine Related Muscles – Identify and Treat Primary Chapman’s Reflex  
a. Test PMS (Liver) and TFL (Colon) – Treat Primary Chapman’s Reflex

Understanding endocrine function depends on grasping the concepts of endocrine interaction. At this point in our assessment, we must consider the following organs: adrenal, thyroid, reproductive, pituitary, and pineal. Immediately following we will address the pancreas and insulin/glucose metabolism that also plays an integral role. We know that a hormone abnormality is due to either too much or not enough of that hormone. If we have excessive hormone activity, we will be led to address the over production by treating the Chapman’s reflex of that organ using IRT with an offender. (Other endocrine assessment and treatment procedures that are not covered in this protocol may be used at this point as well as long as both hypo and hyper function of each organ are considered.)

We may find one endocrine organ that is primary by cross-check TL. If found, this is the gland to which we address nutritional and manipulative efforts at this visit. We have already assessed the biochemical pathways (CAC, ETC) for energy production that are necessary for any organ’s optimal function, so we proceed with assessing hormones, glandulars, and herbals that will be most appropriate for support of the patient’s system in the now present state.

Excess hormone can also be due to decreased breakdown / detoxification of the substance that primarily takes place in the liver. We recognize that, in the most general sense, the liver’s main job is to detoxify the bowel. So we must assess liver – large intestine function and interaction at this point. By this time we will have identified EFA and antioxidants required for proper liver function as well as B vitamins in the CAC and other substances needed for ATP synthesis that are also necessary for liver detoxification pathways. Therefore, we proceed by evaluating the interaction of the liver and gut, and their interaction with the endocrine system.

Most often, cross check TL will identify a primary endocrine gland and a primary organ (liver and large intestine.) Sometimes, however, there will be one organ that, when its VRP is stimulated or its Chapman’s reflex is Tled, will resolve all of the other related muscle inhibitions. It is important to recognize that unfriendly flora in the colon can produce glucuronidase enzymes that break off (deconjugates) estrogen from its conjugation with glucuronic acid, allowing it to be reabsorbed into the portal circulation and return to the liver. When the liver or the large intestine are involved, it is sometimes necessary to jump ahead in the protocol (to the next step for liver detoxification or to the next section for the large intestine) to obtain optimal results.

As previously mentioned, centering the spine patterns reflect endocrine and autonomic functions, presumably mediated via hypothalamic connections to the parabrachial nucleus pattern generator cells in the mesencephalon. Spinal flexion is, of course, associated with sympathetic “fight or flee” activity that is often described as the scared cat arching its back. Conversely, parasympathetic activity is seen in the person who is “laid back.” Lateral flexion patterns relate as mentioned above: Left convex lateral flexion represents increased steroid activity (adrenal and reproductive steroids) and the right convex lateral flexion represents increased thyroid activity. Right and left spinal torques are generated by gait patterns that are produced by increased pituitary / decreased pineal (right foot forward gait) or increased pineal / decreased pituitary (left foot forward gait) functions.

Many systemic muscular patterns will resolve instantly following one specific endocrine correction related to centering the spine. Of course, adaptations to injuries will also cause

centering the spine problems, but these postural and visual (such as EID) responses to trauma will have long since been corrected by IRT, NSB, and/or SP techniques.

Too often doctors work piece-meal at balancing structural function by addressing one local problem after another when there is a single underlying systemic centering the spine problem at the root of all of the individual muscle imbalances. One example of this is body into distortion (BID) patterns that are resolved upon making corrections related to centering the spine.

Another example of this is the use of percussion techniques to many areas of the body to achieve normal spinal and extremity ranges of motion. Although the multiple application percussive treatment is effective, it is time consuming compared to the simple centering the spine corrections. It is also questionable whether or not the multiple percussion therapies achieve changes in the underlying systemic endocrine imbalances (or injuries) that have thrown the spine off-center in the first place. At this point, the use of percussion therapies to multiple areas is appropriate, as long as the causal endocrine imbalance (and/or injury pattern) is addressed as well.

19. Test PMS – Rub and Pinch Liver VRP area – If Positive:

- a. Test Liver Detoxification Nutrients
- b. Challenge Liver Chapman's Reflex with Offenders – If Positive:
  - i. IRT Liver Chapman's Reflex with Offenders
- c. Challenge PMS with Cholesterol – If Weakens: Go to 8c
  - i. Rub Liver Chapman's Reflex with Cholesterol in Mouth.

In fact, a complete liver evaluation may or may not be necessary to correct endocrine imbalances. There may not even be any endocrine significance, but under any circumstances, if the liver VRP is active, now is the time to do a more in depth evaluation. Assessing liver detoxification may include screening with a number of related nutrients, most of which are needed for other important functions as well. Recurrence of liver VRP activity should include in-depth evaluation of the potential nutrients related to the faulty pathway, and this alone will cover most, if not all of the nutrient needs of your patient. The few exceptions will be picked up in the next step when looking at the pancreas or the next section when looking at the GI tract.

Prior to checking the liver for hormone detoxification, we first want to improve any sluggish hormone production. Following treatment (rubbing Chapman's reflexes) to correct an endocrine hypofunction, the levels of circulating hormone would be expected to rise. Therefore, the time to evaluate liver function for hormone detoxification is following endocrine stimulation. The liver might not have any difficulty keeping up with a lowered level of circulating hormones, but the normal circulating levels might challenge a low liver reserve. Therefore, we evaluate liver function after finding, and often treating, the primary endocrine gland.

The same may be said for immune function. It is obvious that we would want to correct immune function prior to checking the liver for cytokine excess that is part of our liver evaluation. If the liver is incapable of keeping up with the demands of detoxifying normal cytokine production levels, and if the immune system is sluggish, the body may not show cytokine excess as a problem. The earlier correction of sluggish immune activity by stimulation of immune Chapman's reflexes will subject the liver to increased cytokine levels thereby increasing the demand on the liver's abilities to handle these substances. In the presence of increased immune function the liver may now be appropriately evaluated. Glycine, which is necessary for cytokine metabolism, is also one of the nutrients necessary for cholesterol metabolism and other liver detoxification. Glycine is also an important inhibitory NT. Be sure to check for glycine and its cofactors at this point if there is a cytokine problem.

20. Pinch Pancreas VRP and Test Biceps Brachii (or Other Upper Limb Flexor)–If Weakens:

- a. Test Chromium, Vanadium, Zinc, Pancreas Tissue, Sesame Seed Oil
- b. Challenge Pancreas Chapman's Reflex with Offender – Offenders include:
  - i. Milk, Cortisol, Bad Fats, NE, Other Allergens

The placement of pancreas endocrine assessment at this point is based as much on clinical observation as it is on biochemical sense. However, since insulin has enzyme blocking effects that impact steroid hormone synthesis, it must be included in the overall evaluation of the endocrine system.

Major offenders to the pancreas are allergens and bad fats. In that light we may have already corrected some or all of the pancreas stress when we addressed allergy-related problems and EFA metabolism. Another common pancreas stressor (driving it to hyper function) is cortisol. So it is only appropriate that we reserve pancreas evaluation until after adrenal function is normalized to get a clearer picture of cortisol's effects. Often, treating a cortisol problem will reveal a pancreas problem not apparent prior to adrenal correction.

When dealing with a patient with joint problems, it may be wise to skip from number 8.d. in the protocol to this step and then return to Step 9. Glucosamine and glucuronic acid, necessary for the production of the mucopolysaccharides that make up connective tissue (hyaluronic acid) and cartilage (chondroitin sulfate), require proper glucose metabolism. Glucose must enter the cell efficiently, which depends on insulin, and it must be metabolized properly via glycolysis (the Embden-Myerhof pathway) in order to be available for polymerization into the appropriate mucopolysaccharides.

Insulin has a significant effect on the autonomic nervous system. Insulin increases sympathetic outflow (which is one of the reasons hyperinsulinism patients often present with hypertension, many times misdiagnosed as “idiopathic hypertension” when it is really secondary to the sympathomimetic effects of the excess insulin.) The neurological SYM response to increased insulin includes the spinal flexion pattern. Spinal flexion is usually accompanied by a spinal torque (gait) pattern as part of normal spinal coupled motion. Repetitive gait in the hyperinsulinism patient frequently leads to a spinal gait torque pattern that results in an iliolumbar ligament IRT pattern. Correcting the hyperinsulinism pattern normalizes the SYM spinal flexion and its concomitant spinal torque rotation. Certainly, if present, this should be addressed prior to checking the iliolumbar ligament as well as prior to administering any spinal and pelvic adjusting procedures. More will be said of this pattern in the discussion regarding gait.

Bilateral upper extremity symptoms in any joint from the shoulders to the fingers are often secondary to hyperinsulinism. The excess response of pancreas insulin production is accompanied by a bilateral triceps over facilitation. Secondary inhibition of the biceps and/or other upper limb flexors results in bilateral upper limb symptoms which are often mistakenly treated as local problems.

Hyperinsulinism patients will often need treatments to both decrease the pancreas response (IRT to the Chapman's reflex with an offender) as well as to increase a tired pancreas by rubbing Chapman's reflex. The latter will be identified when evaluating gait following a challenge of the pancreas VRP.

## GASTROINTESTINAL TRACT

21. Challenge for Hiatal Hernia / GERD
22. Challenge Ileocecal Valve – Open or Closed
23. If Digestive Problem – Rub and Pinch Visceral Referred Pain area(s)
  - a. If Rubbing Strengthens: Rub Chapman's Reflex for that Organ
  - b. If Pinching Strengthens: Use VCT – IRT Chapman's Reflex with Offender
  - c. Challenge with Fat for Ileal Brake (Closed ICV)
  - d. Challenge with Sugar for Open ICV
  - e. 3-Step Challenge for Gastrocolic Reflex

Summary: The GI tract is influenced by 1) autonomic (sympathetic and parasympathetic) function, 2) local factors of the digestive environment including digestive secretions and the gut immune system, and 3) the enteric nervous system (ENS). Dietary allergens and bad dietary fats have been addressed previously as has systemic adrenal stress affecting the gut immune system and many nutritional factors related to the health of the gut. Each digestive organ (including the ICV) must not be treated as a separate entity, but rather must be analyzed in the context of the entire GI tract. In this context, the hiatal hernia / GERD must be addressed initially. Subsequent assessment and treatment relate to systemic SYM and PS activity, individual organ function (as part of the digestive system), and ENS factors.

GI tract problems are assessed and treated in the context of the entire digestive system. Embryologically, the GI tract is a tube of ectoderm that eventually twists and turns and sprouts off the digestive organs as we know them. Yet the GI tract still maintains the interconnectedness of the original tube. Three primary factors must be considered: 1) Systemic SYM and PS activities influence the entire gut. 2) Dietary excesses and irritants also affect the entire gut and improper digestive function in the early stages of digestion will have impact on the later stages including the GI tract flora. 3) The enteric nervous system (ENS) plays its own role in addition to these other factors. Effective treatment of the digestive system includes addressing all of these in the proper sequence, since one factor can hide another from the clinician's view.

Injuries must be corrected prior to investigating GI tract (or any autonomic) function because one of the three spinal cord effects of nociception is sympathetic activation at the intermediolateral columns (IML.) Nociception also activates the spinothalamohypothalamic pathway and its systemic autonomic (SYM) outflow. Muscle reactions to injuries that are maintained in an IRT pattern will cause cerebellar adaptation. The cerebellum fires directly into autonomic centers (parasympathetic) in the vital centers in the pontomedullary reticular formation (PMRF) including the nucleus of the tractus solitarius, dorsal motor nucleus of the vagus, and glossopharyngeal nerves and also affects the mesencephalon which has systemic autonomic consequences. Therefore, any autonomic evaluation in the presence of an IRT injury can be misleading because the real autonomic status may be misinterpreted due to the multiple effects arising from local or systemic autonomic reaction to the injury.

In the application of this clinical protocol, we will already have addressed some GI problems when we correct imbalances related to allergies, bad fats, etc. Allergic reactions and reactions to bad fats will almost always include the small intestine (as well as other various organs) and will have been previously addressed with visceral challenge technique (i.e. IRT to a Chapman's reflex in the presence of an oral offender.) It is important to note that allergies and bad dietary fats will have contributed to the depletion of the gut immune system (GALT.)

We will also impact the GI tract when we correct endocrine problems. Liver – bowel interactions will have been investigated and corrected as part of our comprehensive treatment of the endocrine system. Further, there are well described (although not as well defined)

relationships between female hormonal fluctuations and bowel function (i.e., the common diarrhea or constipation that parallels the menstrual cycle in many women.)

The triad of chronic stress described by Selye includes the development of stomach and duodenal ulcers as well as inhibition of immune system function. The GI immune system depends on adequate levels of DHEA and can be suppressed by excess cortisol levels. Since at least 50% of the human immune system is found in the gut (GALT), normalizing adrenal reactions to stress is important in promoting optimal function of the GI immunocytes. This includes adequate production of secretory IgA (SIgA) necessary to kill gut pathogens and maintain proper flora balance. The GALT is also sensitive to other effects of chronic stress (e.g., high cortisol to DHEA ratios are implicated in the turnover of gut mucosal cells and thinning of the mucus layer.)

It is possible that treating the adrenals to increase cortisol levels will suppress GI tract function just as it does systemic immune system function. However, it appears that patients can tolerate this possibility if they have first been relieved of the GALT stresses from allergies and bad fats.

## 21. Challenge for Hiatal Hernia / GERD

The hiatal hernia / GERD pattern can actually be an IRT pattern that does not manifest itself unless the hiatus is challenged. It will usually not be found during the AF screening for IRT and this can present a problem. If the patient has typical HH / GERD symptoms, consider the challenge and correction with IRT earlier in the protocol – at the same time that you investigate the effects of other injury patterns. Otherwise, the HH / GERD pattern must be corrected prior to addressing any other GI tract issues. If the stomach is compromised by a HH or GERD, the rest of the GI tract will adapt. To get a clear picture of GI autonomic and enteric activity, we must resolve the HH / GERD first. In the situation where the HH / GERD pattern is identified during IRT screening in step 4, traditional concomitant findings such as the dorsolumbar fixation and psoas muscle imbalance will usually be corrected by protocol procedures performed between the IRT correction and this step. If, at this point in the protocol, asymmetrical diaphragm excursion, dorsolumbar fixation, and uneven foot turn-in (indicative of psoas imbalance) are present, they should be corrected.

## 22. Challenge Ileocecal Valve – Open or Closed

### 23. If Digestive Problem – Rub and Pinch Visceral Referred Pain area(s)

- a. If Rubbing Strengthens: Rub Chapman's Reflex for that Organ
- b. If Pinching Strengthens: Use VCT – IRT Chapman's Reflex with Offender

Anything that affects autonomic function should be corrected prior to addressing GI issues. This includes injuries and heart-focused procedures which will correct some emotional stress related problems. The specific emotional recall techniques considered later can also affect the GI tract, and vice versa. If one checks for emotional recall weakening prior to addressing GI dysfunction and finds a GI tract acupuncture head point and/or Chapman's reflex, correction of the GI tract involvement will often clear emotional recall weakening. This suggests that GI tract dysfunction can contribute to emotional stress. Clinical observations show that this is often the case.

Neurologically speaking, aberrant visceral sensory activity will impact the ipsilateral cerebellum (via the GI muscle imbalances) and the contralateral cortex and can contribute to right – left brain imbalances and the emotional recall weakening effects. Cerebellar adaptations to long standing GI tract related somatic muscle imbalances can also cause changes in axial (old, midline cerebellum) sensory feedback from the cerebellum into the limbic system (old, midline

cortex) and aggravate or enhance emotional perceptions. For this reason, the emotional recall techniques are placed after GI tract correction in the clinical protocol. This allows for correction of numerous secondary emotional recall problems during GI tract treatment.

For the purposes of the patient treatment protocol, we can consider a positive ICV open or closed challenge as just another weak digestive system muscle. (In fact, the open ICV is accompanied by a weak right iliacus and the closed ICV will usually be associated with inhibition of one or both quadriceps.) When there is an ICV open or closed, we will address other clinical factors (e.g., SYM or PS status, aggravating dietary substances, etc.) rather than treat it as a separate entity since the ICV is an integral part of, and can't be divorced from, the rest of the GI tract. When we look at the autonomic and enteric effects on the GI tract, the ICV must be considered in this light.

Food allergies, dietary fats, and dysbiosis are common causes of digestive problems including ICV open and closed. Some of these will be corrected much earlier in our protocol. However, there may still be digestive problems present that must now be addressed.

23. If Digestive Problem – Rub and Pinch Visceral Referred Pain area(s)
  - c. Challenge with Fat for Ileal Brake (Closed ICV)
  - d. Challenge with Sugar for Open ICV
  - e. 3-Step Challenge for Gastrocolic Reflex

There are only 200 preganglionic parasympathetic nerve fibers in the vagus nerve at the point the vagus enters the abdomen. In contrast, there are over one hundred million nerve cells in the small intestine enteric nervous system (ENS.)<sup>19</sup> Although these numbers might suggest otherwise, the influence of autonomic (SYM and PS) activity on the ENS function is significant and must be considered. Systemic autonomic imbalances must be corrected prior to investigating the ENS as some ENS problems will be as a result of adaptation to SYM or PS imbalances, and others may be causing the SYM or PS imbalance. In either case it is necessary to correct any SYM / PS problems in order to clearly assess the ENS picture.

ENS dysfunction is frequently at the core of recurrent ICV syndromes and other digestive problems. Left uncorrected, the ENS will create adaptations in SYM / PS autonomic function. These adaptations will often distort the clinician's view of ENS problems, which is why they must be corrected prior to ENS assessment.

Additionally, a frequent finding is that fixing one ENS problem will reveal the presence of another, especially the ileal brake and the sugar-induced open ICV challenges. Using these challenges, there is no way to predict the order of correction. Just know that after identifying and correcting one of these ENS faults, you must check for the other.

Again, correction of digestion problems (of all types) is important prior to the emotional stress recall techniques. It has been thought by some (including this author) that the reason that seemingly equal emotional stressors sometimes result in the need for emotional recall corrections while other times they do not is related to the impact from other sensory inputs at the time of the emotional trauma.

There is a necessity of addressing psychological reversal prior to other emotional therapeutics, as discussed originally by Callahan.<sup>20</sup> Callahan described psychological reversal as a problem with the small intestine acupuncture meridian system and recommends tapping SI-3 bilaterally. Our observations agree with those of Callahan with the addition that normalizing the small intestine by therapies discussed above will normalize the psychological reversal just as does tapping SI-3.

When there is a psychological reversal, there is also a physiological reversal with a switching type effect on muscle testing findings. It is logical (and correct) that any type of switching should be corrected earlier in the course of treatment than at this point. The protocol

addresses this by screening for allergies and bad fats very early on when screening with anti-histamines and aspirin, ibuprofen, acetaminophen mix, respectively. Most of the time, there will be small intestine involvement in both allergies and bad fat ingestion and correction will eliminate both psychological and physiological reversals at this point early in the treatment. There are other cases of small intestine involvement that do not cause these reversals and these will be corrected during other GI tract treatment in this section.

Heart focused activity will clear some GI tract dysfunctions (often many) and some emotional stress disturbances (often many). It may be well noted here that the early correction of psychological and physiological reversal, prior to heart focused activity, will clear the way for more effective HF activity. In eight years of performing HF activity, there have been four negative responses noted by this author. They were all in patients who had small intestine problems that had not been corrected first.

Emotional recall patients frequently demonstrate stomach circuit involvement, but any circuit may need to be treated in emotional stress cases. Considering that the majority of patients will have some GI tract circuit needing treatment for the emotional stress correction, it is prudent to have already addressed the GI tract before embarking on emotional recall techniques. Failure to correct GI tract problems prior to assessing emotional techniques results in several undesirable patterns: 1) There is an excellent chance for recidivism of the emotional recall activity; 2) There will often be positive recall of multiple emotional recall events, all related to the same GI tract circuit misleading both the doctor and the patient to think that there are more severe emotional involvements than are truly present; and 3) Uncorrected small intestine problems will result in confusing clinical presentations and temporary results to any emotional techniques applied in its presence.

## EMOTIONAL STRESS

### 24. Perform Emotional Recall Challenge – If Positive: Do Emotional Recall Quick Fix.

Summary: Many emotional stress related weaknesses will be corrected by applying the factors already addressed in this protocol. Those that are not can now be addressed using emotional recall quick fix or any other emotional related technique in the doctor's armamentarium.

It is acknowledged that emotional stress related techniques play an important role in treating patients. Therefore, the placement of the assessment of emotional stress related issues at this late point in the protocol may be perplexing, even disconcerting to those who give high regard to the importance of the emotional aspects of health. If this is your position, consider that we have already corrected the following factors that can influence emotional stress and the ability to cope with it, biochemically and neurologically:

- IRT and other pain and its impact on right and left hemispheric activity
- Allergies and the effects of histamine on neurotransmission and cognitive function
- Nutrients that will provide higher NT availability
- Adrenal stress issues that both compound and are compounded by the effects of emotional stress
- Other endocrine issues, recalling that right cortical activity is associated with steroids (adrenal and reproductive) and left cortical activity and is associated with thyroid
- Heart-focused activity
- GI tract involvements that appear to increase the vulnerability for stressful events to make an emotional recall problem

Experience has shown that if one identifies an emotional recall induced weakness (and/or positive TL to the emotional Bennett's neurovascular reflexes on the forehead) prior to applying this protocol, and then rechecks that very same emotional recall pattern along the course of the protocol, the weakening effects will often cease to exist somewhere along the way. For this reason, emotional stress related problems should be addressed at this time, and not earlier, except as they may be dealt with by HF activity.

Emotionally based therapies that are performed in the presence of right – left cortical imbalances or in the absence of adequate NT nutrition are predisposed to failure. Recurrence is common, as is the tendency to identify multiple emotional recall events positive on emotional evaluation.

The possibility of treating a neurologically exhausted (metabolically compromised) neuron pool also exists. Over stimulation of such neurons can aggravate the patient's problems, or even lead to neuronal cell death by apoptosis as previously mentioned. Treating immune system problems prior to emotional recall also has an inhibitory protective effect on the mesencephalon – a source of many emotional problems via mesolimbic connections.

Heart-focused activity is also a powerful remedy for many emotional stressors. Before-and-after muscle testing with emotional stressors will show most to be relieved by HF activity. Patients who are trained in HF activity as home therapy seem to demonstrate far fewer emotional recall events (i.e., are far less likely to have TL to the emotional neurovascular points) in the office setting.

At this juncture, patients who still demonstrate positive TL to the emotional Bennett's neurovascular reflexes on the forehead should have the emotional factors assessed. Emotional recall quick fix is often effective for simple day-to-day emotional stressors. However, the fact that this protocol is an open system and a framework for applying any clinical tool implies that other emotional stress related techniques could also be used, and used most optimally, at this time.

## LOCAL PROBLEMS

25. Check Chapman's Reflexes for Weak Muscle(s) – If Positive: Treat by Rubbing
26. Check Fascial Sheath Shortening
27. Check Iliolumbar Ligament
28. Check Pelvic Categories - Iliac & Sacral Fixations
29. Check Spine (and Feet) using FRA activity:
  - a. Challenge Vertebra (or Foot) to Determine Direction of Correction
  - b. Add Spinal Position to Determine Optimal Coupled Position for Spinal Adjustment
  - c. If Uncoupled Mechanics: Look for Source of Uncoupling
30. Challenge Extremities and Adjust as indicated

Summary: Having already addressed systemic factors creates an environment for an amplified response when treating remaining dysfunction directly related to local muscle and ligament involvement, and adjusting spinal, pelvic, and extremity subluxations.

The area of a local problem is the starting point for most practitioners of any discipline. However, by employing this protocol, most, and sometimes all of the patient's symptoms will be improved, if not eliminated, prior to local assessment. When there are still persistent local symptoms, it is time to investigate local muscle weaknesses and find and correct the "local" source(s) of inhibition.

Recall that a weak muscle, as seen in AK, represents a net inhibition at the anterior horn motorneurons for that muscle. The protocol has comprehensively addressed the various factors that will impact the spinal cord AHMNs from both somatosomatic and viscerosomatic sources and from all major descending and ascending pathways. What remains to be treated are problems associated directly with the muscle(s) used to perform the symptomatic activity. This includes spinal cord reflex activity arising from the iliolumbar ligament, pelvic categories, spinal fixations and subluxations, and extremity subluxations. It also encompasses the application of therapies tied directly to a muscle such as Chapman's reflexes, fascial release technique, and even isolated IRT corrections to specific muscle and/or ligamentous tissues.

The concept of spurt muscles and shunt stabilizer muscles provides additional perspectives (biomechanical as well as neuromuscular) regarding the organization of this protocol. Any muscle initiating an action (a spurt muscle) must have a stable base to pull from provided by a shunt stabilizer muscle or risk unsafe mechanical stresses to the joint. Many local problems recur due to treating the muscle at the point of the problem (the spurt muscle) while ignoring the shunt stabilizer muscle. A muscle may be a spurt muscle for one movement and a shunt stabilizer for another. Shunt stabilizer activity can be traced back to extensor muscles that connect to the spine and inevitably the spinal intrinsic muscles themselves. The spinal intrinsic muscles are under unconscious control through descending pathways originating in the cerebellum. (See next paragraph.) Note that the latissimus dorsi and the trapezius (upper, middle, and lower) combine to provide connection (stabilization) to the shoulder joint from the entire spine. It is interesting to note that when we correct immune system problems related to the spleen, we are providing shunt stability for many shoulder problems by normalizing middle and lower trapezius function.

Taking a larger view, centering the spine corrections (flexion-extension, lateral flexion, spinal torque or gait pattern) will provide intrinsic spinal muscle stability that originates in the pattern generator cells in the mesencephalon. The feedback from muscle spindle and joint receptors activated by centering the spine correction will fire into the midline cerebellum which then fires back into the brain stem and down into the intrinsic spinal muscles for central shunt stability. Correcting anything that impacts these pattern generator cells including factors mediated through the hypothalamus-mesencephalon reticular formation connections will ultimately provide shunt stability for virtually any movement in the body. This includes immune problems, endocrine problems, systemic autonomic problems, emotional stresses, and others. Therefore, at several steps along the course of this protocol, corrections have been made to provide for spinal shunt stability for virtually any movement. The concept of spurt versus shunt muscles provides additional perspective on how correcting the systemic problems first will often clear up local problems, or at worst provide an environment for local treatment to create rapid improvement and long-standing correction.

It may be wrongly assumed that any origin and insertion injury would have been corrected during IRT and other pain relief corrections. Occasionally, the origin-insertion of a local muscle will need IRT and this will elude the early screening with AF. It is wise to have the patient TL to the origin and insertion of muscles associated with local problems as well as nearby ligaments. Use TL to identify therapeutic need to muscle origin(s), insertion(s), and ligaments, but do not correct until after TLing to Chapman's reflex for the muscle and the associated spinal areas. It is easier to identify these and other factors (see immediately below) with the muscle weakness present. Correct OI and Chapman's reflex one after the other.

## 25. Check Chapman's Reflexes for Weak Muscle(s) – If Positive: Treat by Rubbing

If a weak muscle is still present, TL to the Chapman's reflex, the muscle's OI, and the associated spinal levels and extremity joints. It is more efficient to TL these factors using the weak indicator muscle prior to correcting any single factor that may be present.

Chapman's reflexes will affect muscles and the associated viscera, if the organ is involved. Although it is likely that any visceral Chapman's reflex will have already been corrected, these reflexes can still be present and related only to the associated muscle, so they should be TLED, and corrected after any other local problems (spinal, extremity, OI) are identified.

## 26. Check Fascial Sheath Shortening

In persistent pain and decreased range of motion, after strengthening any local muscle weakness, check for and correct fascial sheath shortening problems. Since many fascial sheath problems are related to the need for vitamin B-12, and its absorption, the patient will likely have had these factors identified and corrected long before getting to this step. However, if there is a local fascial sheath problem and response to oral B-12 has not been checked, it should be checked now. If B-12 negates the fascial sheath challenge, then digestive factors associated with B-12 activation and absorption should be investigated. It is possible that a B-12 need has been missed (e.g., supplementation with folic acid can obscure the laboratory indications of a simultaneous need for B-12.)

Laboratory assessment (CBC with differential, etc.) may be indicated and reassessment for B-12 (including fascial sheath shortening and small intestine evaluation) should be performed earlier in the protocol (Step 8b) during subsequent visits to ensure proper use of this important nutrient. Fascial sheath stretching manipulation should not have to be performed more than once in any patient. Recurrence suggests some other underlying factor (e.g., injury, intestinal malabsorption, or subluxation) is at fault.

## 27. Check Iliolumbar Ligament

IRT to the IL ligament is the most common finding in my practice. Often it will be missed on initial evaluation for injuries because its presence may be only evident by AF in the weight-bearing position. IRT IL ligament problems are so important that it is recommended that they be checked for during initial injury screening, whether by testing for AF in the weight-bearing (sitting or standing) position or by direct challenge with the patient recumbent using the cephalward talus pressure challenge.

IRT IL ligament problems are usually related to gait asymmetries, but like the chicken and the egg, it is difficult to say which one causes the other. If there is no IRT ligament evident during initial IRT / AF screening, it should be tested for directly with talus challenge at this point and corrected, if indicated, usually with the patient prone. IL ligament problems are often a result of a repeated minor repetitive injury arising from structural manifestations of body chemistry imbalances (off-centering of the spine) and visceral disturbances. These should be negated by this point, with the exception of endocrine (and occasionally other visceral) problems that will be picked up during the gait assessment below.

Most Category 1 and some Category 2 and Category 3 pelvis problems will be eliminated by IRT IL ligament correction. Some other spinal problems will also resolve with IL ligament correction due to the unwinding (re-centering) of the spine associated with these corrections. So it is essential to check and correct the IL ligament prior to looking for any spinal and pelvic problems.

In this regard, IRT of the sacrospinous (SS) and sacrotuberous (ST) ligaments can also create pelvic Category 3 problems. SS and ST ligament IRT involvement will also elude AF screening unless the patient is weight-bearing, but may be challenged at this time.

It was previously mentioned that hyperinsulinism creates a SYM effect including spinal flexion and secondary spinal torque (gait) patterns. This sympathomimetic effect of insulin results in repetitive asymmetric insult to the IL ligament, hence, chronic hyperinsulinism frequently creates an IL ligament IRT pattern. This pattern becomes ingrained in the patient's nervous system and is often mistaken for the commonly found chiropractic neurology pattern referred to as hemisphericity.

Treating a patient for a hemisphericity lesion when the problem is really a neuromuscular pattern secondary to hyperinsulinism not only addresses the wrong problem, the treatment usually causes the patient to respond by a change in symptoms, rather than true correction. That is, since the underlying hyperinsulinism pattern remains, neurological therapies and rehabilitation procedures drive the adaptation (to the insulin problem) to another area of the body, sometimes creating new symptoms, sometimes not, but allowing the underlying hyperinsulinism to go untreated. This is detrimental to the patient's overall good. Following the protocol will help to avoid this potential pitfall and subsequent misdiagnosis

28. Check Pelvic Categories, Iliac & Sacral Fixations
29. Check Spine (and Feet) using FRA activity:
  - a. Challenge Vertebra (or Foot) to Determine Direction of Correction
  - b. Add Spinal Position to Determine Optimal Coupled Position for Spinal Adjustment
  - c. If Uncoupled Mechanics: Look for Source of Uncoupling
30. Challenge Extremities and Adjust as indicated

The bread and butter of the chiropractor, pelvic and spinal adjusting, is left to a point near the end of the protocol. This is the reason why: If you want the maximum response from your adjustment; if you want it to impact the most neurons in the spinal cord, brainstem, cerebellum, and cortex; then you want to make sure as many neural pathways as possible are operating as open channels to carry the consequences of restoring normal afferentation to the most distant and isolated areas of the nervous system. You also want to be certain that the necessary neurochemistry is in place for optimal signal transmission.

Said differently: Get the entire smokescreen out of the way before taking aim at bombarding the nervous system with such an important input as the spinal adjustment. Or clear the static from the radio before trying to hear every piece of an orchestral movement that is being played. Just like getting the static cleared from the radio signal so we can hear all of the parts equally, so do we want to clear the nervous system from aberrant signals so that the normalizing message from the adjustment gets through loud and clear and penetrates the entire nervous system.

Pelvic Categories 1, 2, and 3 are often corrected by treatment of IRT to the IL ligament and/or the SS and ST ligaments as mentioned above. Correction of centering the spine factors (which includes IRT injuries and endocrine imbalances) also allows for self-correction of many pelvic problems, especially Category 1 lesions. When one follows the protocol, one encounters far fewer structural faults (subluxations and fixations.) However, the impact of their correction is significantly magnified.

Identifying subluxation correction hierarchy by the flexor reflex afferent (FRA) subluxation nociceptive challenge is usually a multi-step procedure. NOC afferents create the FRA (flexor withdrawal response.) The nervous system can only respond to one NOC input at a time. In fact, any FRA inhibits all other FRA afferents. Hence the "worst" FRA overrides the

“lesser” FRAs and causes its specific pattern of muscle withdrawal preferentially. Certain other non-NOC sensory pathways also create an FRA response. These include receptors in joints and secondary muscle spindle cell receptors (from the nuclear chain) which would fire in the presence of a subluxation.

Adding nociception (by mild pinching) over the next subluxation to be corrected will yield an FRA response of weakening any extensor muscle. Pinching over any other subluxation will not override the more powerful FRA response, so no muscle weakness will be induced except by pinching over the next subluxation to be corrected. Following correction of the primary subluxation, another subluxation becomes primary and pinching over it (and only it) will result in extensor muscle weakening. The optimal order for correction of the spine and feet is indicated by pinching over the vertebrae (and feet) until a weakness of an extensor muscle is found, identifying the next segment to adjust. FRA activity (pinching over the vertebrae and feet) is continued until there is no extensor weakness induced by nociception over any vertebra or either foot.

Spinal corrections should be made using coupled mechanics. Sources of uncoupled mechanics include injuries, centering the spine problems, cranial faults, TMJ faults, visceral referred pain patterns, sclerotogenous referred pain patterns, and Lovett reactor vertebra subluxation patterns. All of these should have already been cleared with the possible exception of sclerotogenous and Lovett patterns. If an abbreviated protocol has been performed, (that is, not all steps are performed on any one visit) which is the most likely case (few practices are set up to take the time to cover the entire protocol in one visit) and an uncoupled subluxation pattern is identified, it is worthwhile to identify and correct the source of uncoupling rather than adjust the uncoupled segment.

Extremity symptoms that are still present (the feet should have been corrected during FRA subluxation activity) may be related to local muscle weakness (even local IRT problems) and extremity subluxations. As with vertebral corrections, extremity adjustments will be more effective since the nervous system is clear of other sources of interference and has the chemistry needed to amplify the effects of the adjustment.

## GAIT ASSESSMENT

31. Check Gait (backward step first)
  - a. If Gait Testing Facilitation/Inhibition ABNORMAL
    - i. Check Iliolumbar Ligament or Pelvic Category or Foot/Ankle Subluxation
  - b. If Gait Testing Facilitation/Inhibition NORMAL
    - i. Pinch Pancreas VRP – If Pinching VRP Disrupts Gait: Test Nutrients
    - ii. Rub Pancreas Chapman’s Reflex
    - iii. Pinch Other VRP’s – If Pinching Disrupts Gait: Rub Chapman’s Reflex

Summary: After all other corrections have been made, assessing the patient in the right and left gait patterns will assure that normal gait will not wind down the effects of the treatment. Gait patterns might remain in the presence of uncorrected structure, uncorrected endocrine hypo function, and/or residual visceral effects, most notably from hyperinsulinism.

There may still be a disturbance in gait, even after addressing all of the previous protocol steps. If there is a major structural fault (pelvis, spine, foot/ankle) or IL ligament that has been missed, it will cause a disturbance in gait that must now be corrected. However, residual gait disturbance is most commonly due to the need for performing the second step in the correction of hyperinsulinism problems – the need for rubbing Chapman’s reflex for the pancreas. It may also be due to residual dysfunction in any other viscera including the endocrine system.

The presence of endocrine hypo function can be seen at this point by assessing TLR patterns during gait. During gait testing, tilting the head so that the ear is pointed toward the ground on the side of extensor (e.g., latissimus dorsi) inhibition should override the gait-induced weakness. If this does not occur, TL to one of the endocrine Chapman's reflexes will temporarily restore this TLR function. Rubbing the Chapman's reflex identified will correct the disturbance noted on challenging gait with TLR activity.

Hyperinsulinism is extremely common in the patients chiropractors see, probably due, in part, to the spinal flexion effects of insulin and the adaptive spinal torque (gait) stresses. The spinal torque will create residual muscle tightness (and often pain) even after all else is corrected. Long term asymmetrical sensory activity from the spine and extremities can create a cerebral cortical hemisphericity that may require neurological assessment and treatment. However, more often, hyperinsulinism will mimic a hemisphericity pattern, and treating Chapman's pancreas reflex at this point can resolve the imbalance by correcting the source.

Many residual muscular symptoms, anywhere in the body, will resolve upon rubbing the pancreas Chapman's reflex in hyperinsulinism patients. This is due to relieving the remaining insulin induced SYM spinal stress and consequent spinal adaptations.

For these reasons it is recommended that gait be checked on any patient prior to the end of the treatment session, regardless of which procedures were performed previously during that session. This will prevent the patient from walking out of the office with a gait asymmetry that will wind down all of the previous mechanical and neurological corrections, including recurrence of imbalances associated with cortical hemisphericity.

## CHRONIC PAIN

### 32. If Chronic or Persistent Pain: Use LQM Technique

Summary: The application of LQM technique will be applicable in chronic pain after all other procedures have neurologically and biochemically paved the way for its effective use.

If there is still pain, especially if the pain is of a chronic nature, then LQM (location, quality, memory of the pain) technique is appropriate. But prior to checking for LQM, restoration of balance to the cortex (and likely the cerebellum) is necessary, both to allow identification of the LQM problem as well as to enhance treatment effectiveness. In fact, some LQM problems will not even show up until all the other pain relief techniques (especially IRT and NSB) have been employed.

The cells involved with LQM are presumably cortical cells associated with each of the brain areas for L, Q, and M. Therefore, this technique is left until the end so that all other more general effects on the cortex and cerebellum, structural, visceral, and chemical, have been corrected. In patients who still have pain of a chronic nature after all of the previous protocol steps have been completed, it is likely that there is an over firing of pain perception neurons in an isolated area related to the patient's problem. It appears that during LQM technique, those over facilitated neurons are activated when the patient thinks of L, Q, and/or M, and the same neurons become inhibited back to a normal state in the presence of tapping of the appropriate acupuncture head point.

After using LQM technique, if there is still persistent pain, other pain relief techniques may be employed. The AK tonification point technique (analysis of pulse points, alarm points, and finally identification of one tonification point) is a good choice, or any other approach known to provide relief. The rare cases that still report pain at this point may just require time for tissues to heal, or there may be a pathology present that must be differentially diagnosed.

## CONCLUSIONS

The protocol presented herein is the result of thirty years of clinical observations presented in the light of modern neurology and functional biochemistry. At some point or other in the protocol, all known essential nutrients will be tested. All fundamental AK principles will have been screened for, either in their original form or with updated methods. Admittedly, there are natural therapies not included herein, most importantly those associated with altered neuronal metabolic function (transneural degeneration) as taught in chiropractic neurology programs. The assessment for and the application of these therapies can be correlated with the procedures discussed in this paper, but their discussion is the stuff of a future presentation.

Virtually all other natural therapies can be appropriately placed in this protocol. AK is an open system. This protocol is a comprehensive, well thought out, application of this open system. It is tight. It is complete. But, it is open to other therapies: past, present, and future.

These therapies may be added to the protocol like decorative ornaments on a Christmas tree. Like the decorated tree, there is an optimal system of placement. Large ornaments are placed on the bottom branches, small ornaments on the higher branches, tinsel on the outer branches, and a star on top. The trunk of the tree is muscle testing as functional neurological and neurochemical assessment. All other procedures discussed have been neurologically and biochemically placed as appropriate.

There is a physiologically optimal system of approaching a patient. I submit that this protocol represents the optimal approach to quality patient care... and the star on top of this skillfully decorated tree is the healthy patient who is the beneficiary of well designed care.

## REFERENCES

- <sup>1</sup> Schmitt, W.H., & Yanuck, S.F. Expanding the neurological examination using functional neurological assessment part II: neurologic basis of applied kinesiology. *Intern J Neuroscience*, 1999, 97, 77-108.
- <sup>2</sup> McCord, KM, and Schmitt, WH, *Quintessential Applications: A(K) Clinical Protocol*. St. Petersburg, Florida: Privately Published, 2005.
- <sup>3</sup> McCord, Kerry M. Clinical response to a neurologically based comprehensive protocol developed by Dr. Walter H. Schmitt. Submitted for inclusion in *Proceedings of the I.C.A.K. - U.S.A. Volume 1, 2005-2006*.
- <sup>4</sup> Belli, Richard. Deep Tendon Reflexes. *ICAK-USA News Update*, 1994.
- <sup>5</sup> Belli, Richard. Autogenic Inhibition. *Proceedings of the ICAK-USA, volume I, 1995-1996*. pp.15-18.
- <sup>6</sup> Schmitt, Walter H., "A Critical New Concept" (Transneural Degeneration) *ICAK News Update*, (1999) 15:4. p. 5-7.
- <sup>7</sup> Schmitt, Walter H., Jr. Transneural degeneration and the links between the nervous system and the body chemistry. *Proceedings of the I.C.A.K. - U.S.A. Volume 1, 1999-2000*. p. 149-156.)
- <sup>8</sup> Schmitt, Walter H., Jr. A neurological rational for injury recall technique. *Proceedings of the I.C.A.K. - U.S.A. Volume 1, 1999-2000*. p. 137-139.
- <sup>9</sup> Gerald R. Polino, personal communication, 1993
- <sup>10</sup> Burt, Alvin M.; *Textbook of neuroanatomy*. Philadelphia: W.B. Saunders, 1993. pp 459-464.
- <sup>11</sup> Schmitt, Walter H., Jr. Applied kinesiological observations of allergic patients - Parts I and II. *Digest of chiropractic economics* 27:1, July/August, 1984 and 27:2, September/October, 1984.
- <sup>12</sup> Goodheart, George J. *Applied kinesiology 1979 workshop procedure manual*. Detroit: privately published, 1979.
- <sup>13</sup> Schmitt, Walter H., Jr. A neurological basis for the effects of cranial manipulation. *Proceedings of the I.C.A.K. - U.S.A. Volume 1, 1999-2000*. p. 133-135.

- 
- <sup>14</sup> Gangemi, Stephen C, A Newly Discovered Muscle-Organ Relationship: The Pectoralis Minor and the Parotid Gland. Submitted for inclusion in Proceedings of the I.C.A.K. - U.S.A. Volume 1, 2005-2006.
- <sup>15</sup> Schmitt, Walter H., Jr. Centering the spine functional neurological and biochemical considerations. in Collected papers of I.C.A.K. - summer 1987. Shawnee Mission, KS:International College of Applied Kinesiology, 1987
- <sup>16</sup> Schmitt, Walter H., Jr. Where is the triad of health? Proceedings of the I.C.A.K.-U.S.A. Volume 1, 1996-97. p. 83.
- <sup>17</sup> Schmitt, Walter H., Jr. The somatic window on neurological function, part 3. mesencephalic transneural degeneration: the cause of many TMJ problems and bilateral joint problems. Proceedings of the I.C.A.K. - U.S.A. Volume 1, 2003-2004. p. 227-233.
- <sup>18</sup> Goodheart, GJ, Zinc and Cranial Faults. Nutri West company literature. 1985.
- <sup>19</sup> Gershon, Michael D. The second brain. New York: HarperCollins, 1998.
- <sup>20</sup> Callahan, R.J. How executives overcome their fear of public speaking and other phobias. Wilmington, De: Enterprise pub., inc., 1987