PROCEEDINGS
OF THE
SUMMER MEETING OF THE
INTERNATIONAL COLLEGE OF APPLIED KINESIOLOGY-U.S.A.

Experimental Observations of Members of the ICAK

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CHICAGO, ILLINOIS

A MESSAGE FROM THE CHAIRMAN

Dr. Philip Maffetone
Chairman, ICAK-U.S.A.

Research is one of ICAK's priorities. The sharing of a member's clinical observations in this publication is the launching of a research idea, and a step up to a path where too few in the health community have ever gone. While not yet ready for the submission process mandated by scientific and medical journals, many of our members have clinical insights to explore. *The Proceedings* is one publication which bridges that wide gap, and we are fortunate to maintain and encourage its existence.

These published works by our members are often a first step in a process which improves our understanding of clinical methods and outcomes. It can also be part of a process which results in enhancing the material through further evaluation or peer input to publish in other journals. The mechanism is set up for just such a process, and we invite you to be part of it.

INTRODUCTION

This thirty-sixth collection of papers from members of the International College of Applied Kinesiology-U.S.A. contains 20 papers by 18 authors. The papers will be presented by the authors to the general membership at the Summer Meeting of ICAK-U.S.A. in Chicago, Illinois, August 25-28, 1994. The authors welcome comments and further ideas on their findings. You may talk with them at the meeting or write them directly; addresses are given in the Table of Contents.

The manuscripts are published by ICAK-U.S.A. as presented by the authors. There has been no effort to edit them in any way; however, they have been reviewed by the Publications Committee for originality and to determine that they follow the "Instructions to Authors" published by the ICAK-U.S.A. The primary purpose of the ICAK-U.S.A. in publishing the Proceedings is to provide an interchange of ideas to stimulate improved examination and therapeutic methods in applied kinesiology.

It should be understood that the procedures presented in these papers are not to be construed as a single method of diagnosis or treatment. The ICAK-U.S.A. expects applied kinesiology to be used by physicians licensed to be primary health care providers as an adjunct to their standard methods of diagnosis and treatment.

There are three divisions of the Proceedings of the Summer Meeting of the International College of Applied Kinesiology-U.S.A. Division I consists of papers for members' information. Division II contains papers inviting constructive comments to be published in future editions of the Proceedings. Division III is for constructive comments on papers published in Division II and for subjects that might be included in "Letters to the Editor" of a refereed journal. Papers will be put in Division I or II at the author's request. It is expected that authors will choose Division I for papers such as anecdotal case reports, thought-provoking new ideas that have not been researched, and other types of papers that are for the membership's general information. It is expected that Division II will include papers that have a research design, or those the author has thoroughly studied and worked with and believes to be a viable approach of examination and/or treatment. Studies to test methods developed by others, often called validation studies, fit well here. This area also lends itself to editorial-type comments about the practice of applied kinesiology and its procedures. The third section is somewhat similar to the "Letters to the Editor" section of refereed journals. It provides a forum for members to comment on research design or other factors in papers previously presented. Its purpose is for us to improve the quality of our presentations and, in some cases, to provide rebuttal to presented material. Comments on papers will only be published in this area if the paper was presented in Division II inviting constructive criticism.

Neither the International College of Applied Kinesiology-U.S.A., its Executive Board, nor the membership, nor the International Board of Examiners, International College of Applied Kinesiology, necessarily endorses, approves of, or vouches for the originality or authenticity of any statements of fact or opinion in these papers. The opinions and positions stated are those of the authors and not by act of publication necessarily those of the International College of Applied Kinesiology-U.S.A., the Executive Board or membership of the International College of Applied Kinesiology-U.S.A., or the International Board of Examiners, International College of Applied Kinesiology.

INSTRUCTIONS TO AUTHORS
PROCEEDINGS OF THE ICAK - U.S.A.

Manuscripts are reviewed for format, technical content, originality, and quality for reproduction. There is no review for authenticity of material.

The ICAK-U.S.A. recognizes that the usual procedure for selection of papers in the scientific community is a blind review. However, the purpose of *The Proceedings of the ICAK-U.S.A.* is to stimulate creative thinking and critical review among its members. These papers are distributed only to the members of the ICAK-U.S.A. for general evaluation, and for the members to put into perspective the validity of the described approaches. The purpose is to put before the membership primary observations that may lead to scientific investigations, new areas of research, and in-depth study, inspiring progress in the field of applied kinesiology.

Statements and opinions expressed in the articles and communications in *The Proceedings of the ICAK-U.S.A.* are those of the author(s); the editor(s) and the ICAK-U.S.A. disclaim any responsibility or liability for such material.

The current ICAK-U.S.A. Status Statement is published with *The Proceedings of the ICAK-U.S.A.* It is recommended that procedures presented in papers conform to the Status Statement; papers that do not will be published and identified in the table of contents as failing to conform. It is recommended that examination or treatment procedures that fail to conform to the ICAK-U.S.A. Status Statement be supported by statistical studies, literary references, and/or any other data supporting the procedure.

Papers are published in three divisions: I) papers intended by the author as informative to the membership and not inviting critical review; II) papers inviting critical and constructive comments from the membership in order to improve the total value of the paper. Comments may be made on such items as research design, methods presented, clarity of presentation, and practical use in a clinical setting. The author must include with his/her paper written indication of desire for the paper to be included in the section inviting critical review or for informative purposes. III) The third section is for review comments on papers published in Division II. These papers are for constructive review. Opinions or editorials with negative connotations only, may be rejected.

Manuscripts are accepted by the ICAK-U.S.A. for consideration to publish with the understanding that they represent original unpublished work. Acceptance of the manuscript by the ICAK-U.S.A. does not necessarily imply acceptance for publishing. The author may appeal any paper rejected to a committee composed of members of the Publications and Research Advisory Committees. The decision of this committee on publishing the paper will be final.

Following are the current requirements for papers submitted for publication:

1) The paper must be an original work and deal specifically with applied kinesiology examination and/or treatment techniques. Various techniques may be discussed if they are correlated with applied kinesiology manual muscle testing examination.

2) Papers that do not include a clearly labeled **Abstract, Introduction, Discussion, Conclusion** and **Reference list** will be returned to the author for revision. Papers that discuss the outcome of a research study must also include separate sections labeled **Materials/Methods** and **Results**. Papers that describe clinical procedures or protocols should include a concise step-by-step outline or flow chart for each procedure described in the paper. The text of the paper, regardless of the subject material, should include numbered references. Note that the standard format for journal and textbook references is reviewed at the conclusion of this article. The only exceptions are papers which are *Commentaries* or *Critical Reviews*. (See explanation listed below.)

3) Quotations must be short, usually no longer than three lines, and should be referenced, giving credit to the original author. All referenced articles, books, or persons other than the author must be properly referenced at the end of the paper. (See examples listed below.)

(Instructions Cont.)

4) Any quotation of copyrighted material that is longer than that noted above must be accompanied by permission to print from the author and/or copyright holder. The permission must specifically note that the material is to be printed in *The Proceedings of the ICAK-U.S.A.*, copyrighted by the International College of Applied Kinesiology-U.S.A.

5) Any material that is copyrighted by the author must include permission for the ICAK-U.S.A. to reproduce the paper and any accompanying graphs, illustrations, etc., at any time and in any manner that the ICAK-U.S.A. so chooses.

6) All art work must be original, or permission to print must be obtained from the author or artist, referenced in the article, and a copy of the authorization sent along with the article at the time of submission for printing in The Proceedings. Photographs must be original black-and-white glossy prints.

7) Terminology or procedures that might be unfamiliar to some readers should be referenced at the end of the paper. Avoid using nontechnical terms such as, "blow-out", "cleared", "fixed", or "TL'ed". Papers that contain unsupported and unsubstantiated claims for efficacy of the therapy will be returned to the author.

8) Each page of the paper should be identified by an **abbreviated** title, the author's **last** name and a page number, all centered at the top of the paper with a 3/4 inch margin.

9) The publication standards for the health care professions typically call for more details for the following types of papers:

Research Studies - An investigation into the clinical efficacy of diagnostic and therapeutic procedures.

Case Reports - An account of the diagnosis, treatment and outcome of an unusual or otherwise significant case.

Case Studies - A comparative assessment of a series of related cases.

Clinical Procedures - Informative papers that review the procedural aspects of diagnostic or therapeutic approach - clinical protocols.

Hypotheses - A theory that explains a set of facts and presents a basis for further investigation.

Clinical Observations - Unique observations that involve manual/mechanical muscle testing and related procedures.

Commentary - Editorial-like, in-depth essays on matters relating to the clinical, professional, educational, and/or legal aspects of applied kinesiology.

Critical Review - A critique or commentary on a paper that previously appeared in Division 2 of The Proceedings.

With the exception of a *Commentary* or a *Critical Review*, all papers must conform to the following format. Note that each section must be clearly labeled.

Title & Author's Name

Abstract: A brief description of the purpose of the study, basic procedures, main findings and principle conclusions.

(Instructions Cont.)

Introduction: Summarize the rationale for the study or observation. Give background material when available and introduce the reader to what was done and why.

Materials and Methods: (for research studies) Describe the subjects and identify the methods and procedures. Present sufficient detail to allow others to reproduce the procedures for comparison of results.

Results: (for research studies) Present results in a logical sequence and summarize the important observations. Include appropriate tables and illustrations.

Discussion: Discuss the implications of the findings and any limitations. Emphasize any new and important aspects of the findings. Discuss how the findings may relate to other relevant studies or observations.

Conclusions: Unqualified conclusions and statements not directly supported by data or observation must be avoided. Make any recommendations that are appropriate and relevant to the subject matter.

Summary of Procedures: Step-by-Step or Flow-Chart style description of diagnostic and therapeutic procedures described in the paper.

References: The numbered references that correspond to the text of the paper.

For journal articles: Author(s), Title in Quote " ", Name of Journal, Vol., No., (Month/Year).

e.g. Schmitt, Jr., Walter H., "Fundamentals of Fatty Acid Metabolism - Part II," *The Digest of Chiropractic Economics*, Vol. 28, No. 2, (Sept.-Oct./1985).

For textbooks: Authors(s), Title, (City of Publication, Name of Publisher, Copyright Date).

e.g. Walther, David S., *Applied Kinesiology, Volume I - Basic Procedures and Muscle Testing* (Pueblo, CO., Systems DC, 1981).

10) The body of the article should be single-spaced on plain paper. No papers typed on office letterhead will be accepted. The manuscript must be an original with dark print, on one side of the paper only, to ensure adequate reproduction in *The Proceedings of the ICAK-U.S.A.* The margins on both sides of the paper must be a minimum of 3/4 inch, and the top and bottom margins must be a minimum of 3/4 inch when relating to 8-1/2 inch x 11 inch letter-size paper. European authors should make note of the copy height of the American standard 11 inch paper size, which relates to approximately 28 cm.

Please reread, in its entirety, the Instructions to Authors to insure that your paper will be suitable for publication.

Manuscripts that do not meet the above qualifications will be returned to the author, with recommendations for bringing the paper under ICAK-U.S.A. guidelines for possible future publication.

The articles to be published should be sent to the Publications Committee in triplicate (the original and two copies), c/o ICAK-U.S.A., P.O. Box 905, Lawrence, KS 66044-0905, (913) 542-1801.

APPLIED KINESIOLOGY STATUS STATEMENT

INTERNATIONAL COLLEGE OF APPLIED KINESIOLOGY-U.S.A.

The International College of Applied Kinesiology-U.S.A. provides a clinical and academic arena for investigating, substantiating, and propagating A.K. findings and concepts pertinent to the relationships between structural, chemical, and mental factors in health and disease and the relationship between structural faults and the disruption of homeostasis exhibited in functional illness.

A.K. is an interdisciplinary approach to health care which draws together the core elements of the complementary therapies, creating a more unified approach to the diagnosis and treatment of functional illness. A.K. uses functional assessment measures such as posture and gait analysis, manual muscle testing as functional neurologic evaluation, range of motion, static palpation, and motion analysis. These assessments are used in conjunction with standard methods of diagnosis, such as clinical history, physical examination findings, laboratory tests, and instrumentation to develop a clinical impression of the unique physiologic condition of each patient, including an impression of the patient's functional physiologic status. When appropriate, this clinical impression is used as a guide to the application of conservative physiologic therapeutics.

The practice of applied kinesiology requires that it be used in conjunction with other standard diagnostic methods by professionals trained in clinical diagnosis. As such, the use of applied kinesiology or its component assessment procedures is appropriate only to individuals licensed to perform those procedures.

The origin of contemporary applied kinesiology is traced to 1964 when George G. Goodheart, Jr., D.C., first observed that in the absence of congenital or pathologic anomaly, postural distortion is often associated with muscles that fail to meet the demands of muscle tests designed to maximally isolate specific muscles. He observed that tender nodules were frequently palpable within the origin and/or insertion of the tested muscle. Digital manipulation of these areas of apparent muscle dysfunction improved both postural balance and the outcome of manual muscle tests. Goodheart and others have since observed that many conservative treatment methods improve neuromuscular function as perceived by manual muscle testing. These treatment methods have become the fundamental applied kinesiology approach to therapy. Included in the A.K. approach are specific joint manipulation or mobilization, various myofascial therapies, cranial techniques, meridian therapy, clinical nutrition, dietary management, and various reflex procedures. With expanding investigation there has been continued amplification and modification of the treatment procedures. Although many treatment techniques incorporated into applied kinesiology were pre-existing, many new methods have been developed within the discipline itself.

Often the indication of dysfunction is the failure of a muscle to perform properly during the manual muscle test. This may be due to improper facilitation or neuromuscular inhibition. In theory some of the proposed etiologies for the muscle dysfunction are as follows:

* Myofascial dysfunction (micro avulsion and proprioceptive dysfunction)
* Peripheral nerve entrapment
* Spinal segmental facilitation and deafferentation
* Neurologic disorganization
* Viscerosomatic relationships (aberrant autonomic reflexes)
* Nutritional inadequacy
* Toxic chemical influences
* Dysfunction in the production and circulation of cerebrospinal fluid
* Adverse mechanical tension in the meningeal membranes
* Meridian system imbalance
* Lymphatic and vascular impairment

On the basis of response to therapy, it appears that in some of these conditions the primary neuromuscular dysfunction is due to deafferentation, the loss of normal sensory stimulation of neurons due to functional interruption of afferent receptors. It may occur under many circumstances, but is best understood by the concept that with abnormal joint function (subluxation or fixation) the aberrant movement causes improper stimulation of the local joint and muscle receptors. This changes the transmission from these receptors through the peripheral nerves to the spinal cord, brainstem, cerebellum, cortex, and then to the effectors from their normally-expected stimulation. Symptoms of deafferentation arise from numerous levels such as motor, sensory, autonomic, and consciousness, or from anywhere throughout the neuraxis.

Applied kinesiology interactive assessment procedures represent a form of functional biomechanical and functional neurologic evaluation. The term "functional biomechanics" refers to the clinical assessment of posture, organized motion such as in gait, and ranges of motion. Muscle testing readily enters into the assessment of postural distortion, gait impairment, and altered range of motion. During a functional neurologic evaluation, muscle tests are used to monitor the physiologic response to a physical, chemical, or mental stimulus. The observed response is correlated with clinical history and physical exam findings and, as indicated, with laboratory tests and any other appropriate standard diagnostic methods. Applied kinesiology procedures are not intended to be used as a single method of diagnosis. Applied kinesiology examination should enhance standard diagnosis, not replace it.

In clinical practice the following stimuli are among those which have been observed to alter the outcome of a manual muscle test:

* Transient directional force applied to the spine, pelvis, cranium, and extremities
* Stretching muscle, joint, ligament, and tendon
* The patient's digital contact over the skin of a suspect area of dysfunction termed therapy localization
* Repetitive contraction of muscle or motion of a joint
* Stimulation of the olfactory receptors by fumes of a chemical substance
* Gustatory stimulation, usually by nutritional material
* A phase of diaphragmatic respiration
* The patient's mental visualization of an emotional, motor, or sensory stressor activity
* Response to other sensory stimuli such as touch, nociceptor, hot, cold, visual, auditory, and vestibular afferentation

Manual muscle tests evaluate the ability of the nervous system to adapt the muscle to meet the changing pressure of the examiner's test. This requires that the examiner be trained in the anatomy, physiology, and neurology of muscle function. The action of the muscle being tested, as well as the role of synergistic muscles, must be understood. Manual muscle testing is both a science and an art. To achieve accurate results, muscle tests must be performed according to a precise testing protocol. The following factors must be carefully considered when testing muscles in clinical and research settings

* Proper positioning so the test muscle is the prime mover
* Adequate stabilization of regional anatomy
* Observation of the manner in which the patient or subject assumes and maintains the test position
* Observation of the manner in which the patient or subject performs the test
* Consistent timing, pressure, and position
* Avoidance of preconceived impressions regarding the test outcome
* Nonpainful contacts -- nonpainful execution of the test
* Contraindications due to age, debilitative disease, acute pain, and local pathology or inflammation

In applied kinesiology a close clinical association has been observed between specific muscle dysfunction and related organ or gland dysfunction. This viscerosomatic relationship is but one of the many sources of muscle weakness. Placed into perspective and properly correlated with other diagnostic input, it gives the physician an indication of the organs or glands to consider as possible sources of health problems. In standard diagnosis, body language such as paleness, fatigue, and lack of color in the capillaries and arterioles of the internal surface of the lower eyelid gives the physician an indication that anemia can be present. A diagnosis of anemia is only justified by laboratory analysis of the patient's blood. In a similar manner, the muscle-organ/gland association and other considerations in applied kinesiology give indication for further examination to confirm or rule out an association in the particular case being studied. It is the physician's total diagnostic work-up that determines the final diagnosis.

An applied kinesiology-based examination and therapy are of great value in the management of common functional health problems when used in conjunction with information obtained from a functional interpretation of the clinical history, physical and laboratory examinations and from instrumentation. Applied kinesiology helps the physician understand functional symptomatic complexes. In assessing a patient's status, it is important to understand any pathologic states or processes that may be present prior to instituting a form of therapy for what appears to be functional health problem.

Applied kinesiology-based procedures are administered to achieve the following examination and therapeutic goals:

* Provide an interactive assessment of the functional health status of an individual which is not equipment intensive but does emphasize the importance of correlating findings with standard diagnostic procedures
* Restore postural balance, correct gait impairment, improve range of motion
* Restore normal afferentation to achieve proper neurologic control and/or organization of body function
* Achieve homeostasis of endocrine, immune, digestive, and other visceral function
* Intervene earlier in degenerative processes to prevent or delay the onset of frank pathologic processes

When properly performed, applied kinesiology can provide valuable insights into physiologic dysfunctions; however, many individuals have developed methods that use muscle testing (and related procedures) in a manner inconsistent with the approach advocated by the International College of Applied Kinesiology-U.S.A. Clearly the utilization of muscle testing and other A.K. procedures does not necessarily equate with the practice of applied kinesiology as defined by the ICAK-U.S.A.

There are both lay persons and professionals who use a form of manual muscle testing without the necessary expertise to perform specific and accurate tests. Some fail to coordinate the muscle testing findings with other standard diagnostic procedures. These may be sources of error that could lead to misinterpretation of the condition present, and thus to improper treatment or failure to treat the appropriate condition. For these reasons the International College of Applied Kinesiology-U.S.A. defines the practice of applied kinesiology as limited to health care professionals licensed to diagnose.

Approved by the Executive Board of the International College of Applied Kinesiology-U.S.A., June 16, 1992.

Status Statement will be submitted to the International Council for review.

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** Material in this paper does not conform with the ICAK Status Statement

DIVISION I - INFORMATIVE PAPERS

APPLIED KINESIOLOGY MANAGEMENT OF REFLUX ESOPHAGITIS

Cecilia A. Duffy, D.C.

ABSTRACT: A case history of a 41 year old male with a 10 year complaint of "heartburn" and 3 year complaint of "food regurgitation" is presented. Diagnosis and treatment with applied kinesiology methods gave immediate symptomatic response. Specific correction of a diaphragm dysfunction and nutritional supplementation is detailed.

INTRODUCTION: Reflux esophagitis is a combination of substernal burning pain and regurgitation of acid, pepsins, or bile caused by an incompetent lower esophageal sphincter that inflames the mucosa of the distal esophagus. Patients will complain of "heartburn" and "regurgitation" with certain foods, large meals, or lying down after meals. Reflux esophagitis can occur in the presence or absence of a hiatal hernia. <1> <2>

Diagnostic examination via barium swallow confirms esophageal reflux. If conservative measures of treatment fail, further diagnostics include: Acid Perfusion Test (Bernstein) which is reproduction of symptoms with perfusion of acid into the distal esophagus; Endoscopy with Esophageal Biopsy; Esophageal Manometry to assess lower esophageal sphincter function; and Esophageal pH Monitoring which reveals the clearing time of refluxed acid. <2>

Traditional treatment includes: weight reduction; removal of conditions that raise intra-abdominal pressure (i.e. tight clothing); avoidance of recumbancy after meals; sleeping with the head of the bed elevated; avoidance of fats, chocolate, peppermint, caffeine, tobacco, and alcohol which decreases lower esophageal sphincter pressure; avoidance of citrus and tomato. <2>

Allopathic treatment includes medication such as antacids, zantac, and others. Surgery would be indicated in a small percentage when the conservative therapy fails. <2>

In applied kinesiology, "heartburn" and "regurgitation" are associated with diaphragmatic dysfunction and hiatal hernia. Correction of these disturbances can result in symptom improvement. <3>

Gastrex is composed of okra, silica, and tillandsia powder with other food concentrates from vegetable and animal sources. <4> It is clinically indicated in symptoms of heartburn and reflux. <5>

CASE HISTORY: A 41 year old male presented with a chief complaint of "heartburn" and "food regurgitation". He stated that the heartburn had been present for approximately 10 years and described it as a burning sensation behind the sternum shortly after eating at virtually all meals. He would periodically consume milk or antacid tablets to relieve the symptom. Approximately 3 years ago, he started to experience

regurgitation of the food into the mouth that would occur with at least one meal per day, and often with two meals. To avoid further irritation of the esophagus, he would spit the regurgitant out. He also continued to experience heartburn with meals if he did not regurgitate. Surprisingly, he had not sought help for these long standing symptoms prior to presentation.

The history was otherwise unremarkable except for passage of a kidney stone 20 years prior. Standard physical examination revealed the following abnormalities: obesity (height 5'10 1/4", weight 237 1/4 pounds); axillary temperature 97.4 degrees; blood pressure supine 120/90, seated 124/94, standing 120/100; pulse seated 76, standing 64; an elevated urinary chlorides via Koensburg test at 40; and diminished right thoracic expansion upon inspiration.

Recommendation for an upper gastrointestinal barium study was denied by the patient in lieu of a conservative trial of treatment prior to further diagnostic testing.

Applied kinesiological findings included: positive therapy localization at the xiphoid with inspiration; weakening of an indicator muscle when lead was placed over the acupuncture points CV24 and GV27; reactivity between the right psoas muscle and the diaphragm; fixation of the thoracolumbar junction and left C7-1st rib (Limbic fixation); and a subluxation of T4. Hiatal hernia challenge was negative. Interestingly, vital capacity and breath holding time were above normal for this patient. He was placed on Gastrex (Standard Process Inc.) <4>, one capsule 15 minutes prior to a meal. He was also instructed to lose weight with diet modification. Dietary excesses included consumption on a daily basis of whole milk, refined carbohydrates/sugars, 2-3 cans of soda, and 4-8 cups of coffee with cream.

The patient was seen one month later and reported only two episodes of heartburn and approximately 3-5 episodes of regurgitation. The following month, he reported 3 episodes of regurgitation, and then reported no heartburn or regurgitation for 8 months. In that 8 month period, he was taken off the Gastrex and was seen on a self schedule basis for other minor musculoskeletal complaints. The following 6 month period he reported 6 episodes of regurgitation. He noted that these episodes all occurred following dietary overindulgence in fatty or refined foods.

This patient was partially non-compliant with dietary modification instruction. He was vague when asked about his diet, but information was gained from his wife which revealed that he had cut down on the excesses in his diet, but still had daily consumption of dairy, refined carbohydrates, and coffee. The patient's weight fluctuated with no real loss.

DISCUSSION: A presumptive diagnosis of reflux esophagitis was made without the aid of a barium swallow, but based on symptoms. Diaphragm dysfunction and hiatal hernia were indicated and examined for. The only positive physical indicator for diaphragmatic dysfunction was seen in the diminished right lateral thoracic expansion upon inspiration. Applied kinesiological findings for the diaphragm dysfunction were positive for therapy localization of the xiphoid with inspiration and general indicator muscle weakening with lead placed over CV24 and GV27. Challenge for a hiatal hernia was negative. Correction of the diaphragm dysfunction was accomplished by reducing the neuromusculospindle cell activity in the reactive right psoas and adjustment of the thoracolumbar fixation. Following these corrections, all positive indicators of diaphragm dysfunction were negative. Prescription of Gastrex and diet modification instruction was indicated to reduce the irritation of the reflux of acids, pepsins, or bile. Since the patient only partially modified his diet (according to his wife), the large reduction in the frequency of the symptoms is presumably due to a combination of the structural correction of the diaphragm and addition of the Gastrex.

It is probable that total resolution of the heartburn/regurgitation would have occurred had the patient complied fully with the dietary modifications and weight loss. However, considering the severity of the symptoms at the time of presentation, the structural treatment based on applied kinesiology diagnosis and short term use of Gastrex provided significant recovery and symptom abatement.

CONCLUSION: Successful management of a severe case of reflux esophagitis is presented. Applied kinesiological diagnosis and treatment directed towards the diaphragm, prescription of Gastrex, and partial compliance in diet modification provided significant relief of the symptoms of heartburn and regurgitation of food.

REFERENCES:

<1> Walker, Hall, and Hurst, Clinical Methods 2nd Edition, Butterworth Publishers Inc., Boston, 1981, pages 106-108.
<2> Schroeder et al, Current Medical Diagnosis and Treatment, Appleton and Lange, Connecticut, 1992, pages 457-459.
<3> Walther, Applied Kinesiology Synopsis, Systems DC, Colorado, 1988, pages 534-544.
<4> Standard Process Inc., 1200 West Royal Lee Drive, Palmyra, WI 53156.
<5> Product Bulletin, reprinted by AK Printing, Geneva, Ohio, page 69.

CASE HISTORY: APPLIED KINESIOLOGY MANAGEMENT OF PEDIATRIC SEIZURE DISORDER AND STRABISMUS

John M. Heidrich, D.C.

ABSTRACT: A case of complex partial seizure disorder and exotropic strabismus in a seven year-old female responds rapidly to applied kinesiology technique including spinal, cranial, and nutritional therapies.

INTRODUCTION: The incidence of childhood seizure disorders is estimated at 0.5% of the population (1). Complex partial seizure disorders begin with emotional, psychic, illusory, hallucinatory, or special sensory symptoms. These are followed by clouding of consciousness with automatic behavior and amnesia. Attacks usually begin with an aura of visual, auditory, or gustatory hallucination followed by apparently purposeful automatic behavior (e.g. picking up objects, walking about,etc.) A complete attack will usually last one to three minutes and originate activity in the temporal lobe. (2) Abnormal psychic symptoms may include vivid memories, fear, and depression. Childhood seizures may be related to congenital or acquired brain lesion, however a large proportion are considered idiopathic. Pharmacological management includes drugs that increase the concentration of gamma aminobutyric acid (GABA) including phenytoin and carbamazipine (Tegretol). (3)

Strabismus, non-parallel eyes, occurs in approximately 2% of children. Exotropia is an out-turning of one eye while both are fixed on an object and most commonly intermittent. In simple cases, treatment is directed at functional correction through exercise. (1)

HISTORY: A six year-old female originally presented to my office with a three month history of intermittent nausea, stomach pain, anorexia, and left frontal headache. On this initial visit general corrections were made to the pelvis (category II), stomach and bowel reflexes, occipital and dorsal manipulations to correct what was seemingly a common complaint in most chiropractic offices. A two week follow-up for this out-of-state patient was not kept and it was assumed that she had recovered. Ten months later the patient presented to my office a second time with a diagnosis of complex seizure disorder after consulting a neurologist. In addition to her original headache and stomach symptoms, she also began to suffer left earache and was misdiagnosed with ear infection and treated with Amoxicillin. Further diagnostics were undertaken as symptoms increased.

She began to experience auditory hallucination two months after her initial presentation (IP) at my office. She repeatedly asked family members if they were able to hear various noises described as "windshield wipers". These episodes increased in frequency and duration with additional auditory symptoms of "buzzing" and visual aura of "bright, unusual colors and objects" that seemed to "float". At four months from IP, her family osteopath ordered an MRI of the head and cervical spine which was negative for organic lesion. When symptoms increased she was referred to the ophthalmologist and neurologist.

At six months from IP, she was tentatively diagnosed by the ophthalmologist as suffering migraine cephalgia. Muscle balance testing showed normal binocular tracking with right gaze, left gaze produced a large exophoria measured at 18 diopters. This problem with convergence was aggravated by near-point focus. This finding correlated with the mother's observation that when the patient responded to her name, she would turn her head to the right and gaze left. This pattern had occurred for several years and the family had dismissed this as a "cute" or "coy" over-the-shoulder glance. The remaining visual exam was unremarkable and follow-up neurologic exam was recommended. A series of "pencil push-ups" (tracking a pencil point from arms length to nose), were given by the ophthalmologist with admittedly poor patient compliance.

At eight months from IP, an EEG was performed with neurological consult. Abnormal sharp waves in the left central brain region suggested the seizure diagnosis. Sensory, motor, cranial nerve, and cerebellar exams were reported as normal . A trial of Felbatol, an anti-epileptic, was suggested by the neurologist with follow-up in three months. The mother was reluctant to use medication and, with symptoms increasing, re-presented her daughter to my office.

During this ten month interim from IP, episodes increased to a frequency of several episodes per day with increased intensity in duration from 5 to15 minutes. Symptoms included vertigo, nausea and vomiting, headache, vivid hallucinations, lethargy, and characteristic feelings of depression, impending doom, and frustration with performing tasks. The mother correlated the attack onset with reading and her daughter became increasingly reluctant to do "close-up" work.

FINDINGS: Posture revealed a gross elevation of the right ear and left rotation of the head, depression of the right shoulder and right iliac crest. Right upper trapezius and neck flexor groups were weak. Motion palpation and passive head rotation with extension showed a severe anterior rotation of atlas on the right. Goodheart describes increased rotation on the anterior atlas side when cervical spine is in extension. (4) The right transverse of the atlas, on palpation, was seemingly anterosuperior against the back of the mandible with noted palpatory pain.

I adjusted the atlas (with a resounding articulation) after correcting neurolymphatic and neurovascular reflexes for the right upper trapezius in addition to an open ileocecal valve. I then decided, in view of improved postural changes, to cautiously wait. One week following, episodes had decreased to approximately 50% in frequency and it was the "first time in months" that she went a full day without a seizure.

Successive treatment included correction of right temporal bulge, internal frontal, basic Category One, right clavicle, and neurological switching. Ocular lock pattern was found by putting her into the left head rotation with right eye laterality (opposite the pattern that she used in her "coy" pattern).

Food allergy testing was positive for egg and corn and these were eliminated from the diet. Additionally she was prohibited wheat and dairy products. Histidine challenge, was negated by Standard Process Antronex. (5) Supplementation was started after testing on the third visit and interestingly, over the next ten days, two of the four attacks occurred immediately after the food allergy testing performed on the third visit.

There have been full resolve of episodes within the past month with no recidivism of cervical and/or cranio-sacral findings. According to the mother, the ophthalmologist reports a "miraculous" return of convergence on testing, which he attributes to the (unperformed) exercises. The mother did not disclose that the child was being treated by a chiropractor. Mother and daughter have noted a sudden improvement in the patient's mood and the child doesn't feel "sad" anymore. She is scheduled for a follow-up with the neurologist in one month.

DISCUSSION: Chiropractic, since inception, has suggested favorable response with some forms of epilepsy. D.D. Palmer states in 1910:

" No case of epilepsy has ever been found that did not present a grave subluxation at stomach place. So far, I have had but one occasion to adjust other than atlas for epilepsy". (6)

It appears that this quote applies in this case, however with due respect to historic premise, we acknowledge other causative factors. Gonstead and others have correlated daytime seizures with upper cervical dysfunction, specifically involving the atlanto-occipital junction, and nighttime seizures with sympathetic nervous system lesion.(7)

With the emergence of the neuropeptide studies by Pert et. al. (8) , the chemistry of psychological stress has been implicated as another triggering factor in seizures, particularly those in the central and frontal brain regions when emotional or memory symptoms are present. (9) In this case, emotional reaction to the situation produced a vicious circle of symptoms with increasing frustration and depression.

Pharmacologically, the prescribed Felbatol acts to increase GABA (gamma- amino butyric acid) inhibitory effect on GABA sensitive neurons and raise the seizure threshold. Schmitt has detailed the various nutritional co-factors related to GABA production and has recommended screening for free radical chemical exposures. (5)

CONCLUSION: In view of potential renal and hepatotoxicity of most anti-epileptic medication, not to mention the emerging emphasis on cost-effectiveness, it is worthwhile to reconsider drug treatment of epilepsy and other "musculovisceral " (10) complaints, until a thorough applied kinesiology evaluation is conducted.

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Case History: Dupuytren's Contracture and Cervical Disc

John M. Heidrich, D.C.

ABSTRACT: A chronic case of Dupuytren's Contracture responds to applied kinesiology management of lower cervical vertebral disc derangement.

INTRODUCTION: Dupuytren's contracture is a relatively common disorder occurring in white males over age 40 and frequently associated with diabetes mellitus, alcoholism, and other systemic disorders. It is manifested by cord-like thickening of the flexor tendons, usually of the palmaris longus muscle. This condition is associated with similar plantar fascial contractions and Peyronie's disease, a painful fibrous contracture of the penis. Although considered idiopathic, a genetic predisposition is suggested. Due to the progressive nature, it is allopathically controlled with triamcinolone (prednisone) injection and surgical release in severe cases of contracture deformity. Amputation of the fifth finger is considered in extreme cases.(1)

CASE HISTORY: A 46 year-old male presented with bilateral palmar contractures of ten years duration. He complained of pain primarily in the flexor tendons at the fourth proximal phalangeal joints. There was lesser contracture and pain in both fifth flexor tendons. The patient, a socially active church minister, was also distressed with discomfort while shaking hands and hinted at being self-conscious with the cosmetic appearance. There was no past history of trauma to the head, neck, or hands, and none of the associated risk factors were positive. Secondary complaints of intermittent neck pain and acute left hip pain were listed at presentation. He had previously sought treatment for the contractures from a "preventative" medical doctor and two other chiropractors.

Initial examination of the hands revealed a flexion deformity of the fourth proximal phalangeal joint at 25 degrees right and slightly worse 30 degrees on the left. The thickened nodules over the flexor tendons were tender to palpation and well localized approximately 1/2 inch proximal to the phalangeal joint. He was unable to actively bring his fourth and fifth digits into extension and passive extension produced pain in the palmar tendon and volar forearm.

Muscle testing revealed a bilateral weakness of the wrist extensors while the patient was supine. There were no deep tendon reflex changes. Cervical range of motion was normal and orthopedic testing was negative. Bicep, deltoid, and tricep tested grade 5/5 bilaterally. C5 challenged for anteriority and lateral radiograph suggested a mild anterolisthesis at that level. This level was adjusted for anteriority en masse while the patient was supine. Goodheart describes a hidden cervical disc correction with prone rotation of the head toward the anteriority with caudal thrust "down" the facet line. (2) This set up was performed supine with the exact same choreography. There was immediate return of bilateral strength in the wrist extensors upon retesting.

Two subsequent treatments revealed recidivism of the cervical disc finding. Interestingly, the wrist extensors now showed in the usual hidden disc pattern of strong "in the clear" with weakening upon application of vertex pressure to the head. An open ileocecal valve was treated and corrected over the following two treatments. Additional findings/corrections included Lovett correction of L1 anterior, left hamstring, Category II posterior ischium, and dietary recommendations to eliminate his "beloved" popcorn. Nutritional supplements included long-term self administration of Brewer's yeast, cod liver oil, a generic multi-mineral tablet, and Standard Process whole spleen previously prescribed, coincidently, by the "preventative" M.D. in our area. Whole dessicated spleen provides a source of the recommended superoxide dismutase. (3)

One month after the third treatment, the patient noted onset of intense itching in the flexor nodules. He was treated two additional times within the following two months with additional corrections to occipital and limbic fixations, recurrent ileocecal valve and a "tune-up" to the C5 anteriority. At the end of this period, he noted marked improvement in pain and hand range of motion with ability to fully place his palm on a flat surface without contracture pain or restriction. Eighteen months have passed with no return of pain or contracture. There has been no change in the palmar thickening.

DISCUSSION: Standard medical and orthopedic evaluation for "true" cervical disc herniation is usually dependent on correlating symptoms of radicular pain, reflex changes, and specific nerve root motor weaknesses. Goodheart has correlated the work of Kabat who contends that remote problems, particularly low back pain, can be influenced by cervical disc lesions. He also references others that utilize wrist muscle strength in assessing the cervical spine. (3) Although wrist extensor weakness can be indicative of C6 nerve root involvement, this case had no other clinical signs of severe nerve compression which usually precede or concur with gross motor weakness. Non-radicular "vertebrogenic" disc derangements are considered much more common than "neurogenic" types which produce mixed cervical pain and neurologic signs. (4,9)

Duffy has outlined the consistency of the ileocecal valve syndrome as a causative factor in most cases of carpal tunnel. (5) He has noted a consistent finding between wrist extensor weakness and the ileocecal valve effect on lymphatic drainage of the carpal tunnel and upper extremity. He emphasizes proper testing of the wrist extensors with the elbow in full extension to avoid false negative findings. (6)

A recent paper by Koffeman (7) gives case report of successful management of Peyronie's disease, found in over 2% of Dupuytren cases. (1) Since these conditions are considered idiopathic or attributable to genetic connective tissue disorders, (8) it may be possible that altered biomechanics, meridian imbalance as suggested by Koffeman, and other acquired dysfunctions in the health triad may aggravate the expression of symptoms in these disorders.

CONCLUSION: This case suggests immediate functional motor change upon correction of a cervical vertebral subluxation and attendant disc lesion. This correction resulted in apparent resolve of a chronic Dupuytren's contracture. It is suggested that Dupuytren's contracture may be influenced by functional derangement in remote areas.

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BIOMAGNETIC KINESIOLOGY PROTOCOL UPDATE

MICHAEL LEBOWITZ D.C.

ABSTRACT:

The present biomagnetic kinesiology protocol is presented with comments on new or updated procedures.

INTRODUCTION:

For many within the field of applied kinesiology, the biomagnetic protocol[1] has been an effective, efficient, experimental method of evaluating and treating the chronic patient. As time has gone on with continuing research by myself and other practitioners, techniques have been streamlined, added to, improved, etc. In this paper I will attempt to briefly elaborate on some of the changes and additions as well as present the complete summary of procedures. In the discussion section we will list these in the order they come up along with comments. In general I have found that treating the patient for positive biomagnetic indications for dysbiosis, food, chemical, and metal sensitivities, etc., almost always will greatly improve the symptom picture of the patient. Interestingly, this will happen no matter which body system is involved.

It appears that when the body is bombarded by micro-organism, xenobiotics, etc., it is very difficult for it to start a self healing process. When we can identify and treat these culprits both internal and external (most have been identified as neurotoxins), the body can then "heal itself". Biomagnetic kinesiology findings can be backed up with lab tests for the patient or practitioner who wishes to do so. Personally I find that it rarely helps me treat the patient while greatly adding to their bill. We must remember though that we are treating biomagnetic indications and it is not technically correct to tell people they have a parasite, lead toxicity, etc. based on a muscle test. It is accurate to say we found a "neurological change on biomagnetic exposure to certain substances which leads us to suspect the following problems...".

DISCUSSION:

1) To minimize false negatives, avoid certain types of switching, etc., before starting the protocol we make sure the atlas is not subluxated (if it is we adjust it). We also rub GV-21 for 30 seconds. If we do these, we find that we can then screen all substances over GV-27. We no longer have to test over dysbiotic pockets, symptomatic areas, etc. Testing over GV-27 is adequate.

2) For children with fungal problems or adults who are sensitive to SF722[2], and Undecyn[2], we screen with and use Coptis[2] .

3) If GV-21 is stimulated, we no longer have to screen foods for both strengthening and weakening. Contraindicated foods will weaken a strong muscle.

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4) We therapy localize the parotid gland on each patient one side at a time. If either side is positive we rub it, which will abolish our positive therapy localization. Then when we screen foods we test them both with and without a simultaneous parotid therapy localization. Some foods only test positive in combination with the positive parotid therapy localization. These are usually negated by either calcium citrate[2], or E-500[2] and appear to be a different type sensitivity, perhaps IgA mediated.

5) For toxic metals there are usually some spots on the cervical lymph chain that will negate a positive test. These are stimulated while the patient is exposed to the metal. Also the herb Salix Wolfii[2] is extremely useful in some cases of toxic metal problems.

6. Screening for and treating histamine and tyramine as in the summary of procedures listed later are valuable additions to the protocol, though the physiology is beyond the scope of this paper.

7) Lymphatic congestion that eludes traditional applied kinesiology analysis is a very common problem and will be discussed in a future paper. We list in the protocol a technique of evaluation and treatment we have found useful.

8) Wearing metal jewelry in about half of my patients can both mask positive neurolymphatic reflexes and hinder proper detoxification. If the patient shows different findings with jewelry off and on they are better off not wearing it.

9) The tap and zap technique is quite useful in patients that you want to uncover findings that are not "showing" under usual analysis. This technique "resets the circuits" and "desensitizes" previously tested positive items simultaneously. This way we see a whole new layer so to speak.

10) These are incredibly brief explanations as this paper is directed towards practitioners who have already attended one of our seminars or watched a seminar video. If you haven't and wish to do this work, we strongly recommend you obtain a video[3], attend a seminar, or study with an experienced practitioner first to gain practical experience and understand the academics of it all.

CONCLUSION:

Biomagnetic kinesiology testing is an experimental procedure that has proven extremely useful to many practitioners. It continues to improve thanks to the input of many clinical researchers.

REFERENCES:

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2. Available from Thorne Research 1-800-228-1966
3. Available from the author 1-303-856-7573

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SUMMARY OF PROCEDURES

1. Find a weak G-2 muscle and a strong intact muscle. Rub GV-21 hard for 60 seconds to eliminate switching and minimize false negatives. Place the following substances under the south pole of a 4x6 or 2x5 magnet [4], one at a time, over GV-27: SF 722[1], Undecyn[1], Artecin[1], Citricidin[1], Entrocap[1], Berbercap[1], Isatis[1], Phytogen[1], Goldenseal[2], Echinacea Supreme[2], or appropriate pharmaceuticals. If any of the above supplements causes a weak G-2 muscle to strengthen, refer to pages & entitled **"FUNGUS CANDIDA PARASITES BACTERIA VIRUS."**

Make sure the atlas is not subluxated before any testing. If it is, adjust it before doing any of the protocol. DO ALL TESTING OVER GV-27 for the most accurate results,

2. Test commonly eaten foods (antigens)[3] to see if they weaken a strong indicator muscle. See page , entitled **"FOOD SENSITIVITIES"** for the protocol.

3. Screen for parotid IgA food sensitivities by having the patient therapy localize each parotid gland, one at a time, to see if either weakens a strong indicator muscle. If so, refer to the bottom of page , entitled **"PAROTID IgA FOOD SENSITIVITY."**

4. Test to see if a weak G-2 muscle strengthens on therapy localization to the pectoralis minor NL. Also, see if the patient tests positive on their sweat or the axilla sniff test. If positive, refer to page entitled **"CHEMICAL SENSITIVITIES."**

5. Test toxic metals to see if they weaken a strong indicator muscle. If so, refer to page entitled **"TOXIC METAL."**

6. Test Tyramine and Histamine to see if either weakens a strong indicator muscle. If either is positive, refer to page , entitled **"TYRAMINE/HISTAMINE."**

7. Test to see if a G-2 weakness is strengthened on exposure to Basic Nutrients IV or V[1]. If so, supplement two capsules, three times daily at the end of meals. We feel these are the best supplements available for replenishing nutrients. If negative, test to see if a G-2 weakness strengthens on the following hypoallergenic nutrients, all from Thorne[1]:

Basic Nutrients III	Tracemins	PICOLINATE form minerals:
Basic B Complex	Citramins	Boron
B Complexes 1,3,5,6,& 12	Pic-Mins	Copper
Pyridoxal 5' Phosphate	Beta-Carotene	Chromium(or Ultrachrome)
Cobamamide		Iron
Folacal	CITRATE form minerals:	Manganese
Ferrasorb	Calcium	Molybdenum
Ascorbic Acid	Magnesium	Selenium
E-500	Potassium	Zinc
Biomins	Cal-Mag effervescent powder	

Supplement with the appropriate nutrients.

8. Test to see if a G-2 weakness strengthens on Super EPA[1], Black Current Oil[1], or Omega Plus[1]. If so, supplement one capsule three times daily.

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9. Test to see if a G-2 weakness strengthens on L-Tyrosine[1], or Iodine-Tyrosine[1]. Supplement one capsule 2-3 times daily.

10. Test for **ELECTROMAGNETIC FIELD SENSITIVITY.** See page 11.

11. Test for **MOLD SENSITIVITIES.** See page 12.

12. Test for **LYMPHATIC CONGESTION.** See page 13.

13. Test for **ELECTROMETALLIC SENSITIVITY.** See page 13.

14. Test for **DYSBIOTIC TOOTH POCKET.** See page 15.

15. Treat the **MASTER SET POINTS** (page) or perform **"TAP & ZAP"** (page) if you wish to attempt to "uncover more layers."

16. Adjust subluxations, give the supplements found and the diet information.

The patient should be rechecked in 21 days, weekly if convenient. 80% of the patients will feel tremendously better in 21 days.

A. Recheck formerly positive findings and retreat as necessary.

B. Recheck the items that were negative on the first visit, as problems are often revealed in layers. This is especially true with toxic metals.

C. Recheck nutrients, repeating steps 7-9. Continue to supplement with all that strengthen a weak G-2. Often the patient's nutrient needs change. Many times Basic Nutrients III, IV, or V will not be found on the initial exam but will show up on subsequent exams. Chronic patients often need it for many months.

If adequate improvement is not achieved quickly, keep running through the protocol until the patient is clear. Don't be afraid to be creative or to call me if you're stuck.

Once the patient is relatively asymptomatic, occasional structural fine tuning and continuing determination of nutrient needs is necessary to maintain this status, as is a good diet, exercise, and a somewhat nontoxic lifestyle. A few patients with Chronic Fatigue Syndrome; history of steroid, estrogen, or recreational drug use; hepatitis; or severe chemical sensitivities are slower to respond, but will if they are compliant and patient.

SOURCES OF NOTED PRODUCTS:

1. Thorne Research 1-800-228-1966, 1-208-263-1337
2. Gaia Herbs 1-800-831-7780, 1-508-456-3049
3. International Biologicals 1-800-345-5719, 1-405-373-3400
4. Mid American Marketing 1-800-922-1744, 1-219-749-6666

BIOMAGNETIC PROTOCOL UPDATE - LEBOWITZ - PAGE 5

FUNGUS, CANDIDA, PARASITES, BACTERIA, VIRUS

1. Test the following substances and see which strengthen a G-2 weakness when placed over GV-27:

a) SF 722[1] and Undecyn[1]- If positive suspect fungus or candida. To differentiate fungus and candida, see if a powdered antigen mix of candida albicans and tropicalis[3] causes universal muscle weakness when tested over the same area the supplement was positive. If it does, suspect candida; if not, suspect another fungus.

b) Artecin[1] and Citricidin[1]- If positive, suspect parasites or occasionally fungus. Black Walnut[2] may also be checked.

c) Phytogen[1], Isatis[1] and Goldenseal(glycerin form)[2]- If positive, suspect bacteria. You might also keep the alcohol form of goldenseal[2] on hand.

d) Echinacea Supreme[2] and Isatis[1]- If positive, suspect virus.

e) Berbercap[1] and Entrocap[1]. Effective against bacteria, parasites, and fungi, but usually indicative of bacteria.

f) SF 734[1]. Test over the stomach, especially in folks that eat a good amount of chicken and/or eggs.

2. Treat the master set points. The G-2 muscle should now be strong.

3. If fungus and/or candida are positive, the patient must abstain from the following foods for three weeks or until the indications are gone: all sweeteners (sugar, honey, maple syrup, corn syrup, etc.), fruit juice, dried fruit, vinegar, cheese, yeast, alcohol, soy sauce, and other fermented products.

4. Supplement with whatever products negated the weakness in the following doses:

a) SF 722 Undecyn	3 capsules, 3 times per day; 1-3x/day.	If both test positive, we give only one, SF 722.

GENERAL NOTE - Occasionally less than three weeks of supplementation is needed. In these cases the patient becomes symptom free after a week or so, only to have fatigue and headaches (intestinal cramping in the case of Berbercap). Discontinuing the supplement will restore them to a symptom-free status.

b) Artecin Citricidin	1-3x/day;	Use whichever negates the weakness or both, if both do.
Black Walnut	10-15 drops in water-3x/day	
c) Phytogen	1-3x/day	
Isatis	3-3x/day	
Goldenseal	10-20 drops, in water, 3x/day	

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FUNGUS, CANDIDA, PARASITES, BACTERIA, VIRUS -con't

d) Echinacea Supreme — 20 drops, in water, 3x/day

e) Berbercap/Entrocap — 1-3x/day (use only one of these)

f) SF 734 — 1-3x/day

6. Warn patients that for up to a week they may experience a temporary aggravation of their symptoms due to die off of organisms and the withdrawal from some foods.

7. Recheck positive findings in three weeks, most often they are negative. If not, consider the following:

a) If fungus is still positive:
   1) In rare cases, it may take six weeks at double normal doses to clear.
   2) Test an air sample taken from the bedroom and the place of employment to see if it causes a G-2 weakness. If positive ozonate[5].
   3) If applicable, you MUST also check and treat their sexual partner.
   4) If the patient is on estrogen, birth control, Provera, Premarin or steroids, they may may not clear until they go off them.

If the patient, once clear, must take antibiotics or steroids, have them take SF 722 or Undecyn (3/day) simultaneously to prevent recurrence.

b) If parasites still test positive, check a sample of the patient's water. If it weakens them and is negated by the antiparasite supplement, filtration will be necessary. Also check the patient's sexual partner, if applicable.

c) If bacteria or virus are still positive, increase the supplement dosage and take the patient off sweeteners. Also check the patient's sexual partner, if applicable.

d) If the formerly positive supplement tests negative, discontinue it, but continue positively tested vitamins, minerals and essential fatty acids.

8. To minimize the chance of recidivism, food sensitivities should be tested and positive foods avoided at the same time as treating the above.

9. Sometimes things show up sequentially, like peeling an onion, and you might be treating fungus one time, parasites the next, etc.

SOURCES OF NOTED PRODUCTS:

[1] Thorne Research 1-800-228-1966, 1-208-263-1337
[2] Gaia Herbs 1-800-831-7780, 1-508-456-3049
[3] International Biologicals 1-800-345-5719, 1-405-373-3400
[4] Mid American Marketing 1-800-922-1744, 1-219-749-6666
[5] NEEDS 1-800-634-1380

BIOMAGNETIC PROTOCOL UPDATE - LEBOWITZ - PAGE 7

FOOD SENSITIVITIES

1. A person is sensitive to a food if the antigen[1] is placed under the south pole of a magnet over GV-27 or the symptomatic area and weakens a strong indicator muscle. Foods can be tested as many as four at a time. Before testing, treat GV-21 with a firm digital pressure for 60 seconds. This eliminates the need to test foods for both strengthening and weakening.

2. Test the positive foods to see if either Basic Nutrients IV or V, Pyridoxal 5' Phosphate, Zinc Picolinate, Copper Picolinate, or B Complex #3 negates the weakening caused by the food. Supplement accordingly using the doses listed below. Supplements may be cross-checked by checking them "in the clear" on a weak G-2 muscle.

3. Treat the master set points. The foods should now test negative.

4. Have the patient avoid the positive foods for three weeks.

5. Supplement[2] dosages:

a) Basic Nutrients IV or V 2 capsules, three times daily
b) Pyridoxal 5' Phosphate 1-3x/day, (1-2x/day if on BN IV or V)
c) Zinc Picolinate 30 mg/day
d) Copper Picolinate 2-4 mg/day
e) B Complex #3 1-2/day, (0-1/day if on BN IV or V)

6. Retest foods in three weeks. If negative (most are), have the patient reintroduce them one per day and watch for symptoms. If there are no symptoms, continue to eat the food. If there is a reaction, avoid the food a little longer.

PAROTID IgA FOOD SENSITIVITY

1. Have the patient therapy localize each parotid gland (wedged between the ramus of the mandible and the mastoid process, in a triangle from the mastoid process, angle of mandible and midpoint of the zygomatic arch), one at a time, and see if either weakens a strong indicator muscle. If negative, skip this protocol. If positive, continue with the protocol found below.

2. Rub the positive parotid(s), as if it were an NL, until it no longer TL's.

3. Test foods, as above, while the patient TL's the parotid. Some foods will only test positive in conjunction with the parotid TL.

4. Test the positive food(s) to see if either Calcium Citrate or E-500[1] negate the weakness. Supplement Calcium Citrate, 1-3x/day, and/or E-500 1-1x/day.

5. Test to see if touching the beginning (CV-1) and end (CV-24) of the conception vessel negates the weakening. If it does, treat both points by rubbing as you would for a NL.

6. Treat the master set points and avoid the foods found for three weeks. Retest and follow the instructions as in #6 above.

SOURCES:
1. International Biologicals 1-800-345-5719
2. Thorne Research 1-800-228-1966

BIOMAGNETIC PROTOCOL UPDATE - LEBOWITZ - PAGE 8

CHEMICAL SENSITIVITIES

1. There is no definitive one-step A.K. screen for chemical sensitivities. A good patient history is important. Are there any odors that make the patient feel ill? Do they get headaches, irritable, and/or fatigued from shopping malls, fabric stores, car rides, freshly painted rooms, perfumes, etc.?

2. Patients with moderate and/or acute sensitivities will often therapy localize to the pectoralis minor NL. They will also often weaken when tested on a sample of their own sweat or upon sniffing under their axilla. The patient cannot be wearing deodorant for an accurate sniff test.

3. Test chemicals by placing a vial of the substance under the south pole of the magnet over GV-27. Chemicals to test include phenol, formaldehyde, gasoline, and chlorine. A positive chemical will cause a strong muscle to weaken. In very sensitive patients, it is advantageous to test everything they apply to their body.

4. Test to see if any of the following supplements[1] negate the weakness: Cysteplus, Selenium Picolinate, L-Carnitine, L-Glycine, Anti-Oxidant, E-500, Taurine, Glutathione and Spirulina.

5. Treat the master set points and if positive, the pectoralis minor NL. The chemicals should now test negative.

6. Doses for supplements:

a) Cysteplus	one, three times per day
b) Selenium Picolinate	1-2x/day
c) L-Carnitine	1-3x/day
d) L-Glycine	1-3x/day
e) Anti-Oxidant	1-3x/day
f) E-500	1-1x/day
g) Taurine	1-3x/day
h) Glutathione	1-2x/day
i) Spirulina	1 tablespoon daily or 12 capsules

7. Have the patient avoid the chemical(s), if possible, to give their immune system time to heal and their overloaded detox systems time to "catch up." On future visits, test their air from home and work and water to see if they are sensitive to them.

8. Increase pure water intake, exercise, and exposure to sunlight.

9. Fixing dysbiosis, food sensitivities, and nutrient deficiencies greatly frees up detox systems and aids greatly in helping patients overcome chemical sensitivities. Do not neglect to treat these.

10. If a patient continues testing positive to their sweat or axilla sniff test, sauna therapy is recommended.

SOURCES:

1. Thorne Research 1-800-228-1966

BIOMAGNETIC PROTOCOL UPDATE - LEBOWITZ - PAGE 9

TOXIC METAL

We routinely test patients for metal toxicity/sensitivity. We prefer the test kit from Pure & Simple, phone number 1-908-271-0275.

1. Test to see if a strong indicator muscle weakens when the metals are placed under the south pole of a magnet over GV-27.

For metals in the patients mouth, including composite fillings (mercury, nickel, zinc, copper, silver, gold, aluminum, barium, etc.), have the patient therapy localize a tooth with the metal in it while you simultaneously test that same metal under the magnet.

2. Many times metals will not show up on an initial visit unless you **TAP & ZAP** (page 12). They will on a later visit, after other things are corrected. Some do not show up for many months. Some may, especially after amalgam removal, recur periodically when the body is capable of eliminating more.

It is possible to have a toxicity to the inorganic form of a metal and a deficiency in the usable form of the same mineral. Ex. iron and copper.

3. Test to see which of the following supplements[1] negate the weakness.

a) Glutathione	one capsule, two times per day	
b) Metaplex	2-3x/day	
c) Cysteplus	1-3x/day	
d) Taurine	1-3x/day	
e) Biomins/Citramins	2-2x/day	
f) Tracemins	1-3x/day	
g) Ascorbic Acid	1 Tsp-3x/day	Occasionally g,h, and b must be
h) Selenium Picolinate	1-2x/day	tested together to show positive.
i) Salix Wolfii	15 drops-3x/day	
h) Medibulk/Herbbulk	1 teaspoon-3x/day	
j) Spirulina	1 tablespoon or 12 capsules/day	

4. Supplement with all that test positive to get the metal out ASAP.

5. Before treating the master set points, find a spot on the cervical lymph chain that negates the metals. Rub it. If metals still test positive, find another spot. Repeat until metals test negative, then treat the master set points.

6. Retest metals frequently. If mouth metals continue testing positive after a few months, changing to a biocompatible material is recommended.

SOURCES:

1. Thorne Research

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TYRAMINE & HISTAMINE

TYRAMINE

1. Place a vial of tyramine[1] under the magnet over GV-27 and test to see if a strong indicator muscle weakens.

2. If so, test to see which of the following negates the weakness:

a) Niasafe[2]	If positive, supplement one capsule twice daily
b) B Complex #3	1-2x/day
c)Basic Nutrients IV	2-3x/day
d)Ascorbic Acid	1500-2000 mg/day

Supplement with the broadest spectrum product above that negates the weakness.

3. Treat the master set points.

4. If tyramine remains positive on subsequent visits, treat again, as above, with a little extra laser time on GV-20. If that does not work, avoid tyramine-rich foods for a few weeks (wine, cheese, sauerkraut).

HISTAMINE

1. Place a vial of histamine[1] under the magnet over GV-27 and test to see if a strong indicator muscle weakens.

2. If so, test to see which of the following negates the weakness:

a)Folacal[2]	If positive, supplement one capsule twice daily
b)Bio-B12	1-2x/day
c)Basic B Complex	1-2x/day
d)Basic Nutrients IV	2-3x/day

Supplement with the broadest spectrum product above that negates the weakness.

3. Treat the master set points.

4. If histamine remains positive on subsequent visits, treat again, as above, with a little extra laser time on GV-20.

SOURCES:

1. Spectrum Chemical 1-800-772-8786
2. Thorne Research 1-800-228-1966

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ELECTROMAGNETIC FIELD SENSITIVITY

1. This condition is fairly common in people who spend a great deal of time around computers, electric typewriters, TV's, waterbeds, electric clocks(within 3 feet of the bed), electric blankets, fluorescent lights, etc. The target organ of these fields is the pineal gland. Symptoms can be similar to those of allergies and dysbiosis.

If six or more metals test positive on a patient there is almost always an EMF problem. You will also find that in patients with EMF sensitivity, your treatments will not "hold" well.

2. There are three steps to test for this problem.

a) Test to see if a strong muscle weakens when the south pole of a diagnostic magnet[1] is placed on GV-20, the pineal reflex. If this is positive and negated by Pineal Plus[2], there is a high probability the patient is EMF sensitive.

b) If step A is negative, see if a strong indicator muscle weakens when a source of high EMF's is turned on near the patient (TV, hairdryer, viewbox, adjusting instrument). Test to see if Pineal Plus negates the weakness.

c) If step B does not cause a weakness, repeat with the patient touching GV-20 with the diagnostic magnet at the same time. Test to see if Pineal Plus negates the weakness.

3. Treat the master set points.

4. If appropriate, supplement with Pineal Plus, one in the morning and one in the evening.

5. Counsel the patient on decreasing EMF exposure. A Tri-Field meter[1] is an excellent instrument to locate high EMF sources on your environment. Remember, EMF's cannot be shielded out.

6. A patient sensitive to a battery powered watch or heart monitor usually only test positive by placing the south pole of a diagnostic magnet on the device while the patient is wearing it. In a sensitive patient, a strong muscle will weaken. Pineal Plus will most often negate the weakness.

SOURCES:

1. Mid American Marketing 1-800-992-1744
2. Thorne Research 1-800-228-1966

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MOLD SENSITIVITES

Mold sensitivities can lead to a fungal dysbiosis or continue after a fungal dysbiosis is cleared.

1. One at a time, place Mold MIxes A, B, C[1] under the south pole of a magnet over GV-27. If any one of them causes a strong muscle to weaken, continue.

2. If any of the positive Mold Mixes is negated by SF 722, Undecyn or the appropriate pharmaceuticals, it is probably part of a fungal dysbiosis. If so, see pages 3 and 4. If not, continue.

3. Test to see which of the following negates the muscle weakness, caused by the mold antigen, when placed under the magnet at the same time as the sample:

a) Selenium Picolinate[2]	If positive, supplement one capsule twice daily
b) SB 313[2]	1-3x/day
c) HMC Hesperidin[2]	1-3x/day
d) Ascorbic Acid-500mg[2]	1-3x/day

4. Treat the master set points.

5. If the mold mixes continue to test positive on future visits, see if an air sample from home or work tests identically as the positive mold antigen. If so, ozonation and/or aggressive cleaning may be appropriate.

SOURCES:

1. International Biologicals
2. Thorne Reasearch

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LYMPHATIC CONGESTION TECHNIQUE

1. Using a strong indicator muscle, gently challenge along the cervical lymph chain in a superior direction, opposite the normal lymph flow. Do one side at a time. See if the muscle weakens. This is positive about 60% of the time.

2. Challenge along the axillary lymph nodes with a three finger sweeping motion, in an superior to inferior direction. Also challenge along the inguinal lymph nodes in a lateral to medial and slightly superior to inferior direction. The direction of the challenge is opposite to the normal flow of the lymph system. If the muscle weakens, the test is positive.

3. Massage Cleavers herb (Gaia) into the positive area and retest. The test should now be negative.

4. Gently massage the positive areas in the normal direction of lymph flow.

5. Instruct the patient to repeat steps 3 and 4 at home, twice daily for two weeks. Occasionally some toxicity reactions might occur.

ELECTROMETALLIC SENSITIVTY

1. Have the patient therapy localize the NL's of the corresponding meridians that have metal encircling them. For example, a wedding ring on the fourth digit encircles the triple warmer meridian. Therefore you would test the adrenal and thyroid NL's. They will most often test negative.

2. Remove the metal object (watch, braclet, ring, etc.) and retest. Often, the NL's will now test positive.

3. If so, treat the NL with the object off and have the patient stop wearing it.

4. If a chemically or metal toxic patient does not weaken on urine or sweat samples, it is often a clue that this phenomena will be positive. Have the patient stop wearing the object(s) and the sample will soon be positive. The metal appears to mask the positive NL and sample and also hinder proper detoxification.

Thumb- Lung NL

Index finger- Large Intestine NL

3rd finger- Circulation-Sex NL

4th finger- Triple Warmer NL

5th finger- Heart, Small Intestine NL's

Watch or braclet with metal band- all of the above NL's

Metal necklace- bladder, conception vessel, governing vessel (GV-1 & GV-27), gall bladder, kidney, large intestine, small intestine and stomach.

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MASTER SET POINTS

The master set points, when treated with a laser[4] will "reset" all positive findings that were pretested during that visit. It is better to treat them before challenging for subluxations.

1. Find a G-2 weakness and a strong intact muscle. Do all the diagnostic work-up in our protocol, determining sensitivities, dysbiosis, supplement needs, etc.

2. After all testing has been completed, place a vial of Pineal Plus[1] under the south pole of a diagnostic magnet over each of the master set points. In many cases this will strengthen a weak G-2. Supplementation will help stabilize the patient.

3. There are four master set points. The first pair is located on the eyebrows, directly superior to the pupils and occasionally slightly more medial. The third and fourth are GV-20 and GV-21. If the patient is chemically sensitive, add GV-19.

GV-19 is on the posterior fontanel near the adrenal NV
GV-20 is the most superior point on the head.
GV-21 is on the anterior fontanel.

4. Treat them with a laser[2] for ten seconds each. 20-30 seconds is needed if you are using them to correct subluxations. Tapping may be done in place of the laser, but it is not as effective.

Everything that was pretested (foods, chemicals, NL's, muscles) should now test negative. Exposure to them during the treatment of the points is not necessary as was previously done, if they were pretested. The G-2 muscle will also now test strong.

5. Supplement with Pineal Plus if indicated, one in the morning and one in the evening.

6. On future visits, the master setpoints can again be used to treat your findings. Rarely do we find a need to use alternative desensitization procedures.

7. Therapy localize the pubococcygeus muscles (part of levator ani that arises from the pubis and runs posteromedially to insert into the anococcygeal ligament and into the pelvis surface and sides of the coccyx) one at a time. If either causes a strong indicator muscle to weaken, perform origin/insertion work to the muscle. This will help stabilze the master set points.

8. *MASTER SET POINTS DO NOT ALWAYS THERAPY LOCALIZE, BUT SHOULD BE TREATED REGARDLESS. TREATMENT OF THE MASTER SET POINTS DOES NOT NEGATE THE NEED FOR SUPPLEMENTS AND DIETARY INSTRUCTIONS.*

SOURCES:

1. Thorne Research
2. Mid American Marketing

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TAP & ZAP

Tap & Zap is a technique to "bring up" more findings in the course of a visit when you feel there is more to find than your tests revealed and/or you would like to treat another "layer" on that visit.

Toxic metals, parasites, emotional findings, etc., often readily appear after this procedure. Treatment then brings gratifying results.

1. After you have run through the protocol and you suspect something that appears negative is actually positive (a particular organism, metal, etc.), perform Tap & Zap: have the patient tap the temporal bones bilaterally while you treat the master set points.

2. If the G-2 muscle strengthens, the protocol for that day is, in fact over.

3. If the G-2 muscle remains weak, you will still find, as in other master set point treatment, that all pretested findings are now negative. Retest portions of the protocol that you suspect, or that were negative earlier in the visit. New findings will be found at this time. Find the appropriate supplements, etc.

4. Tap & Zap again. The treatment continues in the same fashion until Tap & Zap strengthens the weak G-2. The number of layers found will vary from patient to patient.

DYSBIOTIC TOOTH POCKET

Subclinical infections at the tooth roots can "feed" gastrointestinal dysbiosis or be the result of it. Checking for it in difficult patients often proves quite valuable.

1. Take the south pole of a diagnostic or stick magnet[1] and place it at each tooth root. Test to see if a strong indicator muscle weakens.

2. Whenever muscle weakness occurs, place the "killer" substances listed under **FUNGUS CANDIDA PARASITES BACTERIA VIRUS** (page 3 & 4) between the south pole and the tooth root which negates it. Supplement as described.

3. Rub out the appropriate NL reflex for the tooth. They are inferior to the clavicle and as far lateral from the sternum as the tooth is from the midline. Laser[1] the tooth root for three minutes.

4. If the tooth root continues testing positive for many weeks, referral to a dentist for evaluation should be considered.

SOURCES:

1. Mid American Marketing

Use of Polyunsaturated Oils as a Screen for Hypothyroid Conditions

by
Kathleen M. Power, D.C.

ABSTRACT

Polyunsaturated oils such as flax and wheat germ oils can be used as a screen for hypothyroid conditions in patients whom you suspect to be hypothyroid but whose teres minor muscles test strong "in the clear". Monounsaturated oils and naturally saturated fats may also help in differentiating several factors involved in hypothyroid patients.

INTRODUCTION

In a recent article in the Townsend Letter for Doctors (also printed in the April Health Freedom News), Dr. Raymond Peat discussed the fact that the thyroid is frequently associated with problems of insomnia and hyperactivity. He distinguished the easy relaxation and sleep of normal, healthy cells from the abnormally excited state of cells which, he stated in more technical terms, did not have enough energy to relax. He stated that he has had excellent results with insomnia and other "hyperactivity"-type cases by treating the thyroid; his therapy is often nutritional, but occasionally he uses hormone preparations containing both T3 and T4.[1] Following his lead, I have begun to advise those patients taking thyroid support -- prescription or nutritional -- and still experiencing sleeping trouble to take their thyroid prescription medication at night; often that has taken care of the problem.

In an earlier article in the Townsend Letter, he had said, "Unsaturated oils interfere with thyroid function in several ways, including blocking the...enzymes involved in the release of hormone from the globulin. Unsaturated fatty acids interfere with binding of the hormone to a transport protein, and with the conversion of T4 to T3 in the liver and in the pituitary...The tissue response to the hormone is inhibited by unsaturated fats in proportion to the number of double bonds in the fat."[2] The same effect does not take place with naturally saturated fats, he added, citing Crile's findings that in the Yucatan, where coconuts are a staple food, metabolism is 125% that of people in the United States.[3]

In reading the works of Dr.Johanna Budwig[4,5], Udo Erasmus[6], and others, one would believe that flax oil, one of the richest electron donors in the food supply, would resolve most metabolic disturbance. Dr. Royal Lee gave polyunsaturated oils (PUFAs) to his hypothyroid patients, and developed what is now Linum-B_6 in part for that condition.[7] We have all noticed that many of the symptoms of hypothyroid patients are the same as those of unsaturated fatty acid deficiencies -- dry skin, increase in weight, constipation, etc.

However, we have to admit that vegetable oil is an unnatural and incomplete food, just as orange juice is unnatural; an extract of a health-giving food may not perform the same way in our bodies as the food itself. Even within the seed or nut, the unsaturated oil acts to inhibit protease, according to Dr. Peat, and this interferes with the transport and action of the thyroid hormones.

I thought about a number of patients who had trouble sleeping, who were hyperactive, had low pulse rates and/or axillary morning temperatures, who had high blood fats, etc., but whose teres minor muscles were strong "in the clear".[8] There are many ways to look for hidden weaknesses, but I decided to see what would happen if I tested the teres minor muscles of these patients using flax oil. I chose flax oil because of its excellent PUFA balance and the fact that an excellent organic product is available for testing. I used other oils as well to confirm my findings or to differentiate the findings using flax oil from those using other oils. I also tested fresh ground flax seed.

MATERIALS AND METHODS

For testing purposes, I used the following sources:

Fresh Flaxseed Oil from Omega
Wheat Germ and Black Currant Seed Oils from Standard Process
Olive Oil from Old Monk
Coconut Oil from Spectrum Naturals
Organic flax seed from a local health food store, ground fresh daily

The patients were either found to have strong teres minor muscles or the muscles were treated with the usual 5 factors of IVF. The muscles were then tested against the oil products. If a weakness occurred in the teres minor(s), other muscles were tested to determine whether or not the response was just localized to the thyroid. A generalized weakness could mean an allergy, an oil-handling problem, a shift away from the appropriate total body fatty acid balance, etc., and was pursued differently from a weakness of just the teres minor.

The weak teres minor muscles were then tested via therapy localization to other organ reflex areas, etc., or insalivation of other nutrients to determine what would negate the weakness. Knowing our patient of course helps us to ask the best muscle testing "questions".

SUMMARY OF PROCEDURES

1. Test and fix the teres minor muscles so they are strong "in the clear".

2. Locate other strong indicator muscles.

3. Have patient insalivate flax oil and retest the teres minor. If it weakens, test other strong indicators to be sure it is just a thyroid effect.

4. Confirm the test and determine the sensitivity of the patient by subsequently testing the other oils and the fresh flax meal.

5. Look for ways to neutralize the weakness with activity of other organ reflexes or other nutrients.

SEVERAL CASES

A 47 year old man with a family history of heart problems and resistant high cholesterol and triglycerides, came in for treatment for back and extremity problems. During the course of treatment, I tested him for flax oil, which weakened his teres minor dramatically; this weakness was neutralized by iodine. His energy is improved and he is now sleeping better.

A 47 year old man with anxiety, difficulty sleeping, fatigue and other systemic complaints weakened with flax oil. Only substances to support the liver neutralized the weakness; iodine weakened him dramatically. He is also sleeping better.

A 49 year old woman, with a family history of hypothyroidism, a consistently low body temperature, high blood fats and severe depression weakened on flax oil and flax seed meal; her weakness was neutralized by a tyrosine product only. Her depression is lifting.

DISCUSSION

Often patients have symptoms which suggest the need to increase the PUFA content of their diets, and we must do what we can to help their bodies handle what they need. These patients present to us with a history of eating bad fats and have varied conditions related to disturbance in fatty acid availability to the tissues, prostanoid imbalances, disturbed calcium distribution, etc.

Sometimes patients with hypothyroid symptoms respond well to PUFAs. In pure iodine deficiencies, Goodheart[10], Peshek[11], and Lee[12] have advised patients to increase PUFAs in their diets. This is due to the fact that the unsaturated fats help to carry the iodine to the tissues. The polyunsaturated fats have iodine numbers which rates them according to their ability to carry iodine.[13]

But when the PUFAs weaken the teres minor, we can use that weakness to determine which other factors are needed by the patient. My experience has been that it is

rarely iodine. Dr. Peat also says that iodine deficiencies are not nearly as common as they once were.[14] Rubel said, "It is necessary that protein and iodine both be present in order to have normal thyroid function."[15] Bland said that trace minerals, especially selenium, are necessary to convert T4 into T3.[16] There are many factors because of the extensive relationship of the thyroid hormones to other hormones and all body tissues. With A.K. principles we are able to diagnose and supply the needs of individual patients by asking individual questions.

CONCLUSIONS

By having patients insalivate unsaturated and saturated fatty acids and then testing the teres minor, we gain an additional tool for helping hypothyroid patients. There are many questions to be answered, but the procedure is so simple that we can apply the questions to our patients and let their bodies answer them.

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Zinc, Sodium, Manganese and Adrenal "Burn-out"

by
Kathleen M. Power, D.C.

ABSTRACT

Many patients test deficient in zinc with the Zinc Tally test, but for some it is unwise to supplement them until other factors involved in the "apparent" deficiency are examined. Sometimes the body has lowered the zinc for a purpose; for these patients, giving zinc may aggravate a metabolic disturbance. Zinc can lower the sodium level while increasing the potassium level via its synergism with potassium in some body functions. Patients whose adrenal glands are exhausted, or who are depleted in sodium for any reason, may not tolerate much zinc. Screen these patients by having them insalivate zinc and test adrenal related muscles to determine whether or not zinc taken alone is contraindicated. If a muscle weakens, see what will neutralize the weakness; often it is manganese.

INTRODUCTION

One of the most frequent findings on new patients in my office is a failure of the Zinc Tally taste test. I have usually taken the time to explain the importance of zinc to these patients and I have tested them for zinc supplementation using a convenient indicator muscle, or at least recommended an increase of zinc rich foods in the diet. I don't want to create therapeutic limitations by not addressing the deficiency of such a critical trace mineral.

I also do hair analysis on some patients as a screen for heavy metal toxicity and unusual metabolic patterns, using Analytical Research Laboratories (ARL), headed by Paul Eck, Ph.D. I have always placed ARL's findings in the context of a good A.K. workup. But some of ARL's conclusions on a hair analysis are difficult to translate into muscle testing "questions".

With the new tool of testing patients for ion exchange resin minerals[1], it has become much easier to test patients and correlate apparent deficiencies and excesses of tissue minerals with those we find on a hair analysis. I have found several problems in testing for the ions as described in Dr. Goodheart's Research Report, however. One is that not all of the minerals are available in the ionic form and we may still miss some mineral factors because of this. Also, I have not always been confident about my use of the leg turn-in test. My only option has been to look at other ways of

testing patients.

DISCUSSION

Since beginning to use the ion exchange resin minerals, I have gained greater respect for the metabolic synergism and antagonism between mineral "pairs". I have been reminded of some of Dr. Eck's work on restoring metabolic energy to the system via balancing the ratios of calcium, magnesium, potassium and sodium. Of all the ratios between these four macrominerals, he feels that the sodium to potassium (Na/K) ratio, when low, is the most significant ratio in the hair test and should guide the initial dietary and supplement management of patients.

Dr. Eck uses the term "inversion" for a low Na/K ratio, less than 2.5:1. Patients with inversions are in a state of adrenal "burn-out"; the lower the ratio the more severe the situation. We know that the adrenals fail to produce enough aldosterone to retain adequate levels of sodium in a burn-out situation and that is the basis for the Koenigsburg test. Sodium to potassium inversions are relatively common.

Many patients seem to be zinc deficient. But, over the years of research at ARL, Dr. Eck found that zinc has a tendency to lower the sodium level -- aggravating an inversion -- by depressing the aldosterone level. According to Dr. Eck, "zinc and sodium levels tend to be inversely related". The body will lower the zinc level to "protect" the sodium level. This is due possibly to the intra/extra cellular polarity between sodium and potassium and zinc's synergism with potassium and magnesium. At any rate, Dr. Eck says, "Attempting to correct the low zinc is not wise in this case, as it will lower the sodium making the sodium reading worse. A better solution is to leave the zinc alone, and use other nutrients to raise the sodium. When sodium rises, the zinc will rise on its own, as the adaptation of lower zinc will no longer be needed."[2]

I decided to ask the patients' bodies what they wanted. I tested their adrenal-axis muscles[3], and then asked them to insalivate sources of zinc, and retested the muscles. Many of them weakened in the adrenal muscle pattern. Sometimes the weakness was dramatic. Other muscles did not necessarily weaken. I then tested to see what, if anything, would neutralize the weakness. Often it was manganese; occasionally other factors were involved.

MANGANESE

Manganese is a trace mineral activator of many enzyme systems in the body. It plays a part in insulin synthesis and degradation. It is a limiting factor in the ability of muscles to sustain a contraction or to continue rapid movement, particularly under stress. The many serious effects of streptomycin toxicity in children are caused by manganese deficiency; these include tinnitus, hearing and visual impairment and many

other symptoms of central nervous system damage. We know that ligaments require a source of manganese, and we know about its role in the maternal behavior of mammals. Deficiencies of manganese mimic many of the symptoms of vitamin E deficiencies; manganese is synergistic to E in many actions. It is a key to the liver's production of arginase.

In the body it is stored in most tissues; within the cell it is located in the mitochondria.

The manganese content of plants is dependent upon the manganese content of the soil, and many artificial treatments to the soil render manganese less available for plant uptake.[4] Many refining and processing techniques remove it from our food. It is poorly assimilated and inactivated by ferric iron. Its need is increased by high phosphates in the diet.

Manganese frequently appears low on a hair analysis, (while zinc often appears in the normal range). Giving manganese, according to ARL, has the effect of raising sodium and serving an important role in the recovery from burn-out.

There is no test for manganese as simple as the Zinc Tally test for zinc. Therefore, we should take advantage of any opportunity to learn about the manganese status of our patients. Weakness created when zinc is insalivated is one such clue.

MATERIALS AND METHODS

The materials I used were:

Zinc Tally from Metagenics
Zinc picolinate from Doctors' Mutual Services
Resin-bound zinc and manganese from Miera
Manganese picolinate from Ecological Formulas
Manganese-B12 from Alta Health Products

SUMMARY OF PROCEDURES

1. Test the adrenal-axis muscles and fix if they are weak "in the clear".

2. Have patient insalivate a source of zinc and retest the muscles. If they weaken, see what reflex points or other nutrients neutralize the weakness.

TWO CASES

A diabetic patient had been taking resin-bound zinc hoping to prevent any acceleration of the disease process. Her sartorius weakened with Zinc Tally; the weakness was neutralized by manganese-B12. Since adding manganese to her protocol, she is

rapidly recovering from a difficult problem in both shoulder joints.

Another lady came in with a painful and chronic foot problem caused by poor shoes. She had been taking zinc, but it weakened her posterior tibialis dramatically and manganese neutralized the weakness. She is recovering well.

A FEW ADDITIONAL NOTES

Jerry Deutsch, D.C., did a paper a few years ago on the slow ligament stretch. He found that manganese was more often needed to support the adrenals than was a source of adrenal tissue in patients in whom he found a positive slow ligament stretch response[5]. A zinc-induced weakness has frequently been found in this type of patient, as well, usually neutralized by manganese.

In his book on the thymus and manganese, Dr. Emanuel Josephson discussed the effect that manganese (as well as glycine and vitamin E) has on sustained muscular contraction. I have had some patients who weakened with a sustained, doctor-initiated, muscle test. Manganese has been helpful with these patients, although I have not tested many this way. It seems to show up more in extensor muscle groups, because they fatigue more easily when manganese deficient, according to Dr. Josephson.[6]

CONCLUSIONS

Mineral balancing in the body is complicated. With Applied Kinesiology we can ask the body only one or a few questions at a time. We have to ask enough questions to be sure we do not throw the body off balance. It is important to remind ourselves that raising one mineral lowers others, stimulating one organ may depress another, and what is low in the body may be low out of physiologic need.

It will be necessary, as a follow through on this mineral testing, to measure adrenal function in ways that give us "hard data": postural blood pressure, endocardiography, etc. I know that Dr. Eck's principles work by observing symptomatic improvements as well as mineral ratio changes on subsequent hair analyses, but it is clinically more efficient to use a muscle test instead.

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A Review of Arginine Function.

James A. Tucker, DC

ABSTRACT

Arginine is a non-essential or "contingent" amino acid. During the growth period it is essential, but In adults it can be synthesized from citrulline in a reaction involving aspartic and glutamic acids. Arginine is important in numerous biological systems. The correct function of it and its enzyme systems is of great importance. Applied kinesiologic evaluation and functional assessment of this amino acid in patients with cardiovascular, immune, and neurological symptoms may lead to new applications of conservative physiologic therapeutics.

INTRODUCTION

There continues to be a large volume of information that is being presented and incorporated into applied kinesiology practices. The advent of provocative neurological testing of biochemical pathways by Schmitt, Goodheart, Lebowitz, etc. has caused an explosion of possible chemical and nutritional challenges. One way to aid in the screening of the patients who are candidates to undergo specific pathway examinations is to understand the pathway and its function. Another aid is to see what researchers have found in relation to the human pathophysiology of that chemical or nutritional component. We can then apply what we know about testing the substances and relate them to the pathophysiology caused by the lack or excess of the substance. In essence, making a bridge between the research and the clinician. Knowing what pathology, signs or symptoms may relate to a specific substance is a basis for applied kinesiology functional assessments. This paper represents a review of the recent research into the amino acid arginine.

DISCUSSION

Arginine is a non-essential or "contingent" amino acid. During the growth period it is essential, but can be synthesized from citrulline in a reaction involving aspartic and glutamic acids. Arginine has potent secretagogue activity, stimulating pituitary release of growth hormone and prolactin.[2] Arginine is glycogenic inducing a marked release of insulin, being the most insulinogenic amino acid.[3] Supplemental arginine has been shown to improve weight gain, nitrogen retention and wound healing.[4]

In L-arginine-supplemented enteral diets, postoperative lymphocyte responses to mitogens were better than those seen with unsupplemented diets in cancer patients undergoing a major operation.[6] Other researchers have concluded that postoperative enteral nutrition with supplemental arginine,

RNA and Omega-3 fatty acids, instead of the standard enteral diet, significantly improved immunologic, metabolic, and clinical outcomes in patients with upper GI malignancies who were undergoing major elective surgery.[8] It is hypothesized that the omega-3 fatty acids would decrease macrophage prostaglandin E2 and cytokine release and stimulate T-cell proliferative responses, and that arginine and RNA would directly stimulate T-cell proliferative responses.[10] It was found that the mean length of hospital stay as well as the percent of patient's who had infections or complications was significantly reduced in those given the above captioned diet.[9]

Other functions of arginine include aiding in liver detoxification, an increase in sperm count, aiding in kidney disorders and promotes wound healing.[23] Arginine and lysine are in competition with each other. Diets high in lysine could create imbalances which would be harmful.[22] This amino acid also has been linked to increasing muscle mass and a reduction of body fat when supplemented.[23]

Dr. Goodheart has outlined some of the various uses of L-arginine as the essential precursor for the formation of nitric oxide. In Dr. Goodheart's research tape #123, he introduced information on nitric oxide and its uses by the body as a messenger molecule. This messenger molecule was found to have a wide range of activities. Nitric oxide is a gas enzymatically synthesized from the amino acid L-arginine. It is a biological mediator in the cardiovascular system. NO serves as a neruotransmitter in the brain and peripheral nervous system. It mediates non-adrenergic and no-cholinergic neurotransmission in the peripheral nervous system. NO plays a role in the pathophysiologic responses in peripheral organs.[1] According to Dr. Goodheart it "mediates blood pressure, helps immune system to kill, stops cancer cells from dividing, can cause large scale death of brain cells that can debilitate people with stroke or Huntington's even though the cells of the brain that make it are immune to it".[13] Goodheart has described several AK procedures to identify the need to increase the metabolic synthesis of NO.[12]

In relation to L-arginine's role in NO synthesis the original research by Furchgott and Zawadzki showed the obligatory role of enothelial cells in the relaxation of arterial smooth muscle by acetylcholine.[13] The relaxing substance produced by the endothelial cells was called endothelium-derived relaxing factor (EDRF). In subsequent research it was found that acetylcholine was not the only substance to produce endothelium-dependent relaxation. Virtually all the major endogenous vasoactive neurotransmitter and immune response stimulatory molecules were shown to produce the vascular relaxation through the EDRF mechanism.[14]

The actual characterization of this molecule as NO was performed by Moncada, et al. They found that NO had a very short half life of only a few seconds. They also found that

the breakdown of NO could be inhibited by SOD and Cu++, and thus lengthen it's half-life. In 1988 they reported that vascular endothielial cells synthesize NO from L-arginine.[15] Of interest was the implication that the superoxide ion contributed significantly to the instability of NO.[16]

Hibbs et al. showed that NO was an activated-macrophage-effector molecule. Hibbs has shown that the activated-macrophage-effector mechanism required L-arginine for its expression as a selective metabolic inhibitor in tumor target cells.[17] Other researchers have found that Arginine supplementation inhibits the chemical induction of tumors.[5]

Stuehr et al. explored additional metabolic pathways for the synthesis of NO in macrophages. They identified NADPH, BH4, FAD and GSH as additional co-factors for full NO synthase activity. The requirement for NADPH was second only to the primary substrate, arginine.[18]

In one study,L-arginine values were significantly lower in hypercholesterolemic patients than in age-matched patients with normal cholesterol. This data suggests that impairment of endothelium dependent relaxation factor in patients with hypercholesterolemia may be in part due to a deficiency of L-arginine, the precursor of nitric oxide.[27] I myself hypothesize that it may have some relation to a primary requirement of NADPH, FAD, GSH, BH^4, calcium and magnesium as cofactors. In the light of this new research the question of the use of niacin (NADPH) in the treatment of cholesterol metabolism problems should be re-examined.

In another study of patient's with hypercholesterolemia, the vasoconstrictive effect of acetylcholine on epicardial segments after intracoronary infusion were similar to those in controls, but the increase in coronary blood flow with acetylcholine in hypercholesterolemic patients was significantly reduced. L-arginine restored the acetylcholine-induced increase in blood flow in patients with hypercholesterol, but did not affect coronary blood flow in controls. The authors concluded that hypercholesterolemia impairs endothelium-dependent dilation of the coronary microcirculation and this can be reversed by short administration of L-arginine, which might be a form of treatment for reversing coronary microcirculation dysfunction in hypercholesterolemic individuals.[28] It is of interest to note that the co-factors such as NAPDPH, and FAD are composed of the vitamins described as major constituients of vitamin "G". "G" has been used for years on the coronary-type patient to aid in the resoration of normal heart muscle circulation.

Garthwaite et al. suggested a role for NO as an intracellular messenger in the brain.[19] Dr. Goodheart has pointed out that the highest level of nitric oxide arginase is in the brain.[20]

Arginine is also of importance in the urea cycle. Ammonia (from protien metabolism) and CO^2 (from the Krebs cycle) will combine to form carbamic acid. Carbamic acid will enter into the urea cycle and thru several steps form arginine which will be split into ornitine and arginine and

the byproduct urea. Proper functioning of this cycle with the necessary vitamins and co-factors is important in the maintenance of arginine levels. Schmitt has shown a model for the Applied Kinesiology evaluation of the urea cycle function.[21] Schmitt has listed excess arginine due to primary arginase inactivity as one of those substances which is most likely to create the subtle toxic effects of switching.[25]

A study of amino acid profiles of patients who are HIV positive, with both ARC and AIDS, showed significantly elevated levels of serum arginine, irrespective of the stage of disease. The elevation of the arginine is said to be indicative of reduced urea cycle activity in the livers of AIDS patients.[26]

CONCLUSION

Arginine is important in numerous biological systems. The correct function of it and its enzyme systems is of great importance. Applied kinesiologic evaluation and functional assessment of this amino acid in patients with cardiovascular, immune, and neurological symptoms may lead to new applications of conservative physiologic theraputics.

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Elimination and/or rotation of foods with high arginine content for the pituitary body-type.

James A. Tucker, DC

ABSTRACT

The dietary requirements for pituitary-dominate individuals, based on body-typing, are high protein intake and avoidance of milk and simple carbohydrates. Protein is required because of the catabolic nature of the dominate gland. It would be wise as a course of therapy to try an elimination and/or a rotation diet of foods high in arginine on the P-types. The ingestion of these foods may overstimulate the pituitary and create recalcitrant and recidivistic symptoms and findings.

INTRODUCTION

Body-typing is based on the premise that glandular function and subsequent dominance determines development. In body-typing one evaluates the basic physiogomy of an individual and determine which gland (ie, pituitary, thyroid, adrenal or gonads) is dominant. Based on this glandular dominance you can recommend diets that will calm down the dominant gland while supporting those glands that may be weak. I refer to the excellent reference and seminar by John V. N. Bandy, DC, DIBAK for further information.

DISCUSSION

Based on recent research into the biochemistry of L-arginine one particular body-type diet, the pituitary, may require modification. The pituitary is basically catabolic in nature. People with this body-type tend to eat a lot. There blood sugar also tends to be unstable. Their dietary requirements, based on body-typing, are high protein intake and avoidance of milk and simple carbohydrates. Protein is required because of the catabolic nature of the dominate gland. Milk and simple carbohydrates tend to overstimulate the pituitary and weaker adrenal glands.

L-Arginine is an amino acid that has been found to be a pituitary secretagogue (ie, it stimulates the pituitary towards hyperfunction), stimulating pituitary release of growth hormone and prolactin.[1] As I have said early in this paper a goal in recommending a body-type diet is to "calm" down the dominate gland. A more effective P-type diet may limit those protein foods that have a higher percentage of arginine in them. The foods that have a higher percentage/mg of arginine are:[2]

peanuts and peanut butter
brazil and cashew nuts
beef
chicken, Turkey
sunflower seeds and meal
soybeans

common beans (navy, pea, white)
lentils, lima beans

A secondary consideration is that arginine also induces a marked release of insulin, being the most insulinogenic amino acid.[3] Thus, if a P-type does show a propensity towards blood sugar instability it would be wise to try an avoidance and/or a rotation diet limiting those foods high in arginine.

CONCLUSION

It would be wise as a course of therapy to try an elimination and/or a rotation diet of these foods on the P-types who exhibit blood sugar problems or persistent weight gain patterns. The ingestion of these foods may overstimulate the pituitary and create recalcitrant and recidivistic symptoms and findings.

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DIVISION II - CRITICAL REVIEW PAPERS

RATS IN SPACE!

The Neurology of Spinal Erection

by
Michael D. Allen, DC, NMD, DAAPM, DIBAK, DACAN, DABCN
Chiropractic Neurologist

ABSTRACT The entire experience of human existence is based upon joint mechanoreceptor stimulation as a result of joint movement, and joint movement is a result of muscle function. The ability to resist the Earth's gravitational field is as a consequence of postural muscles, through cerebellar reflex pathways. There is not one function of human existence which is independent of joint mechanoreceptor potentiation.

INTRODUCTION

During the first part of November, 1994, the Orange County Register ran a story entitled, "Space Rats Shed Light on Muscle Mechanics". This story takes place at about 250 miles above the earth in the Space Shuttle, Columbia.

Researchers at the University of California at Irvine, closely monitored five rats which spent two weeks in low earth orbit. The scientists wanted to understand more about the results of short duration space flight on endurance muscle function. The researchers say their findings might mean an end to the idea that we are made of just two types of muscle -- one for endurance activities and one for quick bursts of activity.

Once the rats were safely returned to earth, they were sacrificed and dissected. The researchers found that the rats lost 30 percent of their muscle during the flight.

The researchers found it interesting that the rats lost mostly slow-twitch (or type II) muscle -- that type of muscle used for endurance activity such as distance running -- and relatively little fast-twitch (type I) muscle -- that used for bursts of activity such as sprinting.

Further, they were also surprised to find that some, but not all of the slow-twitch muscle began to transform into fast-twitch muscle during the flight. One of the researchers was quoted as saying, "We need to explore further why some can change and the others cannot". Characterizing how muscle loss occurs is the first step toward understanding how to treat it, they said. The understanding of this type of change can benefit not only astronauts, but also the infirm and elderly, who often suffer tremendous muscle loss.

One of the researchers was quoted as saying, "We need to learn a lot more about how we can offset these changes, but first we need to know the types of changes that are occurring."

DISCUSSION

Muscles are distinguished histochemically into two basic types. One type corresponds to the fast-twitch (type I) fibers; the predominant metabolic enzymes present in these muscle fibers are those of anaerobic (glycolytic) metabolism. The other type corresponds to the red slow-twitch (type II) fibers, which rely primarily on oxidative metabolism.

Besides being defined as fast and slow twitch fibers, muscles can also be categorized by their ability to resist fatigue. Slow twitch muscles are aerobic in nature and possess a high degree of endurance character. They function to resist gravity; they are generally extensor or postural (shunt) muscles. They contain more mitochondria and therefore more mitochondrial electron acceptors (MEA's), more sarcoplasmic reticulum and a higher population of contractile proteins. Aerobic muscles also have a greater ability to produce ATP and replicate protein than that of anaerobic muscles.

As a consequence of a decreased level of joint mechanoreceptor stimulation, aerobic muscles adapt to become more like anaerobic muscles. They lose their mitochondrial electron acceptors, they contain less sarcoplasmic volume, and they have a diminished ability to aerobically produce ATP and replicate proteins at a high rate.

The ATP necessary for aerobic muscle function is generally produced via the Krebe citric acid cycle which is readily found in the mitochondria of type II muscles. When the essential components for the successful completion of the cycle are available, there are about 36 molecules of ATP produced. However, if this cycle cannot be completed because of the unavailability of the requisite factors, not only does the total number of ATP molecules wane, but also the ATP production must be shifted to anaerobic (the Emden-Myeroff) pathways leading to an overall decrease in the production of energy. Not only do the anaerobic pathways yield fewer molecules of ATP, but they also lead to an increased production of lactic acid, which is a well known tissue irritant.

Under normal circumstances, during muscular exercise, the muscle blood vessels dilate and blood flow is increased so that the available oxygen supply is increased. Up to a point, the increase in oxygen consumption is proportionate to the energy expended, and all the energy needs are met by aerobic processes. However, when muscular exertion is very great, aerobic resynthesis of energy stores cannot keep pace with their utilization. Under these conditions, phosphocreatine is used to resynthesize

ATP. Phosphocreatine resynthesis is accomplished by using the energy released by the anaerobic breakdown of glucose to lactic acid. This use of the anaerobic pathway is self-limiting, because in spite of rapid diffusion of lactic acid into the blood stream, enough accumulates in the muscles to eventually exceed the capacity of the tissue buffers and produce an enzyme-inhibiting decline in pH. However, for short periods, the presence of an anaerobic pathway for glucose breakdown permits muscular exertion of a far greater magnitude than would be possible without it. Without this pathway, for example, walking or running at a slow jog would be possible but sprinting and all other forms of short-term, violent exertion would not.

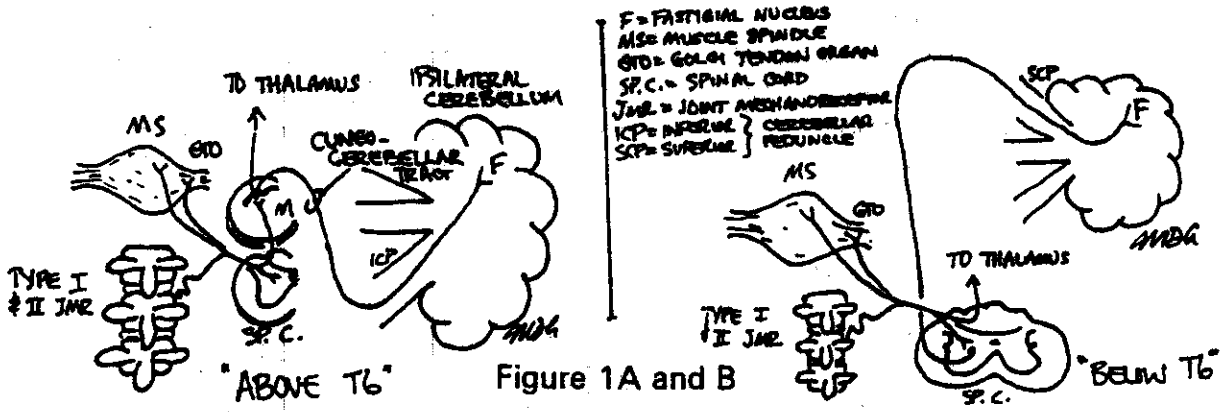
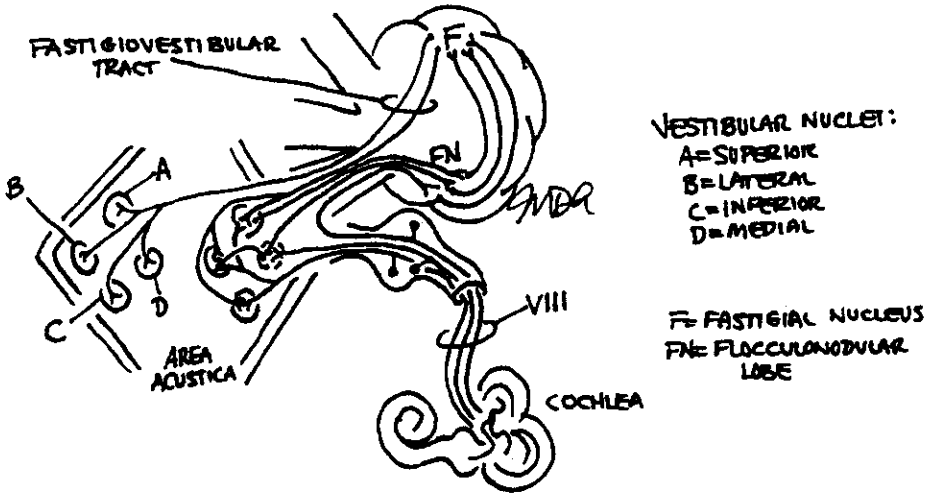


Figure 1A and B

The cerebellum innervates paraspinal muscles through subconscious means. It gets its stimulation almost completely as a result of afferentation from large diameter, type Ia afferent axons which arise from muscle spindles, and type I and II joint mechanoreceptors. These receptors are organized with a higher population and priority, oriented more rostrally than caudally. Therefore, these receptors are more sensitive to spinal, costosternal, costotransverse and costovertebral articular motion than other types of joint activity. Their axons rise to the level of the cerebellum through spinocerebellar and cuneocerebellar pathways, and terminate in midline cerebellar centers (see figures 1A and B). From here, efferent axons travel via the fastigiovestibular tract to all four vestibular nuclei in the area acustica in the floor of the fourth ventricle (see figure 2). These nuclei send fibers both rostrally and caudally to coordinate higher centers with joint motion (see figure 3). The caudal projecting fibers make up the vestibulospinal tracts, both medial and lateral. These nuclei are at their central integrative state as a result of cerebellar afferentation and cause paraspinal muscle stimulation and therefore spinal extension and upright posture. There is no way to consciously move one vertebra relative to another. Each of the paraspinal muscles is subconsciously innervated via the cerebellum and vestibular nuclei. The rostral projecting fibers arise from the superior, medial and inferior vestibular nuclei, to make up the medial lemniscus and terminate in the oculomotor (III), trochlear (IV) and abducens (VI) nuclei to coordinate extraocular movements with spinal function.

The cerebellum drives the mesencephalic centers, particularly the red nucleus and reticular formation through the dentatorubroreticulothalamic tract, and from there to all cortical centers through the corona radiata (see figure 4). These centers together with those fibers from the thalamus drive the extrapyramidal cascade via the thalamohypothalamoreticulospinal pathway (see figure 5) which synapses in the brainstem to drive cranial nuclei III, VII, IX and X, pontine and medullary reticular formation through the fastigioreticular tract (see figure 6) as well as the medullary respiratory and many other centers. That is to say, that as a result of spinal and rib mechanics, the receptor potential from the articulations causes stimulation of higher centers which return motor response to all the areas they innervate, and cause a reaction based upon that receptor input. So, it is reasonable to say that the process of respiration is dependent upon mechanoreceptor afferentation. Without mechanoreceptor afferentation, respiratory centers would be dysfunctional; and that is exactly what happens!



THE VESTIBULAR SYSTEM

Figure 2

Further, the thalamohypothalamoreticulospinal tract functions to inhibit the anterior muscles above T6 and the posterior muscles below T6. This provides a modulation of the strong facilitory effect to these same muscles produced by the corticospinal tract.

Generally, a muscle will stay as it is unless its metabolic needs change due to a change in demand, i.e., undergoes modified G-forces, joint subluxation, disuse or overuse, overtraining, suffers other degenerative changes, or the essential components for repair are no longer available, etc. Moreover, a muscle will only produce contractile proteins relative to their need. In a zero-G environment, there is

a decreased need for any type of muscle function, especially for those of the antigravity type. Therefore, any change in demand which modifies the spinal extensor muscle's ability to function normally causes the juxtaposed muscle(s) to undergo atrophic changes. The muscle adapts from the type II muscle and takes on an appearance much the same as that of a type I muscle. That is, they are less able to sustain endurance activity and more willing to perform short burst (spurt) endeavors; they change from an aerobic to an anaerobic type. This is accompanied by an inability to function as originally designed, which leads to:

Neuropathophysiology -- global deafferentation; immune and inflammatory responses;
Myopathology -- muscle atrophy;
Histopathology -- connective tissue and vascular pathology;
Kinesiopathology -- pathological changes in muscle function;
Biochemopathology -- deficiency of the essential components for repair;
Bioelectropathology -- inability to propagate an adequate neurophysiological impulse.

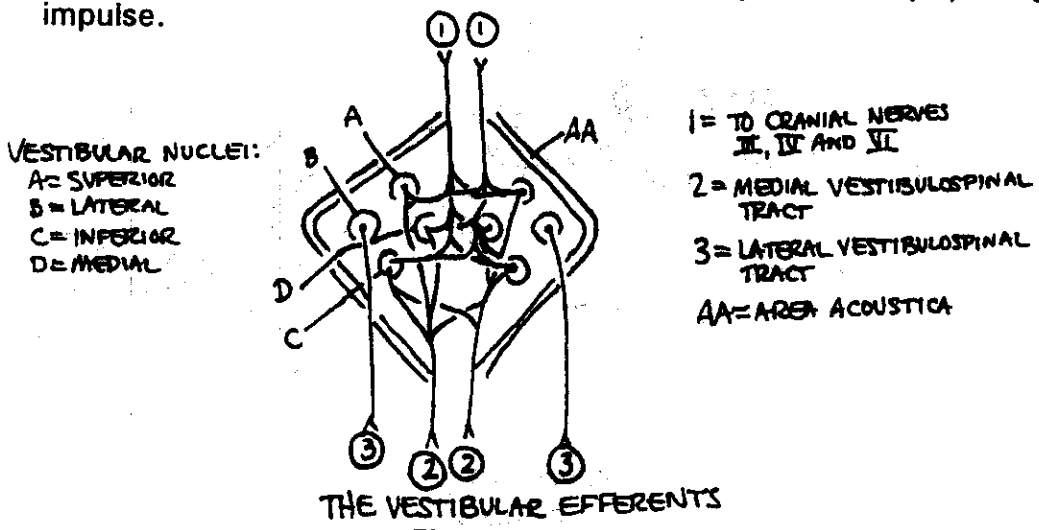


Figure 3

When talking about fast and slow twitch muscles, it is important to also discuss shunt and spurt characteristics of those muscles. Each has a particular function with regards to joint stabilization. Shunt muscles are those muscles that provide joint stability. By definition, a shunt muscle is one whose origin is closer to the joint than its insertion, giving it a greater ability to stabilize a joint (see figure 7). A spurt muscle, on the other hand, is a joint mover. Its origin is further away from the joint than its insertion; it acts as a lever. It is possible for a muscle to act as a shunt muscle in one activity and as a spurt muscle in another type of activity. An example of this is the stability produced between the spine and scapula in order for the first 90 degrees of upper extremity abduction to occur. In this instance, the spine and scapula become stabilized by shunt muscles and provide the stability necessary for

spurt muscles to produce the activity of upper extremity motion. In the second 90 degrees of upper extremity abduction, the spinal shunt muscles act individually in order to provide stabilization of the vertebrae while those muscles which previously provided scapular shunt stability now function differently to provide the spurt activity of scapular and upper extremity movement.

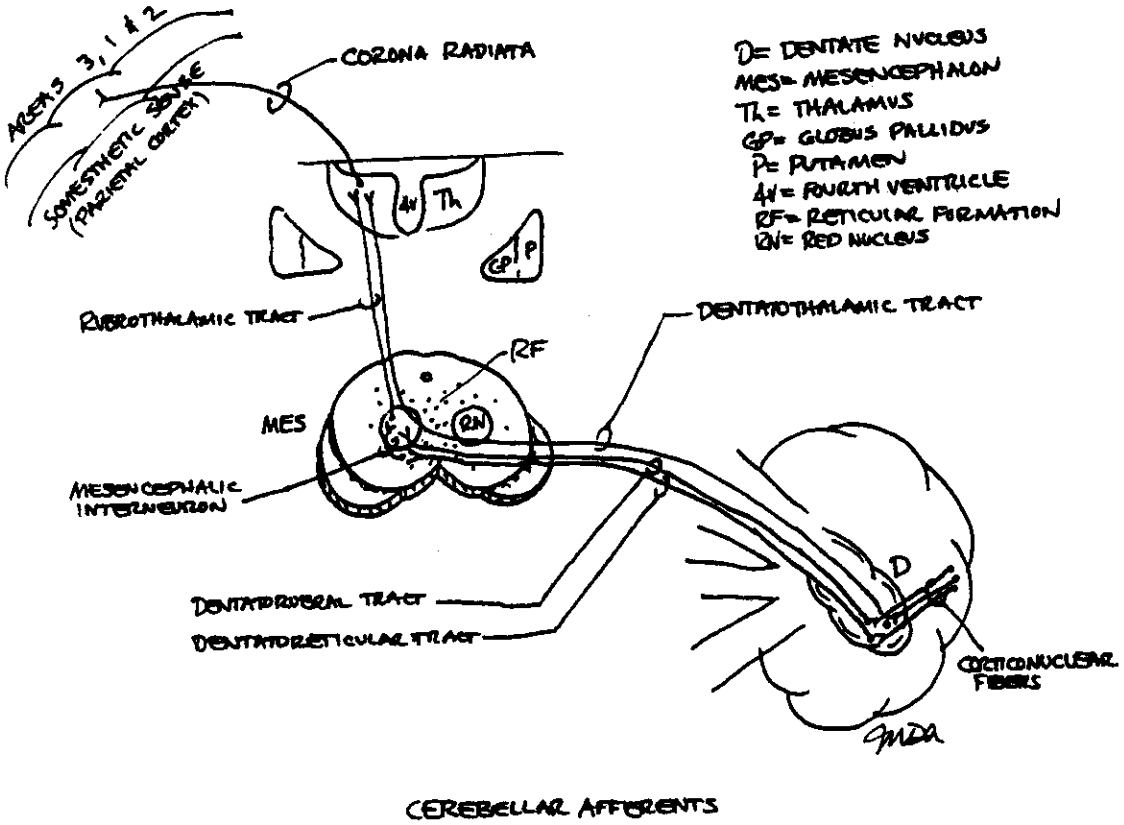
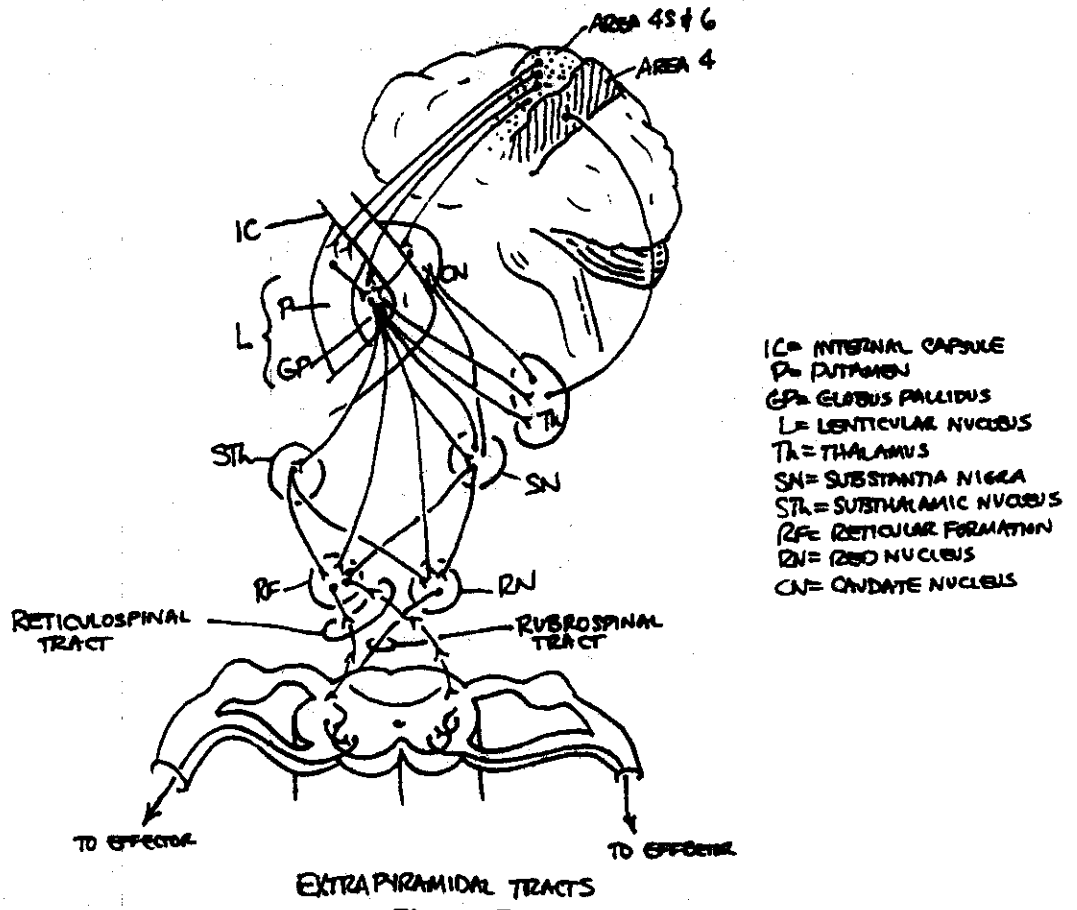


Figure 4

As muscles atrophy, shunt muscles lose their aerobic ability to stabilize a joint; they lose their capacity to endure. When spurt activity is superimposed upon an unstable joint, it causes the breakdown of the polyanionic glycosaminoglycans of joint cartilage, leading to degenerative joint disease and further deafferentation of that joint. Further, the endurance activity wanes in favor of a short burst modification, resulting in a change from aerobic to anaerobic metabolism. If this breakdown happens in the vertebral zygapophyseal joints, the cerebellum and dentatorubroreticulothalamic tract become deafferentated as well as higher centers of the mesencephalon and thalamus, and therefore the thalamohypothalamoreticulospinal tract, leading to a decrease in the central integrative state of cranial nerves III, IV, VI, VII, IX and X, and oxygen utilization, as well as bringing about a change in pH to a more acidic level concomitant with a drive toward potassium equilibrium potential in these areas.

Further, since the thalamohypothalamoreticulospinal tract acts to inhibit the ipsilateral anterior muscles above T6 and the ipsilateral posterior below T6, the hyperpolarization of this inhibitory tract as a result of deafferentation of the afferents ultimately causes a facilitation of that function, and the extremities do that which they are normally inhibited from doing. That is, the ipsilateral upper extremity flexors and lower extremity extensors become facilitated, creating the neurologic equivalent of a stroke antalgia.



EXTRAPYRAMIDAL TRACTS

Figure 5

An atrophied slow twitch muscle fiber can take on the characteristics of a fast twitch fiber. When a muscle loses its optimal length which happens as a result of a zero-G environment or a subluxation, the type Ia afferents are shortened, and the muscle's ability to perform work decreases. In a zero-G environment, postural muscles are unnecessary, leading to a decreased need for ATP. Actually, ATP is not needed for muscles to contract; it is only needed to release a contraction. To change atrophied slow twitch muscle fibers to normal slow twitch fibers, you must increase mitochondrial substrate and sarcoplasmic volume. In order for a muscle to replicate, it requires tetany in that muscle, which leads to increased protein replication.

Rehabilitation of spinal joints requires cerebellar depolarization through stimulation of Ib afferents via fast stretch coupled manual manipulation to the structures demonstrating a resistance to motion, and resultant stimulation to the types I and II joint mechanoreceptors. This stimulates cerebellar function and a drive toward sodium equilibrium potential and a resultant increase in the central integrative state of the vestibular nuclei, with consequential increase in supraspinal control over paraspinal musculature. The more stimulation these muscles receive, the more work they can do. The more muscle strength created, the more tension can be produced, and the more binding sights can be developed. This leads to an increased stability of joint function and resultant enhancement of the central integration of this afferentation.

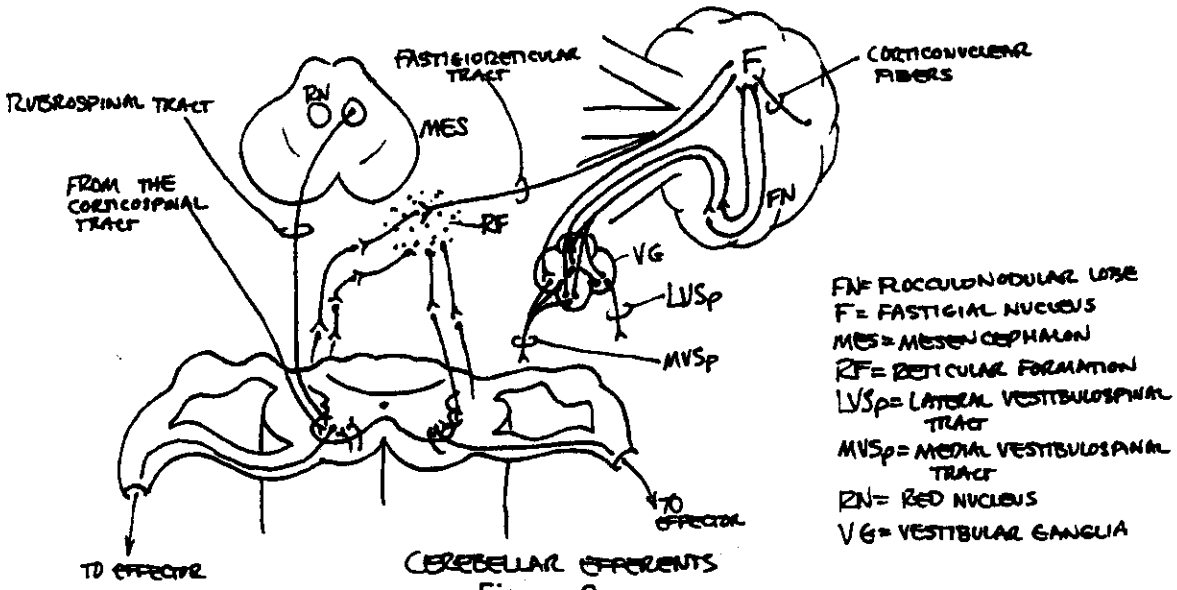


Figure 6

If a vertebrae is truly misaligned or subluxated, so that the amplitude of receptor potentials in that joint is decreased as a component of less mechanical stress, then we really are existing in a "gravitational storm" as far as the detrimental effects of gravitational loading on the joint are concerned. This "Rats in Space" model is appropriate and only reinforces our dependence on Earth and our ability to resist its call via the utilization of central integration of afferentation as a consequence of the gravitational field; i.e., all other things being intact, the ability to stand erect is directly proportional to the amount of joint mechanoreceptor stimulation.

With the foregoing in mind, consider that it only takes six to ten days for a muscle to lose half of its ability to work. In the next six to ten days, half of what is left is lost and so forth until after 30 days, up to 90% of the muscle's function can be lost. This relates equally to zero gravity and a spinal subluxation. The longer they exist, the

more problems they produce and the longer ut takes to fix them, if at all. The rehabilitation of an atrophic muscle is possible if no joint degenerative changes have occurred. If they have begun, full rehabilitation is less likely due to the resultant deafferentation of the joint.

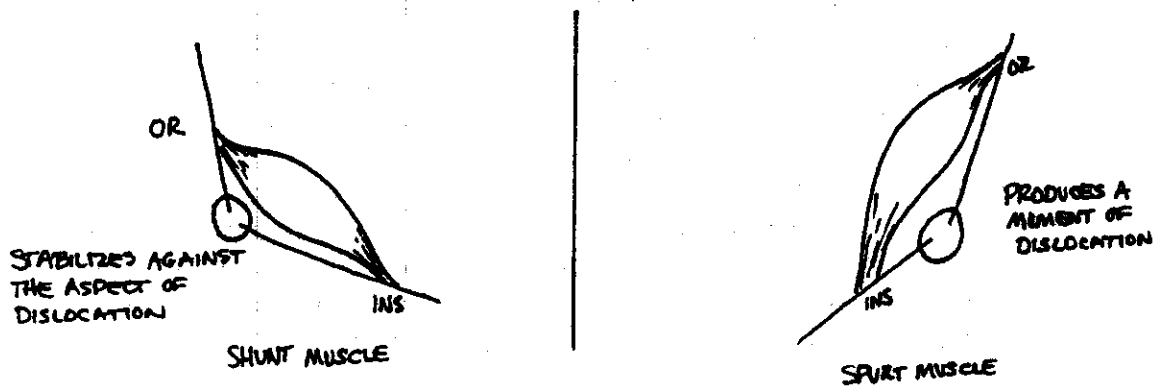


Figure 7

The "Space Rats" showed us a 30% adaptation in endurance muscle function after just two weeks. In humans, we know this number to be approximately 50% in six to ten days. The difference is that rats are quadrupeds, not bipeds. Their cerebellar functions are less developed than that of humans.

The last sentence of the last paragraph of the newspaper article reads, "We need to learn a lot more about how we can offset these changes, but first we need to know the types of changes that are occurring". It is sad that with all the subluxations out there that we have to send billion dollar mice up into space to demonstrate what the general chiropractor has realized in the office for almost a hundred years.

CONCLUSION

In summary, the first step of spinal rehabilitation is to reestablish joint position sense in the shunt muscles by working axially and moving peripherally. Once joint stability has been established, then direct attention to the spurt muscles. It is imperative to reestablish the metabolic function of the shunt muscles which have adapted to become spurt-like. They must regain the shunt function they once enjoyed; i.e., the ability to use oxygen, increase the number of mitochondria and mitochondrial electron acceptors, expand the ability to replicate proteins at a high rate, and to boost the sarcoplasmic reticulum.

The researchers are looking at the zero-G environment of low earth orbit and asking the question, "Why did we see type II muscle fibers change to look like type I fibers, but the type I fibers did not change to look like type II fibers?". There are so many

variables. It is not simply the result of zero-G. There are many neurological components that must be considered. Without putting them into the equation, this question is one of futility.

Chiropractic has innately known the importance of proper spinal function for nearly a century. Our philosophies have been cramped not as a consequence of its inadequacies, but as a consequence of the inadequacies of science and technology heretofore. The medical researcher looks at this research and asks, "Why?". The chiropractic neurologist looks at the exact same research and says, "Because!". Chiropractic is not medicine and cannot be compared to it. The comparison serves to make us sound like we want to be just like them. To compare unequal modalities is an effort in futility. The basic medical model of comparing two vegetables and drawing conclusions is not appropriate for the interpolation of the wonders of specific modulation and the evocation of those things which are always changing.

ZINC TASTE TEST AND A.K. ORAL NUTRIENT TESTING

Katharine M. Conable, D.C.

ABSTRACT

A sequential sample of 76 different patients in a chiropractic office were tested for ability to taste a 0.1% solution of Zn sulfate heptahydrate. Subject grades from 1 (no taste) to 4 (a strong and unpleasant taste is noted immediately) at 10 and at 30 seconds were compared to changes in manual muscle tests with oral insalivation of zinc. Muscles tested were pectoralis major sternal division and quadriceps femoris. There was no significant difference in ZTT scores between those who responded with change in muscle strength to insalivating zinc and those who did not. There was no significant difference in muscle response to zinc between those with grades 1 or 2 on ZTT - "zinc deficient"- versus those with grades 3 or 4 - "zinc sufficient". There was a significant difference in ZTT scores at 10 seconds and at 30 seconds, demonstrating that before the ZTT can be relied upon or studies compared, the test protocol must be standardized.

INTRODUCTION

In 1970 Goodheart[1] reported that in some instances muscles found weak or neurologically inhibited on manual muscle testing responded with immediate strength upon tasting specific nutrients. This observation has been found clinically useful by many applied kinesiologists as part of the evaluation of the nutritional needs of patients. Muscle response to tasting nutrients has never been recommended by the International College of Applied Kinesiology (I.C.A.K.) as the sole diagnostic criterion for giving nutritional supplementation. The 1992 Applied Kinesiology Status Statement of the International College of Applied Kinesiology - USA states in part,

> "During a functional neurologic evaluation, muscle tests are used to monitor the physiologic response to a physical, chemical, or mental stimulus. The observed response is correlated with clinical history and physical exam findings and, as indicated, with laboratory tests and any other appropriate standard diagnostic methods. Applied kinesiology procedures are not intended to be used as a single method of diagnosis. Applied kinesiology examination should enhance standard diagnosis, not replace it" [2]

A very extensive review of the studies and theoretical background for oral nutrient testing was included in Walther's *Applied Kinesiology Synopsis* .[3]

Various attempts of differing scientific quality have been made to demonstrate or refute the significance of change in muscle strength on tasting a nutrient. Triano's

study of A.K. oral nutrient testing started with several weak muscles and administered various nutrients before a retest of the muscle. He reported random results. There was no attempt to evaluate whether the patient needed the nutrient in question.[4] Kenny, Clemens, and Forsythe's[5] study did attempt to address this issue by doing lab testing, but the oral nutrient muscle testing protocols used did not represent usual A.K. procedures. Standard applied kinesiology oral nutrient testing is done using specific muscle tests and oral insalivation of a nutrient for which there is a clinical indication according to history, symptoms, laboratory work, and physical findings.

This study is an attempt to compare oral nutrient testing for zinc with a measure of functional zinc deficiency which has been used by various investigators and clinicians.

ZINC TASTE TEST (ZTT):

Russell et al.[6] described the evidence to date on zinc deficiency and hypogeusia. There are definitely zinc-responsive hypogeusias, especially in conditions known to cause zinc deficiency. There are also idiopathic hypogeusias which are not zinc-responsive. Russell mentions a zinc metalloprotein isolated from saliva which was termed "gustin", although its role in taste had not yet been defined at the time of his writing.

In 1984 in two letters to *The Lancet*, Bryce-Smith and Simpson[7,8] described a test for predicting whether an anorexic patient would be likely to respond to zinc supplementation, based on the decreased taste acuity which accompanies early zinc deficiency. They used a solution of zinc sulfate, which has a rather strong taste to most people. The patient that they described could not taste the solution at all at first. Following zinc supplementation for four months she was able to taste the solution. This return of her taste sensation was accompanied by marked improvement in her anorexia nervosa, demeanor, and body weight. Bryce-Smith and Simpson specified a test solution of one gram of zinc sulfate heptahydrate in 1 liter of distilled water. The subject tastes 5-10 ml. of solution. They described a 4 point scale for grading the zinc taste test:

> "(1) No specific taste or other sensation is noticed, even after the solution has been kept in the mouth for about ten seconds. (Some people even find a solution of twice the above strength to be tasteless.)
> (2) No immediate taste is noted, but after a few seconds a slight taste variously described as "dry", "mineral", "furry", or (more rarely) "sweet" develops.
> (3) A definite though not strongly unpleasant taste is noted almost immediately, and tends to intensify with time.
> (4) A strong and unpleasant taste is noted immediately. The subject normally grimaces."[13]

Bryce-Smith[9] postulates that a zinc-dependent salivary protein, such as "gustin" mentioned by Russell, may mediate the hypogeusia of zinc deficiency. He suggests that the intensification of taste while holding a zinc solution in the mouth seen in some patients (see Grade 2) may represent an activation of such a metalloprotein.

Since publication of these articles, ZTT solutions have been marketed commercially in the U.S. and Britain, and the ZTT has been used by many nutritional practitioners. It is attractive as a simple, convenient, and cost-effective method of in-office monitoring of zinc status.

Two studies by A.G. Schauss using the ZTT were reported in a chapter in *Nutrients and Brain Function* in 1987[10]. The test solution in these studies was Metagenics Zinc Tally which is considerably stronger than that originally described by Bryce-Smith. This solution contains 1.35 g. of $Zn\ SO_4.7H_20$ per liter of water. Bryce-Smith's grading scale was used.

In the first study, 9 known anorexics were tested double blind with ZTT solution and distilled water as a placebo.

A second study was done on 158 healthy, non-eating-disordered subjects. The test was double-blind with distilled water as placebo. These subjects were compared to the known anorexics and the differences were considered statistically significant.

In a letter to *Chemistry in Britain*, Dec. 1989 Delves et al.[11] criticized the ZTT as not correlating well with plasma zinc. Their data are not included in their letter. However, Howard[12] describes a correlational study of serum, leucocyte, sweat, and hair zinc levels with results demonstrating that sweat zinc is the sample of choice, and that leucocyte zinc is nearly as useful. Serum/plasma zinc is considered unreliable except in severe deficiencies.[13,14]

Eaton et al.[15] compared the zinc taste test with sweat zinc analysis in 1990. Subjects were 21 patients with known food intolerance recruited from allergy clinics. These subjects were thought likely to have borderline zinc deficiency. Daily ZTT scores were averaged over a period of one month. The concentration of the test solution was as described by Bryce-Smith. Eaton used a modified grading system of O - 4 in which Grades 1 and 2 overlap with Bryce-Smith's Grade 2, and Grade 3 is not defined as being a "definite" vs. "slight" taste as it is in Bryce-Smith's system, but rather an immediate rather than delayed taste. Some patients were given zinc supplementation midway and the changed ZTT and sweat scores were recorded. Eaton found a moderate correlation between the ZTT and the sweat analysis, and concluded that the ZTT was somewhat less accurate but clinically useful. He felt that the broad scatter of results on the ZTT might be due to patient difficulty in grading the test. He also suggests that Grade 4 may be the normal level, and that Grade 3s should be verified with sweat mineral analysis before supplementation. This is the

only study to date to use an averaged ZTT grade, which may be a useful approach in the future.

MATERIALS AND METHODS

The present study compared patients' grades on the ZTT to changes in manual muscle tests with oral insalivation of zinc.

Subjects were a sequential sample of 76 different patients at the author's chiropractic office in St. Louis, MO. The subjects were tested over a period of 8 consecutive working days in July of 1991. Patients who were under 10 years old or in severe or acute pain were excluded, due to difficulty obtaining accurate muscle tests. Patients in the office for their first visit were excluded for reasons of patient flow and consideration for the needs of the new patient in a new situation. No patient was tested twice. Ages ranged from 15 to 84. There were 62 females and 14 males.

The examiner, who was the patient's regular doctor, recorded the patient's age, sex, and the date on a numbered data sheet. Informed consent was obtained verbally regarding the subject's willingness to undergo extra muscle testing and taste a solution. If a patient asked, he or she was told that the solution was a nutrient, but not its specific content. No patients objected. The wording for informed consent and patient instructions during the study was written down and used consistently with each patient.

The examiner then tested the pectoralis major, sternal division and rectus femoris muscles in the manner described by Walther[16]. The muscles were tested bilaterally during held inspiration and held expiration. The patient was positioned, instructed as to respiration, and and then instructed to push against the examiner's hand in the direction indicated. This was a "patient initiated" test, resulting in a slight initial concentric contraction against resistance. Each test was graded as "strong" or "weak". A "strong" grade corresponds to Kendall and Kendall's "normal" and "good" grades in which the subject is able to maintain the limb in the test position against the examiner's moderate to maximal test pressure. "Weak" corresponds to grades "Good minus" and lower in Kendall and Kendall's system, where the subject cannot maintain the test position against more than minimal test pressure, cannot move the extremity into the test position against gravity, or worse.[17] This style of muscle testing, with emphasis on the muscle's ability or inability to maintain a contraction against the examiner's gradually increasing test pressure is described in detail in Walther[18]

Results were recorded on the data sheet. The order of testing the four muscles was pre-randomized. Whether inspiration or expiration was tested first was randomized.

The examiner then placed one 25 mg. tablet of zinc as picolinate on the subject's

tongue. While this was being tasted, the examiner retested all of the muscles previously tested, in the same order. Results were recorded on the data sheet and the doctor left the room.

An assistant came into the room and administered a zinc taste test by a standardized protocol. The solution was Nutridyn Zinc Status which is a 0.1% solution of ZnSO4.7H2O in water, as described by Bryce-Smith. The assistant rated the patient's description of the taste of the solution at 10 seconds and at 30 seconds using the 1-4 scale described by Bryce-Smith and Simpson[6,7,8]. The patient was not told the nature of the test substance or the significance of its taste. Results were recorded on the data sheet, which was then slid under the unused sheets on the clipboard.

Following this, the doctor returned and proceeded with the patient's normal office visit.

RESULTS

Zinc Taste Test scores were:

GRADE	10 SEC.	30 SEC.
1	34 subjects	26 subjects
2	39	38
3	3	11
4	0	1

There is a statistically significant difference in the distribution of "zinc deficient" and "zinc normal" results when the ZTT is graded at 10 seconds versus 30 sec. Chi square = 5.99, p = 0.014. Hence, the protocol for grading the ZTT is an important source of variability in studies using it.

31 subjects had at least one muscle graded "weak." Of these 22 had at least one muscle which became "strong" on tasting the zinc supplement. One additional subject showed a change from strong to weak. In 20 of the subjects responding to zinc, all weak muscles in that subject responded and in 2 only some responded.

Of the 73 patients who classed as "zinc deficient" on the ZTT (Grades 1 and 2) at 10 seconds, there were 51 who did not respond to oral zinc and 22 who did respond. There were 3 "zinc sufficient" subjects - ZTT Grades 3 & 4 at 10 sec. Of these, there were 2 no responses and 1 response. Chi square = .014, p = 0.906 demonstrating no significant difference between the zinc sufficient group and the zinc deficient group.

At 30 seconds grading, the difference between groups was closer to but did not reach significance. Sixty four subjects classed as "zinc deficient", of whom there were 48 who did not respond and 16 who did respond to zinc. Twelve subjects were "zinc sufficient" with 11 no responses and 1 response. Chi square = 1.616, p=0.203

When only subjects who initially had at least one weak muscle are considered, there is still no significant difference between the two groups. Of the 31 subjects who had at least one muscle weak on the initial test, the results were as follows:

10 sec: Zinc deficient (grade 1 & 2) Zn responders 20 (69%)
Zinc non-responders 9 (31%)
Zinc sufficient (grade 3 &4) Zn responders 2 (100%)
Zinc non-responders 0
Chi square = 0.875, p = 0.3497

30 sec: Zinc deficient (grade 1 & 2) Zn responders 20 (74%)
Zinc non-responders 7 (26%)
Zinc sufficient (grade 3 &4) Zn responders 2 (50%)
Zinc non-responders 2 (50%)
Chi square = 0.98, p = 0.3222

Looking at the Zn responders vs. the Zn non-responders, of those who had at least one muscle weak initially:

10 sec: Zn responders - 20 grade 1 or 2 (90.9%)
2 grade 3 (9.1%), 0 grade 4
Zn non-responders - 9 grade 1 or 2 (100%)
0 grade 3 or 4
Chi square = 0.73, p = 0.46

30 sec: Zinc responders - 20 grade 1 or 2 (90.9%);
1 grade 3, 1 grade 4 (9.1%)
Zn non-responders - 7 grade 1 or 2 (77.7%);
1 grade 3, 1 grade 4 (22.2%)
Chi square = 0.69, p = 0.35

DISCUSSION

Zinc Taste Test: From the literature, although the ZTT is promising as an inexpensive and convenient method of monitoring response to supplementation in patients with hypogeusia due to zinc deficiency, a single positive ZTT is clearly not diagnostic of zinc deficiency in and of itself. Other clinical evidence of zinc deficiency must be taken into account before supplementation of any particular patient. Clinical indications that zinc supplementation may be appropriate are: poor growth in childhood, impaired immune function, low sperm count, poor wound healing, dietary deficiencies including anorexia nervosa, acne, acrodermatitis enteropathica, etc.[9] The resources available for this study precluded using leukocyte zinc, sweat zinc, or another laboratory standard. This might be interesting for a future study. In light of Russell's data[5] on non-zinc-responsive idiopathic

hypogeusias, we would expect that some patients with low ZTT scores will not respond to zinc supplementation with improved ability to taste. Even if zinc deficiency could be demonstrated to correlate perfectly with muscle response to tasting zinc, we would still expect that some subjects with idiopathic hypogeusia might show a low ZTT score and no muscle response. In other words, the specificity of the ZTT for zinc deficiency is not yet fully defined. The incidence of other types of hypogeusia will have to be taken into account to evaluate this.

According to Eaton[14], while the ZTT correlates moderately well with sweat mineral analysis, it is less sensitive. The ZTT may be most useful when other clinical indications of zinc deficiency are present. Muscle response to zinc may turn out to be one such indicator, but further study will be required to verify this.

The author investigated the literature on the ZTT and corresponded with Professor Bryce-Smith and several nutrition companies which make zinc sulfate solutions for the ZTT. Two very different concentrations are being marketed as the "standard" ZTT solution. Lamberts Dietary Products in England, Thorne, Nutri-West, and Nutridyn all market a solution which conforms to Professor Bryce-Smith's original recommendations in the 1984 Lancet letters - 0.1% Zn sulfate heptahydrate. That is, 1 g. of ZnSO4.7H20 in 1 liter of distilled or deionized water. Professor Bryce-Smith continues to recommend this concentration. Metagenics markets Zinc Tally, which is actually a 0.135% solution (1.35 g. per liter) and has a very much stronger taste. They maintained that this is Bryce-Smith's concentration, which by simple mathematics, it certainly is not. The author has attempted without success to find out the origins of this stronger solution. Various explanations have been offered, however no specific references in the literature were found. It is interesting to note that 2 of the papers published in the *ICAK Collected Papers* about ZTT used the stronger Metagenics solution, as does Dr. Goodheart.

In this study the solution used was the 0.1% solution per Bryce-Smith. It would be very interesting to redo the protocol with the stronger solution. A very small number of subjects scored grades 3 or 4 on the ZTT, which made it difficult to compare the groups statistically. It is possible that the stronger solution would yield more significant results. It would be enlightening to discover the origins of the stronger concentration.

Previous ICAK papers which compare ZTT and muscle testing responses, while each flawed for various reasons, do give an interesting view of the varied distribution of ZTT grades using different test solutions and times of grading. Zatkin[19] retrospectively studied 102 consecutive new patients in his chiropractic practice, reporting ZTT results using Metagenics Zinc Tally. The test was graded at 30 seconds. He found 16 Grade 1, 32 Grade 2, 31 Grade 3, 23 Grade 4.

Lebowitz[20] studied a convenience sample of 100 patients with complaints which

were "not purely musculoskeletal" over a 3-month period, recording ZTT scores for each, using Metagenics Zinc Tally and grading the test at 10 seconds. He found 16 Grade 1, 63 Grade 2, 17 Grade 3, and 4 Grade 4.

Hestalden[21] did a ZTT on 35 patients who were thought by other indicators to require zinc. He found 27 Grade 1, 6 Grade 2, 1 Grade 3 and 1 Grade 4. Nutri-West's "Zinc Taste Test" solution was used, which is formulated at Bryce-Smith's 0.1% concentration, and the test was graded at 10 seconds.

Our study with the 0.1 % solution found 34 Grade 1, 39 Grade 2, 3 Grade 3, and 0 Grade 4, in a sequential sample at 10 sec. At 30 sec, there were fewer Grade 1's and more Grade 3's.

As might be expected, the stronger solution, and later grading results in more subjects falling into the "sufficient" categories on this test.

A company representative stated that only Metagenics autoclaves their solution and that others are contaminated with bacteria which make the zinc unavailable or interfere with the taste. While the microbiology professor at Logan College of Chiropractic found it unlikely to suppose that ZnSO4 in water would support bacterial growth, he kindly cultured a sample from one of the test bottles which I used during this study. At the time it was tested the bottle had been unsealed for over 2 weeks. Nothing grew in the culture.

It should be noted that several slightly different ZTT testing protocols are described in the literature. Some grade at 10 seconds and some grade at 30 seconds. One author has the solution in the mouth for 10 seconds, has the subject swallow, and then has the subject describe the taste at 30 seconds[22]. This should be standardized to facilitate comparison of results between different studies.

As the flavor often increases over time, we had the subject hold the solution in the mouth for 30 sec. The assistant timed the test and told the subject to note the taste at 10 sec. and at 30 sec. The subject then spit the solution out and described both tastes. No subject had difficulty doing this. The assistant then categorized the descriptions on Bryce-Smith's scale. We had pretested several variants of the procedure and this seemed easiest for the subjects, rapid, and gave the most consistent grades.

We chose to do the zinc sulfate taste-test after the muscle testing, to ensure that no zinc was swallowed or remained in the mouth to alter the subsequent muscle testing. This is especially important since in pretesting the ZTT protocols we noted that many people continued to taste the solution for up to 15 minutes after the solution had been expectorated.
In this sample there were very few subjects scoring grades 3 or 4, an unexpected result.

This may have been the result of chance, a factor of geography, or a matter of the potency of the test solution (0.1% vs. 0.135%). It seems unlikely to assume that 96% of the sample were actually zinc deficient. St. Louis, where the study was done, is not known for unusually high incidence of zinc deficiency, and our tap water is quite alkaline, rather than acid, making copper excess from plumbing very infrequent.

It should also be noted that ZTT originally was proposed as an assessment of zinc status in anorexics. Schauss, using the 0.135% solution found that none of 9 known anorexics could taste it (grade 1), while all but 1 non-eating disordered subjects could (grades 2 and above). It may well be that there are factors with regard to zinc status and taste in anorexics which are not applicable to the population at large. It may also be that the 1.35g/liter solution is in fact more useful for identifying significant zinc responsive hypogeusias.

Respiration and muscle response to zinc:
A large number of subjects (23 of 76 total = 30%, and 23 of 32 with weak muscles = 72%) showed a change in muscle function during different phases of respiration. This phenomenon has been reported in A.K. texts and papers for over 20 years, and is used clinically to analyze function of the cranio-sacral system[23,24]. This finding serves to emphasize previous observations that controlling respiratory phase is essential for repeatability of manual muscle testing.[25] Otherwise it is quite likely that sequential muscle tests will not be in the same respiratory phase and hence may appear to vary for no reason, or a respiratory variation in strength could spuriously be attributed to another variable.

In this study, of the 23 subjects with change in muscle strength on respiration, 18 responded to zinc. In the 23 subjects who responded to zinc, 21 had a respiratory pattern of weakness initially. This may represent a relationship which could be explored further.

Discussion of Procedures:
Considerations in the choice of test muscles included the following:
Zinc deficiency is associated with problems in sugar metabolism, HCl production (via zinc-dependent carbonic anhydrase), essential fatty acid metabolism, reproductive problems, copper toxicity, depressed immune function, lack of taste and smell, emotional depression and anorexia.[26] Hence, muscles traditionally associated with these factors might be expected to be most responsive to zinc. Muscles considered were latissimus dorsi (insulin, pancreatic enzymes), gluteus muscles, adductors, piriformis (reproductive), pectoralis major clavicular division (HCL), infraspinatus (thymus relationship - immune function), lower and mid trapezius (spleen association - immune function), pectoralis major, sternal division (liver, essential fatty acid metabolism, serum protein maintenance). It should be noted that the 3rd highest concentration of zinc in the body is found in skeletal

muscle, kidney, and liver, so any muscle might conceivably be affected by a zinc deficiency.

For time considerations and to eliminate confounding effects of postural changes, we decided to use only muscles which could be tested simply with standard A.K. tests in the supine position.

Repeatability of muscle tests was an additional consideration. Of those listed, the latissimus dorsi test is, in the author's experience, the least repeatable. It is often used to demonstrate poor muscle testing by varying the timing of the test or altering the angle of pull. Although it has been used in prior oral nutrient testing studies, for the above reason we did not use it. It would have theoretically represented pancreatic function. In the auathor's experience the other muscles listed are rather easily repeatable, although some might argue about pectoralis major, clavicular division. Pectoralis major, clavicular division is a rather small muscle and muscle test results can be altered by slight variations in the angle of test pressure. Rectus femoris was included as a muscle which is traditionally regarded as difficult to overwhelm in a muscle test. This muscle is considered to be small intestine-associated, and thus related to digestion and absorption, factors which both affect and are affected by zinc status.

After open pretesting a number of the above muscles for frequency of weakness and frequency of response to respiration, we chose to work with pectoralis major, sternal division, and rectus femoris, which were each found weak in about one third of the patients screened, about half showing a change of strength to respiration.

A potential criticism is that the patient's treating doctor did the testing. She theoretically had prior information regarding the patient's diet, supplementation, patterns of muscle weakness, and symptoms. Having anyone else do the testing would have made a sequential sample of office patients impractical due to time and the relative inexperience of alternate testers available to us. However, she was blind to the result of the ZTT and the subject was not informed of the significance of the muscle testing, the contents of the tablet, or ZTT results. None of the subjects had been tested using the ZTT previously.

CONCLUSION

This study did not show a positive correlation between the ZTT and muscle testing response to tasting zinc. This may be due in part to the small numbers of subjects in this sample with grades 3 or 4 on the ZTT, and in part due to weaknesses in the ZTT itself. The author did observe changes in some muscle tests while the zinc tablet was in the mouth, as has been observed clinically by applied kinesiologists for many years. No conclusion can be made on the theory that this phenomenon represents a

clinical need for the nutrient being tested.

There is clearly a need to account for respiratory phase in any studies of muscle testing repeatability and in clinical practice.

In clinical practice oral nutrient testing is never done in the kind of double-blind vacuum that this study attempted to establish. It is hoped that nutritional recommendations are never made without knowledge of the patient's diet, symptoms, history, and other clinical findings. It is certainly possible that this knowledge has some effect on the tester's finding of changes in muscle response on oral nutrient testing in clinical practice. I look forward to further research in the area.

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DETERMINING THE PRIMARY SUBLUXATION VIA SPECIFIC MUSCLE TESTING

René Espy, D.C. & Nancy L. McBride, D.C.

Abstract: The purpose of this study is to determine if specific muscle testing is a viable method of discovering the primary subluxation as well as a guide to determine an appropriate method of treatment of an aberrant physiological pattern. The basic procedure used was specific testing of the muscles involved in the aberrant physiological pattern displayed by the patient. Muscles can be evaluated according to Neck, Shoulder, Low Back, Pelvis, Thigh, Knee, Calf and Ankle. 1. As stated in the papers presented at the annual meetings of the ICAK - USA 1992-93 and 1993-94. 2., by the authors, each muscle has a specific frequency that allows the body to display its hologramic relationship. In this paper we have demonstrated only the muscles of the neck. Development of a computer program that analyzes multiple muscle frequencies allowed us to determine the primary subluxation, related to the specific muscles tested, quickly and efficiently. The hypothesis being that if the subluxation is primary, all weak muscles would now test strong once the frequency of the primary subluxation is hologramically applied to the body.

The outcome of this study indicates that subluxations, if primary, have specific patterns of abberant physiologic relationships. The primary subluxation appears to inhibit a specific energetic pattern that becomes the basis for future physiologic inhibition of normal function and may be the basis for pathology, the origin of which is difficult to define.

Introduction: The Vertebral Subluxation Complex (VSC) is defined by the Mercy Guidelines as "an aberration of normal spinal biomechanics, usually involving a restriction or loss of normal **movement** of a **motion** segment, and associated aberrations in the tissues which support articular **motion** (e.g., nerve, muscle, connective and vascular)." The guidelines also define the subluxation syndrome as "the clinical signs and symptoms thought to relate to pathophysiology of dysfunction of spinal **motion** segments or to peripheral joints that may be amenable to manipulative/adjustive procedures."3.

The words **motion** and **movement** have been highlighted to bring attention to the limiting tenet that treatment of the VSC has been limited to adjustment of the spinal vertebrae. Since movement of the spinal segment is dependent on muscle action could it not then be important to determine if there is muscle aberration creating a significant interference in normal spinal motion? We all know that muscles move bones yet we somehow often adjust the same segment numerous times until we fatigue the body and it adapts, possibly developing a new VSC in order to create some semblance of homeostasis. We have found that when we adjust a spinal segment, in the proper direction and with the proper thrust, we only have to adjust it once, in relationship to the area being treated, as the innate intelligence of the body apparently accepts our correction and continues the healing process.

How many times has a patient presented themselves in our office with serious orthopedic and neurological symptoms yet all orthopedic and neurological tests are within normal limits? Numerous times the standard tests (orthopedic, neurological, X-Ray, laboratory) give no data that will justify care, yet the patient has symptoms. When we perform standard chiropractic testing procedures (static and motion palpation, range of motion and posture analysis, etc.,) many findings concur with the patient's complaints. Having noted this during many years of practice and being frustrated with the need for documentation of patient complaints, procedures were developed to assist the need for chiropractic documentation.

For example, if a patient is involved in an automobile accident and sustains a whiplash injury, a common significant spinal involvement is C5. Adjustment of this area has alleviated many of the severe symptomatology patterns that the patient experienced. However, the question must be asked, "Why is the norm to adjust the same area for numerous visits?" And why is it that some cases do not respond even after a lengthy program of C5 adjustments? Is it not possible that the body is trying to tell us to look elsewhere and find a correlative irritation that is continually interfering with the normal function of C5 ? An analogy might be a door that will not stay closed. We can continue to close the door only to have it fly open again and again. We can put a barrier up trying to keep the door closed, or we can check further to see why it continually reopens. Upon inspection we see that the latch is broken and once fixed the door stays closed.

If we look beyond the spinal segment C5 and test other areas that are involved in the movement and function of C5, we may be able to correct its biomechanical aspect more quickly. If we test the muscles related to the myomere (direct nerve supply to the muscle) and the muscles related to the vertebral level (the reflex arc), we may find the answer to the continual misalignment of the same segment. The muscles that have been found to be related are as follows:

MYOMERE LEVEL C5 (MOTOR)

Scalenus Posterior (284)
Serratus Anterior, Superior Division (402)
Supraspinatus, Spine Division (420)
Supraspinatus, Fossa Division (422)
Rhomboid Major (400)
Rhomboid Minor (398)
Deltoid, Anterior, Scapular Division (456)
Biceps Brachii Longhead (466)
Diaphragm, Left Lumbar Division (662)

VERTEBRAL LEVEL C5 (SENSORY)

Coracobrachialis, Coracoid Division (444)
Pectoralis Major, Clavicular Division (460)
Serratus Anterior, Superior Division (402)
Deltoid, Middle, Posterior Division (452)
Infraspinatus, Superior Division (424)
Extensor Carpi Radialis Longus, Extensor Division (516)
Tibialis Anterior, Dorsiflexor Division (898)
Abductor Digiti Minimi Pedis (974)
Flexor Hallucis Longus, Fibular Division (902)
Serratus Posterior, Inferior Division (652)

As shown in the papers presented at the annual meetings of the ICAK - USA 1992, 1993. 4. by the authors, each muscle has been found to have the following potential components: a myomere, a vertebral level, a cranial bone, a foot bone, two organs/tissues, an acupuncture point and a nutrient that may be directly related to it. See table at the end of this paper for the possible interrelated components of the C5 myomere and the C5 vertebral level. (The number in front of the correlations relates to the main frequency of the muscle and to the table above.)

With the awareness of the possibilities related to C5 we can now see why patients with a whiplash injury complain of such varied symptoms. With the above data we as Chiropractors can be more holistic practitioners and demonstrate to the patient why they feel the way they do. With measured Chiropractic care we now have a greater variety of possibilities to correct their problem.

Materials: The materials necessary for this study are:

1. Biostim 200 E
2. Integrated Chiropractic Systems (ICS) computer program
3. IBM compatible computer
4. Body Relationship Muscle Testing Manuals vol's. 1-6
5. Diagnostic and Therapeutic Manuals vol's. 1-10

Method:

1. Test muscles of the specific area of complaint, such as muscles of the neck (see volume 1 Body Relationship Muscle Testing Manual)
2. Enter the results of the muscle tests into the ICS computer program.

3. Analyze the correlations displayed to help determine the primary subluxation.
4. Determine the primary muscle frequency related to the primary subluxation.
5. Apply the determined frequency to the body using the Bio Stim 200 E. The primary frequency includes a main frequency, a carrier wave, an intensity, and a specific wave form.
6. Retest all previously tested muscles. The frequency is the primary frequency if all previously tested muscles now test strong including those in the group which tested strong initially.
7. Treatment consists of any or all of the following (sequence determined by carrier frequency and wave form modulation):
    a. Myomere adjustment
    b. Vertebral adjustment
    c. Cranial adjustment
    d. Foot adjustment
    e. Muscle acupuncture therapy
    f. Organ/Tissue reflex therapy
    g. Nutritional support
    h. Neurovascular therapy
    i. Neurolymphatic therapy
    j. Emotional support

Results: When a cervical muscle examination was performed on ten patients it was found that the primary subluxation was the same for all patients given the same muscles tested weak. If there were variations of weaknesses in the group of patients tested, a variation of primary subluxations were observed within the group. See Tables 1-10.

Patient 1 - Cervical exam - Primary Subluxation - C 2 Frequency - 346

Patient 2 - Cervical exam - Primary Subluxation - C 3 Frequency - 332

Patient 3 - Cervical exam - Primary Subluxation - C 3 Frequency - 346

Patient 4 - Cervical exam - Primary Subluxation - C 2 Frequency - 346

Patient 5 - Cervical exam - Primary Subluxation - C 3 Frequency - 308

Patient 6 - Cervical exam - Primary Subluxation - C 2 Frequency - 346

Patient 7 - Cervical exam - Primary Subluxation - C 3 Frequency - 276

Patient 8 - Cervical exam - Primary Subluxation - C 3 Frequency - 308

Patient 9 - Cervical exam - Primary Subluxation - C 3 Frequency - 276

Patient 10 - Cervical exam - Primary Subluxation - C 3 Frequency - 332

The legend for the table is as follows:

Num = Muscle frequency number
MM = Myomere
VL = Vertebral Level
Cranial = Related Cranial Bone
Foot = Related Foot Bone
MAP = Muscle Acupuncture Point
Nutrient = Related Nutrient

Organ = Related Organ/Tissue

Discussion: As stated above, orthopedic and neurological tests of a specific area may give no findings that relate to the patient's complaint. Specific muscle testing of an area of complaint may display numerous indications of direct and indirect possibilities of physiological interference. Further studies that either duplicate or enhance the above findings can lead to the possibility of specific muscle testing becoming a standard of a complete Chiropractic examination.

Conclusions: A primary subluxation can be determined using specific muscle testing. Analysis of the correlations of a specific muscle group (i.e. neck muscles) indicates a primary frequency that is extremely helpful in restoring the strength to all the weak muscles related to the area being tested. The above data poses a wide range of research questions that warrants an extensive research design. The potential result could be that specific muscle testing would become an acceptable standard for Chiropractic examination, included within the philosophical principle of a subluxation complex and the therapeutic application of Chiropractic practice.

C 5 MYOMERE RELATIONSHIPS

Freq.	Muscle	Organ/Tissue	MM	VL	Cranial	Foot	MAP	Nutrient
284	Scalenus Posterior	Kidney/Urethra	C5	T12	Ethmoid	1st Cuneiform	Tw 12	Spore-X
402	Serratus Ant., Sup. Div.	Lung/Ovary-Testicle	C5	C5	Parietal Bulge	Talus	St 33	Core Thyro
420	Supraspin., Spine Div.	Thymus/Maxillary Sinus	C5	T7	Mandible	Prox. Phal. Grt. Toe	St 43	Thym-X
422	Supraspin. Fossa Div.	Anterior Pituitary/Esophagus	C5	T8	Ethmoid	1st Cuneiform	B 60	Thym-X
400	Rhomboid Major	Liver/Mammary	C5	T8	Vomer	2nd Metatarsal	St 36	Core Liver
398	Rhomboid Minor	Posterior Pituitary/Liver	C5	T7	Frontal, Internal	Prox. Phal. 3rd Toe	Lv 8	Core Liver
456	Deltoid, Ant., Scap. Div.	Lung/Adrenal	C5	L2	Sphenoid	3rd Cunei., Lateral	G 31	Core Carbo Gest
466	Biceps Brachii Longhd.	Kidney/Spleen	C5	T12	Styloid	Dist. Phal. Grt. Toe	Sp 9	Core Folic Acid
662	Diaph., L. Lumbar Div.	Spleen/L.Legs & Ing Node/L. Pancreas	C5	T12	Maxillary (M-L)	3rd Metatarsal	LG30 RSp21	Spore-X

C 5 VERTEBRAL LEVEL RELATIONSHIPS

Freq.	Muscle	Organ/Tissue	MM	VL	Cranial	Foot	MAP	Nutrient
444	Coracobrach., Cor. Div.	Kidney/Nose	C7	C5	Styloid	Dist. Phal. Grt. Toe	Lv 10	Core Inositol
460	Pect. Major, Clav. Div.	Uterus-Prostate (D. P.)/Heart	C7	C5	Parietal Bulge	Talus	G 30	Core Folic Acid
402	Serratus Ant., Sup. Div.	Lung/Ovary-Testicle	C5	C5	Parietal Bulge	Talus	St 33	Core Thyro
452	Deltoid, Mid., Post. Div.	Spleen/Lung	C6	C5	Sphenoid	3rd Cunei., Lateral	Lv 3	Core Lung
424	Infraspinatus, Sup. Div.	Thyroid/Lymph. of Lung	C6	C5	Nasal	Dist. Phal. 2nd Toe	Cx 1	Splen-X
516	Ext. Car. Rad. L., Ext. D.	Stomach (Pyloric Antrum)/Kidney	C7	C5	Parietal Descent	Dist. Phal. 3rd Toe	B 2	Core Niacin
898	Tibialis Ant., Dorsi. Div.	Spleen/Ovary-Testicle	L5	C5	Frontal, External	Navicular	Lu 4	Spore-X
974	Abd. Digitus Min. Pedis	Nose/Posterior Pituitary	S2	C5	Occiput, Lateral	5th Metatarsal	St 33	Core Vitamin A
902	Flex. Hall. Lon., Fib. Div.	Esophagus/Kidney	S1	C5	Inferior Conchae	Prox. Phal. 4th Toe	B 58	Core Selenium
652	Serratus Post., Inf. Div.	Nose/Jejunum	T10	C5	Frontal, External	Navicular	Lu 7	Core Magnesium

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3. Haldeman, Scott, Chapman-Smith, David, and Peterson, Donald M., " Guidelines for Chiropractic Quality Assurance and Practice Parameters. (Gaithersburg, Maryland, Aspen Publishers. Inc., 1993.)

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TABLE 1 - PATIENT NUMBER 1

Muscle Correlations: Total Accumulated Muscle Groups.
Patient Name - Last: First:

Num	MM	VL	Cranial	Foot	MAP	Nutrient
052	Cr VII	T9	Styloid	Dist. Phal. Great Toe	XA 3	Core Thyro
270	C2	L2	Occiput, Lateral	5th Metatarsal	B 58	Core Selenium
272	C3	T10	Maxillary, M-L	3rd Metatarsal	St 10	Core Calcium
274	C2	T8	Occiput, Lateral	5th Metatarsal	Cx 2	Core Selenium
276	C3	T6	Glabella	4th Metatarsal	Lv 6	Core Niacin
278	C4	L1	Parietal Bulge	Talus	Sp 10	Core Kidney
282	C4	L4	Occiput, Universal	Calcaneus	Cx 9	Core Carbo Gest
284	C5	T12	Ethmoid	1st Cuneiform	Tw 12	Spore-X
314	C1	T6	Temporal, Internal	Cuboid, Inferior	Tw 3	Core D-Tox
322	C2	C3	Temporal, External	Cuboid, Lateral	Tw 16	Core Potassium
326	T1 R	C7	Occiput, Lateral	5th Metatarsal	Lv 7	Core Folic Acid
332	C3	T4	Temporal, External	Cuboid, Lateral	K 9	Core Potassium
334	T2	T12	Mandible	Prox. Phal. Great Toe	Tw 16	Core Thyro
346	C2	T10	Occiput, Lateral	5th Metatarsal	G 40	Core Rutin
350	C6	L5	Parietal Bulge	Talus	St 37	Core Thiamine

Muscle Correlations: Total Accumulated Muscle Groups.
Patient Name - Last: First:

Num	First Organ	Second Organ
052	Gallbladder Duct	Thyroid
270	Colon (A-D)	Eye
272	Thymus	Ear (Internal)
274	Pancreas (Sugar)	Gallbladder
276	Lymph. of Submandibular	Larynx
278	Ovary/Testicle	Bladder
282	Lymph. of Submandibular	Gallbladder
284	Kidney	Urethra
314	Anterior Pituitary	Uterus/Prostate
322	Anterior Pituitary	Pancreas (Protein)
326	Adrenal	Heart
332	Spleen	Vagina/Penis
334	Salivary Gland (Sublingual)	Uterus/Prostate
346	Spleen	Lung
350	Colon (A-D)	Stomach (Fundus)

TABLE 1 - PATIENT NUMBER 1

MM Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: First:

MM	Num Assoc MM
Cranial Nerve Total	1
Cr VII	1
Cervical Spine Total	12
C1	1
C2	4
C3	3
C4	2
C5	1
C6	1
Thoracic Spine Total	2
T1 R	1
T2	1

VL Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: First:

VL	Num Assoc VL
Cervical Spine Total	2
C3	1
C7	1
Thoracic Spine Total	9
T4	1
T6	2
T8	1
T9	1
T10	2
T12	2
Lumbar Spine Total	4
L1	1
L2	1
L4	1
L5	1

TABLE 1 - PATIENT NUMBER 1

Cranial Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: First:

Cranial	Num Assoc	Cranial	Num Assoc
Ethmoid Total	1		
Glabella Total	1		
Mandible Total	1		
Maxillary Total	1		
Maxillary, M-L	1		
Occiput Total	5		
OCCIPUT, LATERAL	4		
Occiput, Universal	1		
Parietal Total	2		
Parietal, Bulge	2		
Styloid Total	1		
Temporal Total	3		
Temporal, External	2		
Temporal, Internal	1		

Foot Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: First:

Foot Bone	Num Assoc	Foot Bone	Num Assoc
Calcaneus Total	1		
Cuboid Total	3		
Cuboid, Inferior	1		
Cuboid, Lateral	2		
Cuneiform Total	1		
1st Cuneiform	1		
Metatarsal Total	6		
3rd Metatarsal	1		
4th Metatarsal	1		
5TH METATARSAL	4		
Phalanges Total	2		
Dist. Phal. Great Toe	1		
Prox. Phal. Great Toe	1		
Talus Total	2		

TABLE 1 - PATIENT NUMBER 1

Organ Summary: Total Accumulated Muscle Groups. Part 1 of 2
Patient Name - Last: First:

Organ	Num Assoc	Organ	Num Assoc
Adrenal Total	1	Larynx Total	1
Adrenal	1	Lung Total	1
Bladder Total	1	Lung	1
Colon Total	2	Ovary-Testicle Total	1
COLON (A-D)**********	2	Ovary-Testicle	1
Ear Total	1	Pancreas Total	2
Ear (Internal)	1	Pancreas (Protein)	1
Eye Total	1	Pancreas (Sugar)	1
Eye	1	Pituitary Total	2
Gallbladder Total	3	PITUITARY, ANTERIOR**********	2
GALLBLADDER**********	2	Salivary Gland (Sublingual) Total	1
Gallbladder Duct	1	Spleen Total	2
Heart Total	1	Stomach Total	1
Heart	1	Stomach (Fundus)	1
Kidney Total	1	Thymus Total	1

Organ Summary: Total Accumulated Muscle Groups. Part 2 of 2
Patient Name - Last: First:

Organ	Num Assoc	Organ	Num Assoc
Tissue Total	2		
Lymphatics Total	2		
LYMPH* OF SUBMANDIBULAR********	2		
Thyroid Total	1		
Urethra Total	1		
Urethra	1		
Uterus-Prostate Total	2		
UTERUS-PROSTATE**********	2		
Vagina-Penis Total	1		

TABLE 1 - PATIENT NUMBER 1

MAP Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: First:

MAP	Num Assoc	MAP	Num Assoc
Bladder Total	1	St 10	1
B 58	1	St 37	1
Circulation - Sex Total	2	Triple Warmer Total	4
Cx 2	1	Tw 3	1
Cx 9	1	Tw 12	1
Gallbladder Total	1	TW 16**********	2
G 40	1	XA 3 Total	1
Kidney Total	1		
K 9	1		
Liver Total	2		
Lv 6	1		
Lv 7	1		
Spleen Total	1		
Sp 10	1		
Stomach Total	2		

Nutrients Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: First:

Nutrients	Num Assoc	Nutrients	Num Assoc
Digestive (System - Enzymes) Total	1	Core Rutin	1
Spore-X	1	Core Thiamine	1
Metabolism Total	2		
Core Carbo Gest	1		
Core D-Tox	1		
Mineral Total	5		
Core Calcium	1		
CORE POTASSIUM**********	2		
CORE SELENIUM**********	2		
Glandular Total	3		
Core Kidney	1		
CORE THYRO**********	2		
Vitamin Total	4		
Core Folic Acid	1		
Core Niacin	1		

TABLE 1 - PATIENT NUMBER 2

Muscle Correlations: Total Accumulated Muscle Groups.
Patient Name - Last: First:

Num	MM	VL	Cranial	Foot	MAP	Nutrient
270	C2	L2	Occiput, Lateral	5th Metatarsal	B 58	Core Selenium
272	C3	T10	Maxillary, M-L	3rd Metatarsal	St 10	Core Calcium
276	C3	T6	Glabella	4th Metatarsal	Lv 6	Core Niacin
278	C4	L1	Parietal Bulge	Talus	Sp 10	Core Kidney
284	C5	T12	Ethmoid	1st Cuneiform	Tw 12	Spore-X
308	C3	T4	Vomer	2nd Metatarsal	XL 3	Core Folic Acid
314	C1	T6	Temporal, Internal	Cuboid, Inferior	Tw 3	Core D-Tox
322	C2	C3	Temporal, External	Cuboid, Lateral	Tw 16	Core Potassium
326	T1 R	C7	Occiput, Lateral	5th Metatarsal	Lv 7	Core Folic Acid
332	C3	T4	Temporal, External	Cuboid, Lateral	K 9	Core Potassium
334	T2	T12	Mandible	Prox. Phal. Great Toe	Tw 16	Core Thyro
346	C2	T10	Occiput, Lateral	5th Metatarsal	G 40	Core Rutin
350	C6	L5	Parietal Bulge	Talus	St 37	Core Thiamine

Muscle Correlations: Total Accumulated Muscle Groups.
Patient Name - Last: First:

Num	First Organ	Second Organ
270	Colon (A-D)	Eye
272	Thymus	Ear (Internal)
276	Lymph. of Submandibular	Larynx
278	Ovary/Testicle	Bladder
284	Kidney	Urethra
308	Ovary/Testicle	Jejunum
314	Anterior Pituitary	Uterus/Prostate
322	Anterior Pituitary	Pancreas (Protein)
326	Adrenal	Heart
332	Spleen	Vagina/Penis
334	Salivary Gland (Sublingual)	Uterus/Prostate
346	Spleen	Lung
350	Colon (A-D)	Stomach (Fundus)

TABLE 1 - PATIENT NUMBER 2

MM Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: First:

MM	Num Assoc	MM	Num Assoc
Cervical Spine Total	11		
C1	1		
C2	3		
C3	4		
C4	1		
C5	1		
C6	1		
Thoracic Spine Total	2		
T1 R	1		
T2	1		

VL Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: First:

VL	Num Assoc	VL	Num Assoc
Cervical Spine Total	2		
C3	1		
C7	1		
Thoracic Spine Total	8		
T4	2		
T6	2		
T10	2		
T12	2		
Lumbar Spine Total	3		
L1	1		
L2	1		
L5	1		

TABLE 1 - PATIENT NUMBER 2

Cranial Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: First:

Cranial	Num Assoc	Cranial	Num Assoc
Ethmoid Total	1		
Glabella Total	1		
Mandible Total	1		
Maxillary Total	1		
Maxillary, M-L	1		
Occiput Total	3		
OCCIPUT, LATERAL**********	3		
Parietal Total	2		
Parietal, Bulge	2		
Temporal Total	3		
Temporal, External	2		
Temporal, Internal	1		
Vomer Total	1		

Foot Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: First:

Foot Bone	Num Assoc	Foot Bone	Num Assoc
Cuboid Total	3		
Cuboid, Inferior	1		
Cuboid, Lateral	2		
Cuneiform Total	1		
1st Cuneiform	1		
Metatarsal Total	6		
2nd Metatarsal	1		
3rd Metatarsal	1		
4th Metatarsal	1		
5TH METATARSAL**********	3		
Phalanges Total	1		
Prox. Phal. Great Toe	1		
Talus Total	2		

TABLE 1 - PATIENT NUMBER 2

Organ Summary: Total Accumulated Muscle Groups. Part 1 of 2
Patient Name - Last: First:

Organ	Num Assoc	Organ	Num Assoc
Adrenal Total	1	Lung	1
Adrenal	1	Ovary-Testicle Total	2
Bladder Total	1	OVARY-TESTICLE**********	2
Colon Total	2	Pancreas Total	1
COLON (A-D)**********	2	Pancreas (Protein)	1
Ear Total	1	Pituitary Total	2
Ear (Internal)	1	PITUITARY, ANTERIOR**********	2
Eye Total	1	Salivary Gland (Sublingual) Total...	1
Eye	1	Spleen Total	2
Heart Total	1	Stomach Total	1
Heart	1	Stomach (Fundus)	1
Jejunum Total	1	Thymus Total	1
Kidney Total	1	Tissue Total	1
Larynx Total	1	Lymphatics Total	1
Lung Total	1	Lymph. of Submandibular	1

Organ Summary: Total Accumulated Muscle Groups. Part 2 of 2
Patient Name - Last: First:

Organ	Num Assoc	Organ	Num Assoc
Urethra Total	1		
Urethra	1		
Uterus-Prostate Total	2		
UTERUS-PROSTATE**********	2		
Vagina-Penis Total	1		

TABLE 1 - PATIENT NUMBER 2

MAP Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: First:

MAP	Num Assoc	MAP	Num Assoc
Bladder Total	1	Tw 3	1
B 58	1	Tw 12	1
Gallbladder Total	1	TW 16**********	2
G 40	1	XL Total	1
Kidney Total	1	XL 3	1
K 9	1		
Liver Total	2		
Lv 6	1		
Lv 7	1		
Spleen Total	1		
Sp 10	1		
Stomach Total	2		
St 10	1		
St 37	1		
Triple Warmer Total	4		

Nutrients Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: First:

Nutrients	Num Assoc	Nutrients	Num Assoc
Digestive (System - Enzymes) Total	1	Core Thiamine	1
Spore-X	1		
Metabolism Total	1		
Core D-Tox	1		
Mineral Total	4		
Core Calcium	1		
CORE POTASSIUM**********	2		
Core Selenium	1		
Glandular Total	2		
Core Kidney	1		
Core Thyro	1		
Vitamin Total	5		
CORE FOLIC ACID**********	2		
Core Niacin	1		
Core Rutin	1		

TABLE 1 - PATIENT NUMBER 3

Muscle Correlations: Total Accumulated Muscle Groups.
Patient Name - Last: a First:

Num	MM	VL	Cranial	Foot	MAP	Nutrient
052	Cr VII	T9	Styloid	Dist. Phal. Great Toe	XA 3	Core Thyro
270	C2	L2	Occiput, Lateral	5th Metatarsal	B 58	Core Selenium
272	C3	T10	Maxillary, M-L	3rd Metatarsal	St 10	Core Calcium
274	C2	T8	Occiput, Lateral	5th Metatarsal	Cx 2	Core Selenium
276	C3	T6	Glabella	4th Metatarsal	Lv 6	Core Niacin
278	C4	L1	Parietal Bulge	Talus	Sp 10	Core Kidney
282	C4	L4	Occiput, Universal	Calcaneus	Cx 9	Core Carbo Gest
284	C5	T12	Ethmoid	1st Cuneiform	Tw 12	Spore-X
314	C1	T6	Temporal, Internal	Cuboid, Inferior	Tw 3	Core D-Tox
322	C2	C3	Temporal, External	Cuboid, Lateral	Tw 16	Core Potassium
326	T1 R	C7	Occiput, Lateral	5th Metatarsal	Lv 7	Core Folic Acid
332	C3	T4	Temporal, External	Cuboid, Lateral	K 9	Core Potassium
334	T2	T12	Mandible	Prox. Phal. Great Toe	Tw 16	Core Thyro
346	C2	T10	Occiput, Lateral	5th Metatarsal	G 40	Core Rutin
350	C6	L5	Parietal Bulge	Talus	St 37	Core Thiamine

Muscle Correlations: Total Accumulated Muscle Groups.
Patient Name - Last: a First:

Num	First Organ	Second Organ
052	Gallbladder Duct	Thyroid
270	Colon (A-D)	Eye
272	Thymus	Ear (Internal)
274	Pancreas (Sugar)	Gallbladder
276	Lymph. of Submandibular	Larynx
278	Ovary/Testicle	Bladder
282	Lymph. of Submandibular	Gallbladder
284	Kidney	Urethra
314	Anterior Pituitary	Uterus/Prostate
322	Anterior Pituitary	Pancreas (Protein)
326	Adrenal	Heart
332	Spleen	Vagina/Penis
334	Salivary Gland (Sublingual)	Uterus/Prostate
346	Spleen	Lung
350	Colon (A-D)	Stomach (Fundus)

TABLE 1 - PATIENT NUMBER 3

MM Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: a First:

MM	Num Assoc	MM	Num Assoc
Cranial Nerve Total	1		
Cr VII	1		
Cervical Spine Total	12		
C1	1		
C2	4		
C3	3		
C4	2		
C5	1		
C6	1		
Thoracic Spine Total	2		
T1 R	1		
T2	1		

VL Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: a First:

VL	Num Assoc	VL	Num Assoc
Cervical Spine Total	2		
C3	1		
C7	1		
Thoracic Spine Total	9		
T4	1		
T6	2		
T8	1		
T9	1		
T10	2		
T12	2		
Lumbar Spine Total	4		
L1	1		
L2	1		
L4	1		
L5	1		

TABLE 1 - PATIENT NUMBER 3

Cranial Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: a First:

Cranial	Num Assoc	Cranial	Num Assoc
Ethmoid Total	1		
Glabella Total	1		
Mandible Total	1		
Maxillary Total	1		
Maxillary, M-L	1		
Occiput Total	5		
OCCIPUT, LATERAL**********	4		
Occiput, Universal	1		
Parietal Total	2		
Parietal, Bulge	2		
Styloid Total	1		
Temporal Total	3		
Temporal, External	2		
Temporal, Internal	1		

Foot Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: a First:

Foot Bone	Num Assoc	Foot Bone	Num Assoc
Calcaneus Total	1		
Cuboid Total	3		
Cuboid, Inferior	1		
Cuboid, Lateral	2		
Cuneiform Total	1		
1st Cuneiform	1		
Metatarsal Total	6		
3rd Metatarsal	1		
4th Metatarsal	1		
5TH METATARSAL**********	4		
Phalanges Total	2		
Dist. Phal. Great Toe	1		
Prox. Phal. Great Toe	1		
Talus Total	2		

TABLE 1 - PATIENT NUMBER 3

Organ Summary: Total Accumulated Muscle Groups. Part 1 of 2
Patient Name - Last: a First:

Organ	Num Assoc	Organ	Num Assoc
Adrenal Total	1	Larynx Total	1
Adrenal	1	Lung Total	1
Bladder Total	1	Lung	1
Colon Total	2	Ovary-Testicle Total	1
COLON (A-D)**********	2	Ovary-Testicle	1
Ear Total	1	Pancreas Total	2
Ear (Internal)	1	Pancreas (Protein)	1
Eye Total	1	Pancreas (Sugar)	1
Eye	1	Pituitary Total	2
Gallbladder Total	3	PITUITARY, ANTERIOR**********	2
GALLBLADDER**********	2	Salivary Gland (Sublingual) Total	1
Gallbladder Duct	1	Spleen Total	2
Heart Total	1	Stomach Total	1
Heart	1	Stomach (Fundus)	1
Kidney Total	1	Thymus Total	1

Organ Summary: Total Accumulated Muscle Groups. Part 2 of 2
Patient Name - Last: a First:

Organ	Num Assoc
Tissue Total	2
Lymphatics Total	2
LYMPH* OF SUBMANDIBULAR********	2
Thyroid Total	1
Urethra Total	1
Urethra	1
Uterus-Prostate Total	2
UTERUS-PROSTATE**********	2
Vagina-Penis Total	1

TABLE 1 - PATIENT NUMBER 3

MAP Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: a First:

MAP	Num Assoc	MAP	Num Assoc
Bladder Total	1	St 10	1
B 58	1	St 37	1
Circulation - Sex Total	2	Triple Warmer Total	4
Cx 2	1	Tw 3	1
Cx 9	1	Tw 12	1
Gallbladder Total	1	TW 16**********	2
G 40	1	XA 3 Total	1
Kidney Total	1		
K 9	1		
Liver Total	2		
Lv 6	1		
Lv 7	1		
Spleen Total	1		
Sp 10	1		
Stomach Total	2		

Nutrients Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: a First:

Nutrients	Num Assoc	Nutrients	Num Assoc
Digestive (System - Enzymes) Total	1	Core Rutin	1
Spore-X	1	Core Thiamine	1
Metabolism Total	2		
Core Carbo Gest	1		
Core D-Tox	1		
Mineral Total	5		
Core Calcium	1		
CORE POTASSIUM**********	2		
CORE SELENIUM**********	2		
Glandular Total	3		
Core Kidney	1		
CORE THYRO**********	2		
Vitamin Total	4		
Core Folic Acid	1		
Core Niacin	1		

TABLE 1 - PATIENT NUMBER 4

Muscle Correlations: Total Accumulated Muscle Groups.
Patient Name - Last: b First:

Num	MM	VL	Cranial	Foot	MAP	Nutrient
052	Cr VII	T9	Styloid	Dist. Phal. Great Toe	XA 3	Core Thyro
270	C2	L2	Occiput, Lateral	5th Metatarsal	B 58	Core Selenium
272	C3	T10	Maxillary, M-L	3rd Metatarsal	St 10	Core Calcium
274	C2	T8	Occiput, Lateral	5th Metatarsal	Cx 2	Core Selenium
282	C4	L4	Occiput, Universal	Calcaneus	Cx 9	Core Carbo Gest
308	C3	T4	Vomer	2nd Metatarsal	XL 3	Core Folic Acid
322	C2	C3	Temporal, External	Cuboid, Lateral	Tw 16	Core Potassium
326	T1 R	C7	Occiput, Lateral	5th Metatarsal	Lv 7	Core Folic Acid
332	C3	T4	Temporal, External	Cuboid, Lateral	K 9	Core Potassium
334	T2	T12	Mandible	Prox. Phal. Great Toe	Tw 16	Core Thyro
346	C2	T10	Occiput, Lateral	5th Metatarsal	G 40	Core Rutin

Muscle Correlations: Total Accumulated Muscle Groups.
Patient Name - Last: b First:

Num	First Organ	Second Organ
052	Gallbladder Duct	Thyroid
270	Colon (A-D)	Eye
272	Thymus	Ear (Internal)
274	Pancreas (Sugar)	Gallbladder
282	Lymph. of Submandibular	Gallbladder
308	Ovary/Testicle	Jejunum
322	Anterior Pituitary	Pancreas (Protein)
326	Adrenal	Heart
332	Spleen	Vagina/Penis
334	Salivary Gland (Sublingual)	Uterus/Prostate
346	Spleen	Lung

TABLE 1 - PATIENT NUMBER 4

MM Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: b First:

MM	Num Assoc	MM	Num Assoc
Cranial Nerve Total	1		
Cr VII	1		
Cervical Spine Total	8		
C2	4		
C3	3		
C4	1		
Thoracic Spine Total	2		
T1 R	1		
T2	1		

VL Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: b First:

VL	Num Assoc	VL	Num Assoc
Cervical Spine Total	2		
C3	1		
C7	1		
Thoracic Spine Total	7		
T4	2		
T8	1		
T9	1		
T10	2		
T12	1		
Lumbar Spine Total	2		
L2	1		
L4	1		

TABLE 1 - PATIENT NUMBER 4

Cranial Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: b First:

Cranial	Num Assoc	Cranial	Num Assoc
Mandible Total	1		
Maxillary Total	1		
Maxillary, M-L	1		
Occiput Total	5		
OCCIPUT, LATERAL**********	4		
Occiput, Universal	1		
Styloid Total	1		
Temporal Total	2		
Temporal, External	2		
Vomer Total	1		

Foot Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: b First:

Foot Bone	Num Assoc	Foot Bone	Num Assoc
Calcaneus Total	1		
Cuboid Total	2		
Cuboid, Lateral	2		
Metatarsal Total	6		
2nd Metatarsal	1		
3rd Metatarsal	1		
5TH METATARSAL**********	4		
Phalanges Total	2		
Dist. Phal. Great Toe	1		
Prox. Phal. Great Toe	1		

TABLE 1 - PATIENT NUMBER 4

Organ Summary: Total Accumulated Muscle Groups. Part 1 of 2
Patient Name - Last: b First:

Organ	Num Assoc	Organ	Num Assoc
Adrenal Total	1	Lung	1
Adrenal	1	Ovary-Testicle Total	1
Colon Total	1	Ovary-Testicle	1
Colon (A-D)	1	Pancreas Total	2
Ear Total	1	Pancreas (Protein)	1
Ear (Internal)	1	Pancreas (Sugar)	1
Eye Total	1	Pituitary Total	1
Eye	1	Pituitary, Anterior	1
Gallbladder Total	3	Salivary Gland (Sublingual) Total	1
GALLBLADDER**********	2	Spleen Total	2
Gallbladder Duct	1	Thymus Total	1
Heart Total	1	Tissue Total	1
Heart	1	Lymphatics Total	1
Jejunum Total	1	Lymph. of Submandibular	1
Lung Total	1	Thyroid Total	1

Organ Summary: Total Accumulated Muscle Groups. Part 2 of 2
Patient Name - Last: b First:

Organ	Num Assoc	Organ	Num Assoc
Uterus-Prostate Total	1		
Uterus-Prostate	1		
Vagina-Penis Total	1		

TABLE 1 - PATIENT NUMBER 4

MAP Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: b First:

MAP	Num Assoc	MAP	Num Assoc
Bladder Total	1	XA 3 Total	1
B 58	1	XL Total	1
Circulation - Sex Total	2	XL 3	1
Cx 2	1		
Cx 9	1		
Gallbladder Total	1		
G 40	1		
Kidney Total	1		
K 9	1		
Liver Total	1		
Lv 7	1		
Stomach Total	1		
St 10	1		
Triple Warmer Total	2		
TW 16**********	2		

Nutrients Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: b First:

Nutrients	Num Assoc	Nutrients	Num Assoc
Metabolism Total	1		
Core Carbo Gest	1		
Mineral Total	5		
Core Calcium	1		
CORE POTASSIUM**********	2		
CORE SELENIUM**********	2		
Glandular Total	2		
CORE THYRO**********	2		
Vitamin Total	3		
CORE FOLIC ACID**********	2		
Core Rutin	1		

TABLE 1 - PATIENT NUMBER 5

Muscle Correlations: Total Accumulated Muscle Groups.
Patient Name - Last: d First:

Num	MM	VL	Cranial	Foot	MAP	Nutrient
052	Cr VII	T9	Styloid	Dist. Phal. Great Toe	XA 3	Core Thyro
274	C2	T8	Occiput, Lateral	5th Metatarsal	Cx 2	Core Selenium
278	C4	L1	Parietal Bulge	Talus	Sp 10	Core Kidney
308	C3	T4	Vomer	2nd Metatarsal	XL 3	Core Folic Acid
326	T1 R	C7	Occiput, Lateral	5th Metatarsal	Lv 7	Core Folic Acid
332	C3	T4	Temporal, External	Cuboid, Lateral	K 9	Core Potassium
350	C6	L5	Parietal Bulge	Talus	St 37	Core Thiamine

Muscle Correlations: Total Accumulated Muscle Groups.
Patient Name - Last: d First:

Num	First Organ	Second Organ
052	Gallbladder Duct	Thyroid
274	Pancreas (Sugar)	Gallbladder
278	Ovary/Testicle	Bladder
308	Ovary/Testicle	Jejunum
326	Adrenal	Heart
332	Spleen	Vagina/Penis
350	Colon (A-D)	Stomach (Fundus)

TABLE 1 - PATIENT NUMBER 5

MM Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: d First:

MM	Num Assoc	MM	Num Assoc
Cranial Nerve Total	1		
Cr VII	1		
Cervical Spine Total	5		
C2	1		
C3**********	2		
C4	1		
C6	1		
Thoracic Spine Total	1		
T1 R	1		

VL Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: d First:

VL	Num Assoc	VL	Num Assoc
Cervical Spine Total	1		
C7	1		
Thoracic Spine Total	4		
T4**********	2		
T8	1		
T9	1		
Lumbar Spine Total	2		
L1	1		
L5	1		

TABLE 1 - PATIENT NUMBER 5

Cranial Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: d First:

Cranial	Num Assoc	Cranial	Num Assoc
Occiput Total	2		
OCCIPUT, LATERAL**********	2		
Parietal Total	2		
PARIETAL, BULGE**********	2		
Styloid Total	1		
Temporal Total	1		
Temporal, External	1		
Vomer Total	1		

Foot Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: d First:

Foot Bone	Num Assoc	Foot Bone	Num Assoc
Cuboid Total	1		
Cuboid, Lateral	1		
Metatarsal Total	3		
2nd Metatarsal	1		
5TH METATARSAL**********	2		
Phalanges Total	1		
Dist. Phal. Great Toe	1		
Talus Total	2		

TABLE 1 - PATIENT NUMBER 5

Organ Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: d First:

Organ	Num Assoc	Organ	Num Assoc
Adrenal Total	1	Spleen Total	1
Adrenal	1	Stomach Total	1
Bladder Total	1	Stomach (Fundus)	1
Colon Total	1	Thyroid Total	1
Colon (A-D)	1	Vagina-Penis Total	1
Gallbladder Total	2		
Gallbladder	1		
Gallbladder Duct	1		
Heart Total	1		
Heart	1		
Jejunum Total	1		
Ovary-Testicle Total	2		
OVARY-TESTICLE**********	2		
Pancreas Total	1		
Pancreas (Sugar)	1		

MAP Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: d First:

MAP	Num Assoc
Circulation - Sex Total	1
CX 2**********	1
Kidney Total	1
K 9**********	1
Liver Total	1
LV 7**********	1
Spleen Total	1
SP 10**********	1
Stomach Total	1
ST 37**********	1
XA 3 Total	1
XL Total	1
XL 3**********	1

TABLE 1 - PATIENT NUMBER 5

Nutrients Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: d First:

Nutrients	Num Assoc	Nutrients	Num Assoc
Mineral Total	2		
Core Potassium	1		
Core Selenium	1		
Glandular Total	2		
Core Kidney	1		
Core Thyro	1		
Vitamin Total	3		
CORE FOLIC ACID**********	2		
Core Thiamine	1		

TABLE 1 - PATIENT NUMBER 6

Muscle Correlations: Total Accumulated Muscle Groups.
Patient Name - Last: d First:

Num	MM	VL	Cranial	Foot	MAP	Nutrient
272	C3	T10	Maxillary, M-L	3rd Metatarsal	St 10	Core Calcium
274	C2	T8	Occiput, Lateral	5th Metatarsal	Cx 2	Core Selenium
284	C5	T12	Ethmoid	1st Cuneiform	Tw 12	Spore-X
308	C3	T4	Vomer	2nd Metatarsal	XL 3	Core Folic Acid
314	C1	T6	Temporal, Internal	Cuboid, Inferior	Tw 3	Core D-Tox
322	C2	C3	Temporal, External	Cuboid, Lateral	Tw 16	Core Potassium
346	C2	T10	Occiput, Lateral	5th Metatarsal	G 40	Core Rutin

Muscle Correlations: Total Accumulated Muscle Groups.
Patient Name - Last: d First:

Num	First Organ	Second Organ
272	Thymus	Ear (Internal)
274	Pancreas (Sugar)	Gallbladder
284	Kidney	Urethra
308	Ovary/Testicle	Jejunum
314	Anterior Pituitary	Uterus/Prostate
322	Anterior Pituitary	Pancreas (Protein)
346	Spleen	Lung

TABLE 1 - PATIENT NUMBER 6

MM Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: d First:

MM	Num Assoc	MM	Num Assoc
Cervical Spine Total	7		
C1	1		
C2	3		
C3	2		
C5	1		

VL Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: d First:

VL	Num Assoc	VL	Num Assoc
Cervical Spine Total	1		
C3	1		
Thoracic Spine Total	6		
T4	1		
T6	1		
T8	1		
T10	2		
T12	1		

TABLE 1 - PATIENT NUMBER 6

Cranial Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: d First:

Cranial	Num Assoc	Cranial	Num Assoc
Ethmoid Total	1		
Maxillary Total	1		
Maxillary, M-L	1		
Occiput Total	2		
OCCIPUT, LATERAL**********	2		
Temporal Total	2		
Temporal, External	1		
Temporal, Internal	1		
Vomer Total	1		

Foot Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: d First:

Foot Bone	Num Assoc	Foot Bone	Num Assoc
Cuboid Total	2		
Cuboid, Inferior	1		
Cuboid, Lateral	1		
Cuneiform Total	1		
1st Cuneiform	1		
Metatarsal Total	4		
2nd Metatarsal	1		
3rd Metatarsal	1		
5TH METATARSAL**********	2		

TABLE 1 - PATIENT NUMBER 6

Organ Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: d First:

Organ	Num Assoc	Organ	Num Assoc
Ear Total	1	Spleen Total	1
Ear (Internal)	1	Thymus Total	1
Gallbladder Total	1	Urethra Total	1
Gallbladder	1	Urethra	1
Jejunum Total	1	Uterus-Prostate Total	1
Kidney Total	1	Uterus-Prostate	1
Lung Total	1		
Lung	1		
Ovary-Testicle Total	1		
Ovary-Testicle	1		
Pancreas Total	2		
Pancreas (Protein)	1		
Pancreas (Sugar)	1		
Pituitary Total	2		
PITUITARY, ANTERIOR**********	2		

MAP Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: d First:

MAP	Num Assoc
Circulation - Sex Total	1
CX 2**********	1
Gallbladder Total	1
G 40**********	1
Stomach Total	1
ST 10**********	1
Triple Warmer Total	3
TW 3**********	1
TW 12**********	1
TW 16**********	1
XL Total	1
XL 3**********	1

TABLE 1 - PATIENT NUMBER 6

Nutrients Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: d First:

Nutrients	Num Assoc	Nutrients	Num Assoc
Digestive (System - Enzymes) Total..	1		
SPORE-X**********	1		
Metabolism Total.....	1		
CORE D-TOX**********	1		
Mineral Total.....	3		
CORE CALCIUM**********	1		
CORE POTASSIUM**********	1		
CORE SELENIUM**********	1		
Vitamin Total.....	2		
CORE FOLIC ACID**********	1		
CORE RUTIN**********	1		

TABLE 1 - PATIENT NUMBER 7

Muscle Correlations: Total Accumulated Muscle Groups.
Patient Name - Last: x First:

Num	MM	VL	Cranial	Foot	MAP	Nutrient
052	Cr VII	T9	Styloid	Dist. Phal. Great Toe	XA 3	Core Thyro
270	C2	L2	Occiput, Lateral	5th Metatarsal	B 58	Core Selenium
272	C3	T10	Maxillary, M-L	3rd Metatarsal	St 10	Core Calcium
276	C3	T6	Glabella	4th Metatarsal	Lv 6	Core Niacin
278	C4	L1	Parietal Bulge	Talus	Sp 10	Core Kidney
282	C4	L4	Occiput, Universal	Calcaneus	Cx 9	Core Carbo Gest
284	C5	T12	Ethmoid	1st Cuneiform	Tw 12	Spore-X
314	C1	T6	Temporal, Internal	Cuboid, Inferior	Tw 3	Core D-Tox
326	T1 R	C7	Occiput, Lateral	5th Metatarsal	Lv 7	Core Folic Acid
334	T2	T12	Mandible	Prox. Phal. Great Toe	Tw 16	Core Thyro
350	C6	L5	Parietal Bulge	Talus	St 37	Core Thiamine

Muscle Correlations: Total Accumulated Muscle Groups.
Patient Name - Last: x First:

Num	First Organ	Second Organ
052	Gallbladder Duct	Thyroid
270	Colon (A-D)	Eye
272	Thymus	Ear (Internal)
276	Lymph. of Submandibular	Larynx
278	Ovary/Testicle	Bladder
282	Lymph. of Submandibular	Gallbladder
284	Kidney	Urethra
314	Anterior Pituitary	Uterus/Prostate
326	Adrenal	Heart
334	Salivary Gland (Sublingual)	Uterus/Prostate
350	Colon (A-D)	Stomach (Fundus)

TABLE 1 - PATIENT NUMBER 7

MM Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: x First:

MM	Num Assoc	MM	Num Assoc
Cranial Nerve Total	1		
Cr VII	1		
Cervical Spine Total	8		
C1	1		
C2	1		
C3	2		
C4	2		
C5	1		
C6	1		
Thoracic Spine Total	2		
T1 R	1		
T2	1		

VL Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: x First:

VL	Num Assoc	VL	Num Assoc
Cervical Spine Total	1		
C7	1		
Thoracic Spine Total	6		
T6	2		
T9	1		
T10	1		
T12	2		
Lumbar Spine Total	4		
L1	1		
L2	1		
L4	1		
L5	1		

TABLE 1 - PATIENT NUMBER 7

Cranial Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: x First:

Cranial	Num Assoc	Cranial	Num Assoc
Ethmoid Total	1		
Glabella Total	1		
Mandible Total	1		
Maxillary Total	1		
Maxillary, M-L	1		
Occiput Total	3		
OCCIPUT, LATERAL**********	2		
Occiput, Universal	1		
Parietal Total	2		
PARIETAL, BULGE**********	2		
Styloid Total	1		
Temporal Total	1		
Temporal, Internal	1		

Foot Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: x First:

Foot Bone	Num Assoc	Foot Bone	Num Assoc
Calcaneus Total	1		
Cuboid Total	1		
Cuboid, Inferior	1		
Cuneiform Total	1		
1st Cuneiform	1		
Metatarsal Total	4		
3rd Metatarsal	1		
4th Metatarsal	1		
5TH METATARSAL**********	2		
Phalanges Total	2		
Dist. Phal. Great Toe	1		
Prox. Phal. Great Toe	1		
Talus Total	2		

TABLE 1 - PATIENT NUMBER 7

Organ Summary: Total Accumulated Muscle Groups. Part 1 of 2
Patient Name - Last: x First:

Organ	Num Assoc	Organ	Num Assoc
Adrenal Total	1	Larynx Total	1
Adrenal	1	Ovary-Testicle Total	1
Bladder Total	1	Ovary-Testicle	1
Colon Total	2	Pituitary Total	1
COLON (A-D)**********	2	Pituitary, Anterior	1
Ear Total	1	Salivary Gland (Sublingual) Total	1
Ear (Internal)	1	Stomach Total	1
Eye Total	1	Stomach (Fundus)	1
Eye	1	Thymus Total	1
Gallbladder_Total	2	Tissue Total	2
Gallbladder	1	Lymphatics Total	2
Gallbladder Duct	1	LYMPH* OF SUBMANDIBULAR********	2
Heart Total	1	Thyroid Total	1
Heart	1	Urethra Total	1
Kidney Total	1	Urethra	1

Organ Summary: Total Accumulated Muscle Groups. Part 2 of 2
Patient Name - Last: x First:

Organ	Num Assoc
Uterus-Prostate Total	2
UTERUS-PROSTATE**********	2

TABLE 1 - PATIENT NUMBER 7

MAP Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: x First:

MAP	Num Assoc	MAP	Num Assoc
Bladder Total	1	TW 16**********	1
B 58**********	1	XA 3 Total	1
Circulation - Sex Total	1		
CX 9**********	1		
Liver Total	2		
LV 6**********	1		
LV 7**********	1		
Spleen Total	1		
SP 10**********	1		
Stomach Total	2		
ST 10**********	1		
ST 37**********	1		
Triple Warmer Total	3		
TW 3**********	1		
TW 12**********	1		

Nutrients Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: x First:

Nutrients	Num Assoc	Nutrients	Num Assoc
Digestive (System - Enzymes) Total	1		
Spore-X	1		
Metabolism Total	2		
Core Carbo Gest	1		
Core D-Tox	1		
Mineral Total	2		
Core Calcium	1		
Core Selenium	1		
Glandular Total	3		
Core Kidney	1		
CORE THYRO**********	2		
Vitamin Total	3		
Core Folic Acid	1		
Core Niacin	1		
Core Thiamine	1		

TABLE 1 - PATIENT NUMBER 8

Muscle Correlations: Total Accumulated Muscle Groups.
Patient Name - Last: d First:

Num	MM	VL	Cranial	Foot	MAP	Nutrient
052	Cr VII	T9	Styloid	Dist. Phal. Great Toe	XA 3	Core Thyro
270	C2	L2	Occiput, Lateral	5th Metatarsal	B 58	Core Selenium
272	C3	T10	Maxillary, M-L	3rd Metatarsal	St 10	Core Calcium
274	C2	T8	Occiput, Lateral	5th Metatarsal	Cx 2	Core Selenium
282	C4	L4	Occiput, Universal	Calcaneus	Cx 9	Core Carbo Gest
308	C3	T4	Vomer	2nd Metatarsal	XL 3	Core Folic Acid
322	C2	C3	Temporal, External	Cuboid, Lateral	Tw 16	Core Potassium
326	T1 R	C7	Occiput, Lateral	5th Metatarsal	Lv 7	Core Folic Acid
332	C3	T4	Temporal, External	Cuboid, Lateral	K 9	Core Potassium
334	T2	T12	Mandible	Prox. Phal. Great Toe	Tw 16	Core Thyro
350	C6	L5	Parietal Bulge	Talus	St 37	Core Thiamine

Muscle Correlations: Total Accumulated Muscle Groups.
Patient Name - Last: d First:

Num	First Organ	Second Organ
052	Gallbladder Duct	Thyroid
270	Colon (A-D)	Eye
272	Thymus	Ear (Internal)
274	Pancreas (Sugar)	Gallbladder
282	Lymph. of Submandibular	Gallbladder
308	Ovary/Testicle	Jejunum
322	Anterior Pituitary	Pancreas (Protein)
326	Adrenal	Heart
332	Spleen	Vagina/Penis
334	Salivary Gland (Sublingual)	Uterus/Prostate
350	Colon (A-D)	Stomach (Fundus)

TABLE 1 - PATIENT NUMBER 8

MM Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: d First:

MM	Num Assoc	MM	Num Assoc
Cranial Nerve Total	1		
Cr VII	1		
Cervical Spine Total	8		
C2	3		
C3	3		
C4	1		
C6	1		
Thoracic Spine Total	2		
T1 R	1		
T2	1		

VL Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: d First:

VL	Num Assoc	VL	Num Assoc
Cervical Spine Total	2		
C3	1		
C7	1		
Thoracic Spine Total	6		
T4	2		
T8	1		
T9	1		
T10	1		
T12	1		
Lumbar Spine Total	3		
L2	1		
L4	1		
L5	1		

TABLE 1 - PATIENT NUMBER 8

Cranial Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: d First:

Cranial	Num Assoc	Cranial	Num Assoc
Mandible Total	1		
Maxillary Total	1		
Maxillary, M-L	1		
Occiput Total	4		
OCCIPUT, LATERAL**********	3		
Occiput, Universal	1		
Parietal Total	1		
Parietal, Bulge	1		
Styloid Total	1		
Temporal Total	2		
Temporal, External	2		
Vomer Total	1		

Foot Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: d First:

Foot Bone	Num Assoc	Foot Bone	Num Assoc
Calcaneus Total	1		
Cuboid Total	2		
Cuboid, Lateral	2		
Metatarsal Total	5		
2nd Metatarsal	1		
3rd Metatarsal	1		
5TH METATARSAL**********	3		
Phalanges Total	2		
Dist. Phal. Great Toe	1		
Prox. Phal. Great Toe	1		
Talus Total	1		

TABLE 1 - PATIENT NUMBER 8

Organ Summary: Total Accumulated Muscle Groups. Part 1 of 2
Patient Name - Last: d First:

Organ	Num Assoc	Organ	Num Assoc
Adrenal Total	1	Ovary-Testicle	1
Adrenal	1	Pancreas Total	2
Colon Total	2	Pancreas (Protein)	1
COLON (A-D)**********	2	Pancreas (Sugar)	1
Ear Total	1	Pituitary Total	1
Ear (Internal)	1	Pituitary, Anterior	1
Eye Total	1	Salivary Gland (Sublingual) Total	1
Eye	1	Spleen Total	1
Gallbladder Total	3	Stomach Total	1
GALLBLADDER**********	2	Stomach (Fundus)	1
Gallbladder Duct	1	Thymus Total	1
Heart Total	1	Tissue Total	1
Heart	1	Lymphatics Total	1
Jejunum Total	1	Lymph. of Submandibular	1
Ovary-Testicle Total	1	Thyroid Total	1

Organ Summary: Total Accumulated Muscle Groups. Part 2 of 2
Patient Name - Last: d First:

Organ	Num Assoc	Organ	Num Assoc
Uterus-Prostate Total	1		
Uterus-Prostate	1		
Vagina-Penis Total	1		

TABLE 1 - PATIENT NUMBER 8

MAP Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: d First:

MAP	Num Assoc	MAP	Num Assoc
Bladder Total	1	XL Total	1
B 58	1	XL 3	1
Circulation - Sex Total	2		
Cx 2	1		
Cx 9	1		
Kidney Total	1		
K 9	1		
Liver Total	1		
Lv 7	1		
Stomach Total	2		
St 10	1		
St 37	1		
Triple Warmer Total	2		
TW 16**********	2		
XA 3 Total	1		

Nutrients Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: d First:

Nutrients	Num Assoc
Metabolism Total	1
Core Carbo Gest	1
Mineral Total	5
Core Calcium	1
CORE POTASSIUM**********	2
CORE SELENIUM**********	2
Glandular Total	2
CORE THYRO**********	2
Vitamin Total	3
CORE FOLIC ACID**********	2
Core Thiamine	1

TABLE 1 - PATIENT NUMBER 9

Muscle Correlations: Total Accumulated Muscle Groups.
Patient Name - Last: x First:

Num	MM	VL	Cranial	Foot	MAP	Nutrient
052	Cr VII	T9	Styloid	Dist. Phal. Great Toe	XA 3	Core Thyro
270	C2	L2	Occiput, Lateral	5th Metatarsal	B 58	Core Selenium
272	C3	T10	Maxillary, M-L	3rd Metatarsal	St 10	Core Calcium
274	C2	T8	Occiput, Lateral	5th Metatarsal	Cx 2	Core Selenium
276	C3	T6	Glabella	4th Metatarsal	Lv 6	Core Niacin
278	C4	L1	Parietal Bulge	Talus	Sp 10	Core Kidney
282	C4	L4	Occiput, Universal	Calcaneus	Cx 9	Core Carbo Gest
284	C5	T12	Ethmoid	1st Cuneiform	Tw 12	Spore-X
314	C1	T6	Temporal, Internal	Cuboid, Inferior	Tw 3	Core D-Tox
350	C6	L5	Parietal Bulge	Talus	St 37	Core Thiamine

Muscle Correlations: Total Accumulated Muscle Groups.
Patient Name - Last: x First:

Num	First Organ	Second Organ
052	Gallbladder Duct	Thyroid
270	Colon (A-D)	Eye
272	Thymus	Ear (Internal)
274	Pancreas (Sugar)	Gallbladder
276	Lymph. of Submandibular	Larynx
278	Ovary/Testicle	Bladder
282	Lymph. of Submandibular	Gallbladder
284	Kidney	Urethra
314	Anterior Pituitary	Uterus/Prostate
350	Colon (A-D)	Stomach (Fundus)

TABLE 1 - PATIENT NUMBER 9

MM Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: First:

MM	Num Assoc	MM	Num Assoc
Cranial Nerve Total	1		
Cr VII	1		
Cervical Spine Total	7		
C1	1		
C2	1		
C3	2		
C4	2		
C5	1		

VL Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: First:

VL	Num Assoc	VL	Num Assoc
Thoracic Spine Total	5		
T6	2		
T9	1		
T10	1		
T12	1		
Lumbar Spine Total	3		
L1	1		
L2	1		
L4	1		

TABLE 1 - PATIENT NUMBER 9

Cranial Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: x First:

Cranial	Num Assoc	Cranial	Num Assoc
Ethmoid Total	1		
Glabella Total	1		
Maxillary Total	1		
Maxillary, M-L	1		
Occiput Total	3		
OCCIPUT, LATERAL**********	2		
Occiput, Universal	1		
Parietal Total	2		
PARIETAL, BULGE**********	2		
Styloid Total	1		
Temporal Total	1		
Temporal, Internal	1		

Foot Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: x First:

Foot Bone	Num Assoc	Foot Bone	Num Assoc
Calcaneus Total	1		
Cuboid Total	1		
Cuboid, Inferior	1		
Cuneiform Total	1		
1st Cuneiform	1		
Metatarsal Total	4		
3rd Metatarsal	1		
4th Metatarsal	1		
5TH METATARSAL**********	2		
Phalanges Total	1		
Dist. Phal. Great Toe	1		
Talus Total	2		

TABLE 1 - PATIENT NUMBER 9

Organ Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: x First:

Organ	Num Assoc	Organ	Num Assoc
Bladder Total	1	Pancreas (Sugar)	1
Colon Total	2	Pituitary Total	1
COLON (A-D)**********	2	Pituitary, Anterior	1
Ear Total	1	Stomach Total	1
Ear (Internal)	1	Stomach (Fundus)	1
Eye Total	1	Thymus Total	1
Eye	1	Tissue Total	2
Gallbladder Total	3	Lymphatics Total	2
GALLBLADDER**********	2	LYMPH* OF SUBMANDIBULAR********	2
Gallbladder Duct	1	Thyroid Total	1
Kidney Total	1	Urethra Total	1
Larynx Total	1	Urethra	1
Ovary-Testicle Total	1	Uterus-Prostate Total	1
Ovary-Testicle	1	Uterus-Prostate	1
Pancreas Total	1		

MAP Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: x First:

MAP	Num Assoc	MAP	Num Assoc
Bladder Total	1	XA 3 Total	1
B 58**********	1		
Circulation - Sex Total	2		
CX 2**********	1		
CX 9**********	1		
Liver Total	1		
LV 6**********	1		
Spleen Total	1		
SP 10**********	1		
Stomach Total	2		
ST 10**********	1		
ST 37**********	1		
Triple Warmer Total	2		
TW 3**********	1		
TW 12**********	1		

TABLE 1 - PATIENT NUMBER 9

Nutrients Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: x First:

Nutrients	Num Assoc	Nutrients	Num Assoc
Digestive (System - Enzymes) Total..	1		
Spore-X.....	1		
Metabolism Total.....	2		
Core Carbo Gest.....	1		
Core D-Tox.....	1		
Mineral Total.....	3		
Core Calcium.....	1		
CORE SELENIUM**********	2		
Glandular Total.....	2		
Core Kidney.....	1		
Core Thyro.....	1		
Vitamin Total.....	2		
Core Niacin.....	1		
Core Thiamine.....	1		

TABLE 1 - PATIENT NUMBER 10

Muscle Correlations: Total Accumulated Muscle Groups.
Patient Name - Last: x First:

Num	MM	VL	Cranial	Foot	MAP	Nutrient
052	Cr VII	T9	Styloid	Dist. Phal. Great Toe	XA 3	Core Thyro
308	C3	T4	Vomer	2nd Metatarsal	XL 3	Core Folic Acid
322	C2	C3	Temporal, External	Cuboid, Lateral	Tw 16	Core Potassium
326	T1 R	C7	Occiput, Lateral	5th Metatarsal	Lv 7	Core Folic Acid
332	C3	T4	Temporal, External	Cuboid, Lateral	K 9	Core Potassium
334	T2	T12	Mandible	Prox. Phal. Great Toe	Tw 16	Core Thyro
346	C2	T10	Occiput, Lateral	5th Metatarsal	G 40	Core Rutin
350	C6	L5	Parietal Bulge	Talus	St 37	Core Thiamine

Muscle Correlations: Total Accumulated Muscle Groups.
Patient Name - Last: x First:

Num	First Organ	Second Organ
052	Gallbladder Duct	Thyroid
308	Ovary/Testicle	Jejunum
322	Anterior Pituitary	Pancreas (Protein)
326	Adrenal	Heart
332	Spleen	Vagina/Penis
334	Salivary Gland (Sublingual)	Uterus/Prostate
346	Spleen	Lung
350	Colon (A-D)	Stomach (Fundus)

TABLE 1 - PATIENT NUMBER 10

MM Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: x First:

MM	Num Assoc	MM	Num Assoc
Cranial Nerve Total	1		
Cr VII	1		
Cervical Spine Total	5		
C2	2		
C3	2		
C6	1		
Thoracic Spine Total	2		
T1 R	1		
T2	1		

VL Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: x First:

VL	Num Assoc	VL	Num Assoc
Cervical Spine Total	2		
C3	1		
C7	1		
Thoracic Spine Total	5		
T4	2		
T9	1		
T10	1		
T12	1		
Lumbar Spine Total	1		
L5	1		

TABLE 1 - PATIENT NUMBER 10

Cranial Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: x First:

Cranial	Num Assoc	Cranial	Num Assoc
Mandible Total	1		
Occiput Total	2		
OCCIPUT, LATERAL**********	2		
Parietal Total	1		
Parietal, Bulge	1		
Styloid Total	1		
Temporal Total	2		
TEMPORAL, EXTERNAL**********	2		
Vomer Total	1		

Foot Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: x First:

Foot Bone	Num Assoc	Foot Bone	Num Assoc
Cuboid Total	2		
CUBOID, LATERAL**********	2		
Metatarsal Total	3		
2nd Metatarsal	1		
5TH METATARSAL**********	2		
Phalanges Total	2		
Dist. Phal. Great Toe	1		
Prox. Phal. Great Toe	1		
Talus Total	1		

TABLE 1 - PATIENT NUMBER 10

Organ Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: x First:

Organ	Num Assoc	Organ	Num Assoc
Adrenal Total	1	Pituitary Total	1
ADRENAL	1	PITUITARY, ANTERIOR	1
Colon Total	1	Salivary Gland (Sublingual) Total	1
COLON (A-D)	1	Spleen Total	2
Gallbladder Total	1	Stomach Total	1
GALLBLADDER DUCT	1	STOMACH (FUNDUS)	1
Heart Total	1	Thyroid Total	1
HEART	1	Uterus-Prostate Total	1
Jejunum Total	1	UTERUS-PROSTATE	1
Lung Total	1	Vagina-Penis Total	1
LUNG	1		
Ovary-Testicle Total	1		
OVARY-TESTICLE	1		
Pancreas Total	1		
PANCREAS (PROTEIN)	1		

MAP Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: x First:

MAP	Num Assoc
Gallbladder Total	1
G 40	1
Kidney Total	1
K 9	1
Liver Total	1
Lv 7	1
Stomach Total	1
St 37	1
Triple Warmer Total	2
TW 16	2
XA 3 Total	1
XL Total	1
XL 3	1

TABLE 1 - PATIENT NUMBER 10

Nutrients Summary: Total Accumulated Muscle Groups. Part 1 of 1
Patient Name - Last: x First:

Nutrients	Num Assoc	Nutrients	Num Assoc
Mineral Total	2		
CORE POTASSIUM	2		
Glandular Total	2		
CORE THYRO	2		
Vitamin Total	4		
CORE FOLIC ACID	2		
Core Rutin	1		
Core Thiamine	1		

Metabolic Aspects of Health - A Summary

Kenneth S. Feder

ABSTRACT:

The following paper is a summarization of the therapeutic uses of the nutritional elements discussed in the book entitled Metabolic Aspects of Health - Nutritional Elements in Health and Disease by Karl Schutte, PhD. and John A. Myers, M.D.

INTRODUCTION:

With the processing of our food, staleness, preservation with heat, and eventually cooking and sterilization, many of the active vitamins and minerals are inactivated and are no longer available to the cell; and so, in varying amounts and at varying times, the respiratory activity of the cell is jeopardized and leads to chronic disease.

DISCUSSION:

The following nutritional elements and information may be helpful in the treatment of various illnesses:

ANTI-ATHEROGENIC AGENT - Iodine fraction in ovary.
ANTIHYPERTENSIVE ELEMENT - Manganese.
ATHLETE'S FOOT - May be helped by Zinc and Cobalt.
BERI-BERI SYMPTOMS - B1 deficiency (also check for Zinc deficiency).
BONE HEALING - Calcium accelerates healing; also influenced by Magnesium, Iodine, and Silicon.
BONE AND TEETH INTEGRITY - Helped by Vandium and Strontium
BRAIN DEVELOPMENT - Influenced by Vanadium, Silver, Manganese, Iodine, Cobalt, Potassium, Magnesium, Copper, Zinc, and Molybdenum.
CHOLESTEROL SYNTHESIS (INCREASED) - Aided by Manganese and Chromium.
CHOLESTEROL SYNTHESIS (DECREASED) - Influenced by Vanadium and Iron (Vanadium inhibits utilization of Mevalonic Acid in cholesterol synthesis and has anti-athero-sclerotic effect).

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COLOR VISION STABILITY - Silver makes colors more vivid and aids in light sensitivity. Adequate amounts of Tyrosine, Tryptophane, and Vitamin A are also necessary.

COPPER DEFICIENCY ANEMIA - Morphologically indistinguishible from Iron Deficiency Anemia.

DEGENERATION OF NERVOUS SYSTEM - Check for Copper deficiency.

DENTAL INTEGRITY - Maintained by Copper, Zinc, Magnesium, Manganese and Chloride.

DENTAL PLAQUE - When the surface of the tooth is dry and there is precipitation of plaque, there is need for Iodine.

DIABETES MELLITUS - Check for Zinc deficiency.

THE WRONG VITAMIN E FORM (Alpha Tocopherol Acetate) - May produce depression in the hypothyroid patient; may also produce cramp in calf muscle.

VITAMIN E DEFICIENCY - Helped by Cobalt.

EYBALL DRYNESS - Cobalt helps eyeballs to feel more at ease and allows eyeball mucous to be more lubricating.

EYEBALL DRYNESS AND TEAR FORMATION - Helped by Cobalt and Copper.

IRRITATED EYES - Cobalt may help red, irritated corners and lid margins.

FETAL DEVELOPMENT - Vitamin E

RECEDING GINGIVAL MARGIN - Helped by methionine, Magnesium, and Zinc.

GUM AND TOOTH PAIN - Helped by Magnesium Chloride, Zinc Iodide, and Methionine.

HERPES SIMPLEX - May be helped by Cobalt (Molybdenum may precipate a fever blister).

IODINE UPTAKE IN THYRONINE FORMATION - Must check for adequate Copper.

IRON DEFICIENCY (SECONDARY ANEMIA) SYMPTOMS - Inflammation of tongue, inflammation of corners of mouth, spoon-shaped nails -- Helped by Ferrous Sulphate.

LIGHT SENSITIVITY - Cobalt, Copper, and Iodine reduce light sensitivity.

LIPOTROPHIC AGENTS - Thyroglobulin, Thyronine, Diiodotyrosine, Triiodotyrosine (the hormones of the thyroid gland) are known to be the

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most effective lipotrophic agents; all of these thyroid functions, with the exception of diiocotyrosine, contain Tyrotine which is also a potent fat mobilizer. The diiodotyrpsonine fraction, being free of Thyroxine, is effective in reducing the liver fat and the cholesterol in the blood plasma. The diiodotyrosine fraction has been proven to be non-toxic when given in large doses and for prolonged periods of time.

MUCOUS MEMBRANE ULCERATIONS - May be helped by Cobalt.

NAIL AND HAIR SUPPORT - Copper and Cobalt support keratin formation.

NAIL GROWTH - Helped by Cobalt and Copper.

NASAL PHYSIOLOGY - Improved function with Thiamin, Iodine, Vitamin A, Vitamin E Tryptophane, and Manganese.

NOSE BLEEDS - Cobalt helps prevent nose bleeds by improving the turgency of turbinates and by strengthening the integrity of blood vessels.

OSTEOPOROSIS - Check for Manganese and Copper deficiency.

PAIN IN AREA OF PANCREAS (Left side and back) - May be helped by Cobalt.

PAIN IN LEFT UPPER BACK AND EXTENDING TO THE LEFT SHOULDER AND NECK, DOWN THE LEFT ARM INTO THE HAND ASSOCIATED WITH NUMBNESS AND TINGLING OF THE EXTEMITY; THE PAIN MAY FOLLOW THE FACIAL NERVE INTO THE LEFT SIDE OF THE FACE AND PRODUCE SORENESS IN LEFT UPPER AND LOWER MOLAR TEETH - Condition may be helped by Iodine and Silver. Symptoms may be made worse by use of Sulfur compounds, particularly Cystine and Methionine.

PERNICIOUS ANEMIA - May be helped by Silver (it is the intrinsic factor in pernicious anemia), while Cobalt is the intrinsic factor.

POSTOPERATIVE HEALING FROM SURGERY - Improved by Vitamins A, C, B6, E, Unsaturated Fatty Acids, Magnesium, Zinc, and Iodine.

PREGNANCY (DIFFICULTY IN CONCEIVING) - Check for B12 deficiency.

PREGNANCY DEBILITATION - Helped by Zinc, Cobalt, and Copper.

PULSE IRREGULARITY - May be helped by Cobalt. (May also need Zinc, Copper, Molybdenum, Manganese, Vanidum, Iodine, Potassium, Sodium, Calcium,and Magnesium).

RBC FORMATION - Affected by Vanadium, Cobalt, and Copper deficiency.

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RED COLOR PERCEPTION - Helped by Cobalt, Copper, and Iodine.
SHINGLES AND HERPES SIMPLEX - Magnesium Chloride (intravenously) and Pyridoxine and Cobalt (by mouth) may aid healing .
SKIN, HAIR & NAIL SUPPORT - Cobalt helps them become stronger and tougher.
STERILITY - Must check for Vitamin E deficiency.
STIES - Helped by Zinc Iodide and Cobalt.
THYROID FUNCTION - Influenced by Magnesium, Zinc, Copper, Cobalt and Iodine.
THYROXINE LEVELS - Influenced by Copper level.
THYROID METABOLISM - Helped by Cobalt.
THYROTOXICOSIS - B12 prevents toxic effects of excess thyroxine. First signs of thyrotoxicosis may be B12 deficiency.
VEIN PATENCY - Cobalt helps flat veins regain their turgency.
WARTS AND "OLD AGE" BLEMISHES - May be helped by Cobalt.
WRINKLES AT MOUTH CORNERS - Helped by Cobalt.

CONCLUSION:

The above material was presented as an aid in the use of various trace minerals and vitamins and was strictly based on the research obtained in the book entitled Metabolic Aspects of Health.

REFERENCES:

1. Schutte, K.H., and Myers, V.A. Metabolic Aspects of Health - Nutritional Elements in Health and Disease, Discovery Press, 1979.

THE CASE FOR SELENIUM DEFICIENCY PROMOTING OXIDATIVE STRESS VIA DYSFUNCTION OF THE GLUTATHIONE CONJUGATING SYSTEM

Timothy D. Francis, D.C., DIBAK

ABSTRACT

Glutathione possesses a high turnover rate in most tissues; and is a cosubstrate for glutathione peroxidase which is selenium dependent. Glutathione plays an important role in the protection of cell constituents against oxidative damage. Selenium is known to be an integral part of glutathione peroxidase; which is responsible for protection against hydrogen and organic peroxides. Without adequate selenium, glutathione peroxidase (GPX) production is hindered, leading to cellular oxidation/reduction imbalances.

I. INTRODUCTION

Glutathione (GSH) is a tripeptide composed of cysteine, glutamic acid, and glycine. (21) Glutathione is important in transport of amino acids across cell membranes, catalyzing disulfide exchange reactions, maintaining SH-groups in proteins, and quenching hydroperoxides and organic peroxides including free radicals formed as reactive toxic intermediates during oxidative metabolism. (1)(99)

Humans as well as other organisms are creatures of dynamic balance. Life at all evolutionary levels maintain a certain constancy of the internal environment and that this constancy (homeostasis) is necessary to the life of the cells and there-fore to the life of the total organism. (2) If a cell becomes overoxidized or underreduced, cellular chemistry will go awry, therefore prooxidant and antioxidant substances should maintain a balance.(78) Since the human body may be thought of as a composite of it's cells (of which there are 100,000 trillion), then it becomes of utmost importance to balance this reduction/oxidation at a cellular level in order to achieve and maintain homeostasis throughout the body.

Glutathione peroxidase (GPX) is the key to understanding the role of GSH:GSSG (reduced glutathione: oxidized glutathione) reactions and the pathology of GSH related deficiency syndromes.(51) Selenium affects the rate of synthesis of GPX protein and selenium deficiency results in a decrease in GPX activity. GPX is the only well-characterized selenoprotein,(37) therefore GPX activity reflects selenium status in deficient and adequate states. (27)(28)(38)

Hydroperoxides alter intracellular thiol homeostasis.(65) Lipid Peroxidation seems to be the primary toxic effect of super oxide radical (SOR) attacks.(57) GPX protects membranes from peroxidation by preventing the initiation of a chain reaction by catalyzing the reduction of hydrogen peroxide to water and corresponding alcohols, blocking lipid peroxidation with high capacity.(72)

This ability to metabolize hydrogen peroxide via the GSH conjugating system is curtailed in selenium deficient red cells and polymorphonuclear leukocytes (PMN). The inability to reduce hydrogen peroxide is corrected upon supplementation with selenium,(35) and selenium deficient animals have been shown to be highly susceptible to injury by oxidant stress.(85)

II. OXIDATIVE STRESS

Oxidation is defined as the loss of an electron.(20)(21)(22) Within biological systems, everytime a molecule is oxidized, there occurs a simultaneous reduction (gain of an electron) in another molecular species. (16) This redox coupling occurs in each and every cell in the body. The free energy exchange is proportionate to the capability of the reactants to donate or accept electrons.(20)(21) The reducing agent is defined as an electron donor, while the oxidizing agent is defined as an electron acceptor.(20)(21)(22) These electron tranfers are essential for cellular chemistry.

Aerobic metabolism as we know it today is a product of evolution. Oxygen is both necessary for life but is also responsible for cell death under acute oxidative stress. The GSH conjugating system is a major defense against oxidative stress.(9) Due to oxygen's lack of octet fulfillment in it's outer orbital, diatomic molecular oxygen (O_2) will preferentially accept an electron to form a highly reactive superoxide free radical (SOR). (16)(18) A free radical is any molecular species with an odd number of electrons.(17) SOR's are produced from electron leaks via the mitochondrial electron transport system and the NADP oxidase system from the surface of PMN's and tissue histiocytes.(16) SOR formation is also involved in autoxidation of hydroquinones, thiols, flavins, catecholamines, tetrahydropterins, ferrodoxins, dialuric acid and hemoglobin.(18)

SOR's may then react with another SOR or an electron, which in the presence of superoxide dismutase (SOD) forms hydrogen peroxide (H_2O_2). (16)(18) SOD is found in both the mitochondrion and cytoplasm, and contains either copper, zinc, or manganese. (18) Hydrogen peroxide may be acted upon by either catalase or GPX to form water; or it may react with another SOR in the presence of ferric ion (Fe^{3+}) to decompose via the Haber-Weiss reaction to form hydroxyl radicals.(16)(18) (See Fig. 1) Catalysis by iron and other transition metals such as copper allows the Haber-Weiss reaction to proceed at a much faster rate via the much debated Fenton reaction.(5)

$$O_2^- + Fe^{3+} \text{ ---------- } Fe^{2+} + O_2$$

$$O_2^- + O_2H. \text{ ---------- } O_2 + H_2O_2$$

$$Fe^{2+} + H_2O_2 \text{ --------- } Fe^{3+} + OH + OH^-$$

It has been found in a study by Westermarck and Santavouri that selemium inhibits the toxic effect of iron.(19)

Excess serum and tissue levels of transition metals not involved in normal redox couples or active enzyme sites use their free d-orbitals to catalyze one electron reactions when in close proximity to mitochondrial respiratory chain leakage.(6) Free radical reactions are initiated continuously throughout cells from enzymatic reactions such as phagocytosis, respiratory chain propagation, and the cytochrome P-450 system as well as non-enzymatic reactions involving oxygen reacting with organic compounds and ionizing radiation.(8) The cell's efforts are directed at prevention of the hydroxyl radical.(16) GSH is a free radical inhibitor and is a co-substrate for GPX which keeps the cellular concentration of hydrogen and organic peroxides low which inhibits the formation of the more toxic hydroxyl radical. (8)(16)

Some of the most unstable organic molecules are phospholipids with unsaturated fatty acid side chains.(4) These phospholipids form cellular membranes. Oxidation of unsaturated fatty acids may predispose this lipid bilayer to degradation, cause enzyme deactivation, and may have teratogenic effects on cell division.(4)

Lipoperoxidation is a chain reaction and once initiated is self-propagating.(4)(5) Chain propagation and branching involves non-heme iron. Chain termination entails a reaction between a radical and an antioxidant, a metal, and/or a reaction between two radicals leading the chain.(4) In addition to attacking membranes, free radicals may also undergo an addition reaction to produce an alcohol or scission and cross-linking reactions which increase cullular rigidity.(18)

The peroxidation process is greatly influenced by lipid bilayer fluidity. Lipoperoxidation increases membrane inelasticity and negative surface charge with an increase in nonspecific permeability and therefore electrical instability.(4)(5) The change in microviscosity inactivates membrane bound enzymes and oxidizes thiols. These enzyme reaction kinetics if altered may lead to cell death or at the minimum disorganization.(4)

Within the mitochondria, increased oxidative stress in the form of peroxidation brings about the phenomena of swelling, uncoupling of oxidative phosphorylation due to an enhancement in membrane permeability and loss of cytrochrome c. Increasing electrical instability predisposes to respiratory chain inhibition. (See Fig. 2)

If the complex mechanism of lipid peroxidation is unbalanced with depletion of regulatory mechanisms such as thiols (GSH conjugating system) or lipid antioxidants (GPX and Vitamin E) then the detrimental effects such as enhancement of membrane permeability, an increase in negative surface electrical potential and enzyme deactivation will predispose the cell to pathology. This pathology may cause the release of lysosomal enzymes, activation of phospholipases, and destruction of tocopherols, steroids, and thyroxine. Other effects may include cell motility inhibition and cell division degeneration.(4)

The initial free radical formation produces local effects, however the secondary radicals and their products may exert effects distant to the site of the original reaction.(16) These oxidation induced changes include an alteration in collagen, elastin, and mucopolysaccharide degradation, chromosome damage, accumulation of ceroid and arteriolocapillary fibrosis.(8)

Lipid peroxides inhibit prostacyclin synthetase. Prostacyclin inhibits platelet aggregation, which is a major event leading to atherosclerosis. Free radicals are involved in reactions leading to neuritic plaques associated with senile dementia as well as lipofuscin accumulation.(8) Lipid proxidation cross-links proteins and affects all aspects of cell organization.(13)

Lipid peroxidation is a problem for all tissue, but especially so for the adrenal medulla and cortex due to their high oxygenase content.(15) Hans Selye, M.D., author of the renowned book <u>The Stress of Life</u>, states that there is a triad of stress. This triad involves adrenal cortex enlargement, followed by atrophy of thymus, spleen and lymphatic structures, and if prolonged, gastric and duodenal ulcers. This stress may be categorized into physical, chemical, mental, and thermal forms.(23) The adrenals possess a high concentration of glutathione and

selenium with elevated GPX activity. If adrenocortical cells become antioxidant deficient, adequate protection is not possible until selenium is added.(15)

The second stage is atrophy of immune function. T suppressor cells maintain tolerance to autoantigens. Free radicals depress T suppressor cell function. This may explain the increase in autoimmune disorders with age.(8) Numerous studies implicate oxygen radicals and lipid peroxidation in various illnesses such as cancer, multiple sclerosis, and Parkinson's disease.(10)

"Aerobic life has evolved an involved system of protection via antioxidants and enzymes to prevent damage from oxidation."(16)(24) Selenium is a component of glutathione peroxidase which decreases free radicals by reducing hydrogen peroxide to water.(8)(10) It has been demonstrated that glutathione peroxidase activity is proportional to the log of selenium concentration in the diet.(18)

Diet affects the concentration of cholesterol and polyunsaturated fatty acids in red cell membranes, thereby affecting the susceptibility of erythrocytes to peroxidation. Adequate GPX levels inhibit damage by free radicals present in the cardiovascular system. "The incidence of cardiovascular disease is low in areas where selenium intake is high and vice versa."(8)

Humans utilize an enzymatic defense mechanism (SOD and GPK) as a first line defense against free radicals such as SOR and H_2O_2 respectively. However, singlet oxygen and the hydroxyl radical require a second line of defense. The most important constituents are ascorbate and the tocopherols.

Ascorbate is an effective scavenger of the hydroxyl radical. However, ascorbic acid may act as a prooxidant as well as an antioxidant.(19) When ascorbate quenches a hydroxyl radical, it in turn becomes oxidized to ascorbate free radical, which may react with another hydroxyl radical to become oxidized to dehydroascorbate. GSH reacts directly with dehydroascorbate to produce ascorbic acid, preparing it for future radical quenching and providing an effective recycling system for ascorbate. (See Fig. 3)

Tocopherols as a whole reduce singlet oxygen and hydroxyl radicals in the hydrophobic layer of the cell membrane.(24) These tocopherols in turn become oxidized to quinones. Glutathione reduces the tocopherol radical which recycles Vitamin E. (See Fig. 4)

In addition to intracellular oxidation protection, tissues are protected against free radicals by extracellular antioxidant systems.(5) Tocopherols are the major fat soluble antioxidants circulating in blood plasma where they provide protection for the hydrophobic segments of proteins and lipids.(24) On the other hand, ascorbic acid which is hydrophilic, acts as an oxidant scavenger extracellularly in the lung as well as in other vital areas such as the adrenal glands, brain, spleen, eye lens, and gastrointestinal tracts.(18)(19)(24) GPX is also found in plasma and has been shown to be selenium dependent.(45)(55)(61)

"There is an optimal redox balance for every organism, between tendencies to donate or accept electrons."(24) A substantial shift in reduction-oxidation potential due to oxidant stress can affect the viability of cells and tissues. Without adequate available reducing

equivalents, oxidized forms of ascorbic acid and Vitamin E cannot become readily reduced. Glutathione is an extremely versatile conjugating agent which can react with a wide variety of reactive electrophiles to avert their potential toxicities.(24)

III. THE GLUTATHIONE CONJUGATING SYSTEM

Disorders in GSH synthesis are known which promote oxidation and cause membrane lysis.(47) The reactions of the gammaglutamyl cycle occurring intracellularly are responsible for the synthesis and degradation of reduced glutathione.(98)(99)

Glutathione production occurs in two steps. The first step involves combining glutamate with cysteine catalyzed by gammaglutamyl-cysteine synthetase. The second step is the addition of glycine by glutathione synthetase. Both steps require potassium and magnesium as cofactors.(3)(47)(98)(99) Glutathione has two characteristic structural features, a sulfydryl group and a gamma-glutamyl linkage.(99) (See Fig. 5)

GSH is synthesized by all tissues.(103) The rate of glutathione synthesis is significantly influenced by the intracellular concentration of cysteine.(99) Selenium deplete cells maintain a higher concentration of intracellular GSH which reflects a greater utilization of cysteine for GSH synthesis in selenium deficiency.(103) Cysteine is a non-essential sulfur containing amino acid which is formed from methionine and serice.(20) Methionine supports GSH synthesis through its conversion to cysteine via the transulfuration pathway.(103) Serine is the carbon skeleton source for selenocysteine in GPX.(37)

Glutamate is the only amino acid in mammalian tissues which undergoes oxidative deamination. These reactions are very important since ammonia is toxic, elevation of it's concentration may produce coma. (20)(104) Synthesis of glutamate from the protein pool is derived from alphaketoglutarate, ornithine, histidine, proline, and lysine. Alphaketoglutarate is a product of the citric acid cycle. Arginine produces ornithine and proline via the urea cycle.

The simplest in structure and the only optically nonactive amino acid is glycine. Glycine is derived from serine, glutamate, alanine, threonine, and choline.

Cysteine, glutamate, and glycine are non-essential amino acids. Their biosynthesis from the body's protein pool requires an adequate intake and absorption of protein, cofactors, and coenzymes. A deficiency of any vitamin or mineral cofactor will produce a limited availability of the amino acid which is dependent upon that nutrient. The mixtures of amino acids generated by digestion via the diet usually are not adequate in the proportions required by the body. It is necessary then to rearrange them metabolically which the body can accomplish unless the diet is lacking some essential nutrient.(104)

Reduced glutathione plays a major role against oxidation of cell organelles, it conjugates electrophilic compounds and reduces hydrogen peroxide and lipid hydroperoxides.(47) GSH regulates its own synthesis by non-allosteric feedback inhibition of gamma-glutamyl-cysteine synthetase.(99) The ratio of recuded to oxidized glutathione in most cells is greater than 500.(3)

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It is the pentose phosphate shunt that provides NADPH for the reduction of GSSG to GSH. This reaction is catalyzed by GSH reductase which is a flavin dependent enzyme. Deficiencies of riboflavin and glutathione reductase are a common finding. GSH removes hydrogen peroxide catalyzed by selenium dependent GPX. This reaction is important since the overall accumulation of hydrogen peroxide may decrease the life span of the red cell by increasing the oxidation of hemoglobin to met-hemoglobin. Lysis then occurs when the cell is exposed to oxidant stressors.(20) Glutathione is effective in protecting hemoglobin from oxidation only if selenium status is adequate.(41)

Released glutathione is carried to the kidney; there it is degraded to glutamate, cysteine, and glycine.(102) Extracellular conversion of GSH into GSSG requires oxygen and leads to formation of hydrogen peroxide. Approximately 10 to 15% of plasma glutathione is in the oxidized form. The hydrogen peroxide form serves as a protective mechanism by destroying microorganisms.(98)

Glutathione serves as cosubstrate for the glutathione peroxidases.(47)

$$2GSH + R\text{-}O\text{-}OH \xrightarrow{GPX} GSSG + H_2O + ROH$$

GPX has a molecular weight of 80,000 daltons with covalently attached selenium atoms.(3)(94) Selenium was found to be present in GPX from all sources at four atoms per mole of protein.(55) Selenium is incorporated through a pyridoxine dependent modification of the polypeptides.(72)

GPX's active site contains selenium ion which has replaced sulfur. The selenosulfide oxidizes glutathione and becomes reduced to selenolate. This selenolate ion reduces hydrogen and organic peroxides to their corresponding alcohols; and in turn becomes oxidized to selenenic acid, becoming oxidized and forming selenosulfide which restarts the cycle.(3) (See Fig. 6)

Selenium is incorporated into GPX and into very few other proteins.(20)(61) The metabolically active form of selenium seems to be as selenocysteine.(55) Since all cells require GPX for their protec-

tion, a decrease in it's production and activity is considered a detriment to cell function.(61) In a selenium deficient state, the metabolism of hydrogen peroxide via GPX is decreased, resulting in increased susceptibility of cellular functions to oxidative stress.(45)

Free radicals are toxic to cells and cause tissue damage by oxidizing DNA, proteins, and lipids. Intracellular defense mechanisms include superoxide dismutase, catalase (more important in prokaryotic cells), glutathione peroxidase and Vitamins C and E. However, phagocytic and endothelial cells produce extracellular superoxide radicals. Plasma peroxidase activity is attributed exclusively to GPX.(61)

Plasma GPX is similar to intracellular GPX in amino acid composition and selenium content, and is synthesized and secreted from hepatocytes.(61) The amino acids of GPX from all sources is derived primarily from aspartate, glutamate, proline, glycine, alanine and leucine. The majority of plasma GSH is metabolized by the kidneys and lung.(70) Low serum selenium is commonly seen in patients with chronic renal failure.(93)

GPX is responsible for 47% of selenium in plasma and 57% in the erythrocytes.(45) Experimental and clinical selenium deficiency result in a decrease in cellular and plasma GPX activity. Selenium supplementation of patients who are deficient increases GPX activity in both cells and plasma.(45) GPX activity returns to normal in two to four weeks in plasma whereas it takes three to four months for red cell GPX activity to return to normal. All of the red cells and a majority of platelet and granulocyte GPX activity is selenium dependent.(45)

Recently a selenium independent GPX has been identified but has much less reactivity towards hydrogen peroxide. This has been named glutathione-transferase. Both selenium dependent and selenium independent GPX function to protect the cell against oxidation.(47) However it has been demonstrated that selenium deficient animals have greatly reduced GPX activity.(45)(72)(98)

IV. SELENIUM

Selenium is about as rare as gold. The name is derived from the Greek word selene, meaning the moon. It occurs in three different allotropic forms; monoclinic and amorphous selenium are red in color, the third is a bluish grey consisting of hexagonal crystals.(100) Schwartz and Foltz discovered selenium is an essential trace element in mammals in 1957.(102)

Selenium closely resembles sulfur in physical and chemical properties; selenates are similar to sulfates and selenites to sulfites.(100) Selenium exists naturally in food as selenomethionine, selenocystine, and selenocysteine.(28) However it is selenomethionine that is more rapidly incorporated into cells. There are three forms of selenium present in plasma: GPX, selenoprotein P, and selenomethionine.(85) Methionine supplementation increases red cell CPX activity in selenium deficiency, but it is selenocysteine that is the ultimate metabolic form of selenium.(95)

The absorption of selenium depends on the solubility of the selenium compound, gastric output, and the ratio of sulfur to selenium in the diet.(81)(100) Selenium has four natural oxidation states, -2,

0, +4, and +6. Selenite (+4) slowly oxidizes in an alkaline medium in the presence of oxygen to selenate (+6).(93) The +6 state is the most common form of selenium in inorganic salts, however sodium selenite (+4) is more effective than sodium selenate (+6) for invitro synthesis of GPX.(36)(94) All forms of selenium supplementation appear to be somewhat effective in raising plasma selenium levels.(93)

Selenium is absorbed in the small intestine and regulated via a homeostatic mechanism.(97) Therefore disease that affects the small intestine such as Crohn's Disease and non-tropical sprue may be expected to cause selenium deficiency.(93) Antagonists, such as zinc and flouride may also compete with selenium.(40) The important forms of selenium are sodium selenite, selenious acid, sodium selenate, and the organic forms selenomethionine and cysteine for prevention and treatment of deficiency.(93) Selenomethionine competes for absorption with methionine.(100) Selenate and selenomethionine were better absorbed than as selenite.(83) Selenomethionine has an absorption of approximately 96% with a half life of about 245 days, whereas sodium selenite has an absorption of approximately 60% with a half life of about 100 days.(77)

The National Academy of Science, Food, and Nutrition Board recommends a daily intake of selenium in the range of 50-200 micrograms/day for adults, and the selenium urinary level should be below 100 micrograms/liter.(94) Selenium levels double from early infancy to adult life with similar changes occuring in plasma GPX activity.(93)

Selenium is deposited in all tissues, with especially high concentrations in the liver, kidney, and heart.(100) In dietary selenium deficiency, the reticuloendothelial tissues selectively retain it. These tissues include the spleen, lymph nodes, and thymus.(24) It has been demonstrated that selenium affects immunity.(93)

Selenium is also increased in the brain, pituitary, thyroid, adrenals, and gonads. This strongly implicates a metabolic role for selenium.(93) During biosynthesis of thyroxine, iodine is oxidized by a peroxidase using hydrogen peroxide as a substrate. In selenium deficiency, lower GPX activity in thyroid allows an increase of hydrogen peroxide, increasing the synthesis of thyroxine. A significant negative correlation was found between selenium and T_3, T_4 levels in hyperthyroid patients whose plasma selenium was lower than controls.(93)

Selenium and Vitamin E are synergistic, therefore this presents a complicating factor in establishing nutritional requirements for both. Selenium spares alpha-tocopherols in that normal pancreatic function requires selenium for digestion and absorption of fats, and aids in the retention of Vitamin E in blood plasma lipoproteins. As a component of GPX, converts peroxides to alcohols, saving the polyunsaturated fatty acids of lipid membranes from oxidation which reduces the Vitamin E requirement for maintenance of membrane integrity.(20) On the other hand, low selenium levels are less likely to result in clinical deficiency in populations which are well-nourished in other nutrients such as Vitamins E and C.(93)

Selenium is excreted primarily in the urine, although significant amounts are also released via the feces, perspiration, and exhaled in air via the lungs.(81)(93)(100) During selenium toxicity, dimethylselenide is excreted via the lungs giving rise to the pathognomonic sign of garlic breath.(20)(90)(100) The methyl donor here is methionine which helps to reduce toxicity to the respiratory tract.(20)(93)

This bimodal effect of selenium has given it the reputation of being one of the most toxic elements. One mechanism by which selenium exerts it's toxic effects is by competing with sulfur for active sites in which sulfur plays vital metabolic roles. In toxic doses selenium also inhibits the activity of succinic dehydrogenase, choline oxidase and proline oxidase.(100) Some of the more common signs and symptoms include pallor, lassitude, irritability, and indigestion.(94) Endemic selenosis in China has also been characterized by a loss of hair and nails as well as impairing reproduction.(98) Excess selenium ingestion during embryonic development has disastrous effects with the greatest disturbance in bone and cartilage formation.(100) In the lens of the eye, a deficiency or excess of selenium may cause cataract formation.(93)

V. SELENIUM, GPX, AND OXIDATIVE STRESS

The availability of selenium regulates the availability of GPX and therefore the ability to adapt to oxidative stress.(24) The toxic derivatives which the antioxidants must neutralize are produced by normal cellular metabolic activity, drugs, toxins, and radiation.(93)

Erythrocytes in the presence of reduced glutathione could prevent the oxidative denaturation of hemoglobin and red cell lysis induced by hydrogen peroxide.(94) In two groups fed peroxides, those supplemented with selenium showed no increase in GPX activity above basal level whereas the second group not supplemented demonstrated selectively increased GPX activity. This indicates that the supplemented groups were capable of detoxifying the peroxides without a need for adaptive augmentation of GPX in certain tissues. The non-supplemented group were unable to completely detoxify the peroxides and suffered toxic effects.(24) Only red cells synthesized in the presence of selenium contain GPX activity.(37)

Cell damage following exposure to ionizing radiation is mediated by hydrogen peroxide and organic peroxides. These reactive molecules may denature cellular components and mediate injury via oxidation. These injuries are expressed by cell dysfunction and/or extinction.(60) The extent of toxicity is related to the amount of depletion of GSH and protein thiols.(68) GPX was found to be more effective against oxidant damage produced by free radicals than either catalase or SOD.(57)

GPX activity was found to be extremely dependent on dietary intake of selenium and offers a very accurate physiological index of selenium status.(25)(32)(41)(46) However, selenium deficiency presents a special problem due to a lack of signs and symptoms unless

the diet is also deficient in Vitamin E and the sulfur-containing amino acids, or the body is exposed to oxidant stressors.(41) Numerous studies suggest that a deficiency in selenium-Vitamin E are responsible for oxidant stress in the body.(9)(13)(14)(19)(51)(57)

It has been found that high oxygen pressure induces toxicity via hydrogen peroxide and not superoxide radicals. SOD is unable to protect cells in a free radical generating system.(57) GPX activity increases in a variety of tissues due to oxidant stressors such as exposure to ozone, Vitamin E deficiency, exposure to peroxidized lipids, ethanol, and various disease states.(41)(43)

VI. DISEASE STATES ASSOCIATED WITH OXIDATIVE STRESS RELATED TO SELENIUM DEFICIENCY AND DYSFUNCTION OF THE GSH CONJUGATING SYSTEM

Structural damage to liver cells may result from peroxidation of lipids in cellular membranes by free radicals. Selenium deficiency is associated with hepatic necrosis and a decrease in platelet GPX activity.(78)(93) Supplementation with selenium to restore GPX activity prevents liver necrosis.

The liver is an important storage organ for selenium and also has the highest GPX activity.(78) It has been demonstrated that selenium prevents liver necrosis in Vitamin E deficient states.(93) Hepatic selenium was low in chronic alcoholics with cirrhosis and in patients with liver disease of non-alcoholic cause.(93) Liver GPX activity responds rapidly to increased selenium intake.(39)

A reduction in GPX activity due to selenium deficiency induces an elevation in tissue lipid peroxide concentration. Selenium regulates prostacyclin synthesis via GPX activity influences the function of various enzymes of the arochidonic acid pathway. A selenium deficient animal during periods of vascular stress has a suppressed endothelial response to platelet activation which predisposes the blood vessels to injury during inflammatory and thrombotic episodes.(75) A deficiency in platelets accelerates thrombotic tendencies which induces fibrosis of the pancreas associated with chronic pancreatitis.(79) Supplementation of humans with selenium increased GPX activity in platelets.(75) Platelets may preferentially retain selenium in deficiency and therefore GPX activity in platelets is a good indicator of selenium status.(73)(83)

Low selenium with resulting low GPX activity may lead to decreased reduction of hydroperoxides and increased accumulation of lipid peroxides with damage to arterial endothelium. Low cellular selenium alters metabolism of thrombocyte prostaglandins and increases platelet aggregability with resulting tendency to thrombosis.(93) Hypertensives have demonstrated lower levels of reduced glutathione and GPX activity and prostacyclin production were increased when selenium was added to the culture medium of pig aortic endothelial cells. Plasma selenium was found to be inversely correlated with the severity of coronary atherosclerosis and with the resultant risk of myocardial infarction.(93)

The status of reduced glutathione in a cell reflects its ability to protect against oxidative damage.(48) GPX activity is low in sickled erythrocytes, leading to enhanced levels of hydrogen peroxide, malondialdehyde, and reduced unsaturated fatty acids.(29)(44) Malondialdehyde is a marker of lipid peroxidation. When there is an increase, this suggests oxidative damage to cells.(49)(57) Membrane lipids are major targets for cellular damage induced by oxygen radicals.(54) Red cells of patients suffering from favism with acute hemolytic crisis have elevated levels of SOD with much lower GPX activity.(53) GPX metabolizes either hydrogen peroxide or lipid peroxides, and thereby provides protection against oxidative damage to red cell membranes which is a crucial event in the onset of hemolysis.(50) Deficiency of selenium decreases GPX activity leading to enhanced production of methemoglobin and hemolysis of the red cell.(26)

A decrease in red cell GPX activity has been used to show that selenium deficiency is severe enough to alter cellular metabolism.(93) Red cells saturated with carbon monoxide or oxygen fall prone to lysis only when a decrease in GPX activity occurs.(56) Erythrocytes of newborn infants demonstrate increased susceptibility to lysis from oxidation due to a low selenium concentration and decreased GPX activity.(36) Therefore a disturbance in selenium metabolism causes the breakdown of the oxidative defense mechanism of the cell which is GPX.(26)(36)(54)

Blood samples for coronary heart disease (CHD) patients demonstrated significantly lower GPX activity than controls, and patients with very low levels of blood selenium run an increased risk of myocardial infarction.(74)(78)(81) There is a strong correlation between serum selenium levels below 0.57 micromoles/liter and occurence of heart disease.(93) Epidemiological studies have shown low selenium intake to be the cause of various cardiovascular diseases.(29)(36)(79)(88)(93)

Under normal conditions, ingestion is the major entrance into the body for selenium.(29) Case reports of cardiomyopathy and myopathy due to selenium deficiency acquired during total parental nutrition (TPN) have confirmed the importance of selenium as an essential nutrient in man.(35)(93) Red cell GPX activity decreased in unsupplemented patients on TPN but not in a supplemented group.(93) Replacement of selenium in TPN patients resulted in an increase in GPX activity in plasma after 6 hours, platelet and granulocytes became normal within the kinetics of their production, and erythrocyte GPX activity returned to normal after approximately three months.(35) Children on TPN suffered profound muscular weakness that was nonresponsive to Vitamin E or carnitine supplementation. Muscle function improved after selenium supplementation.(93)

Extreme selenium deficient areas of China have an endemic form of cardiomyopathy known as Keshan disease. This disease results in dilatation of all four chambers as well as severe pancreatic atrophy. Supplementation with selenium is extremely effective as a treatment and as a preventive measure.(93)

GPX is an appropriate indicator of human selenium status in populations with below normal exposure to selenium, as activity of this en-

zyme is saturated at low levels. Therefore GPX has priority for selenium over other systems.(87) There seems to be a threshold effect for selenium; at serum levels less than 100 micrograms/liter, GPX activity is suboptimal.(84)

Decreased selenium and GPX may impair the ability of the liver to detoxify oxidative products of estrogen metabolism and induce cholestasis.(93) As pregnancy advances, estrogens increase and selenium concentration decreases, with selenium dropping to 84% of control values while GPX activity falls to 68%.(93) This usually does not cause a problem if the storage of selenium is adequate from a previous normal dietary intake of selenium. These excess estrogens disturb intracellular biliary metabolism and increase the requirement for hepatic antioxidative capacity, which results in functional and structural damage to hepatocytes resulting in cholestasis.(31) Intrahepatic cholestasis of pregnancy is most common in Scandinavia where there is low dietary intake of selenium.(31)(93)

GPX activity increases in the fetus prior to delivery as a protective factor against exposure to higher oxygen concentrations post partum.(52) If children are subject to severe or prolonged oxidative stress, Vitamin E concentration is inadequate to cope with the antioxidant requirements.(28) GPX may be involved in protecting human milk lipids from oxidative stress and consequently infant tissues.(33)(59) There is a positive correlation between GPX activity and selenium content in human milk.(59)

There is a good correlation between asthma and GPX levels. Patients with the most severe asthma had the lowest GPX levels.(52)(73) Airway inflammation is fundamental to the pathophysiology of asthma. SOR's mediate this response.(24) Low selenium levels exacerbate the mucusal inflammation in asthma by reducing GPX activity.(73) A low concentration of selenium in plasma and whole blood has also been associated with an increased risk of developing asthma.(73)

Increase in GPX activity in the lung due to oxidative stress is modulated by selenium availability. Those tissues which are least able to retain selenium are those which are most easily compromised by oxidative attack.(24) The lung is under heavy oxidative stress due to normal cellular metabolism. During added stress, such as exercise, tobacco smoke, and inflammation in association with the depletion of GSH, acute cytotxicity occurs.(69)

The only known condition of generalized GSH deficiency is in HIV-sero positive individuals.(70) Deficiency of selenium can contribute to a depressed immune response whereas GSH and selenium enhances immune function.(8)(62)(70) Selenium affects both cellular and humoral immunity.(79)(93) GSH is necessary for function of natural killer cells and for lymphocyte mediated cytotoxicity.(70) Exposure of lymphocytes to extracellular oxidants breaks DNA strands which suppresses the ability of lymphocytes to proliferate. Extracellular GSH protects cells of diverse tissue from oxidative insult.(70)

Selenium deficiency acts by reducing resistance to viral infections which cause myocardial necosis. Aids patients typically demonstrate low plasma, red cell, and whole blood selenium levels. These

patients typically develop nonobstructive myocardiopathy. The hearts become enlarged with dilatation of all chambers. GPX activity is decreased in these patients. Supplementation with sodium selenite resulted in a return to normal left ventricular shortening fraction.(93)

Lymphocytes require extracellular cysteine to maintain intracellular GSH levels where it functions as an antioxidant.(70) GSH is the major transport form of cysteine. In patients with leukemia who were supplemented with selenocysteine there were definite clinical improvements such as a decrease in the abnormal size of the spleen as well as immature leukocytes disappearing from the blood.(100) Selenium may act as a modulator of phagocytosis, T-lymphocyte activity and beta-cell synthesis of immunoglobulins; functions that are relevant to inflammatory and immunological processes.(73)

Free oxygen radicals have been shown to be involved in the pathogenesis of rheumatoid arthritis (RA).(30)(60) SOR's are produced by the respiratory burst of PMN's, biosynthesis of prostaglandins and leukotrienes, and by the autoxidation of arachidonic acid and other polyunsaturated fatty acids,(80) Granulocytes fed a selenium deficient diet were impaired in their ability to generate superoxide radicals.(63) Low content of antioxidant enzymes in granulocytes may cause a vicious cycle of inflammation in rheumatoid arthritis due to accumulation of oxygen radicals inside the cell after the respiratory burst.(30)(62)(80)

Selenium and GPX levels were found to be significantly lower in red cells, serum and granulocytes of rheumatoid arthritis patients versus controls. An increase in GPX activity with a decrease in symptoms was noted upon supplementation with selenium.(30)(80)(81)(93) Serum selenium levels were found to be inversely proportional to the severity of rheumatoid arthritis(80)(93)

Oxygen radicals play a central role in the inflammatory process of autoimmune diseases.(65)(71) After lipid peroxides are formed by primary inflammatory lesions, they are released into the serum and trapped by the liver where they are metabolized by GPX. This reflects the high concentration and rapid turnover rate of GPX in the hepatocyte. Therefore, GPX activity plays an integral part to tissue histiocytes functioning under increasing oxidative stress.(64)(65) GPX may be inactivated in conditions of severe stress as occurs in rheumatoid arthritis.(30)

Hydrogen peroxide produces a powerful inhibition of the chondrocyte biosynthetic system. Any disturbance in the glutathione conjugating system affects the rate of proteoglycan synthesis in chondrocytes during inflammation which predisposes cells to peroxide mediated damage.(71) Also, the protection of fibroblasts from oxidative damage depends almost entirely on the glutathione redox cycle.(49)

Kashin-Beck Disease (KBD) is an endemic osteoarthropathy characterized by atrophy, necrosis of cartilage, muscular atrophy, and joint stiffness with swelling. Metaphyseal lesions improved upon supplementation with selenium.(93)

Human myeloid cells were grown in a selenium free medium. GPX activity decreased thirty-fold when compared to the control group.

Selenium was then added and after one week removed again. GPX activity increased during the first week and subsequently declined in a time dependent manner. The researchers concluded that GPX activity is controlled by an external supply of selenium.(101)

Patients with active multiple sclerosis (MS) had higher levels of lipid peroxidation than controls.(32) Epidemiological, experimental, and clinical studies suggest that free radical production and decreased antioxidant defenses in the nervous system are involved in the etiopathogenesis of multiple sclerosis. The underlying mechanism is probably lipid peroxidation and oxidative membrane damage of myelin sheaths. (25)(50)(70) The myelin sheath is approximately 70% lipid.(76)

Selenium intake is low with a corresponding decrease of GPX activity in MS patients.(50) Currently there are some physicians advocating selenium supplementation as treatment for multiple sclerosis.(76)

Dystrophic muscle has elevated free radical activity. Patients with duchene muscular dystrophy have abnormal selenium metabolism with lower concentrations and a higher turnover rate. Supplementation with sodium selenite at approximately 1 mg/day increased GPX activity in plasma and erythrocytes.(97)

GPX activity has been shown to be decreased in a variety of dermatological disorders such as eczema, psoriasis, vasculitis, mycosis fungoides, dermatitis herpetiformis, and acne. Treatment with selenium raised GPX activity levels substantially with good clinical response. (24)(93)

"A subacute nutritional deficiency existing for a period of time may induce a metabolic and histopathological change which could foster a disease at some future time when a frank deficiency is not detectable."(34) A correlation was found between patients suffering from myocardial infarction and cardiovascular disease and a subacute selenium deficiency.(34)

Accumulation of lipofuscin pigments are a function of oxidative stress and antioxidant deficiency with a resultant increase in lipid peroxidation. These pigments accumulate with age.(43) Neuronal ceroid lipofuscinosis (NCL) patients exhibit low GPX activity which is responsive to selenium supplementation.(19)(33)

Glutathione is an important compound in protecting the brain against oxidative damage.(32) Patients with Parkinson's disease had substantially lower GSH levels in the substantia nigra portion of the brain compared to normal subjects.(67)

Plasma selenium and GPX activity are consistently altered in Down's Syndrome (DS). (24)(73) There is increased oxidation and elevated levels of lipid peroxides. GPX activity is increased due to the oxidant stress in this disease process. This hypothesis is supported by the positive correlation between I.Q. scores and red cell GPX activity in DS patients. (24)(42)

GPX activity may also be genetically determined.(77) The intracellular enzymic defenses against hydrogen peroxide; catalase (more important in prokaryotic cells) and GPX are of prime importance in protecting DNA.(27) The human GPX gene is regulated post-transcriptionally by selenium. A selenocysteine transfer-RNA has been identified which supports this hypothesis.(101)

There are approximately fifty studies that have confirmed an inverse epidemiological relationship between selenium intake and neoplasm.(40)(89)(93)(94) Case studies have consistently demonstrated low blood selenium in patients with cancer.(93) The probability is doubled in people with low selenium intake for developing a neoplasm.(40)(90)

Free radical and lipid peroxidation are involved in the initiation and production of cancer. Malondialdehyde which is a marker of lipid peroxidation was found to be higher in tumor than in non-tumor tissue.(90) A deficiency of selenium and lowered GPX activity has detrimental effects on the structural and biochemical function of cellular membranes.(40)(90) Reduced selenium levels exhibit a pro-tumor effect by a numerical decrease in GPX activity which predisposes the cell to damage by free radicals.(63)(89) Lipid peroxide concentrations were elevated with decreased selenium and GPX activity in greater than half of the patients with cancer.(40)

Marked decreases in selenium concentration and GPX activity were found in patients with squamous cell carcinomas of the head and neck, malignant lymphoma, hepatocellular carcinoma, and stomach cancer.(24)(58)(63)(90) Supplementation with selenium is associated with a decrease in the mutagenic activity of known carcinogens.(89)

"The activity of GPX is reflective of selenium availability."(90) Supplementation of both animals and humans with selenium results in an increase in cellular GPX activity.(37)(39)(82)(85)(87)(94)(97) GPX activity responds in a logarithmic fashion to increasing levels of selenium up to 1.27 micromoles/liter. At this point GPX activity reaches a plateau.(38)(93) Therefore, GPX activity can be considered a functional index of selenium deficiency since it is decreased in selenium deficiency states.(34)(42)(51)(93)

"Selenium dependent GPX activities are affected by sex, age, iron deficiency anemias, oxidant stressors and other toxicants."(39) Copper and iron increase the rate of lipid peroxidation. Selenium has been shown to inhibit this toxic effect.(19) GPX activity increases in times of oxidant stress and physical activity. Bioavailability of selenium sources and transition metals which interact with selenium has a direct bearing on GPX activity.(86) An optimal level of GPX activity is a balance between these factors and selenium intake.(39)

VII. CONCLUSION

Selenium concentration in blood has been adopted as a measurement of both selenium status and intake, however it is still not clear how this is related to body content.(83) An interlaboratory study revealed a coefficient of variation of 55% for blood selenium.(93) A study by Jackson et al,found no correlation between high dose selenium supplementation and GPX activity in muscle.(97) However, selenium in muscle is an inaccessible pool with a slow turnover rate and is not in equilibrium with other pools in the body.(39) Also, GPX activity as an indicator of selenium status appears to be limited to cases of selenium deficiency due to the enzymes threshold effect at levels of 1.27 micromoles/liter or higher of serum selenium.(5)(38)(93)

Levander has shown that platelet GPX activity was very responsive to selenium repletion in Finns and is therefore a sensitive indicator of changes in selenium status.(92) Since no specific GPX inhibitors are known and platelets have low non-selenium dependent activity, platelet GPX responds more rapidly to changes in selenium supplementation than plasma or red cell GPX and is therefore the best assessment of the bioavailability of selenium.(39)(71)(79)

A point of controversy centers on the absence of symptoms in populations who have low selenium levels and corresponding GPX activity. New Zealanders, for example, demonstrate no noticable oxidative damage or compensatory changes.(5)(93) However, in long-term selenium deficiency the isoenzymes of glutathione transferase (some refer to this as selenium independent GPX) become elevated and partially compensate for the lowered selenium dependent GPX activity. Add to this an adequate intake of Vitamin E and this explains the apparent lack of symptoms. Also, if one were to classify New Zealanders on Mertz's scale of trace element deficiency, they may have only reached stage one which is initial depeletion and may not have fallen into stage two involving a compensated phase of impaired biochemical function.(83)

GPX is the only metabolically active selenoprotein in human tissues, albeit 14 different selenium containing proteins have been identified.(27)(33)(60)(73)(78) Although some researchers state that there are additional forms of GPX, no non-selenium GPX activity was detected in red cells, platelets, or muscle.(33)(39)(73)(90) The so-called selenium independent GPX (glutathione transferase or ligandin) can only reduce hydroperoxides, not hydrogen peroxide. These ligandins are a family of proteins that bind hydrophobic molecules.(101)(103)

The distribution and levels of both GPX's. GSH reductase, and selenium are the main factors in the susceptibility of tissue to oxidant injury.(90) To maintain GPX activity at optimum, it is required that all constituents of the glutathione redox cycle operate adequately.(30) Glutathione reductase is riboflavin dependent.(1) Production of cysteine from methionine requires pyridoxine. It has been shown that Vitamins B_2 and B_6 enhance the antioxidant effect of selenium.(19)

The ability of organisms to respond to oxidative stress by increasing the activity of GPX is the primary function of this protective system.(24) Normal tissues contain a large excess of GSH.(101) Increased oxidative stress increases GPX activity and exacerbates selenium deficiency.(33) Selenium deficiency increases liver arginase and GSH synthesis and causes release of reduced GSH into the blood. This release of GSH is not due to passive diffusion but is to protect cellular membranes from oxidative damage.(52)(59)(73)(101) Although the safety margin for selenium between a state of deficiency and toxicity is very narrow, increasing the level of selenium increases GPX activity in plasma, platelets, and red cells.(59)(92)

"Today it is assumed that an insufficient amount of selenium leads to pathological changes in the human organism."(26) Selenium has only one documented function in mammals, and that is as a component of GPX.(34)(77)(80) GPX activity correlates closely with selenium concentra-

tion and influences the basal tissue GPX activity even in the absence of oxidative stress.(24)(30)(41)(92) "Signs of selenium deficiency can be explained by a lack of the only known biologically active selenoprotein in mammals; GPX."(33)

VIII. PROCEDURE

1. Find any weak muscle.
2. Test against strengthening of oral insalivation of selenium.

A P P E N D I X

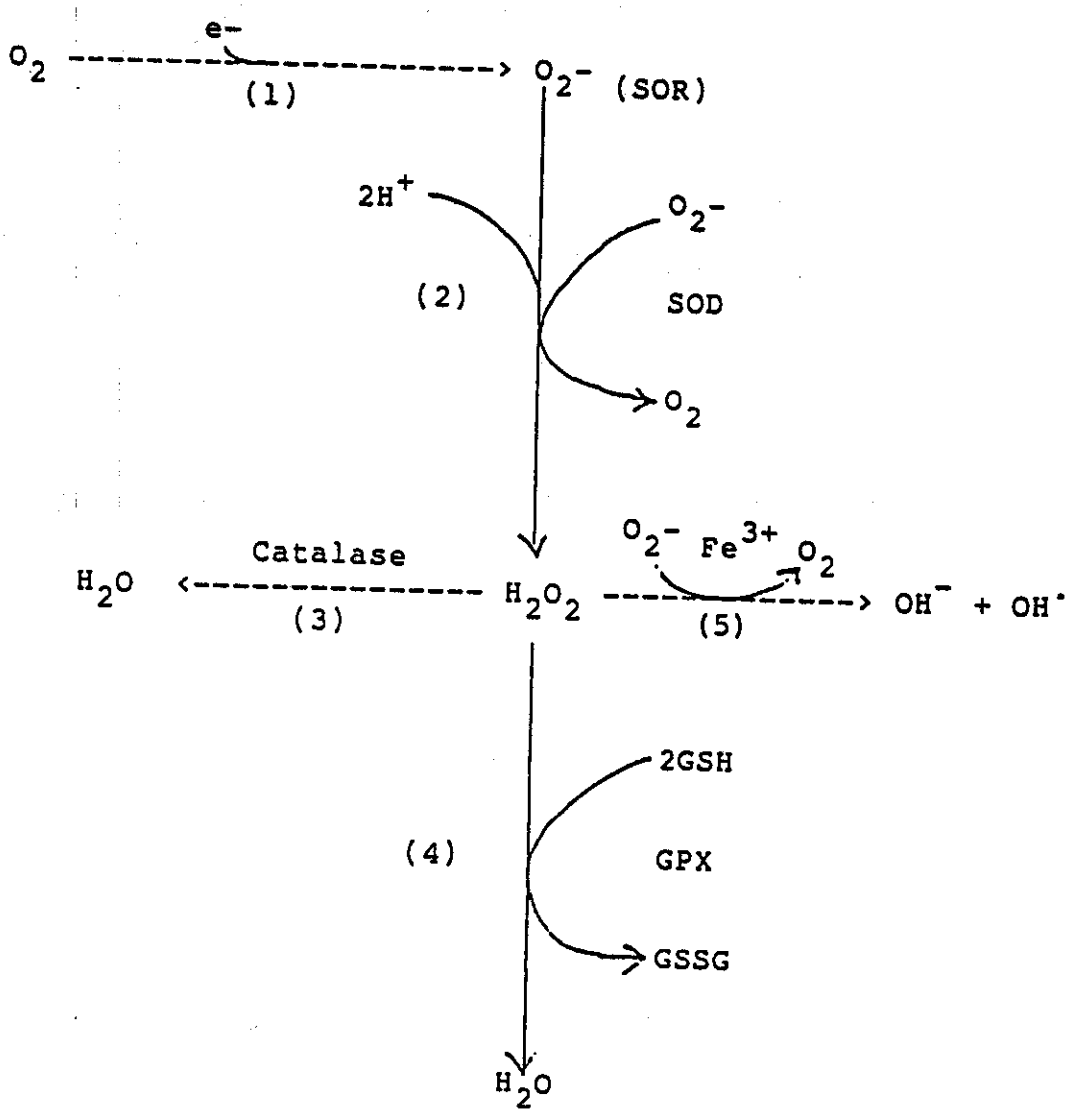

FIG. 1: 1. Reaction (RX) facilitated by electron transport, drugs, enzymes, pollutants, and radiation; 2. Superoxide dismutase (SOD); 3. Catalase; 4. Glutathione peroxidase (GPX); 5. Haber-Weiss reaction catalyzed by Ferric Iron (Fe^{3+}).(18)

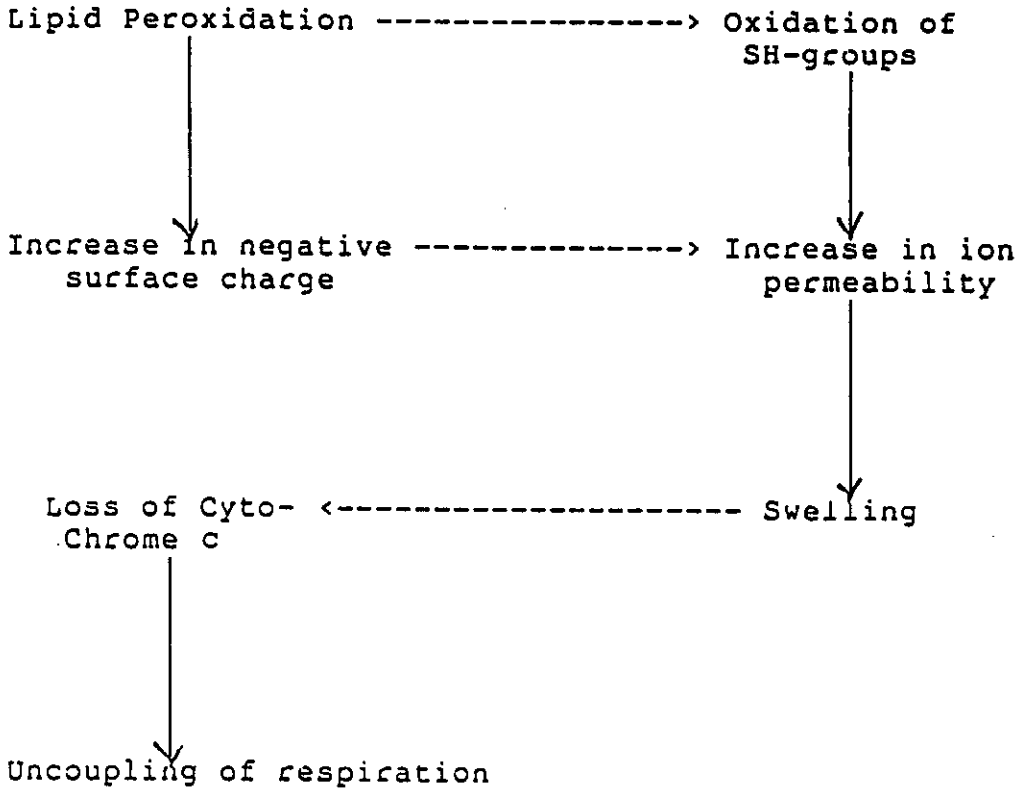

FIG. 2: Effects of lipid peroxidation on mitochondria. (4)

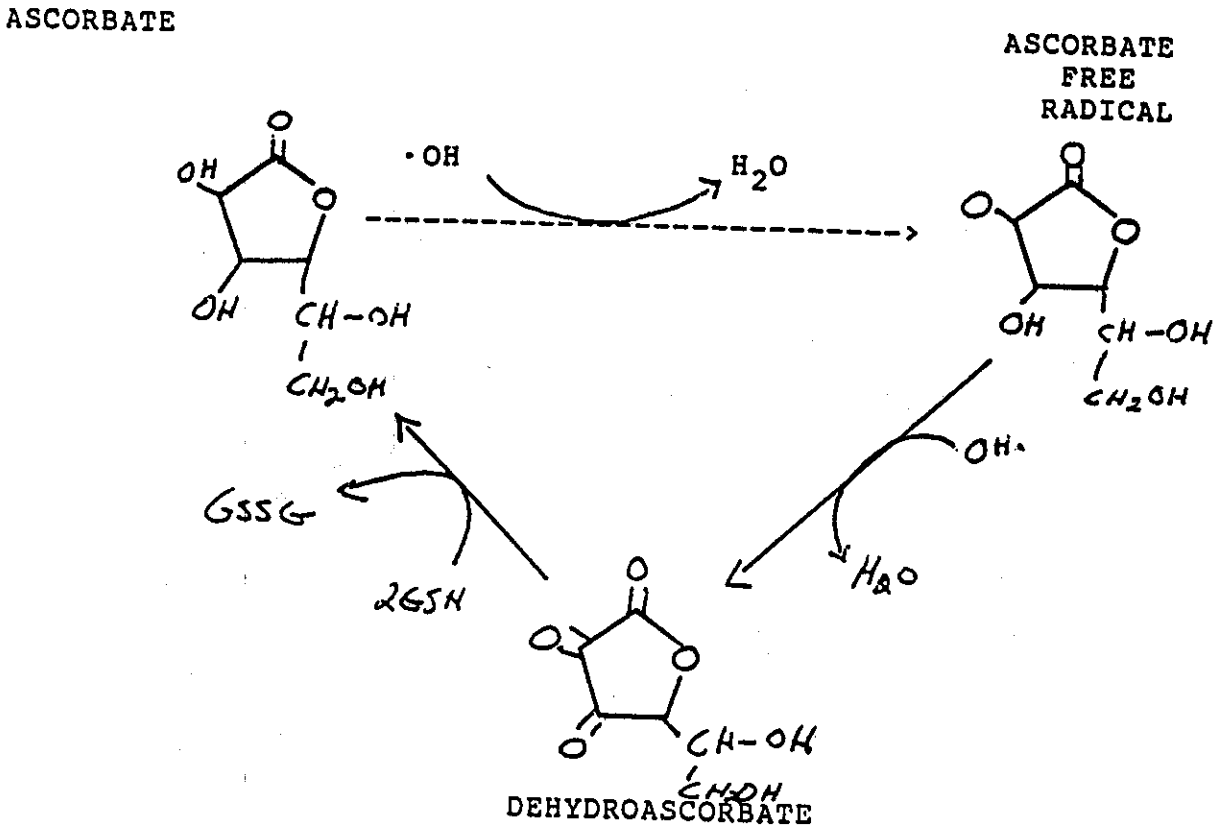

FIG. 3: Reduction of dehydroascorbate by GSH to ascorbate.(18)

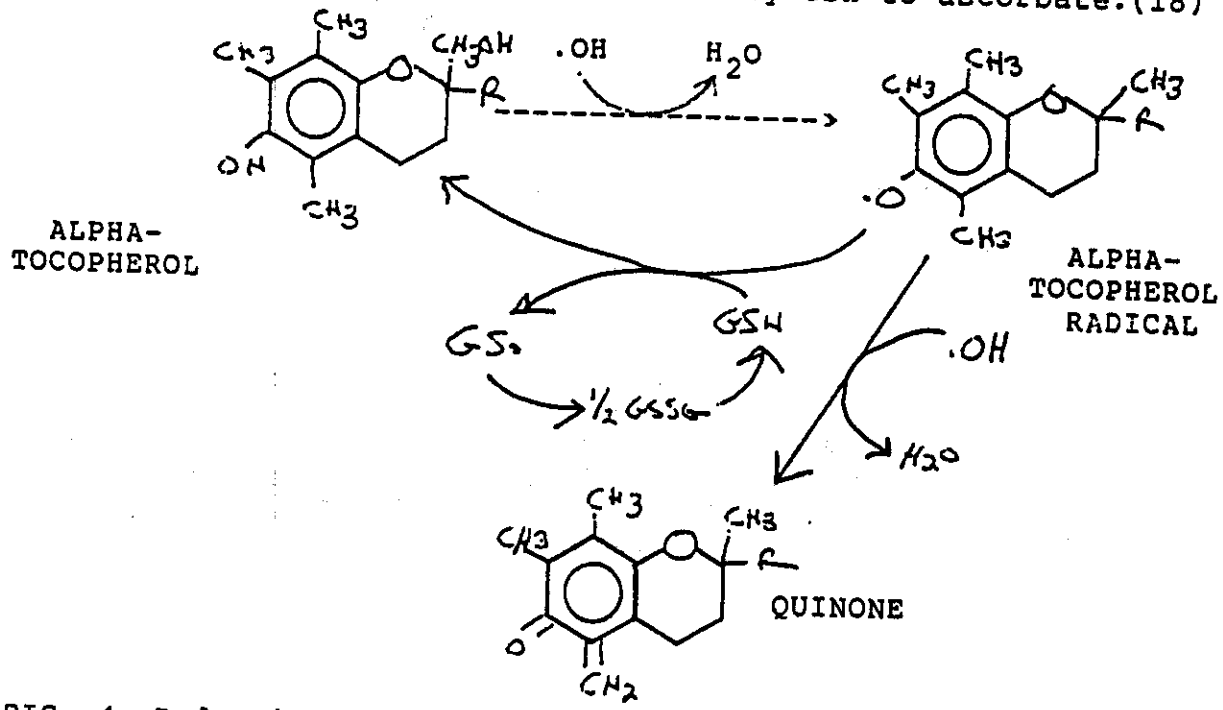

FIG. 4: Reduction of tocopherol radical by GSH.(18)

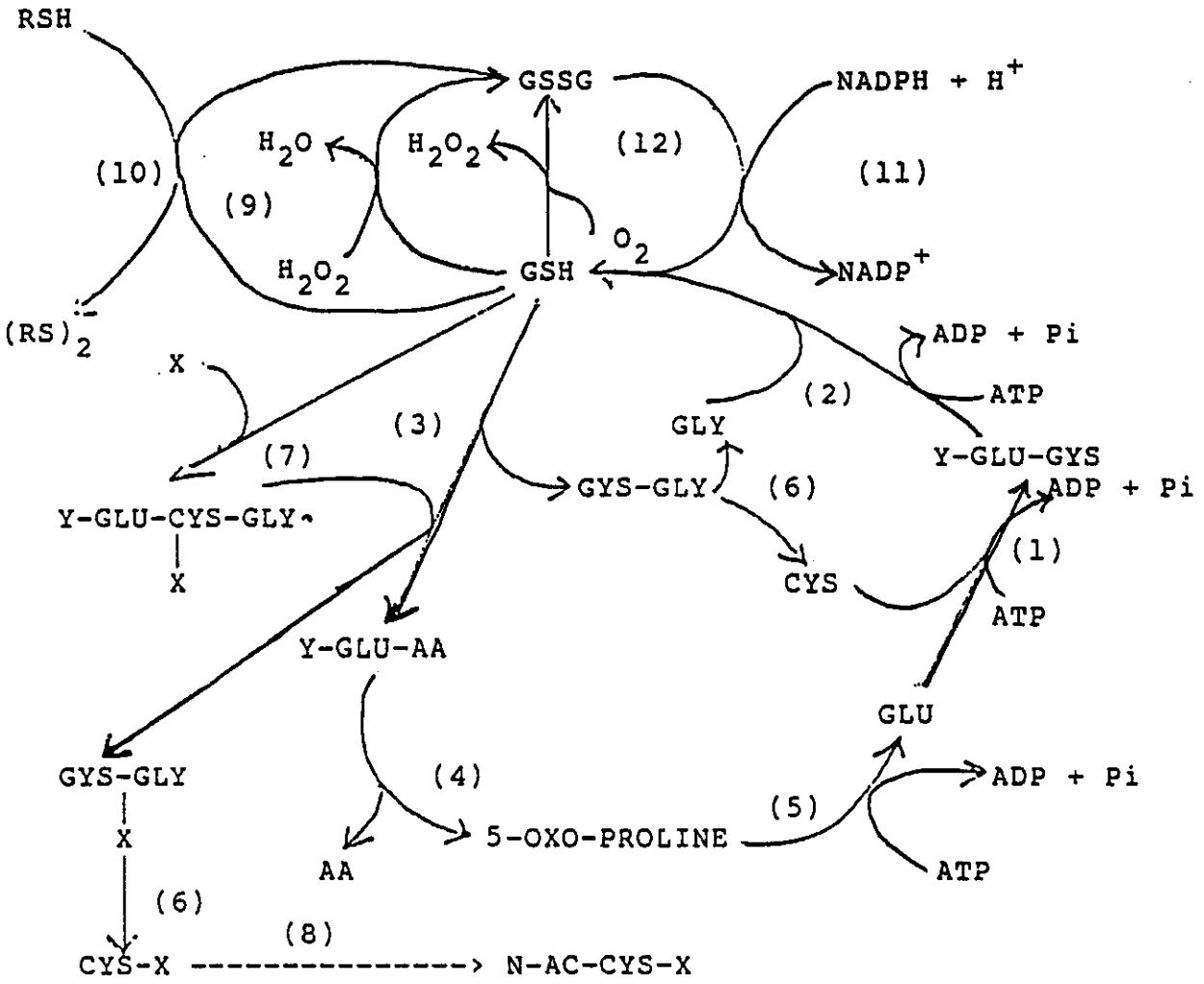

FIG. 5: Overall summary of GSH metabolism: 1. Gamma - glutamyl-cysteine synthetase; 2. Glutathione synthetase; 3. Glutamyl transpeptidase; 4. Gamma glutamyl cyclotransferase; 5. 5-oxoprolinase; 6. Dipeptidase; 7. Glutathione-S-transferase; 8. N-acetylase; 9. Glutathione peroxidase; 10. Transhydrogenase; 11. Glutathione reductase; 12. Oxidation of GSH by H_2O_2.(98)

- Conversion of GSH to GSSG is also mediated by free radicals.

X = eletrophic compound

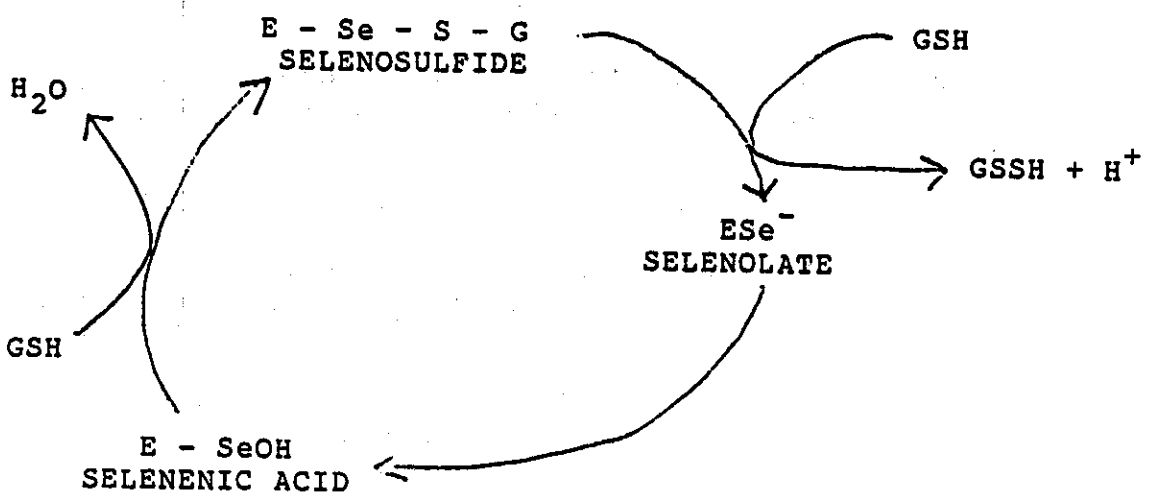

FIG. 6: Catalytic mechanism of glutathione peroxidase (GPX).(3)

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The Efficacy of Applied Kinesiology Protocols in Correcting Peripheral Nerve Entrapment Associated With Carpal Tunnel Syndrome An Inter-Examiner Study

By James D.W. Hogg, D.C.

Abstract: A study was conducted by this author and Dr. Dennis Hageman among sixty five patients suffering signs and symptoms of carpal tunnel syndrome. Statistics reflect a combination of a records search of past carpal tunnel patients as well as patients who were seen during the term of the study. Information was gathered regarding areas of nerve entrapment as well as overall effectiveness of treatment and total costs.

Introduction

Carpal tunnel syndrome is a problem that is increasing in frequency. Industries that put unusual stresses on the wrists such as meat packing and assembly line work as well as the repetitive microtrauma common in data processors have created whole new areas of ergonomically induced injury. Other professions that are traditionally prone to carpal tunnel injuries include carpenters, plumbers, waiters, massage therapists and, of course, chiropractors.

Medical approaches to this problem, including surgical decompression of the carpal tunnel may run several thousand dollars and have failure rates of up to 50%. One possible reason for this failure may be the frequency of multiple areas of nerve entrapment commonly found in "carpal tunnel syndrome". This so-called "double crush syndrome" hypothesis has been demonstrated in studies by Hurst, Weissberg and Carroll as well as Upton and McComas [1,2,3]. Briefly stated, the idea behind the double crush syndrome in carpal tunnel problems is that the discomfort and disability associated with carpal tunnel syndrome arise from compression of the median nerve and it's contributors not only at the carpal tunnel but at various more proximal locations. These locations may include entrapment at the pronator teres muscle, the elbow, lacertus fibrosus of the biceps brachii tendon, ligament of Struthers, the shoulder (due to imbalance of rotator cuff members) and the spinal nerve root[1,4]. In addition to mechanical considerations at the above locations, nutritional support in the form of $B6^5$ or it's activated from, pyridoxal-5-phosphate[6] should be considered.

As opposed to standard medical treatment, it has been my experience (and that of many colleges) that AK procedures have been very effective in treating carpal tunnel syndrome. As is often the case, however, no studies have been easily available to determine exactly how effective the AK approach is. At Dr. Hageman's suggestion, the two of us decided to gather statistics on previous and current carpal tunnel cases to determine effectiveness of treatment as well as frequency of peripheral nerve entrapment at some of the above areas. Patients selected for this study exhibited at least one of the following symptoms or signs: noticeable loss of grip strength, pain in the wrist with pain in the median nerve sensory area of the hand (first 3 1/2 fingers), numbness and/or tingling in the median area of the hand, dermatitis with a "glove" distribution, atrophy of the thenar eminence, sleep disturbance caused by hand pain. In addition, subjects selected all demonstrated weakness of the opponens policis muscle on manual

muscle testing.

The factors tracked included incidence of involvement of distal radius and ulna separation, lunate subluxation, nutritional need, pronator quadratus weakness, proximal radius or ulna (elbow) subluxation, involvement at the shoulder, lower cervical subluxation and upper thoracic subluxation. We also tallied the number of office visits necessary to eliminate symptoms, the number of visits to eliminate all positive AK signs and the cost involved in each category. Neurologic involvement in the different areas was determined by therapy localizing[4] the suspect areas and observing any change in the previously weak opponens policis on manual muscle testing. For example a patient with weak opponens policis might experience temporary facilitation of that muscle while therapy localizing to the carpal tunnel area, the proximal radius, the shoulder and the first thoracic. We would then list all to these areas as being involved neurologically for that patient. The need for B6 or other nutrition (sometimes raw bone meal or some other nutrient would show) was determined by having the patient taste the nutrient in question to see if it would result in temporary refacilitation of the opponens policis. When therapy localization to osseous components produced facilitation, the bone in question was challenged[4] to determine direction of subluxation.

A summary of findings is contained in table one, below.

TABLE 1	Total Hogg	%	Total Hageman	%	Total combined	%
Patients	48	74	17	26	65	100
Distal Radius/ulna	47	98	7	41	54	83
Lunate	20	42	12	70	32	49
Nutrition	21	44	3	18	24	37
Pronator Quadratus	43	90	0	0	43	66
Elbow	17	35	0	0	17	26
Shoulder	13	27	0	0	13	20
Lower cervical	5	10	16	95	21	32
Upper thoracic	29	60	10	59	39	60

Therapy for the patients in this study involved manipulation of involved osseous components as indicated by challenge, spindle cell or golgi tendon technique for pronator quadratus involvement (Pronator quadratus was not

usually a primary site of nerve entrapment but was often involved in a separation of the distal radius and ulna which resulted in median nerve entrapment between the flexor retinaculum and the carpal bones) and spinal manipulation to indicated cervical and thoracic vertebrae. If distal radius/ulna separation was indicated, a non-elastic (canvas or leather) brace was used after manipulation to help prevent re-separation. When therapy localization to the shoulder was positive attention was paid to the various rotator cuff elements as well as other muscular components of the gleno-humoral joint. Various AK procedures were employed to restore balance to the involved shoulder muscles. Nutritional support was used as indicated. The most common dose of B6 or P-5-P was 50mg two to three times/day.

A summary of therapeutic results is contained in table two.

Table 2	Hogg	Hageman	Combined
Visits to eliminate symptoms	6.73	7	6.78
Cost	168.25	161.00	164.63
Visits to eliminate signs	8.6	8.7	8.6
Cost*	215.00	200.10	207.55

* The cost category had been adjusted to reflect current fee schedules.

The range of treatments needed was from one for a few simple cases to twenty five for some of the post surgical cases. The overall success rate, (patients who became sign and symptom free for a minimum of six months) was 84%. Subjects were counted as failures if we were unable to completely eliminate their positive symptoms and signs. Patients who dropped out of care before completing care were considered failures if they showed positive signs or symptoms at their last visit. The study also included several patients who had previous ineffective carpal tunnel surgery. If patients who dropped out of care after only four or five visits and post surgical patients are eliminated from the statistics, the success rate is closer to 95%. It is interesting to note that one patient who had dropped out after three visits and was counted a failure returned to my office four years later. She was symptom free after her last visit and saw no reason to continue care, returning only after re-injuring her wrist!

Discussion

The above statistics indicate some variation in the findings obtained by Dr. Hageman and myself. For instance I found many more cases with separation of the distal radius and ulna, pronator quadratus and nutritional need while Dr. Hageman found more involvement of the lunate and lower cervical spine. On the other hand we both found about the same amount of upper thoracic involvement. Our bottom line statistics of visits and cost to negative symptoms and signs were almost identical, with Dr. Hageman's lower figures for cost reflecting a slightly lower fee per visit. The variations may reflect the fact that Dr. Hageman's study sample was less than half of my own and that the overall study size (65) was fairly small. With

a larger sampling things may have evened out somewhat. Another factor may involve patient population. A large portion of Dr. Hageman's study group were meat cutters at a nearby meat packing plant who tended to stress their hands and wrists in a way that differs from the general population. A final consideration is differences in what we each look for. Although we are both trained in AK, our findings are going to be affected by our individual preferences. For example Dr. Hageman practices in Iowa where nutritional therapy is discouraged while I practice in Illinois and have an interest in nutrition that predates my interest in chiropractic.

Dr. Hageman and myself entered into this study with the objective of general information gathering. This study is also several years old. As a result there was only minimal effort made to standardize our information gathering. About one half to one third of our stats were generated via record searches of cases completed before we had conceived of this study and any associated standardization. It would be useful to repeat this study with a well defined testing and recording protocol and limited to current cases.

Just as an aside, you may have noticed that I footnoted several references that no one in the ICAK would need to look up. I was recently asked to participate as a reviewer for a referenced chiropractic journal that is starting up locally, a project of Burl Pettibon, D.C. and some of his colleges. The purpose of this journal is to publish papers from a wide spectrum of chiropractic techniques and I was asked to review any AK papers. So far there has been only one AK paper, but in reviewing it I noticed that the author was using a lot of AK-specific terminology that would be unfamiliar to the average chiropractic reader. Along with corrections involving technique and clarity, I suggested that the author footnote the numerous references that would be unfamiliar to the average reader. Since one of our goals as an organization is to have our papers (for those of you who feel that "paper" is not an accurate term for works published in this volume feel free to substitute "article", "report", "ramblings" etc.) available to a larger audience I felt it would be a good idea to get in the habit of footnoting references for that larger audience.

Conclusion

This informal inter-examiner study suggests that AK protocols are effective in reducing nerve entrapment common in carpal tunnel syndrome. Despite some differences in frequencies of level of peripheral nerve entrapment, one consistent finding was that multiple levels of such entrapment are quite common. Considering the effectiveness in terms of overall correction and cost I feel that the AK approach to carpal tunnel syndrome can confidently be positioned as an excellent alternative to standard medical approaches.

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A NEW PROCEDURE FOR IDENTIFYING NEUROLOGIC DYSORGANIZATION AT THE CORTICAL LEVEL

BY DAVID A. KUBICEK, D.C.

ABSTRACT

This paper discusses neurologic dysorganization (N.D.) and describes a new procedure that determines its presence at the cortical level.

INTRODUCTION

The terms N.D. originally coined by Doman and Delacato,[1,2] and switching used in Applied Kinesiology[3] are often used loosely and without complete understanding. Because of this, their paramount significance is not realized or appreciated. Our level of neurologic organization (N.O.) to a great extent determines our ability to perceive and respond to our world and ourselves. What could possibly be more significant than that?

DISCUSSION

The term switching or switched originally referred to the aberrant muscle test response that occasionally occurs in direct opposition to what is observed in postural analysis.[4] For example: Postural analysis reveals a weak left latissimus dorsi, but upon testing, it remains strong, while the right latissimus dorsi tests weak. The patient is then said to be switched. Remember, postural analysis never lies, but muscle testing can, especially when the patient is switched. We rely on accurate information from the patient in order to diagnose and treat. When the patient is switched the information gained cannot be relied upon to be accurate. The only information that can be relied upon is that which abolishes the switching. Therefore no diagnosis or treatment should be rendered until the presence of switching is denied or confirmed and corrected.

How does switching correlate or compare to Neurologic Dysorganization? To answer this and better understand N.D., let's first define N.O.. "N.O. is the process whereby the organism subject to environmental forces, achieves" and maintains "the potential inherent in its genetic endowments."[5] In Man, it is a developmental process that begins intra-uterine and continues in stages up to approximately the age of 8 or 9. This is basically accomplished by the gradual myelination of the nervous system beginning with the spinal cord and proceeding up through the brain stem into the cortex. Each stage is characterized by a specific set of abilities or functions both sensory and motor.

According to Doman and Delacato,[6,7] the potential physical genetic endowments for the average human can be divided into six categories. Three reflect sensory endowments or functions and three motor. The three sensory categories are: Visual competence, Auditory competence and Tactile competence.

Visual competence begins with the simple light reflex that should be present at birth and hopefully proceeds through various stages to its ultimate potential of reading words using a dominate eye consistent with the dominant hemisphere.

Auditory competence begins with the startle reflex and ends with the understanding of complete vocabulary and proper sentences with proper ear.

Tactile competence begins with Babinski's reflex and ends with tactile identification of objects using a hand consistent with the dominant hemisphere.

The three motor categories are: Mobility, Language, and Manual competence.

Mobility begins with movement of arms and legs without bodily movement and ends with using a leg in a skilled role which is consistent with the dominant hemisphere.

Language begins with the birth cry and crying, and ends with complete vocabulary and proper sentence structure.

Manual competence begins with the grasp reflex and ends with using a hand to write which is consistent with the dominant hemisphere.

N.O. can also be defined as having the complete ability to receive all incoming stimuli (physical, chemical, and mental) as it exists in reality; process it as it exists in reality and respond to that stimuli (physically, chemically and mentally) in an appropriate manner. In order to achieve this, the afferent, central and efferent parts of the nervous system respectively must function properly. (For simplicity the autonomic nervous system will be arbitrarily included as part of the efferent system).

N.D. can now be defined as a dysfunction in any one, any combination of, or all 3 of these areas. It can also be defined as a dysfunction at any level and in any category of the developmental process.

Switching is not N.D., but only identifies its presence. Switching is merely the expression, manifestation or one of the symptoms of N.D., like pain and swelling are the symptoms of tissue injury. I believe N.D. is a more accurate term for this phenomena.

Any imbalance or dysfunction of the body regardless of the system or systems involved effects or at least is reflected in the nervous system. We who use muscle testing can verify and appreciate this the most. Muscle strength is ultimately controlled by the nervous system regardless of the other forces (physical, chemical or emotional) that may be influencing or modifying its function.

When treating a patient the physician must have a source of reference, or a goal in which to refer or achieve. Optimal health is the ultimate goal or reference, however this is much too broad or encompassing to give one a firm place to begin. Therefore, the physician must begin with what he/she feels will influence the patient's health the most, or bring them closer to that ultimate goal of optimal health. For the cardiologist it may be a strong heart, for the gastroenterologist a clean bowel, for the chiropractor a properly functioning spine, etc.. But if one looks at the hierarchy of the body, the nervous system sits at the top. If

the nervous system is dysfunctional it is highly unlikely that any other system will function to its full potential. This hierarchy is based on a systems value in maintaining life. Some structures such as the spleen, gallbladder, uterus, prostate, appendix, etc., can be removed without mortally effecting the life process, most however cannot. A structure's value can be determined by how long life can be sustained without its presence. Death will arbitrarily be defined as a complete cessation of all nervous activity. Any definition less than this would leave room for argument. Without the spleen, gallbladder, or appendix life can be sustained for years; without the liver or kidneys, hours to days; without the heart - minutes; but without the nervous system life ceases immediately. If all areas of reception and motor functions both internal and external are eliminated then death occurs. If the nervous system is of paramount importance, then treating it should influence health more than any other system. Therefore, I propose that our frame of reference or immediate goal should be a properly functioning nervous system. Or in other words neurologic organization.

The presence of N.D. can be used as an incredibly accurate tool in identifying and prioritizing the cause of a patients illness. This is especially important when faced with multiple complaints and the dysfunction of multiple systems upon examination. It separates the cause from the compensations. It identifies where the treatment should begin. By identifying the lowest level of N.D. and determining what abolishes it, one determines the cause and prioritizes the treatment.

After the causes have been eliminated and N.O. has been restored it may be necessary to mop up some structural compensations or effects such as subluxations/fixations, neurolymphatic reflexes, meridian points etc., and any other dysfunction that is not yet significant enough to effect N.O.. I believe that all dysfunctions if allowed to progress will eventually reach a threshold and create N.D..

A child's as well as an adult's level of N.O. can be determined by their ability or function. A person is complete at a particular level only when they can perform all of the functions characterized by that level. By knowing the functions of each level a patient can be tested and their level determined.

Doman and Delacato[8,9] studied hundreds of well children to determine what functions were normal at each level. Each level is dependent on the previous one. A person ideally begins the next level when all the functions of the present level have been mastered. This then signals the next level to begin. Environmental stimuli usually initiates this development. The child eventually through trial and error responds to that stimuli appropriately which re-enforces the reception of more stimuli etc.. In most cases this development proceeds uninterrupted and proper N.O. is achieved. However, this can be interrupted by brain damage (e.g. trauma or disease), decreased environmental stimuli (e.g. poor living conditions, neglect), physical restriction of the motor response (e.g. restrictive clothing, injuries that restrict motion) or encouraging the child to perform functions of a later level before they have mastered their present function (e.g. encouraging a child to walk before they learn to crawl). Not all functions, motor or

sensory, of a particular level may be involved. That is why all functions of each level should be evaluated. When a particular function is lacking, all higher levels associated with that function will also be effected. The lowest level where dysfunction is determined is the level where treatment should begin. Sometimes just treating this level will eliminate the interference and allow the rest of the developmental process to proceed uninterrupted. Other times each level must be treated independently. Proper neurologic development is not guaranteed or automatic but instead depends on normal, structure physiology and environmental factors.

The following is a brief summary of the developmental process.[10,11,12] It will describe the level involved and its primary functions. If further detail of functions and standard clinical examinations are desired, the author refers you to Dr. Walther's Applied Kinesiology Volume I and Synopsis and works by Doman, Delacato, and LeWinn.

Intra-uterine-16 weeks

The first level begins with functions belonging to the spinal cord and medulla. These functions should be present at birth and consist of the basic reflexes (light, startle, Babinski and grasp), the birth cry and crying, and movements of the extremities and torso without mobility.

16 weeks-6 months

The second level involves the pons and consists of homolateral motor, visual and auditory functions. The tonic neck reflex is present and can best be observed while the child sleeps.

6 months-1 year

This involves the mid-brain and is the level where you will observe the most dysfunction in your clinical practice. You will rarely if ever see patients with involvement's of the first 2 levels because of their extreme disabilities. This level consists of bilateral cross-crawl motor pattern and bilateral visual and auditory function.

1 year-5 years

This involves early cortical function and consists of bilateral cross-pattern walking and perfection of bilateral visual and auditory functions.

3 years - 8 years

This involves cortical hemispheric dominance and consists of performing skilled activities with the leg, hand, eye and ear which are consistent with the dominant hemisphere. Mixed dominance suggests N.D.. Hemisphere dominance is hereditary and can be determined early by observing the child's tonic neck reflex. The position most often assumed reveals the pre-determined side.[13] Forcing a child to switch handedness will

almost always cause N.D.. This level is unique to Man and gives us the ability to communicate and understand language. It should be noted that all lower functions remain, but are superseded or over shadowed by the higher functions.

Doman and Delacato[14,15] believe that hyperactivity, stuttering, dyslexia, autism, mental retardation, etc. are not independent entities or conditions, but all one and the same. They are all simply manifestations of N.D., and they differ only in their degree. Meaning one is closer to or farther away from N.O.. Their positive results in treating these so called different conditions using virtually the same treatment procedure seems to support that thinking. Doman and Delacato originally based their developmental treatment procedure on brain injured children. They determine what area of the brain is involved and the lowest level effected by taking a history and performing a physical examination. Treatment of motor dysfunction consists of imposing the deficient function onto the child in hope that it will facilitate the nervous system to form alternate pathways around their damaged area or activate undamaged areas to take on new functions. This is called patterning. Treatment of sensory dysfunction consists of imposing specific environmental stimuli in significantly greater amounts than would randomly occur in nature. Theoretically affecting the nervous system in a similar way. These procedures are also used to treat children and adults who are not brain damaged, but failed to fully develop due to adverse environmental and/or health factors. Their success does not depend upon the extent of the patients's disabilities, brain damage or level of involvement because some with greater, disabilities, brain damage or lower levels of involvement fared better than those with less. They are extremely successful with some and only moderately or not at all with others. I believe the reason for this lies in their failure to identify and/or treat the dysfunction (structural, chemical or emotional) that originally caused the N.D.. If the cause remains the results of patterning are bound to be incomplete, temporary or completely unsuccessful. The dysfunction or disease that originally caused the N.D. must be resolved in order for patterning to be successful. The majority of our patients will be adults who developed normally, but began to exhibit symptoms of N.D. due to an illness or physical or emotional trauma. With these patients the treatment of the cause almost always eliminates the N.D. and patterning is usually not necessary.

There are several procedures for identifying switching. The most common is the therapy localization (T.L.) of various meridian points such as cross K27,[16] K27, GV27, CV24, etc..[17] From my clinical experience, most if not all forms of switching are eliminated with the correction of N.D..

It is believed that a cross K27 weakness is analogous or associated with mid-brain and early cortical dysfunction because virtually every time a cross K27 weakness was present the patient would also weaken to the cross crawl pattern. This appears to be true, however I suggest you use the cross crawl weakness as the indicator because it frees the hands to T.L. the cause.

Initially, I thought the K27 weakness was analogous with cortical (hemispheric dominance) dysfunction because occasionally after the cross K27 weakness was eliminated

a K27 weakness would appear. This could not be confirmed because there was no procedure like the cross crawl pattern in which to associate or compare. I have recently developed a procedure using muscle testing that appears to identify cortical dysfunction. In comparing this procedure with K27 it appears the association is not 100% valid. Cortical dysfunction is often present without K27, but not vice-versa.

Please realize that rubbing K27 and the umbilicus is not a cure for N.D.. It was used only because a better method was not known at that time.

"Ocular lock"[18] is another method for identifying N.D.. This evaluates dysfunction in the visual category at the mid-brain/early cortex level. Remember, not all of the categories or functions of a particular neurologic level may be involved. Fortunately however, for the vast majority of patients only a few key procedures are usually necessary to evaluate for N.D.. For the more difficult patients a complete evaluation may be required and should probably be left to a qualified specialist. The following is a list of those key procedures and the level to which they belong:

Pons: homolateral crawl
Mid-brain/early cortex: cross crawl
Cortex: (R) or (L) unilateral crawl

For most practical purposes the mid-brain and early cortex can be considered 1 level. The main difference is that the cross crawl pattern of the mid-brain is quadrupedal where as in early cortex it is bipedal. This distinction is mostly academic and is usually not needed in the following procedures.

METHODS AND PROCEDURES

1) Find a strong lower extremity indicator muscle to both patient and doctor initiated tests.[19]

2) While supine, have the patient perform 10 cycles of cross crawl without head turn (because 99% of patients with N.D. will exhibit dysfunction at the mid-brain/early cortex level or higher).

   a) If the indicator weakens to only a doctor initiated test, which is extremely rare, it almost always represents a structural dysfunction consisting of subluxations and/or fixations of the spine, pelvis and extremities, NL reflexes, or meridian imbalances involving points below the head. The cross crawl weakness will last for several minutes and give you plenty of time to T.L. the spine, pelvis and extremities.

      Lower extremity subluxations are by far the most common cause for this type of weakness. If one of these areas abolishes the weakness simply adjust it. If not, then use the traditional meridian pulse points to identify the organ/gland that requires structural correction. The structural correction usually consists of rubbing

its NL reflex and/or stimulation of its tonification point. T.L. the meridian pulse points to determine which one abolishes the weakness. Then T.L. the alarm points related to the active pulse point to determine the exact meridian involved. I do not recommend using light and firm pressure on the pulse points to differentiate. Then T.L. the involved meridians related NL reflexes and its tonification points. Simply treat the area that abolishes the weakness.

3) If the cross crawl creates both a doctor and patient initiated weakness, which is much more common, this indicates an emotional or chemical imbalance.

   a) T.L. the emotional neurovascular (NV) points. If it abolishes the weakness, use whatever emotional technique with which you are familiar. [The value of emotional techniques such as Dr. Scott Walker's Neuro Emotional Technique (N.E.T.)[20] appears to be in its ability to break the psychosomatic connection which keeps the emotion from causing physical dysfunction. However, it is ultimately the individual's responsibility to transform the emotion itself.] If you are unfamiliar with N.E.T. or any other emotional techniques, just holding the emotional NV points can also be used.

   b) If these points do not abolish the weakness, T.L. over the glabella which this author believes represents pineal, hypothalamus, or pituitary dysfunction. If this abolishes the weakness it is important to return the patient back to their original strength or condition prior to the cross crawl. To accomplish this you can do 1 of 2 things. You can have the patient perform 10 cycles of homolateral crawl or find out what phase of respiration abolishes the cross crawl weakness and adjust the cranium accordingly. From my clinical experience, the supraspinatus appears to be associated with all 3 and will usually test weak. First test the weak supraspinatus against the emotional NV points. [Emotional trauma can effect N.O. directly by actually creating N.D., or indirectly by creating a dysfunction in an organ/gland, etc...that ultimately leads to N.D.]. If the emotional NV abolishes the weak supraspinatus use step 3a. If not, then challenge for the pineal cranial fault[21] to differentiate it from the hypothalamus and pituitary. If positive, do not correct it until you have tested the weak supraspinatus against the therapeutic supplements (dietary, herbal, homeopathic, etc...) that you normally use for the pineal. The only criteria that must be met is that the supplement abolishes both the supraspinatus and the cross crawl weakness. You will often find a supplement that will abolish the offending organ/gland, but not the cross crawl. This product should not be used for this condition because it will almost always create iatrogenic effects and will not completely correct the condition. I have virtually eliminated these effects by sticking to this protocol. When the supplement strengthens the offending organ/gland, keep it in the patient's mouth and have them again perform 10 cycles of cross crawl. If it is the proper supplement they will no longer weaken to the cross crawl. If they still weaken, try again with other supplements associated with the organ/gland until you find the one that satisfies the protocol. This criteria should be used when giving any supplement for any reason. Not only can a

supplement abolish N.D., but it can also create it. Always check a supplement against the cross crawl and right or left unilateral crawl to assure its effectiveness. If it creates a weakness with either of these, it should not be used regardless of what it may have strengthened.

If the pineal cranial fault challenge is negative, evaluate the supraspinatus against supplements for the hypothalamus and pituitary to differentiate between them. Simply provide the positive supplement.

c) If neither the emotional NV points or glabella abolishes the cross crawl weakness, then use the meridian pulse points to determine the organ/gland dysfunction that is effecting N.O. This is by far the most common occurrence. Find the one pulse point that abolishes the weakness and then T.L. its related alarm points to identify the one meridian involved. Return the patient to their original strength using one of the two methods previously mentioned. Then evaluate the muscles related to that meridian. They will usually test weak. First test them against the emotional NV points. [To reiterate emotional trauma can effect N.O. directly by actually creating N.D., or indirectly by creating a dysfunction in an organ/gland, etc... that ultimately leads to N.D.]. If the emotional NV points abolish the weakness use step 3a. If not, then test them against whatever supplements you use associated with that organ/gland in the same manner as described for the pineal, hypothalamus and pituitary. For most of the meridians the organ/gland association is self explanatory. However, several meridians are associated with more than one system. The circulation sex (CX) meridian is associated with the reproductive organs and the adrenals, the spleen (SP) meridian with the spleen and pancreas, the triple heater (TH) meridian with the thyroid, thymus[22] and parathyroid.[23] When one of these meridians is involved you must differentiate to determine the exact organ/gland. After the patient is returned to their original strength only the muscles associated with the offending organ/gland will weaken and not all of the muscles associated with that meridian.

If the cross crawl does not create a weakness then immediately proceed to the evaluation of the cortex. If it does, then wait until the dysfunction at that level is resolved. When the dysfunction is structural or emotional the resolution is usually immediate. When it is chemical allow 3 to 4 days of supplementation before re-evaluation.

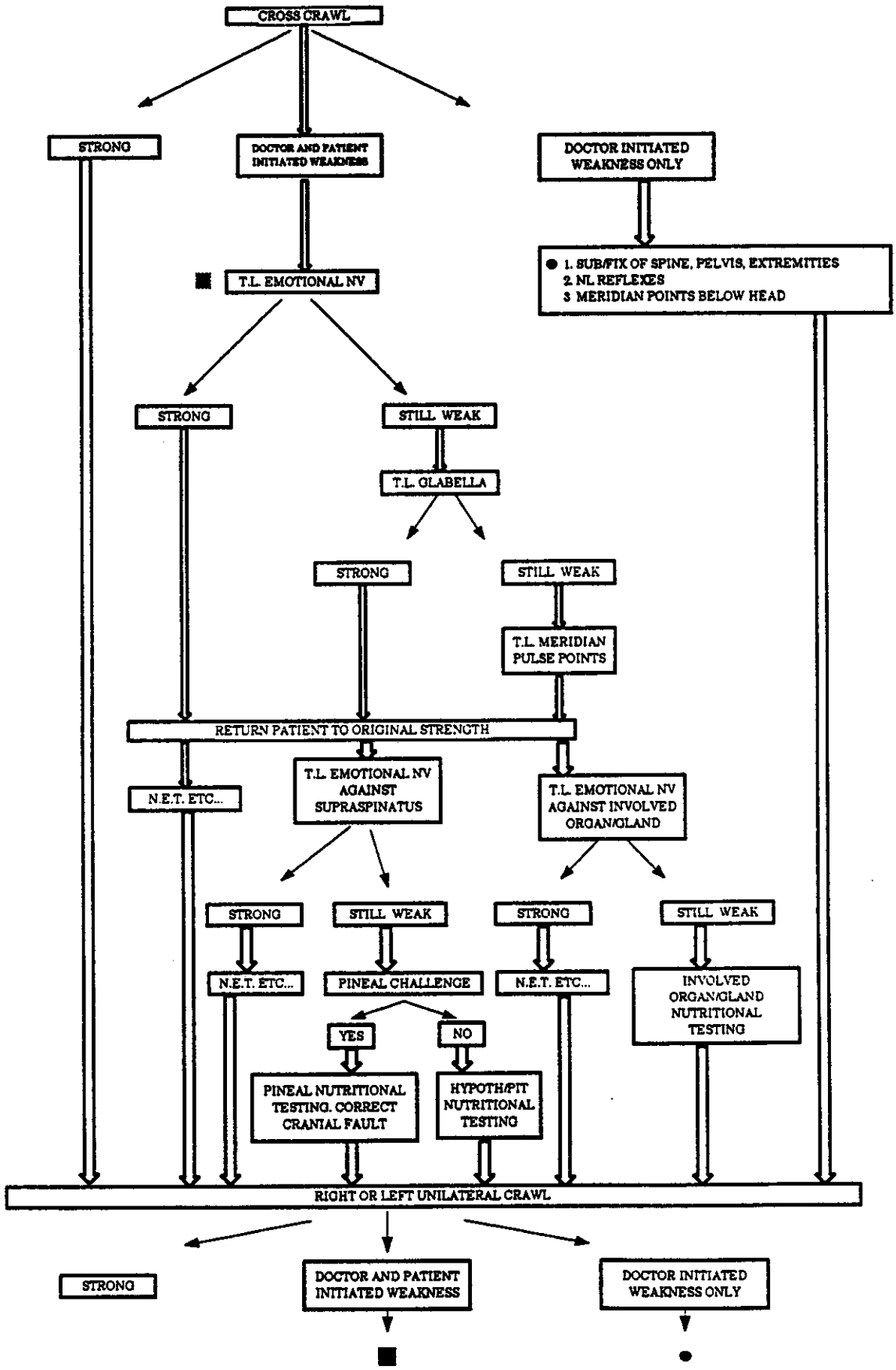
4) To evaluate for N.D. at the cortex one can use what I call the right or left unilateral crawl. First ask the patient with which hand they write. If right, then have them cover their left eye with their left hand, first making sure it does not T.L. Then have them raise and lower their right leg and arm simultaneously about 10 cycles while keeping their right eye open. This activates 3 (leg, hand and eye) out of the 4 (leg, hand, eye, and ear) areas that are used in a skilled role which are consistent with the dominant hemisphere. In this case the left hemisphere. It is usually not necessary to evaluate the auditory function unless there is an obvious speech

disability. If they write with the left hand, then the procedure is simply reversed. The right hand is over the right eye, while they raise and lower their left leg and arm simultaneously; keeping their left eye open. If the unilateral crawl creates doctor initiated weakness only, then use step 2a. If both a doctor and patient initiated weakness then use step 3a or b or c. The only difference between this level and mid-brain/early cortex is that the cross crawl is used instead of the homolateral crawl as one of the two methods for restoration of the patient's original strength.

CONCLUSION

This procedure has been extremely effective in dealing with problems of all kinds; including learning disabilities, digestive disturbances, endocrine imbalances, recurrent structural problems, immune deficiencies, cardiovascular problems, allergies, headaches, back and neck pain, etc. It is extremely accurate in identifying the cause and prioritizing the treatment. And can easily be incorporated into your practice utilizing the supplements in which you are already familiar. In fact it is a treatment in itself and should only take 5-10 minutes.

NEUROLOGIC DYSORGANIZATION AT THE CORTICAL LEVEL - KUBICEK - PAGE 10



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A PROPOSED TESTING PROCEDURE FOR DIETARY LECTIN INCOMPATIBILITY

Herbert Kuehnemann, RRT, DC

ABSTRACT

Dietary lectin incompatibilities found on the D'Adamo Serotypes Polymorphisms (DSP-1) and applied kinesiology screening procedures are examined. Oral challenge testing and therapy localization demonstrate a correlation with dietary lectin incompatibility.

INTRODUCTION

Blood typing is performed by placing a drop of the sample blood onto a slide and mixing anti-A or anti-B substance with it and observing an agglutination reaction for a positive response or no agglutination for a negative response (1,3). Kidney bean Phaseolus vulgaris extract is used as a phytohaemagglutinin in anti-A agglutination reactions. Other food substances are used for different blood typings (2). These substances which choose different blood group cells are termed lectins (3). Lectins are cell surface recognition markers comprised of glycoproteins and glycolipids that protrude through the outer cell wall. They can be thought to act like a velcro strip surrounding the food cell wall that sticks to the cell wall of incompatible blood and tissue types . In the alimentary tract, agglutination occurs with non-compatible cells and distorts the cytoarchitecture of the intestinal wall which may contribute to malabsorption conditions (4). Certain lectins have an affinity for gastrin-secreting cells and parietal cells (5,6). Other lectins bind to various areas in the alimentary-tract and non-alimentary areas (2). This investigation was limited to lectin reactions in the stomach.

PROTOCOL

Serum and saliva specimens were obtained and sent for analysis (7). The DSP-1 [Sero-sort] report results were explained to the patients. Food samples on the "avoid or minimize" list were brought into the office for comparison. The patients were also instructed to bring in any foods that they wanted screened for possible lectin incompatibility problems. Patients were evaluated for cranial-respiratory, TMJ, switching, cervical subluxations, and treated using applied kinesiology procedures. The immune system challenge procedure of Dr. Schmitt was performed (8). This consisted of placing different foods on the tongue and therapy localizing (TL) to the upper sternum thymus neurolymphatic reflex (NL), lower sternum (pectoralis minor NL), liver NL, and spleen NL. If the challenge was positive, the patient performed the cross crawl ten times while therapy localizing (TLCC) to the involved NL with the food still on the tongue. This was done to counteract any known anti-nutritive effects of food on the physiology. The patient was then instructed to therapy localize (2.5 centimeters below the xiphoid process) over the stomach, or to the stomach NL or neurovascular (NV) reflex points.

Sample foods on the DSP-1 "avoid or minimize list" were insalivated, chewed, and retained on the tongue by the patient. The strong indicator muscle became weak. After the food was swallowed, the stomach no longer therapy localized. Therapy localization with cross crawl had no effect upon the lectin challenge. The lectin challenge could be repeated on the same patient to reproduce the testing response.

DISCUSSION

According to Freed (9), lectins can affect many bodily functions including gut immunity, allergies, and auto-immunity. It was observed that on more than one occasion, after ingesting certain lectin incompatible foods that the patients became switched. The patients then complained of headache and fatigue consistent with food allergies. There appeared to be a quantitative immune response with different offending foods. One subject with A2/Rh positive blood had mild allergic symptoms after eating grapefruit. It was listed on the DSP-1 "avoid or reduce" list. The grapefruit challenge did not produce a positive therapy localization to the stomach, but did trigger a delayed switching response. Beef and pork were listed by D'Adamo on the "reduce or avoid list" for the A blood groups because of paleoserological data (7). He believes the A blood groups are deficient in hydrochloric acid production and cannot properly digest these heavier proteins. Beef and pork were tested on these patients, and they did not therapy localize using this procedure, nor did they demonstrate any switching response or symptoms. The positive lectin challenges usually responded quickly, but a few subjects demonstrated that chewing and insalivation took a few seconds before the strong indicator muscle weakened. Due to anatomical variations in subjects, effective therapy localization required mild pressure 2.5 centimeters inferior to the xiphoid process in a posterior and superior direction to be certain that contact was made over the stomach.

CONCLUSION

D'Adamo organized his testing based upon Dr. Nathan Sharon's work from the 1970's (7). D'Adamo suggests that this test be done at birth to give the parents a guide to proper dietary choices for their children for their whole lives . The adverse lectin effects never change because these are physical-chemical reactions not under the modulation of the immune system (3). Freed has written a classic review article explaining the potential broad range of lectin effects on the human body (9). According to Freed (2), lectins are causes in search of diseases. He also suggests that lectin effects can be blocked by administering the proper sugar inhibitors (3). This therapy has not been investigated using applied kinesiology methods by the author. Freed's peer reviewers comment that many of these observations occur *in vitro*, and have not been observed *in vivo* (9). Applied kinesiology testing methods initially appear to have a high correlation *in vivo* with the DSP-1 report results. Larger controlled studies are needed to obtain valid comparison statistical data.

Lectins and AK: testing procedure, page 3, (Kuehnemann)

The proposed lectin challenge procedure may be of significant value in the clinical setting. The doctor can confirm the results of DSP-1 quickly and easily. This serves as a valuable teaching tool for patient education and compliance. Additional foods can be tested for inclusion or exclusion from the patient's diet. More AK research needs to be done to develop protocols for determining the non-allergic effects of lectins on pathophysiology. It is this author's opinion that the now obscure DSP-1 should be upgraded to a more routine component of the applied kinesiology evaluation.

SUMMARY OF PROCEDURES

1. Screen the patient for cranial-respiratory, TMJ, switching, cervical subluxations and treat using applied kinesiology procedures.

2. Perform immune system challenge procedure of Schmitt. Place sample foods on the tongue and TL to thymus NL at the upper sternum and lower sternum, liver NL, and spleen NL. If challenge is positive, have patient perform cross crawl 10 times while therapy localizing the involved NL (TLCC). It is not necessary to turn their head to the side. Retain the sample food in the subject's mouth.

3. TL to the stomach or NL or NV. A positive lectin challenge will weaken the strong indicator muscle.

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Common Nerve Entrapments of the Lower Extremity

David Leaf

Abstract: This paper will discuss the most common nerve entrapment syndromes encountered in the lower extremity. It will cover quick diagnostic tests to aid in the finding of the condition and offer some treatment options to speed the recovery of the patient.

The lower extremity presents a number of locations that peripheral nerve entrapments can occur. These can cause great confusion in the diagnosis of exactly where the root cause of the patients symptoms is coming from.

There are basically three areas where nerves become entrapped. They are in the pelvis, around the knee and in the foot.

In the pelvis, the fifth lumbar nerve root can be entrapped by the iliolumbar ligament, the inguinal ligament can effect the femoral nerve, the piriformis muscle the sciatic, inferior gluteal , inferior gluteal nerves and the obturator nerve can become entrapped as it exits the obturator foramen.

Around the knee, the tibial nerve can be entrapped as it passes by the popliteus muscle and the peroneal nerve can be stretched as it traverses around the head of the fibula.

In the foot, nerve entrapments occur in the tarsal tunnel, the anterior tarsal tunnel and under the metatarsal heads.

Each of these conditions has its specific muscular and sensory findings. Basic knowledge of the underlying neurology will aid in your diagnostic skills.

In all of these conditions, you will generally find that injury has been sustained by the local muscles, ligaments, osseous structures, and skin. Proper treatment requires a consideration of all of these factors. In general, you will find that a chronic weakness of a muscle will result in laxity of the ligament that performs the same task as the involved muscle. As part of the defence nature of the body, another muscle will overcontract becoming hypertonic in an attempt to stabilize the joint. It is this muscle weakness, ligament laxity and muscle hypertonicity that is the underlying cause of most of these entrapment syndromes.

In each case, treatment must be rendered to the supporting structures. These include the supporting muscles and ligaments. Skin reflexes need to be tested for. Muscle coordination problems as evidenced by reactive type weaknesses will need to be tested and corrected. In most of these conditions, support will need to be given to the joints to prevent a return of the problem.

Lumbosacral - Iliolumbar Ligament Syndrome

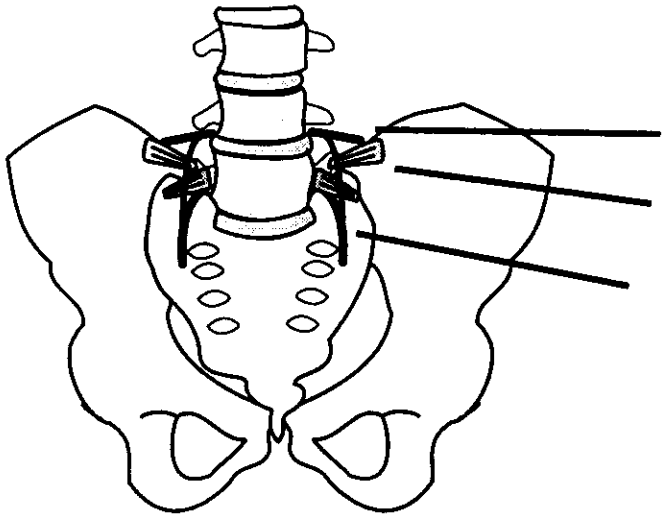
The fifth lumbar nerve root loops anterior after leaving the intervertebral foramina. It then traverses under the iliolumbar ligament and superior to the sacrum. The superior surface of the sacrum and the inferior surface of the iliolumbar ligament form a tunnel. Pecina, Krmpotic-Nemanic and Markiewitz term this the lumbosacral tunnel syndrome (12). After passing through this tunnel the fifth lumbar nerve joins the fourth lumbar nerve, which has traversed over the anterior surface of the ligament, to form the lumbosacral trunk. Peripheral nerves that are partially derived from the fifth lumbar nerve root include the superior and inferior gluteals, the sciatic, the common peroneal and the tibial nerves as well as the sacral plexus.

Entrapment of the nerve root is a result of alterations in the length of the iliolumbar ligament. Goodheart, based on the work of Illi, described a condition in which the iliolumbar ligament could effect the coordination of the body in a gait configuration. Basically, his test for involvement of the iliolumbar ligament was the failure of muscles to be properly inhibited in a gait position when the patient stepped backwards. In these cases, treatment was rendered as to shorten the ligament.

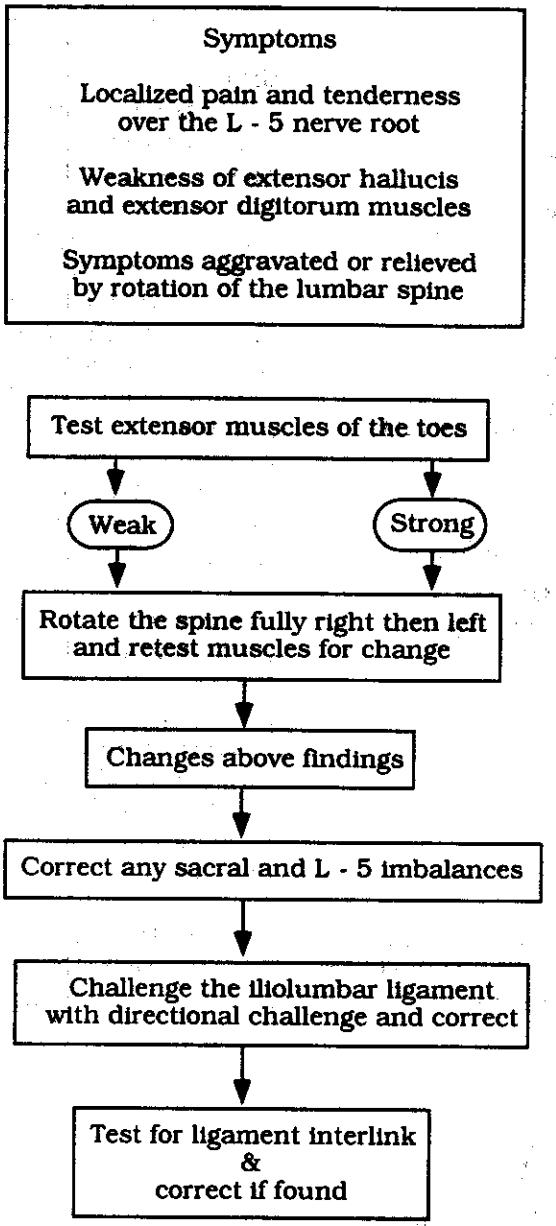
Localized injury to the iliolumbar ligament, usually from a rotational type force, can cause localized swelling that causes entrapment of the fifth lumbar nerve (6).

To test for this involvement, the patient may have to twist or lateral bend in a sitting position to create the entrapment. Conversely, rotation or lateral bending may relieve the entrapment causing a relief of symptoms and/or strengthening of any related structures. As the fifth lumbar nerve is part of the superior gluteal, inferior gluteal and sciatic nerves, almost any muscle of the lower extremity with the exception of the psoas, iliacus, quadriceps, sartorius and adductors can be used for testing.

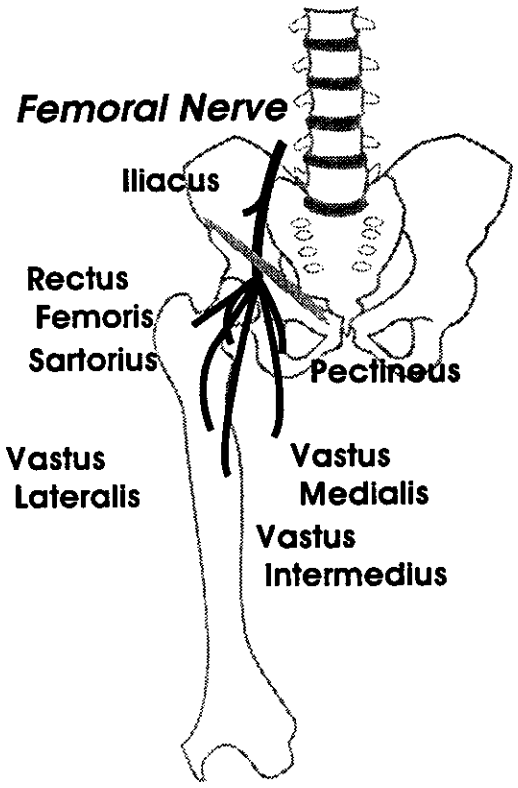
An interesting observation is that if the ligament is shortened and needs to elongated, the opposite gait inhibition pattern will be found. If the ligament is lengthened and needs to be shortened, the original Goodheart finding, the patient must step back in a gait pattern to disclose the failure to inhibit the gait muscles. If the ligament is shortened and needs to be lengthened, the patient will fail to show inhibition in the normal gait pattern.



Notice how the fifth lumbar nerve root descends under the iliolumbar ligament and then is joined by fibers from the fourth lumbar nerve root.


Symptoms
Localized pain and tenderness over the L - 5 nerve root
Weakness of extensor hallucis and extensor digitorum muscles
Symptoms aggravated or relieved by rotation of the lumbar spine
Test extensor muscles of the toes
Weak
Strong
Rotate the spine fully right then left and retest muscles for change
Changes above findings
Correct any sacral and L - 5 imbalances
Challenge the iliolumbar ligament with directional challenge and correct
Test for ligament interlink & correct if found

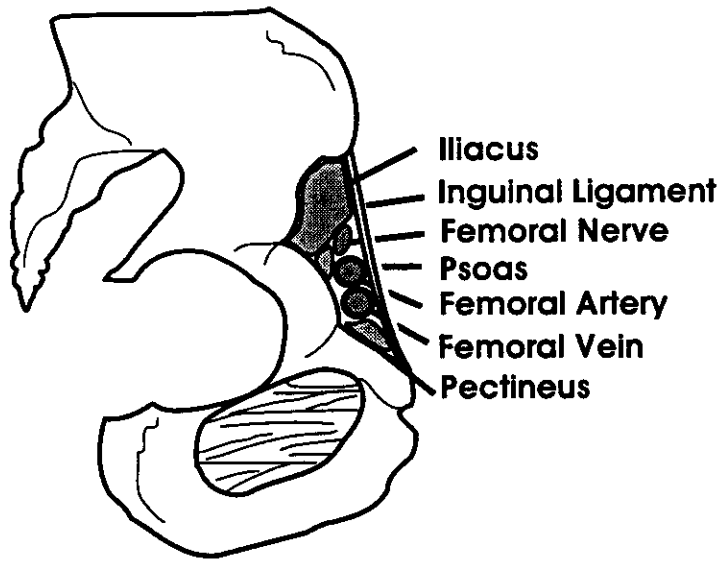
Iliopsoas - Inguinal Syndrome



The femoral nerve and the femoral vascular structures accompany the iliopsoas as its passes beneath the inguinal ligament. The inguinal ligament attaches to the anterior superior iliac spine and extends down in an arc to its attachment on the pubic tubercle. It has a firm fascial connection to the fascia lata and the iliac fascia. An aponeurosis of the ligament forms a support for the spermatic cord. When viewed from an oblique angle, the inguinal ligament forms the superior margin of a tunnel like structure with the border on the innominate forming the inferior margin of the tunnel. In this tunnel is the iliacus, the psoas, the femoral nerve, the iliopectineal fascia, the lumboinguinal nerve, the femoral artery and vein, the femoral ring and the lacunar ligament. The femoral ring may be of clinical importance because it is just anterior to this that the spermatic cord or the round ligament of the uterus are found.

The femoral nerve descends between the fibers of the psoas and the iliacus to exit inferior to the inguinal ligament. It supplies the iliacus superior to the inguinal ligament. The psoas is supplied by fibers from the second and third lumbar nerves. The first muscles innervated after the nerve clears the inguinal ligament are the pectineus and the sartorius. Inferior to this, the nerve supplies the entirety of the quadriceps muscle.

The physical signs of femoral nerve entrapment are obvious. The patient will have weakness of the quadriceps muscle with strength of the iliacus. This condition should be suspected in any patient that has had localized trauma over the inguinal ligament, lower abdominal surgery or increased pain when the femur is extended. For example, when testing the gluteus maximus, the patient states that pain is increased in the groin area. The patient will have difficulty climbing stairs first and then arising from sitting. The quadriceps muscle will become atrophied.



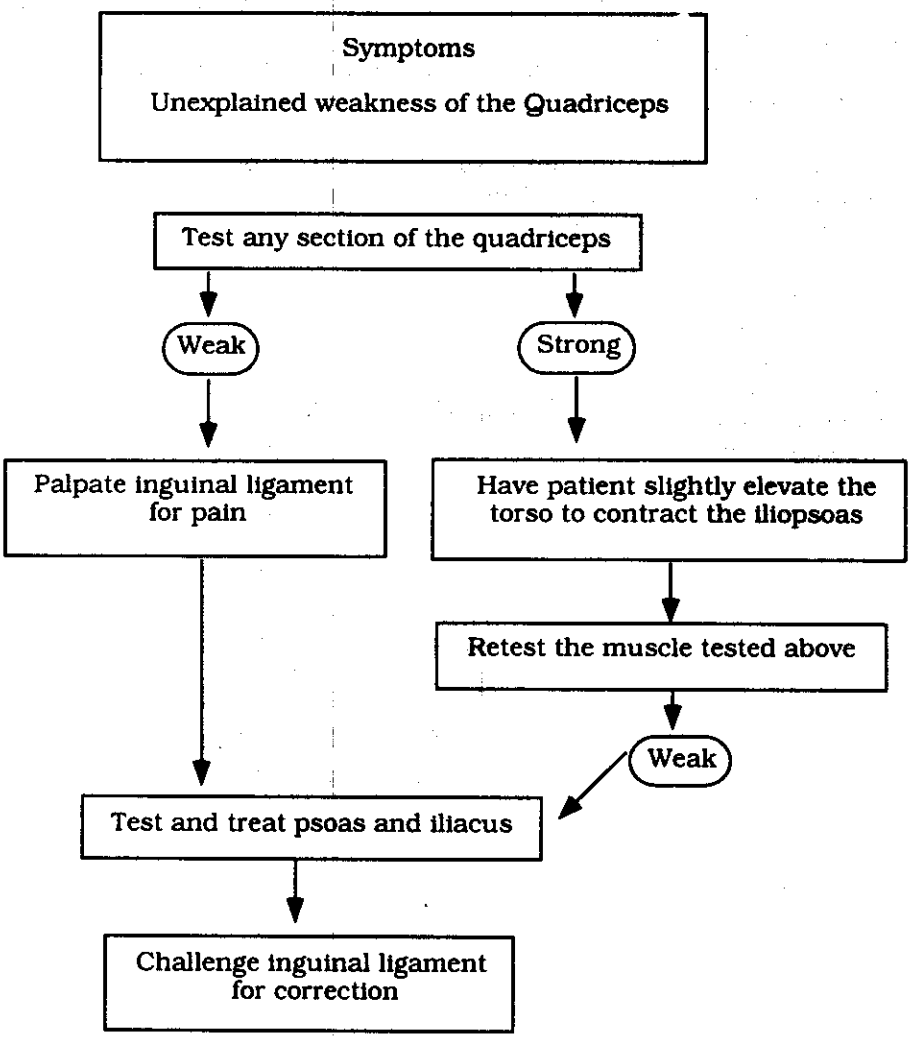
Injuries which can cause the iliacus or the psoas to enlarge or cause a shortening of the inguinal ligament can cause entrapment of the femoral nerve. This is fairly easy to diagnose if you keep in mind that the iliacus is supplied above the ligament and the quadriceps below.

Ask the patient to perform a task which would use the psoas and ili-

acus. Immediately test for weakness of the rectus femoris or the vastus muscles. If weakness is found, challenge the inguinal ligament for shortening by pressing on the ends of the ligament and pulling towards the attachments of the ligament. If this stops the weakness pattern, continue to treat the ligament and balance all muscles that attach to the ligament.

An example of this condition was a patient who was a highly competitive runner on the international scene. She moved her household goods from one location to another and within two weeks of the move developed knee pains. She consulted numerous other practitioners about the leg pains which continued to worsen to the point where she could not run over 100 meters without leg pain. Prior treatment had included manipulation to the pelvis three times a week for six weeks, myofascial release techniques to the quadriceps, three sets of orthotics and four different anti-inflationary medications.

Examination revealed no signs of weakness when the patient was first examined. She was placed on treadmill and her walking gait was observed. She exhibited excessive hip rotation on walking which indicated to me that she was not using her psoas to bring her femur anterior. Testing at this point still showed no weakness pattern. The treadmill was increased in speed so that she could jog. After only one minute, the rectus femoris and the vastus muscles tested weak. Further testing revealed localized tenderness over the inguinal ligament and a positive testing for shortening of the ligament. Questioning revealed that she had rested heavy boxes against the ligament during the move that proceeded the symptoms.



After correcting the ligament, she was instructed to perform the elongation of the ligament at home for the next week. Within ten days, she returned to training with no pain.

Obturator Syndrome

The obturator nerve is the motor supply to the adductor muscles. This nerve is composed of three sections from the second, third and fourth lumbar nerve roots. It descends along the medial posterior border of the psoas. Then it crosses the sacroiliac joint and then superior to the pubes traverses to enter the obturator foramen. The obturator tunnel is composed superiorly by the pubic bone and inferiorly by the internal and external obturator muscles. The tunnel holds the obturator artery and vein as well as the obturator nerve.

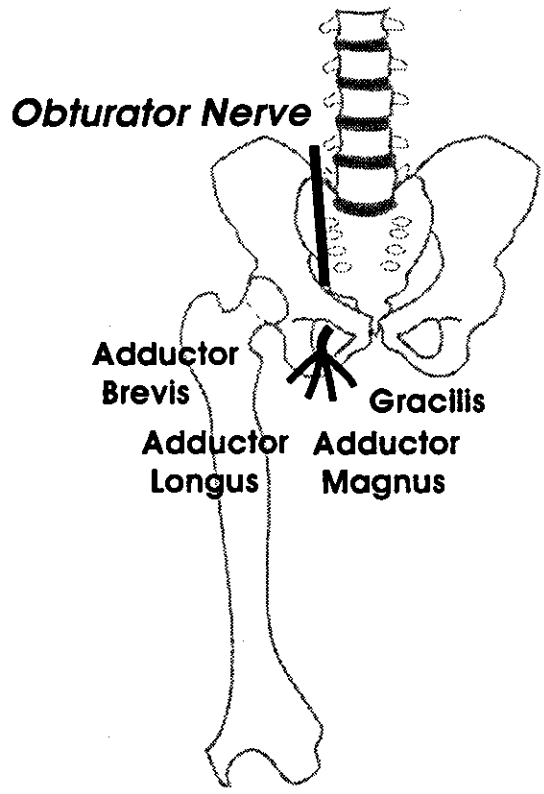
This nerve entrapment syndrome is commonly found during pregnancy, localized trauma like horseback riding and lower abdominal surgery (12).

The symptoms can range from localized numbness to pain radiating down the medial aspect of the thigh to the medial aspect of the knee. The adductors will test weak and there maybe visible atrophy of the muscle. The patient will begin to walk with the leg away from the midline with a noticeable lateral sway of the body.

It is this last finding that will help you to find this condition. If you suspect that the adductors are weak and you cannot find the weakness, have the patient bear down as in the Valsalva test and test the adductors. The increased abdominal pressure may cause increased pressure on the obturator nerve as it exits the pelvis. This is common in cases of visceroptosis.

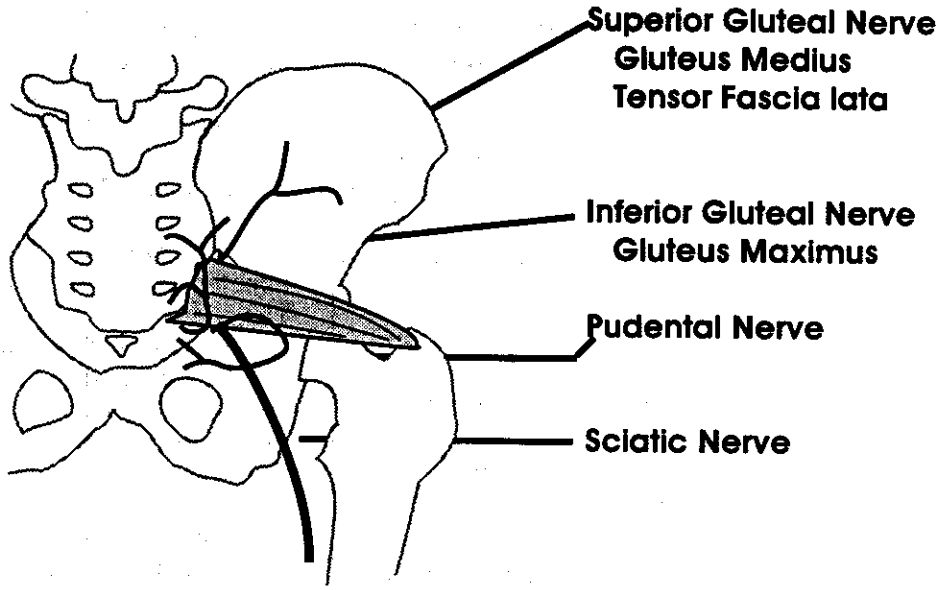
In general, the treatment consists of supporting the abdominal contents. Evaluation of the sacral base angle should be made and any alterations from normal stabilized. The integrity of the various sections of the abdominal muscles must be evaluated and corrected.

One of the uncommon causes of obturator neuropathy is pelvic cancer. Rogers and Borkowski reported, in the August 1993 issue of Neurology, on cases of pelvic cancer that had produced obturator nerve symptoms as the sole presenting symptom. Half of the patients had ipsilateral leg edema accompanying the nerve findings. The tumors included lymphoma, cancer of the bladder, pelvic papillary carcinoma and cancer of unknown origin (13).



Piriformis Syndrome

The author first wrote about the piriformis syndrome in 1992. The following is a brief review of that paper. For a more in-depth discussion, you are referred to that article (10).



The piriformis is a thick muscle that arises from the anterior surface of the sacrum by separate digitizations which attach between the first, second, third and fourth anterior sacral foramina. Attachments are also possible at the sciatic foramen, the capsule of the sacroiliac joint and the sacrospinous ligament. The muscle attaches to the greater trochanter of the femur on its medial superior surface by means of a tendon that is joined by the tendons of the obturator internus and the gemelli muscles.

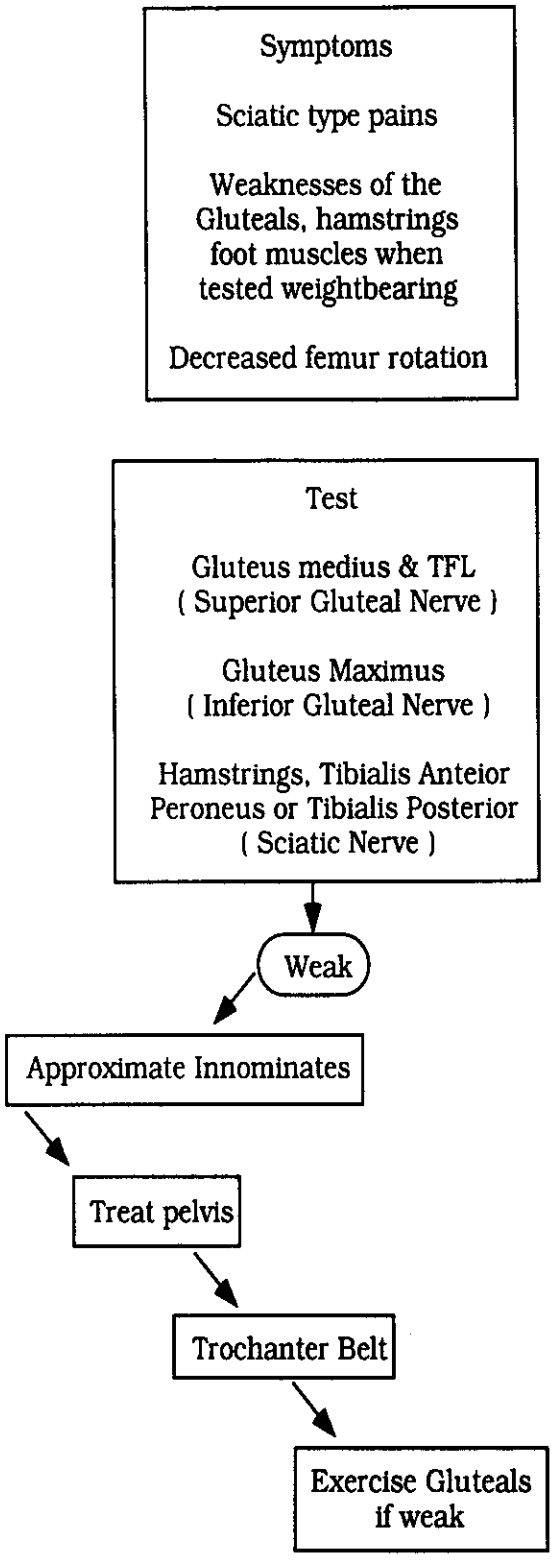
The greater sciatic foramen is bounded superiorly and anteriorly by the ilium. The posterior portion is formed by the sacrotuberous ligament and the inferior margin by the sacrospinous ligament. Within this foramen pass nerves and blood vessels as well as the piriformis. Superiorly, the superior gluteal nerve and blood vessels pass between the ilium and the superior margin of the piriformis. The gluteal nerve supplies the gluteus medius, the gluteus minimus and the tensor fascia lata muscles. Inferior to the piriformis and adjacent to the greater sciatic foramen pass the sciatic nerve, the pudendal nerve and blood vessels. Additional nerves which pass through the notch with the piriformis include the inferior gluteal, the posterior femoral cutaneous and those which supply the quadratus femoris, the obturator internus and the gemelli muscles. The inferior gluteal nerve is important in that it supplies the gluteus maximus muscle.

Any weakness of the gluteus maximus causes over contraction of the piriformis creating entrapment of at least some of the structures discussed above.

If weakness of any of the structures that are innervated by the nerves that pass through this area are found, either have the patient reduce the weight on the pelvis (if testing in a sitting position) or belt the

pelvis using a trochanter type belt to approximate the sacroiliac joints. In either case, the related muscle weakness patterns will strengthen.

One of the difficulties in diagnosing this condition is the fact that the piriformis changes its role as a rotator of the femur by 180 degrees when the femur is raised above 90 degrees. This presents problems when the patient is tested in a sitting position. If the patient leans forward, they can change the angle between the femur and the pelvis enough to cause the function of the piriformis in femur rotation by 180 degrees. This phenomenon is easily demonstrated by finding a weak piriformis with the femur flexed to 85 degrees. Raising the femur to 100 degrees flexion will result in a strong test. Reversing the test angle so that you are pressing on the lateral side of the lower leg and pressing the leg towards the midline will reveal the weak piriformis.



Fishman and Zybert describe a testing procedure for use when performing an EMG test on the sciatic nerve. In this test they adduct and flex the affected leg and then internally rotate the leg. They use this to apply pressure on the nerve from the piriformis. This same position can be used to aid in diagnosing the piriformis condition by testing for a weakening of a strong muscle that is innervated below the piriformis while holding this leg position (4).

The major problem is correcting this condition is the relative strength of the gluteus maximus muscle. Weakness of a muscle will result in chronic weakness of the underlying ligaments. This combination of weak muscle and ligament laxity results in overcontraction of another muscle attempting to stabilize the joint. This is a common finding throughout the body. Proper treatment requires correcting the muscle, correcting the ligament, correct manipulative procedures and possible bracing of the joint.

Popliteal Syndrome

In the posterior knee, the popliteus and the soleus muscles form an arch through which the popliteal artery and vein and the tibial nerve pass.

Due to the softness of the borders of the popliteal and soleus muscles, entrapment of the tibial nerve is rare. The major symptoms are of entrapment of the vascular structures.

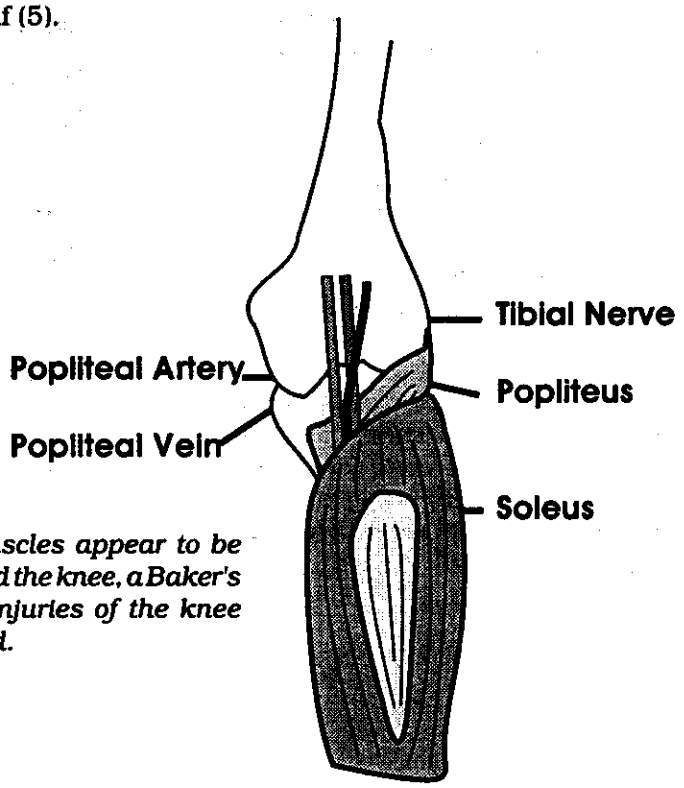
Intermittent claudication may occur in well-conditioned athletes because of an unusual form of popliteal artery entrapment that results from overtraining. These patients complain of calf muscle cramping, rapid limb fatigue, and occasional paresthesias on the plantar surface of the foot when running on inclines or when repetitive jumping is performed.

Intermittent claudication following activity is the most common finding. The patient may complain of numbness, leg cramping or feelings of cold. Intermittent claudication may occur in well-conditioned athletes because of an unusual form of popliteal artery entrapment that results from overtraining. These patients complain of calf muscle cramping, rapid limb fatigue, and occasional paresthesias on the plantar surface of the foot when running on inclines or when repetitive jumping is performed.

Palpation of the dorsal pedis pulse may show variations of the pulse when the foot is plantarflexed or dorsiflexed. If nerve entrapment is suspected, test for weakening of foot muscles with the foot forcefully plantarflexed and with the lower leg fully internally rotated contracting the popliteus muscle. The most important physical finding is local tenderness over the area of suspected entrapment.

The history will usually reveal a hyperextension injury of the knee or a rotational type injury. The physical findings will reveal trigger points in the popliteus that will respond to either the strain counterstrain procedure or the spray and stretch technique.

Geissler, Corso and Caspari have reported, in the British Journal of Bone and Joint Surgery, that the popliteus can become ruptured and create a palsy of the tibial nerve. This was due to pressure on the neurovascular bundle in the proximal calf (5).



Suspect this condition if the calf muscles appear to be hypertrophied, localized trauma behind the knee, a Baker's cyst or in cases of hyperextension injuries of the knee where the popliteus has been injured.

Peroneal Tunnel Syndrome

In the superior portion of the popliteal fossa, the common peroneal nerve is formed. The nerve then wraps around the head of the fibula and then descends to pass inferior to the attachment of the peroneus longus muscle. The nerve then passes through the peroneal tunnel at the level of the neck of the fibula.

In cases of lateral deviation of the fibula caused by stretching of the ligaments that hold the head of the fibula against the tibia, the nerve becomes stretched.

The first muscles that will test weak are those innervated by the deep peroneal nerve, especially the peroneus tertius. As the condition worsens, all of the muscles supplied by the superficial and deep peroneal nerves will test weak. There will usually be palpable tenderness in the heads of the gastrocnemius. The weakness and the tenderness will be relieved when the head of the fibula is approximated against the tibia.

This condition is more common than you would expect. Common findings unrelated to the peroneal nerve are increased tenderness in the gastrocnemius and possible weakness in the lateral hamstrings.

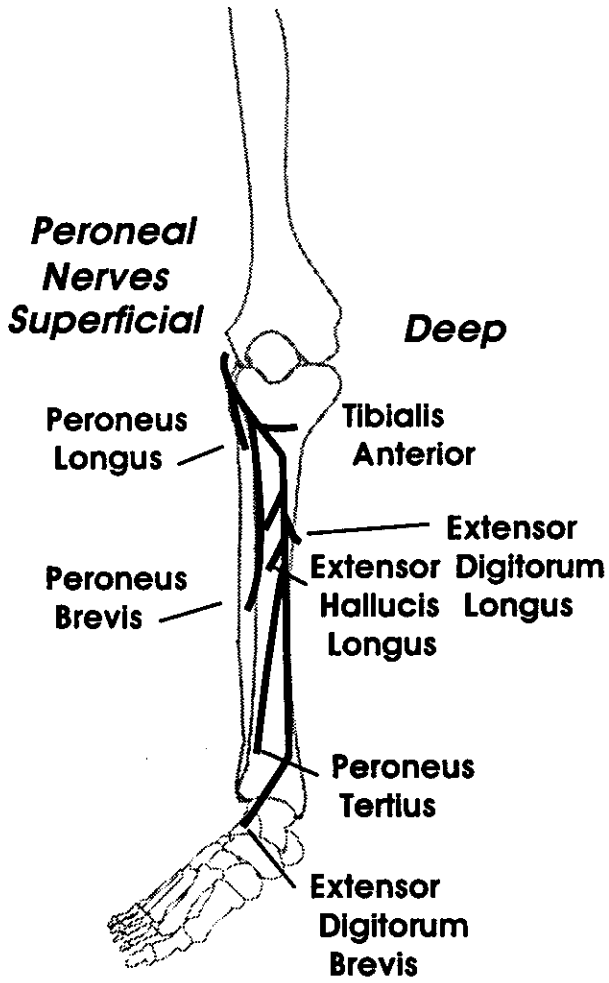
You will see the failure of the peroneus muscles to control the foot in the walking pattern and the muscles will test extremely weak. If the patient is asked to approximate the fibular head, there will be an immediate lessening of the palpable pain in the gastrocnemius and a dramatic increase in strength of the peroneal muscles.

The fibular head must be treated in a similar fashion to the radial head in elbow problems. The joint must be approximated, the ligaments treated and the joint stabilized by a support.

It is common for this to take four to six weeks for the ligament to gain the strength to support the fibular head.

Care must be taken to examine the gait so that the person is not landing with a flat foot. Even though the fibula is supposedly a non-weightbearing bone, a common finding is to find that the patient is very heavy on their feet causing excessive jarring of the lower leg when they walk.

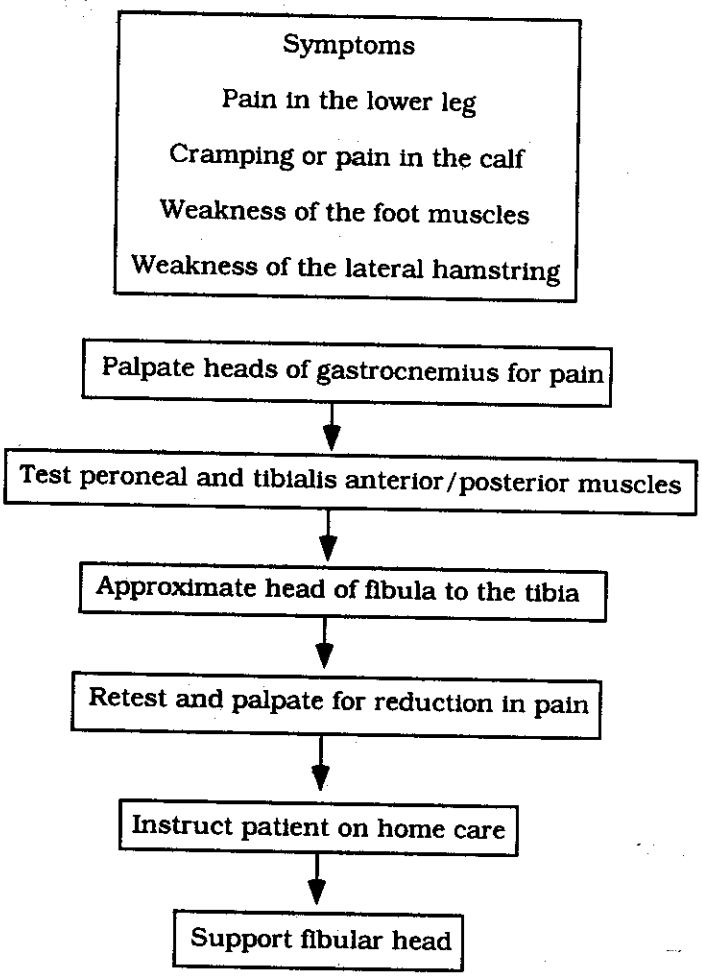
In extreme cases, a compression wrap around the lower leg has aided the healing. This is accomplished by using a three to four inch cloth wrap that the patient applies each morning before walking. It is worn throughout the day and removed only at bedtime.

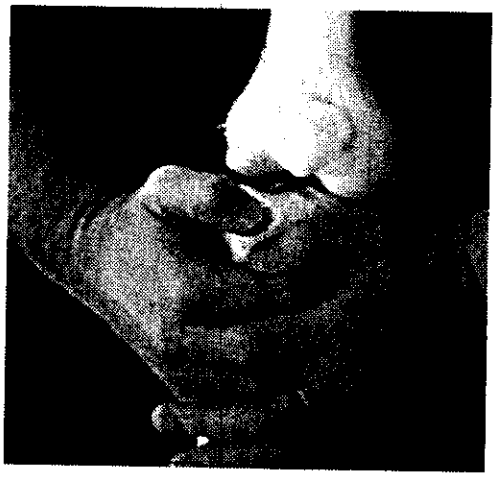


This condition is commonly misdiagnosed as a lumbar disc problem. The symptoms include lower leg pain as well as muscular weakness that make it appear like a chronic disc problem. In fact, one patient was scheduled for disc surgery and found to have this type of nerve entrapment.

The patient was a male nurse, approximately 71 inches in height and 375 pounds in weight. He had chronic leg problems and was scheduled for disc surgery to relieve the pain he had in his lower right leg. Examination revealed palpable pain in the gastrocnemius and weakness of the foot muscles that increased in strength on approximation of the fibular head. It took eight weeks of taping and support and his symptoms have not returned in two years.

Another patient had fallen skiing and torqued the right knee. There was localized tenderness around the head of the fibula. She had been treated by a well known orthopedist who specializes in knee problems and placed on crutches for three months. After being on the crutches, she began to trip over the smallest objects. Examination showed that she was not lifting her foot prior to heel strike. She had weakness of the peroneal muscles that strengthened on approximation of the fibular head.





This picture shows the position that I have the patient place their hands to approximate the fibular head. The base of the thumb is placed posterior to the fibular head and they squeeze their hands together. This moves the head medially and slightly anterior.

Anterior Tarsal Tunnel Syndrome

This syndrome occurs when the deep peroneal nerve is entrapped as it passes beneath the inferior extensor retinaculum. The floor of this tunnel is formed by the fascia overlying the talus and the navicular. Within the tunnel, beside the nerve, are four tendons, an artery and a vein. The retinaculum is a strong band and can easily cause excessive pressure on the deep peroneal nerve (13).

Symptoms

Pain or neuralgia over the dorsum of the foot

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Palpate dorsum of foot for pain

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Have patient plantarflex foot and test extensor digitorm brevis muscles

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Challenge talus for an anterior subluxation

↓

Manipulate

↓

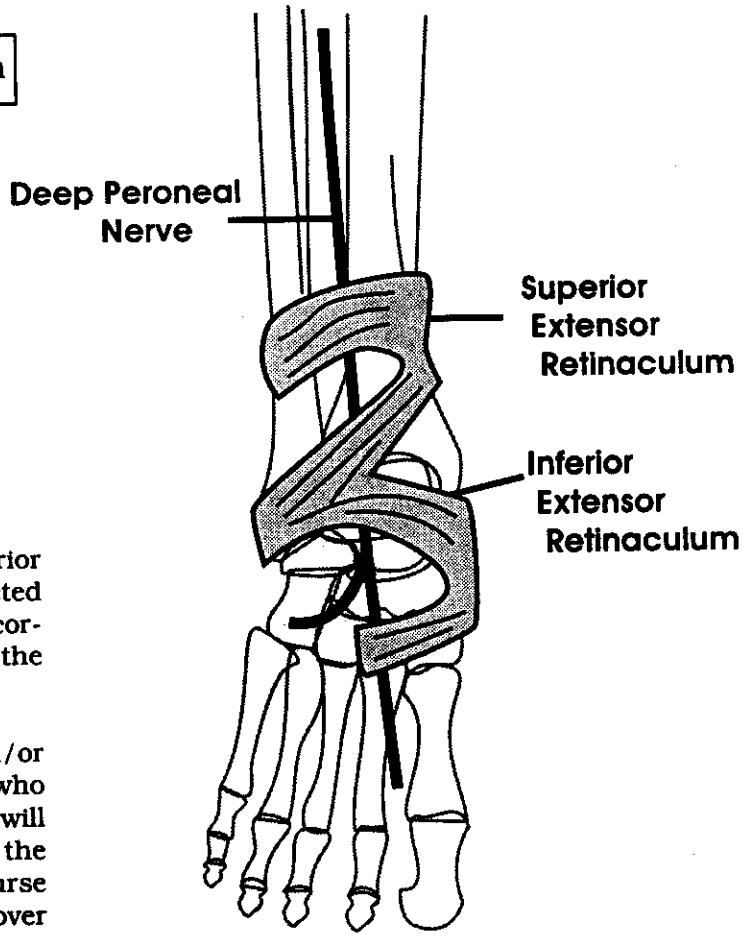
Test for reactive pattern between tibialis anterior & gastrocnemius

The most common finding will be an anterior displacement of the talus. This is easily corrected with proper manipulative techniques. After correction, the ligaments and skin overlying the talus should be challenged for involvement.

Examine the patient for their walking and/or running style, you will usually find a person who is landing on their toes. This excessive force will cause the talus to slip anterior. Instruct the patient on landing on their heels. This is of course impossible to do if the person wears heels over one inch in height.

Anterior slippage of the talus is the initiating factor in this condition. This is common in people who wear elevated heels on their shoes or boots. Localized trauma should also be suspected.

The primary sensory symptom is one of pain between the first and second toes or a deep ache through the dorsum of the foot. The motor weakness is most prominent in the extensor digitorum brevis muscle. The foot may have to placed in full plantarflexion to find this weakness pattern (1).



Tarsal Tunnel Syndrome

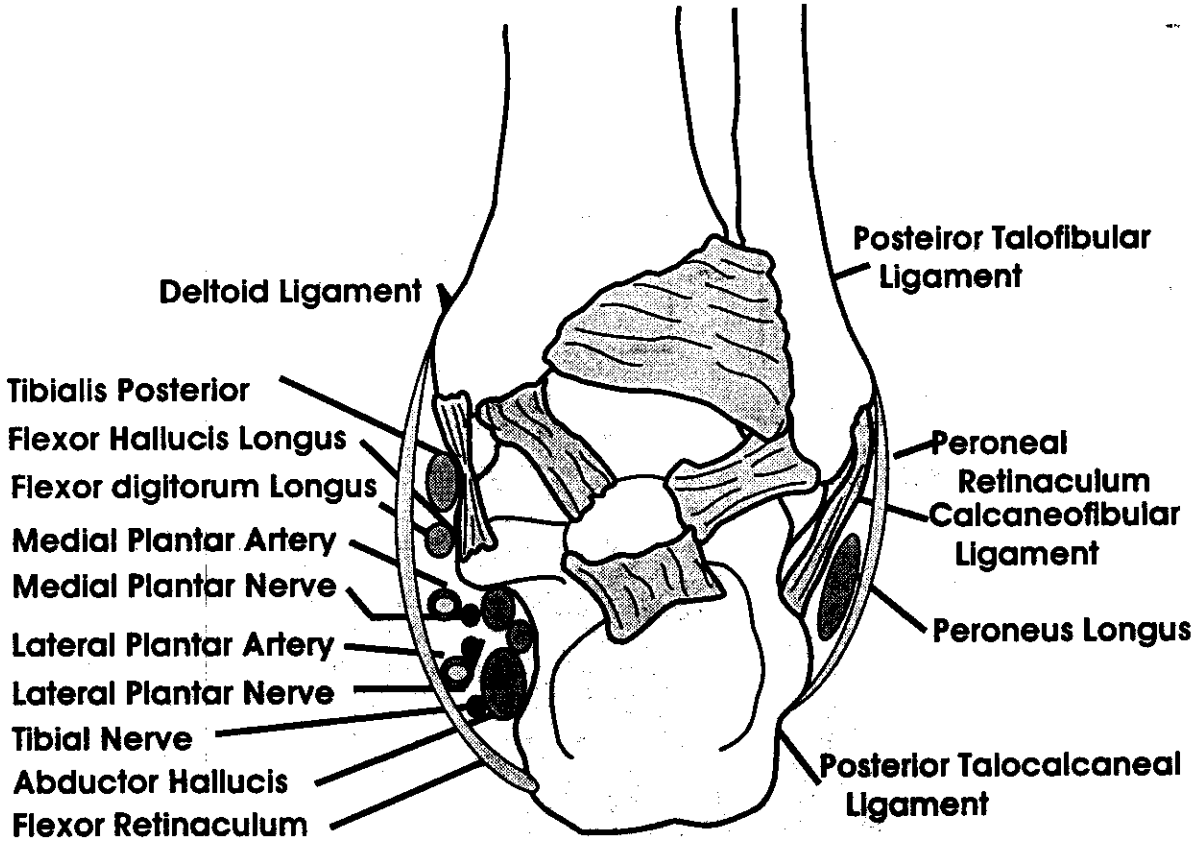
The tarsal tunnel is found inferior and posterior to the medial malleolus. The lateral boundary of the tunnel is formed by the bones and the medial boundary by the flexor retinaculum. Through this tunnel pass the tibial nerve, the medial and lateral plantar nerves, the medial and lateral peroneal artery, the tibialis posterior, the flexor hallucis longus and flexor digitorum longus muscles.

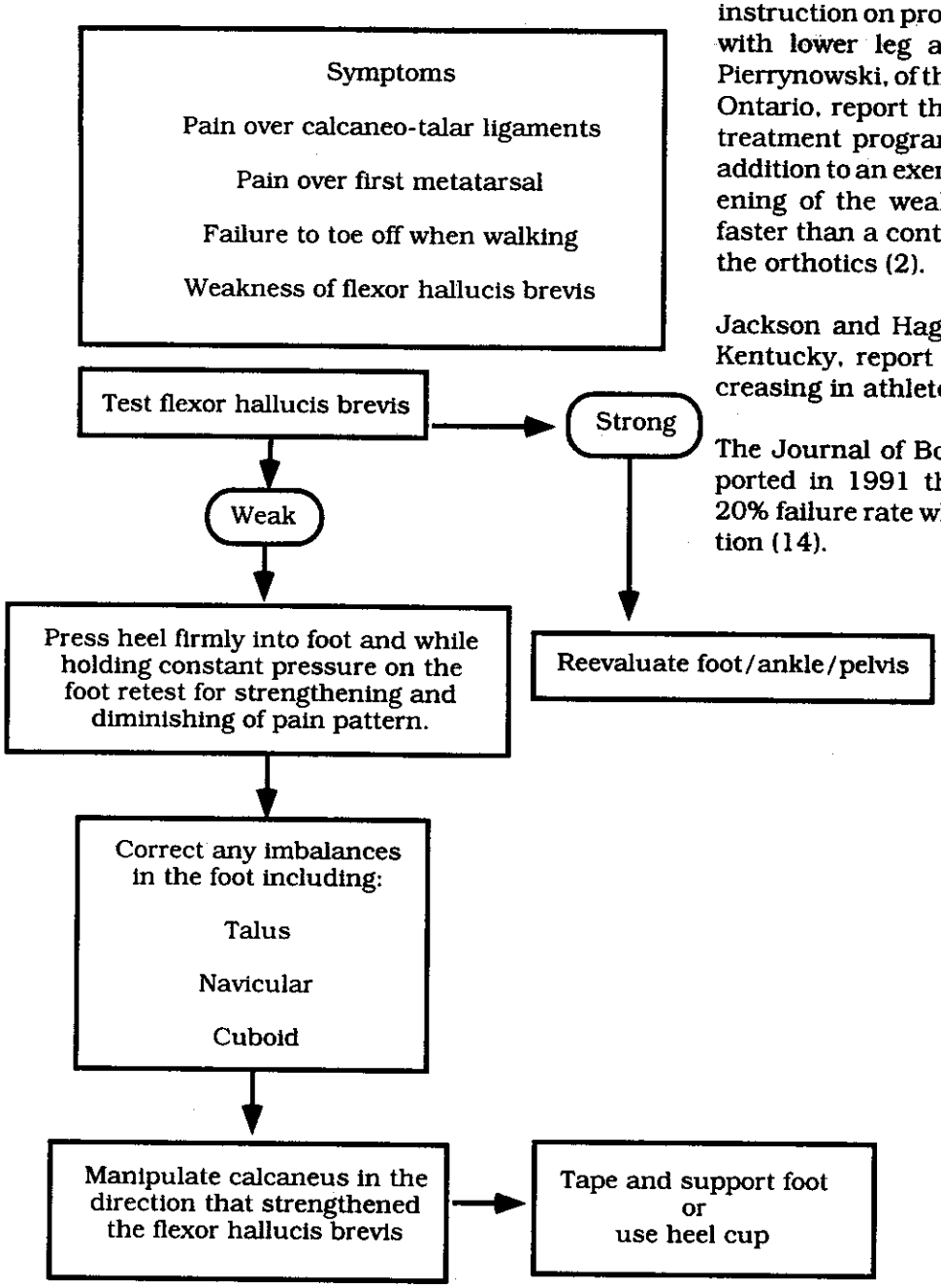
The flexor hallucis brevis is the major muscle innervated after the tarsal tunnel. Entrapment of the structures in the tunnel will cause alterations in the vascular supply to the foot and weakness of the flexor hallucis brevis. Chronic involvement leads to changes in the plantar muscles that will cause hammer toes - or a claw toe. While some consider this condition to be infrequently found, this condition will be found to accompany almost every case of bunion formation and chronic pronation.

This condition commonly is found when the navicular has dropped inferior. This causes the calcaneus to be moved in a posterior direction causing increased pressure on the flexor retinaculum. Other localized problems can produce a narrowing of the tunnel. These can result from chronic injury to the area, gouty formation of crystals, edema, etc. (3).

A common finding is the patient who lands on their foot in a horizontal position and does not use the big toe to push off. This causes excessive stress on the ligaments and muscles of the foot that slowly causes the calcaneus to move in a posterior direction. In general, improper stride and mechanics of landing on the feet are common causes of this condition.

Correction requires supporting the arch of the foot, normal function of the tibialis posterior, manipulative procedures to normalize the position of the talus, navicular and the calcaneus and

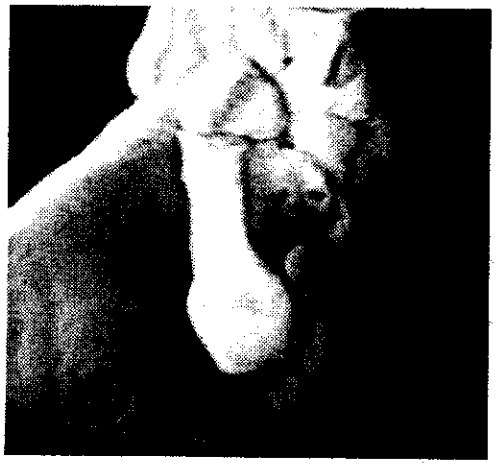




instruction on proper gait. In a paper dealing with lower leg and knee pain, Eng and Pierrynowski, of the University of Waterloo in Ontario, report that patients that were in a treatment program using soft orthotics in addition to an exercise program for strengthening of the weakened muscles recovered faster than a control group that did not use the orthotics (2).

Jackson and Hagland, of the University of Kentucky, report that this condition is increasing in athletes (7).

The Journal of Bone and Joint Surgery reported in 1991 that surgery resulted in a 20% failure rate when applied for this condition (14).



The calcaneus must be challenged in an anterior direction to approximate the calcaneus back into its normal physiological position. Usually you will find that there is tenderness over the first metatarsal that will be dramatically diminished when the calcaneus is compressed into the foot.

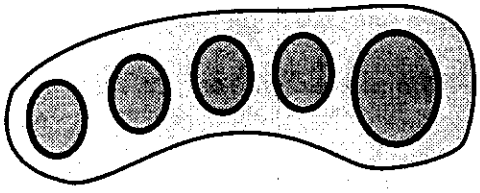
Metatarsalgia

The metatarsal tunnels lie between the superficial and deep transverse metatarsal ligaments. Within these tunnels lie the medial and lateral plantar nerves, arteries and veins.

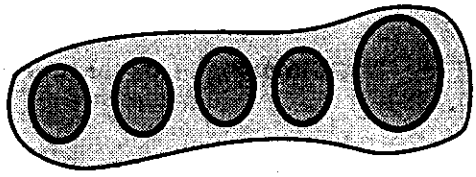
Entrapments of the nerves in these tunnels in known by many names. The most common name being Morton's neuroma. In general, entrapments are caused my misalignments of the metatarsals. Other causes include edema, fractures, inflammatory conditions, sesamoid bones, etc..

Pain can easily be palpated for over the dorsum of the foot. When this is found, place tongue depressors under the metatarsal arch elevating it. Repalpate the dorsum of the foot for a reduction of pain. There will also be weakness of the extensor muscle of the great toe. This is easily tested with the patient standing. Have the person bring their weight forward over the front of the foot and test for weakness of the toe extensors. This weakness pattern will also be relieved if the metatarsal arch is elevated. In cases where the patient complains of pain when wearing shoes, grasp the foot and compress the metatarsals and palpate for pain.

Correction involves correcting all foot imbalances and elevating the metatarsal arch.

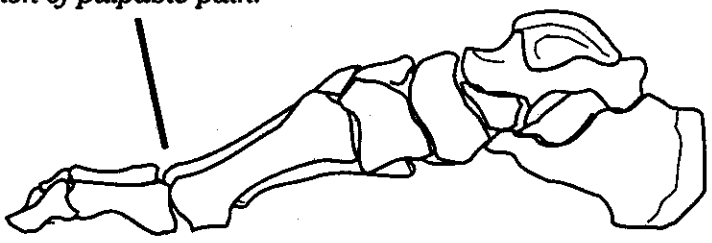


Normal alignment of the metatarsals



Dropping of the metatarsals.

Location of palpable pain.



This paper has attempted to summarize the possible nerve entrapments of the lower extremity. While orthopedists are inclined to treat these conditions with cortisone injections and surgery, conservative care will many times result in complete alleviation of these conditions. Care needs to be taken in considering the existence of these conditions when evaluating patients with sensory and motor weaknesses in the lower extremity.

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DYSBIOSIS

MICHAEL LEBOWITZ D.C. & HARRY LEFKOWITZ D.C.

Abstract: This paper is a discussion and a description of dysbiosis. It will discuss what dysbiosis is and its etiology. Various types of dysbiosis will be addressed, and the symptoms involved will be presented.

Dietary habits may lead to changes in the intestinal flora over a period of time. Also, drug interactions may have an adverse effect on the intestinal environment.

A method of diagnosis and treatment of the syndrome is presented. Treatment with natural antifungal substances, herbs, and vitamins and minerals is presented. Also, the treatment of the syndrome by treating acupuncture points and neurolymphatic reflexes is discussed.

Introduction: Recognition that the intestinal flora has a major impact on health first developed with the birth of immunology in the late 19th century. A normal healthy relationship with the indigenous gut flora is termed "eusymbiotic," meaning a state of living together that is beneficial. Metchnickoff popularized the idea of "dyssymbiosis" or dysbiosis, a state of living together with the intestinal flora that had harmful effects.[1]

He postulated that toxic amines produced by bacterial putrefaction of foods were the cause of degenerative disease and that ingestion of fermented foods containing lactobacilli could prolong life by decreasing gut putrefaction.[2] Although Metchnikoff's ideas have been largely ignored in the United States, they have influenced four generations of physicians in Europe. Dysbiosis is a state in which the normal flora of the intestines is altered to the point where toxic metabolites are produced and are interfering with normal chemical pathways, thus producing a state of ill health.[3]

Effects of Normal and Abnormal Flora in the Gut: It should be made clear that dysbiosis is not a disease. It is a theory that attempts to explain the breakdown of health in the human body.[4] There may be one symptom or many symptoms. They may be acute and frequent or chronic and infrequent. The symptoms may be confined to one system or may be multisystemic. It may be that a symptom or group of symptoms may be classified as a "disease" such as Irritable Bowel Syndrome, Chronic Fatigue Syndrome, or Sinusitis. However, the focus of identifying and treating dysbiosis is to help heal the whole body, not to treat a particular disease. Dysbiosis puts the body into a state of dis-ease which may later manifest into a disease. It is important to keep this concept in mind as we discuss dysbiosis. The stool of the healthy human being consuming a western diet is approximately 85% microorganisms. Twenty species comprise 75% of the total number of colonies. Anaerobes predominant over aerobes by a ratio of 5,000 to 1.[5] In the colon, the predominant microorganisms are spirochetes and fusiform bacteria and anaerobic rods such as Eubacterium, Bacteroides and Bifidobacterium. The ileum is predominant in Coccobacilli. The stomach and small intestine are predominant in Lactobacilli and yeast.[6]

The benefits of the normal gut flora are the following:

1. Synthesizes vitamins - biotin, cobalamine, pantothenic acid, pyridoxine, riboflavin, and vitamin K.
2. Synthesis of fatty acids.
3. Degrades toxins and xenobiotics.
4. Prevents columnization by pathogens.
5. Stimulates maturation of normal immune system response.
6. Demethylation of methyl mercury.
7. Synthesis of antibiotics.[7]

Gorbach in a study in 1986 presented research which suggests that a diet high in meat leads to significantly higher fecal counts of anaerobic bacteria and rods. People eating a diet high in complex carbohydrates and low in meat have higher counts of aerobic bacteria and lower counts of certain anaerobic bacteria which have pathogenic capability.[8] Other factors that may affect intestinal flora are hydrochloric acid and digestive enzyme production. A deficiency in production of these in the body may lead to incomplete protein digestion with accompanying putrefaction and overgrowth of pathological anaerobic bacteria.[9] Lack of adequate fiber intake and increased transit time also lead to putrefaction and overgrowth of pathological organisms.[10]

Some of the common bacteria in immune testings are Eubacterium lentum, Bacteroides, Clostridia, Klebsiella and Bifidobacteria. Lactobacillus is present mainly in the small intestines and stomach while the colon contains spirochetes and fusiform bacteria.[11] Small bowel bacterial dysbiosis may cause abdominal distension, carbohydrate intolerance, malaise and fatigue. The symptoms may be very similar to intestinal Candidiasis.[12]

Toxic metabolites of GI flora may include ammonia, phenol, carcinogenic lithocholates, nitrites, nitrosamines, ethanol and acetone.[13] Leakage of these metabolites into the systemic circulation may cause irritation of various tissues.[14] Dysbiosis has been implicated in vitamin B12 deficiency, steatorrhea, irritable bowel disease, autoimmune disease, eczema,[15] cystic acne and genesis of colon and breast cancer.[16] Two decades ago exaggerated immunological responses to the components of the normal fecal flora were proposed as a possible mechanism in the etiology in inflammatory bowel disease.[17] In one study, 30% of patients hospitalized for Crohn's disease had bacterial overgrowth of the jejunum.[18] In ulcerative colitis, damage from passage of metabolites of bowel acids has been suggested.[19] Immunological responses to gut flora have been advanced by several authors as important factors in the pathogenesis of inflammatory joint diseases. In some cases, bacterial antigens have been found in synovial cells and may enter the circulation because of increased intestinal permeability.[20] Strong evidence suggests that various arthritides such as ankylosing spondylitis and rheumatoid arthritis are related to immunological responses to the gut flora.[21] Due to changes in the gut wall anatomy, invasive organisms may have access to the systemic circulation and cause immune reactions in the joints.[22]

Yeast and Fungus: Yeasts are small elliptical cells which make up a small percentage of gut flora. There are 81 strains of yeast. Candida albicans is the most common. The gut bacterial flora keeps yeasts in check. Antibiotics, steroids, estrogens birth control pills can stimulate the overgrowth of yeasts. This is then worsened by diets high in refined carbohydrates, especially sugars and corn syrup. Yeasts can then transform into the fungal or mycelial form in which they develop hyphae which may attach to the intestinal wall. This in turn leads to increased intestinal permeability.[23] The patient may then become hypersensitized to Candida albicans and its metabolites. This then leads to sensitivity to food sources of yeast, fermented products and refined carbohydrates which cause further growth of yeast.[24]

Vaginal dysbiosis is the result of persistent colonization of the vagina from dysbiotic fecal flora that migrate from the anus and peri-anal area into the vaginal and urethral area.[25] Some things associated with yeast and fungal overgrowth are atopic diseases, urticaria, asthma, irritable bowel syndrome, chronic fatigue syndrome, myalgia, PMS and vaginal dysbiosis. Increased intestinal permeability, which may be a secondary stage or the mycelial stage, can lead to leakage of pathogens which then initiate an immune system response. This brings into play abnormal immune system responses which may lead to the genesis of the arthritides, recurrent febrile episodes, and autoimmune disorders.[26] The stronger the patient's defenses, the longer it takes for the bowel wall to become leaky and for the immune system disorder to develop.

Intestinal Parasites: According to Galland, there is a high prevalence of intestinal protozoan infection in the United States. There are three major classes of Protozoa in the lumen of the intestines. These are amoebae, flagellates, and ciliates. The organisms of greatest clinical interest are the flagellates, Giardia lamblia, and the amoebae, Entamoeba histolytica. Other pathogens of importance are Cryptosporidium, Blastocystis hominis,[28] Endolimax nana, and Trichomonas hominis. Giardia lamblia is the most common parasitic disease in the United States. Almost 20% of urban North American adults harbor antibodies to Giardia lamblia. The U.S. Department of Health, Education and Welfare reports that 90% of Giardia lamblia infections are acquired from drinking contaminated water or exposure to diaper age children attending childcare centers. Classic risk factors such as foreign travel and male homosexuality play a relatively small role in the epidemiology of chronic giardiasis.[29] Common symptoms associated with protozoan infections are fatigue, colon disorders, asthma, urticaria, myalgias, headache, allergies and rheumatologic disease.[30] Often the patient with protozoan infection will be diagnosed with irritable bowel syndrome. The mechanisms of how protozoans produce disease is largely unknown. Postulated theories are (1) T-lymphocyte response to parasitic infection, (2) production of toxic metabolites by parasites which leads to local irritation and systemic symptomatology, (3) RNA virus transfer by parasites which may act as hosts for these viruses suggests that they may account for chronic viral symptoms in patients with protozoan infections.[31]

Discussion: The technique to discern, monitor, and correct dysbiosis has been evolving over a number of years. Before, we would test stool, and mucus samples biomagnetically.[32] If they weakened a strong indicator muscle, by seeing which

antifungal, antibacterial, and antiparasitic substances negated the weakness we could get an idea of the type of condition we were dealing with, and a rational course to pursue. The inconvenience of this, plus the reluctance of the practitioner and patient to handle the samples made us seek alternative methods.

We found that if we took the appropriate "killer" substance (antibacterial, antifungal, antiparasitic, etc.) and tested it biomagnetically over an area where the organism was causing "problems" that a weak G-2 muscle would strengthen. As a rule we search over hollow areas such as the intestines, sinus cavities, vagina, etc. where the organism is more likely to inhabit. We find in some cases the organism appears to be systemic in the sense that the "killer" strengthens no matter where it is placed, while in other cases it might just strengthen over one small area such as a four inch segment of the transverse colon. This led us to believe there are pockets of dysbiosis. Other practitioners in the natural health care field have since lent their support to this idea of "dysbiotic pockets". Doing this type of testing over time has also helped us to find the highest quality most broad spectrum products to use.

Supplementing appropriately, while taking people off foods they showed neuro-muscular hypersensitivities to, plus doing a neurological desensitization (e.g. master set points), brings wonderful clinical results in most cases. You must be careful how you communicate to the patient since you are really not making a diagnosis of fungus, bacteria, or parasite infestation. Dysbiotic syndrome may be an appropriate term based on the patient history and exam findings. We have seen hundreds of chronically ill patients become asymptomatic doing this technique in combination with treating food sensitivities and other environmental factors,[33] as well as replenishing their nutrients. Energy levels rise, weight drops in the obese, brain fog and headaches clear, chronic subluxations and joint pains cease to be a problem, etc. Any musculoskeletal problem that does not respond very quickly to basic applied kinesiology is checked for the "dysbiotic syndrome" as it can cause swelling to settle into traumatized areas. We have seen all kinds of bizarre "idiopathic" problems resolve. For interesting cases refer to issues of the "Sproadic Newsletter".[34]

Conclusion: Correcting the "dysbiotic syndrome" is critical in the chronic patient. We consider it possibly the most important thing you can do. Testing for it and treating it is fairly simple though in a small minority of patients resolution can by quite difficult. We have barely touched the surface in this paper but welcome further questions if we can be of service.

Summary of Procedures:

1. Take the following substances and see which strengthen a G-2 weakness when placed over the colon, small intestine, lungs, sinus, bladder, reproductive organs, etc. Some may be negative, some may strengthen all over, some just over a small infectious or dysbiotic pocket:

    a) SF722[35] or Undecyn[35] - if positive suspect fungus or candida. To differentiate between candida and other fungi, see if a powdered antigen mix of candida albicans and tropicalis[36] causes universal muscle weakness when tested

under the magnet over the same area the positive supplement was tested. If it does suspect candida, if not suspect another fungi.

b) Artecin[35] or Citricidn[35] - if positive suspect parasites (occasionally a fungus will be negated by these)
c) Phytogen[35] or Goldenseal[37] - if positive suspect bacteria
d) Echinacea Supreme [37] - if positive suspect virus
e) Berbercap[35] or Enterocap[35] - effective against bacteria, parasites, and fungi, but usually indicative of bacteria.

2. Massage the dysbiotic pocket if appropriate and the corresponding NL. This can be quite uncomfortable.

3. Treat the master set points. The G-2 muscle should now be strong.

4. If fungi or candida were positive the patient must abstain from the following foods until fungal findings are clear: all sweetening (sugar, honey, maple syrup, corn syrup, etc.), fruit juice, dried fruit, vinegar, cheese, yeast, alcohol, soy sauce and other fermented products. The patient must also avoid all foods they are hypersensitive to.

5. Supplement with whatever products negated the weakness in the following doses:

a) SF722- two capsules 3x/day or Undecyn one cap 3x/day. We give only one of these products if both test positive (I prefer SF722). Occasionally less than three weeks of supplementation is needed. In these cases the patient becomes symptom free after a week of so, only to have fatigue and headaches recur after two or more weeks. Leaving off the supplement will restore symptom free status at that time (the same goes for the products listed below).

b) Goldenseal- 10-20 drops in water three times daily

c) Phytogen- one cap three times daily

d) Echinacea Supreme- 20 drops in water three times daily

e) Citricidin, Artecin- one cap 3x/day; use whichever negates the weakness (use both if both positive)

f) Berbercap or Enterocap- one cap 3x/day (use only one of these)

6. Warn patients that for up to a week they may experience a temporary aggravation of symptoms due to die off or withdrawal.

7. Recheck positive findings in three weeks (most often they are negative).

a) If fungus is still positive, you need to see if the patient weakens on an air sample taken from the bedroom or place of employment. If positive ozonate.[38] You must also check and treat if needed their sexual partner. If the patient is on steroids or estrogen they may not clear until they have been

off of them for six weeks. We also double our antifungal dose if it is still there. If the patient ever needs to take antibiotics or steroids after they are clear, tell them to take SF722 or Undecyn simultaneously (3 per day) to prevent recurrence.

b) If parasites still test positive check a sample of the patients water. If it weakens and is negated by the anti-parasite supplement, filtration will be necessary.

c) If bacteria or virus is still positive, increase supplement dose, take the patient off sweetening also.

d) If the formerly positive supplements test negative, discontinue it, but continue appropriate vitamins, minerals, etc. as testing dictates.

8. To minimize the chance of recidivism, food sensitivities should be tested for and positive foods avoided at the same time as treating all of the above.

9. Sometimes things show sequentially and you might be treating fungi one visit, parasites the next, etc.

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ORGAN DYSFUNCTION: A NEW PROCEDURE

by MICHAEL LEBOWITZ D.C.

ABSTRACT: Evaluating organ dysfunction via applied kinesiology analysis is a useful experimental procedure most of us use. Procedures we feel enhance this along with treatment procedures are discussed.

INTRODUCTION: One thing that we all have learned over the years of doing applied kinesiology is that as we put the body into stress via torques, exposure to noxious substances, focusing on certain thoughts, etc. etc., not only do we find more "hidden" subluxations, positive reflexes, etc., but also that correcting these findings while the body is under the stress often brings dramatic results. We have been told to "fix what we find" and in most cases the more we find and fix the better our results. According to the syntonic optometrists as well as many of the pioneers in color therapy looking at or through the color red either stimulates the sympathetic nervous system or inhibits the parasympathetic nervous system. Conversely looking through or at blue stimulates the parasympathetic or inhibits the sympathetic nervous system. They have clinically used these principles claiming very good results. We decided to use this information to "irritate" patients with already unbalanced nervous systems and see if it enhances applied kinesiological findings.

DISCUSSION (PROCEDURE): Taking a strong indicator muscle a patient would therapy localize the following neurolymphatic reflexes (anterior ones if available) and we would see which would cause the strong muscle to weaken: adrenal, thyroid, reproductive, pancreas, stomach, large intestine, small intestine, liver, pineal (GV-20), pituitary, spleen, heart, lungs, gall bladder, bladder. We would record our findings.

The patient would then either put on red lenses or look at red construction paper (to the exclusion of everything else) and we repeated the "organ screening". In 80% of patients between 2-12 more neurolymphatic reflexes would test positive during this procedure. Results were recorded.

The patient would then either put on blue lenses or look at blue construction paper (to the exclusion of everything else) and we repeated the tests. In 10% of patients this procedure would "bring out" more positive reflexes. Results were recorded. In almost all cases (90%) either red or blue (very rarely both) brought out many more findings than testing without using color as a stressing agent.

Looking through or at the stressing color we then use two point therapy localization to find one organ neurolymphatic reflex that would therapy localize to the rest of the positive ones. We feel this is the major organ weakness. Then therapy localizing this reflex (still looking at the color) we would find a nutrient

that negated the reflex. This nutrient was almost never a glandular (with the exception of Pineal Plus[1] when pineal was major). The liver or adrenals were the major organ in the vast majority of patients. S.A.T.[1] for the liver or B Complex #5[1] for the adrenals were the nutrients that show up most frequently.

We then stimulate the reflex while the patient looks at the stressing color and have the patient do follow-up treatment twice daily at home for one week as well as take the supplements.

Patient response to this procedure appears to be very positive with good clinical change. Occasionally on subsequent visits a different pattern occurs and additional treatment is needed. The procedure also appears to lower the patients total load as many previously positive findings become negative. You can also use that "stressful color" to find many more muscle weaknesses, subluxations, etc. and treat them in a similar manner.

CONCLUSION: Stressing the nervous system via syntonic optometry procedures is a very useful technique to both evaluate and treat most patients.

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Functionally Weak Muscle Response After Visceral Manipulation

Thomas A. Rogowskey, D.C., DIBAK

Abstract

Functionally weak muscles can be displayed and facilitated using a combination of visceral manipulation, Dr. Walter Schmitt's injury recall technique and his set point theory. Food and chemical substances which may prove noxious to a person, and do not cause a muscle to be functionally weak, can cause a muscle to weaken when visceral manipulation is performed and set points are simutaneously activated.

Introduction

Visceral manipulation has been introduced as an effective and useful technique for body work in many disiplines such as osteopathy, applied kinesiology and sacro-occipital technique.(1,2) Previously, muscle tests were not reliable indicators of the need for visceral manipulation. The use of set points increases their reliability.

Dr Schmitt introduced the injury recall technique (IRT) in combination with the use of set points as a technique to help facilitate functionally weak muscles and their associated organs.(7,8) It is thought that strengthening functionally weak muscles will have a positive effect on those muscles and their associated organs. I have used this technique as a foundation for my recent work and have found ways of eliciting weaknesses in specific muscle groups. Some of these techniques have been developed in conjunction with Dr. Erik Carlin.(3) In addition, foods and other substances that had not previously elicited a weakness did so when this technique was used. The results of this display of functional weakness often confirm subjective patient observations of chemical sensitivities.

Definition: **set points:** Set points are the yang meridians' beginning and end points and are located on the face.(5) Each point represents two coupled meridians. For example, the large intestine (LI) and lung (Lu) set point is located bilaterally next to the nares of the nose and is the last point on the LI meridian(LI20). In other words, the LI/Lu meridians are represented by this particular set point. The other coupled meridian set points are represented by the gall bladder/liver(GB1), the bladder/kidney(B1), the triple warmer/circulation sex(TW23), the stomach/spleen-pancreas(ST1), and the small intestine/heart(SI19).

Dr. Schmitt teaches a combination of set point and injury recall technique (IRT) as part of an initial clearing of the patient.(8) This technique is as follows. First find a positive g2sub-max (**g2sm**) muscle.(8) A g2sm is a muscle test in which the

g2sub-max (**g2sm**) muscle.(8) A g2sm is a muscle test in which the patient initiates the resistive motion used in a standard muscle test, and the practitioner immediately applies pressure to oppose that action. Apart from the fact that the patient initiates the test, this muscle testing technique is the same as that normally used to isolate a muscle. A positive g2sm displays weakness when the test is performed in this manner. Beginning with a weak g2sm muscle, each set point is challenged or therapy localized by the patient. When a change in strength occurs with the g2sm muscle, that set point is therapy localized by the patient and a search is performed to locate an organ reflex that will two point. A **two point** is a reflex point that will change the strength of a muscle when a second area is being challanged or therapy localized. To date, this organ reflex has been related to one of the two coupled meridians associated with that set point; if it is a bilateral reflex, it will be ipsilaterally found. The talus on the side of the set point is then gently tugged in an inferior direction. When a set point is found for an organ with unilateral reflexes (e.g., the pancreas) and the set point is contralateral to that organ reflex, gently tug the talus in an inferior direction bilaterally. The only exceptions are the TW meridian points. When one of these is found, the patient is turned prone. A challenge to the sacro-tuberous ligament is performed in a superior direction, the ipsilateral talus is likewise pushed in a superior direction, and a strong muscle is tested for change. When a change is elicited, tug the talus in an inferior direction on that side. The opposite side is often positive on challenge at this time and is corrected in a similar manner.

To summarize the procedure, first a g2sm muscle is located and by definition, will be initially weak. A set point is then found that will facilitate that g2sm muscle. Next, an organ reflex associated with that set point is located that inhibits the g2sm muscle again. This is called two-pointing. We are looking for the reflex that two-points to that set point. Then the talus on the side of the set point is gently tugged in an inferior direction. This inferior tug of the talus will be referred to as the **IRT**. Most likely, the g2sm muscle is now strong. If not, perform the above procedure again. Using the now strong g2sm muscle, each set point is therapy localized. If a weakness is found, an associated organ reflex point is located that two-points, and an IRT is performed.

Organ Area Pump

The organ area pump is a combination of IRT and CMRT and some inventive visceral techniques. CMRT stands for chiropractic manipulative reflex technique and is a technique developed by founders of Sacral Occipital Technique. **Initially, the set points should all test strong when therapy localized; the**

clearing technique described above should be completed. These points are therapy localized while the area of each organ is challenged, massaged, manipulated, or pumped and an g2sm indicator muscle is tested for a change in strength. A change in the muscle strength indicates a positive finding and the appropriate therapy can be administered. The therapy can consist of any of the normal responses to a challenge: chemical, physical or electromagnetic treatment. Typically, I have found most therapies will still require an IRT release.

Great caution must be observed when performing any therapy in the abdominal region. Attention must be paid to any abnormal findings and appropriate referrals should be made. It is important to be very gentle when performing these manipulations to avoid iatrogenic problems. **It is contraindicated to perform these maneuvers over an area of inflammation or infection.** When any indication is made to an organ, it must be understood that it refers only to the function of that organ and there is no pathology implied. If pathology is suspected or present these techniques should not be performed and, if necessary, appropriate referrals should be made.

Large Intestine\Lung

The large intestine can be treated by any method of treating dysfunctional organs. Alternatively, the organ area pump can be first performed then followed by a check for further treatment indicators. The patient therapy localizes both LI set points with one hand; the g2sm muscle is tested and it should be noted whether it is in a strong or weak phase. If the clearing technique described above is completed, all set points will not therapy localize, and the muscle will be strong. If the g2sm muscle tests weak while therapy localizing the set point, find a corresponding neurolymphatic or visceral organ reflex for the large intestine or lung that strengthens the g2sm muscle and perform an IRT to the same side as the set point to clear the circuit. With the patient still holding the set points, start at the cecum area, and begin to apply pressure in the direction of the ascending colon; retest your g2sm muscle. A positive finding is a weakening of the g2sm muscle. At this point, treatment can be directed in many ways. Standard procedure for the organ pump is to continue to apply pressure to the area over the colon. Using the weak g2sm muscle, find a two-point with a large intestine reflex, and perform the IRT or talus distraction. The reflex associated with the tensor facia lata (TFL) muscle in applied kinesiology procedure is the most likely reflex involved. The colon reflexes in applied kinesiology reflect over the bilateral TFL neurolymphatic.(9) The cecum is the most proximal portion of the right side TFL. Proceeding distally down the right TFL, the ascending and right half of the transverse colon is represented. Going from the most distal to proximal on the left TFL, the left transverse colon continues followed by the

descending and sigmoid portions of the colon. The area of the colon being pumped can be located on the TFL accordingly. Remember that a great deal of force is unnecessary and is probably contraindicated.

As an alternative to the injury retrieval technique, when you elicit a change in the g2sm muscle, investigate the three major categories for the appropriate therapy (chemical, structural, or electromagnetic).

The lung area pump is performed while continuing to therapy localize the LI set point bilaterally. Pressure is directed on the anterior and posterior aspects of the ribcage simultaneous with an expiration. This maneuver is performed three to four times, and the g2sm muscle is tested. With a positive response, find a two-point to a lung reflex. When the positive therapy localization is found, perform an IRT. The anterior reflex of the deltoid muscle in applied kinesiology is recommended as the first reflex to test. After completing the pump on one side, check the opposite side as well.

The levator scapula muscle, which is associated with the lung meridian, has been related to the function of the parathyroid gland. I have the patient therapy localize the LI/Lu set point and then pump the area over the parathyroid and thyroid gland. With a positive response, find a two-point to a parathyroid reflex area and perform an IRT. In this case, the levator scapula reflexes are appropriate to use.

Small Intestine/Heart

The small intestine area pump will be addressed next. The patient therapy localizes his or her small intestine (SI) set point as the doctor probes the abdomen ipsilaterally, primarily in a semicircle around the umbilicus. The weakening of the previously strengthened g2sm muscle is a positive response. Two-point to a small intestine reflex point and perform an IRT ipsilaterally. The most common small intestine reflex point is the neurolymphatic associated with the quadricep muscle group in applied kinesiology. Other areas of the abdomen can be investigated for the need to perform this procedure by continuing to therapy localize the SI set point and probing the areas of the abdomen ipsilaterally over the location of the small intestine. A weakening of the g2sm muscle after a probe indicates the need to two-point a small intestine reflex while performing an IRT. After one side of the abdomen is complete, the same procedure is repeated on the opposite side.

The heart area pump can be activated under the left ribcage from the xiphoid process down the costal cartilage about three to four inches. I have only had occasion to use this procedure a few times. This pump is indicated by a weakening of the g2sm muscle while therapy localizing the SI set point and probing under the left ribcage. A two-point to a heart reflex is then established and an IRT performed. I have used the neurolymphatic

reflex related to the subscapularis muscle in applied kinesiology.

Triple Warmer/Circulation Sex

As in the LI/LU procedure, the set points (TW23) are therapy localized simultaneously. There are many areas that can be pumped with these set points. The first is the thyroid area. Initially, I found that milking the area over the thyroid often displayed a neurolymphatic reflex that had not previously indicated a need for treatment. Utilizing the set points indicated a greater need for treatment. Dr. Erik Carlin often found it necessary to milk or goad the area inferior to the thyroid to elicit the indicators. As with the other area pumps, two-point a reflex point for the thyroid, and perform an IRT.

The adrenal area is very difficult to pump directly; direct pressure must be applied under the ribs in a superior, posterior direction. The opposite hand lifts the ribs toward the other hand on inspiration. I have elicited reflexes attributed to the adrenal gland after performing this maneuver. It is initially unilateral, but can display the opposite side after one side is completed. Often an initial slight thrust toward the adrenal gland can create an indication that more pumping is required. Thrust once in the direction indicated above and with the set points therapy localized, test the g2sm muscle for a change in strength. If a change is noticed, proceed to pump the indicated side several times, two-point an adrenal reflex and perform an IRT.

The sternum area over the thymus gland can be pressed in a posterior direction several times, and the g2sm muscle can be tested while the TW set points are therapy localized. With a positive response, two-point a reflex area associated with the thymus gland and perform an IRT. A commonly occurring reflex is the neurolymphatic associated with the infraspinatus muscle in applied kinesiology. This is the area beneath the right breast just lateral to the mid-breast line.

In females, while continuing to therapy localize the TW set points, the area above the pelvic brim can be pushed in all directions while checking for a change in muscle strength. With a change in strength, the other hand two-points an ovary reflex as an IRT is performed.

Again with females, the area of the uterus can be gently pushed in various directions while therapy localizing the TW set points. With a positive muscle response, the other hand therapy localizes a uterine reflex point that two-points, and an IRT should be performed.

In males, the area of the prostate can be directly or indirectly pumped while therapy localizing the TW set points. To indirectly pump the prostate, have the patient perform the Kegel

maneuver several times. The Kegel maneuver requires the patient to contract the muscle he uses for stopping the urine flow. With a positive muscle response, two-point a prostate reflex point with the remaining hand and perform an IRT.

Again in males, the testicles should be gently (!) pumped by your patient while he therapy localizes his TW set points. Test the g2sm and with a positive response, two-point the reflex area associated with the gonads, and perform an IRT.

Stomach/Spleen-Pancreas

While therapy localizing the set point ST1, the midline from the xiphoid to the umbilicus is pushed in a posterior direction, one section at a time. The area directly over the stomach should also be pumped following the curve from the most superior aspect on the left side to the right side moving in an inferior direction to the area of the pylorus. Again, you are looking for a change in muscle strength. With the appropriate change, two-point a reflex associated with the stomach and perform an IRT.

Continuing to therapy localize the stomach set point, palpate along the abdomen over the pancreas, testing for a change in muscle strength. When found, pump along the area over the pancreas from the left to the right side. Two-point a reflex associated with the pancreas, and perform an IRT.

The third area to be pumped is over the area of the spleen. Place one hand on the ribcage over the area of the spleen while the other hand gently applies pressure up and under the ribcage in the general direction of the spleen. An initial challenge of gentle thrust under the ribcage will often demonstrate the need to perform this maneuver. When the appropriate change occurs in muscle strength, two-point a spleen reflex and perform the IRT.

Another muscle associated with the stomach meridian is the sternocleidomastoid (SCM) muscle, and it is related to the sinuses. Therapy localize the stomach set point and pump the area over the various sinus locations; the frontal bone and the maxilla bones. When a change occurs after the pumping, two-point to a sinus reflex point, and perform an IRT. The neurolymphatic reflexes for the SCM usually two-point after the sinus area pump.

Liver/Gallbladder

As in CMRT work, the gallbladder should precede the liver pump. I have often found that the abdominal area over the duct from the gallbladder to the duodenum will respond to a challenge when therapy localizing the GB set points. Therapy localize both GB set points simultaneously. Gentle pressure is applied to the area directly inferior to the rib cage and the gallbladder. Limit the challenge to one point of pressure each time the g2sm muscle is tested, and proceed in an inferior direction, challenging down a line located about one or two inches right of the body's mid line. You may find only one area of the entire

course to challenge, or the entire area over the common bile duct may respond to a challenge. When a positive muscle response is elicited, have the patient two-point a gallbladder reflex point and perform an IRT. The reflex I have found most commonly is the neurolymphatic located at the fifth intercostal space from mid-mammillary line to the sternum. This is the neurolymphatic reflex associated with the popliteus muscle in applied kinesiology procedure. After performing an IRT when appropriate, pump directly over the gallbladder and retest a muscle while continuing to therapy localize the GB set points bilaterally. Again, two-point to a gallbladder reflex point, and perform an IRT when appropriate. Another area that may challenge with a dysfunctional gallbladder is over the CMRT area for the gallbladder. It is located three to four inches inferior to the umbilicus and one inch lateral to the right of the centerline. As in all these pumps, great care must be used when performing the manipulation. If this area does not respond to pumping with the gall bladder set point, try pumping while therapy localizing the small intestine set point.

After the gallbladder area pump has been performed, the liver area pump may be performed as in CMRT. Therapy localize the GB set points bilaterally. A light pressure under the right ribcage may be used as a challenge to determine if this pump is necessary. Press under the right rib cage with one hand while the other hand is pressing on the anterior ribs over the area where the liver is located. The second part of the procedure is to move the rib pressure hand to the right posterior ribcage, again over the liver, while pumping under the ribcage with the other anterior hand. Pressure in both cases is upon inspiration in an attempt to gently compress the area of the liver. Again use light pressure when performing this procedure. When the pumping is complete, perform an IRT while the patient two-points a liver reflex area. The reflex most commonly two-pointing to the GB set point is the neurolymphatic associated with the pectoralis major muscle, sternal division in applied kinesiology. It is located at the right fifth intercostal space from the mid-maxillary line to the sternum.

Bladder/Kidney

A bladder area pump can be indicated by applying slight pressure to the area of the abdomen directly over the bladder while the bladder set points are therapy localized bilaterally. Test the g2sm muscle for a change in strength. If the response is positive, pump the area over the bladder several times, two-point a bladder reflex, and perform an IRT. The most common bladder reflex is the neurolymphatic point associated with the peroneus muscle group in applied kinesiology. This point is located over the pubis bone.

The kidney area is pumped similarly to the adrenal area pump already described. The posterior hand is placed over the

ribcage approximately where the kidney is located. The anterior hand applies pressure in a posterior and superior direction on several inspirations. As with the other pumps, when the g2sm muscle weakens after pumping the area, two-point a reflex point for the kidneys, and perform an IRT. To perform a challenge, the bladder set points are therapy localized bilaterally, and a gentle push on the abdomen is performed in a similar manner to pumping the kidney area. The most common reflex point is the neurolymphatic associated with the psoas muscle in applied kinesiology.

This completes the organ area pump procedure as related to each set point and the appropriate follow-up procedure to a positive finding.

Food and Chemical Challanges

Often food and chemical substance that one suspects to be problematic for the patient, do not display a functional muscle weakness when tested. Although we cannot say a functionally weak muscle response indicates sensitivity to a substance, we can say there is a negative physical response to this substance. Clinically this indicatesd a substance that potentially provokes problems in a person who contacts or consumes them.

The proceedure is to introduce the food or substance to the patient's body. Historically, oral or nasal introduction has been the method used. With individuals who are hypersensitive or with substances potentially noxious to ingest, other methods may be used. Using the south side of a powerful magnet has been relabile in these cases.(4,6) The substance is placed between the body and the south pole of a powerful magnet. Once the substance is introduced to the body, a muscle previously tested, is retested and it is noted if there is a change in muscle's response. When there is a change from a strong muscle to one that is functionally weak, the substance has provoked a reaction in the muscle. Historically, this reaction in applied kinesiology literature has been interpreted as one that indicates a potenial problem for the patient. My clinical experience has confirmed the same.

Some foods and chemicals do not indicate a functionally weak muscle when tested in the above manner. This is a case where the organ area pump can be used. Use the same procedure is as in the organ area pump but introduce the substance as well. The doctor is looking for the same functional weakness as in other provoked tests.

For example, suppose a patient reports a reaction to wheat every time he or she ingests it. However, no muscle reaction is found after the wheat is introduced. Wheat is introduced to the patient and a set point is therapy localized. The organ areas associated with that set point are then manipulated, and an indicator muscle is tested. If a positive response is noted by the weakening of a strong indicator muscle, remove the wheat, and

make sure the organ area pump does not weaken the muscle as well. If the manipulation produces no weakness without the wheat, you can confirm the patient's observation about wheat causing a reaction when ingested. In addition, an area of their body has shown to need treatment. Reintroduce wheat again and as the patient therapy localizes the set point. Do the organ area pump or other necessary visceral manipulation that had produced a weakness. Therapy localize the reflex point that two points with the patient's other hand, and perform an IRT. Note the area that shows the response, for that area is probably is functionally weak and may be an area of future treatment focus. If one set point and organ area manipulation does not show a change in strength of the indicator muscle, it may be necessary to try the other set points and organ areas to evoke an response.

You may also try other therapies when the patient displays the weakness to support the organ/set point weakness. For example, after pumping an organ area with the patient still exposed to wheat, the indicator muscle will test weak. You may now attempt to negate that weakness with digestive enzymes or other nutrients.

Observations

The two areas that appear most frequently when the organ area pumps are performed, are the gall bladder and the colon. Perhaps it is the high fat content of the New York diet or even the greater fat content of the average American diet that causes a very high degree of gall bladder dysfunction in the patients I treat. I also suspect that widespread dysbiosis increases its frequency. It is probably the low fiber diet or perhaps, functionally low thyroid conditions that contribute to such a high frequency of colon dysfunction. There is no automatic correlation between organ dysfunction and a positive finding when performing the organ area pump. It is necessary to investigate correlating information relating to the function of the organ before making that determination.

The adrenal area pump is frequently required. Functional hypoadrenia is the most prevalent condition I treat. One must consider that the majority of patients treated with this technique are residents of New York City. (One newspaper reporter observed that when he was in Toronto it took him 6 cups of coffee to make him feel like he was in New York!) Further observations on adrenal function are contained in a future paper.

I have observed that patients have symptoms of a particular organ dysfunction and have not shown related muscle or organ related reflexes until an organ area pump was performed. This would seem to indicate the value of performing the organ area pump under these conditions. One could also infer from this fact that future dysfunction may be prevented by provoking hidden problems in this manner. I believe the organ area pump to be very beneficial for patients' health. It is, however, impossible

to isolate the impact of the organ area pump given its imbeddedness in a comprehensive treatment plan.

The organ area pump is also useful in eliciting responses to food and chemicals not previously evoked, and may be another step in treating these responses.

Conclusion

Dr. Schmitt's technique of using set points and organ reflexes has been helpful in determining a patient's functional assessment as well as an aid to removing some of the blocks in the body's attempt to heal itself. The organ area pumping technique presented in this paper is a further attempt to do the same as Dr. Schmitt's good work. It is not the final step in the patient's continuing care, but I have observed that it is an effective step in the continuing effort to facilitate our patients' health.

Summary of Clinical Procedures

Injury Retrieval Technique

1. Determine a weak g2sub-max (g2sm) muscle.
2. Therapy localize a set point that strengthens the g2sm muscle.
3. Two-point to an organ reflex point associated with the organ of the meridian or the coupled meridian of the set point that weakens the g2sm muscle.*
4. Perform an IRT by gently pulling the talus in an inferior direction on the side(s) of therapy localization.
5. Repeat until no set points therapy localize.

* When the TW set points therapy localize, the patient is turned prone; a challenge to the sacro-tuberous ligament is done in a superior direction; the ipsilateral talus is pressed also in a superior direction; a strong muscle is tested for change. When a change is elicited, perform the IRT. The opposite side is often positive on challenge at this point and is corrected in a similar manner.

Organ Area Pump Technique

1. Pump areas over the organs associated with the set points currently being therapy localized, and test the g2sm muscle after each pump or challenge to the area.
2. When the g2sm tests weak, two-point an organ reflex associated with the pumped/set point area.
3. Perform an IRT.

Provoking Food and Chemical Response

1. Introduce the food or chemical to the patient's system.
2. If there is no response with the g2sm muscle, therapy localize a set point sumultaneously. If there is still no response to any of the set points being therapy localized, pump organ areas associated with the set point while the food or chemical is still introduced to the patient's system and look for a weakness in the g2sm muscle.
3. When a weakness is evoked in the g2sm muscle, look for the reflex area that two points.
4. Perform an IRT. You may also try other therapies to negate the muscle weakness and then perform the IRT.

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Pathway Specific Stimulation
with
Immune Challenge

Variations on an Original Theme of Schmitt's Immune Challenge Technique

by Samuel F. Yanuck, D.C.

ABSTRACT

This is a report of clinical observations based on a short series of patients, with consistently promising initial results. Patients were treated using Immune Challenge Technique (ICT), as described by Schmitt (1), in conjunction with activation of specific afferent or efferent neurologic pathways suspected of being involved according to the patient's complaint. The intent of this approach is to create a context in which the impact of immune reactivity on pathway-specific neurologic function might be revealed. Immune circuit involvement in pathways for vibration and for temperature were treated, for instance, in a patient with multiple sclerosis. Pathways for joint position sense and motor pathways were treated in patients with stiffness and/or pain in the joints. Significant persistent improvement was seen in virtually all the patients involved in the series.

INTRODUCTION

It is common to see changes in function occur with changes in neurologic state. Accordingly, applied kinesiologists often attempt to recreate specific states of the patient's nervous system in which a problem might be revealed. Identifying such contexts with a high degree of specificity is often crucial, because correction of the problem may require that the corrective measures are introduced into the specific context in which the problem occurs. Such was this author's intent in combining the activation of specific neurologic pathways with activation of the immune system. If, for example, a neurologic context could be created emphasizing an MS patient's autoimmune reaction to specific neurologic pathways, then more specific corrections might be made to reduce that autoimmune reactivity.

METHODS

Patients were treated with applied kinesiological methods intended to organize functional neurologic parameters prior to utilization of the technique presented here. Centering the Spine (2), correction of tonic labyrinthine reflexes (3), spinal adjusting, and other techniques geared toward increasing accuracy of central neurologic integration were used

Methods, cont'

on each patient. In the classical terminology of applied kinesiology, the patients were "unswitched" to the fullest extent possible.

In each case, an intact indicator muscle, preferably an extensor, was used as a reference. The patient then made digital contact (so-called therapy localization or TL) in succession over the Angle of Louis, lower sternum, and left 7th rib space (the so-called "three immune circuits") to see if any of these contacts would induce a failure of the previously intact muscle to resist the tester's pressure. If this occurred, other treatment would be introduced, until such time as these contacts alone did not induce failure of the muscle.

Once these three contacts had no effect on their own, they were combined with other neurologic input, to find a neurologic context in which the failure of the intact muscle might be induced.

In the multiple sclerosis patient, the complaint was left arm paresthesia. Contact to each of the three immune circuits was combined with vibration by placing a tuning fork on the left radial styloid, ulnar styloid, carpals, or phalanges. Each of these tuning fork contacts, when combined with contact over one of the immune circuits, would create failure of the previously intact muscle. Treatment consisted of tractioning the head gently forward while holding the tuning fork over the involved bone (each of the above mentioned bones was treated) while the patient held each of the three immune circuits. Though I had treated the patient before, addition of this treatment gave persistent relief from symptoms in her arm.

In patients with joint pain or arthritic stiffness, contact to each of the three immune circuits was combined with activation of joint position sense. The doctor moved the involved joint while the patient contacted each of the immune circuits. During or immediately after this activity, the head was tractioned gently forward into flexion. In cases of hand stiffness either post-injury or with arthritis, it was necessary to do the procedure with passive movement of each joint of the hand or finger to ensure that joint position receptors in all joints were fired. Increased range of motion and decreased pain were immediately observed.

Patients with joint pain or stiffness were also treated by combining immune circuit contact with motor pathway activation. The patient moved the involved joint actively while contact was made over each of the immune circuits. The head was then tractioned forward.

In patients with burning sensations in the feet, the same approach was used, with activation of temperature pathways by placing a warm pack on the soles of the feet.

Methods, cont'

The following types of stimulation have been useful. Other effects are likely relevant, though this author has not had opportunities to identify all of them.

Motor:
- active movement of a specific joint, i.e. the elbow, by the patient.
- active movement of groups of joints, i.e. repeated flexion/extension of the rib cage.

Sensory:
- vibration (tuning fork)
- temperature (hot or cold pack)
- pain (pinwheel or pinching - may be especially useful over visceral referred pain areas)
- joint position sense (passive movement of the joint)
- deep digital pressure

Special Senses:
- far to near gazing.

DISCUSSION

The intent of this procedure is to create a neurologic context which is sufficiently noxious to induce a flexor reflex afferent (FRA) withdrawal response (4). In the arms and legs, this response strongly activates flexors, so the limb is pulled in to the body and away from pain (pulling the hand away from a hot stove, for instance). In the head, the withdrawal response is extensor, so the face moves back and away from pain. This FRA effect can be observed as an induced failure of a limb extensor muscle, in response to strong flexor withdrawal activation.

Schmitt (5) has previously described Injury Recall Technique (IRT). Briefly, this involves activating a specific neurologic context which induces a withdrawal response, then opposing that withdrawal response by tractioning the limb axially, or by tractioning the head into flexion. Both of these maneuvers oppose the withdrawal response, and in doing so appear to have the effect of eliminating the noxious content of that specific neurologic context. After treatment, the same set of inputs no longer activate FRA effects.

The approach described here appears to be a useful application of Immune Challenge and Injury Recall Techniques, intended to create contexts combining immune and neurologic elements. The relevant observation is that pathway-specific sensory and/or motor stimulation may be important in creating neurologic contexts that are sufficiently specific to create FRA responses, and therefore provide the opportunity to make IRT corrections that bring those FRA responses to extinction.

As is often the case, getting the problem to show up can be the key to fixing it.

SUMMARY OF PROCEDURES FOR

Pathway Specific Stimulation with Immune Challenge

1. Immune circuit TL does not induce failure of an intact limb extensor.

2. Activation of a neurologic pathway does not induce failure of an intact limb extensor.

3. Performing #1 plus #2 simultaneously does induce failure of an intact limb extensor.

4. Maintain #1 and #2 actions above while tractioning the head mildly into flexion.

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DIVISION III - COMMENTS ON PUBLISHED PAPERS

DR. GEORGE J. GOODHEART
RESEARCH REPORT

DR. GOODHEART'S RESEARCH TAPES
TAPE 127
-1-

-WHEN we eat WHAT we eat has a lot to do with how much good we get from it.

-The proper combination of foods is a big subject and an important one. Much has been said on it and there has been some controversy. A close study of the known facts and informed opinions leads inexorably, in GJG's opinion, to one conclusion. GJG will set them forth here and let them speak for themselves.

-GJG knows that in doing so he will encounter resistance from two quarters. First from those to whom the idea is a new one, those to whom eating bread and potatoes with meats seems so eminently natural that they are loath to accept the thought that such a combination is a bad one. Many of our oldest habits are unsound and should be changed, not lightly or for a whim, but when solid convincing reason is brought forth for doing so.

-As a foundation for my observations, my theory of dietetics is based upon the hypothesis that inadequate absorption of food causes degeneration of tissue, and that for perfect metabolism we must not combine high starches and high proteins and fats in the same meal. It is, of course, absolutely impossible not to combine proteins and carbohydrates in the same meal. Practically all foods have some protein, some carbohydrate or some fat. However, a meal can be predominantly protein or predominantly carbohydrate. No one will ever dispute that statement.

-Briefly stated, his contention is that a combination of high protein and high starches effectively inhibits the complete absorption of all the nutritive factors of foods and places an unnecessary burden upon the entire digestive apparatus.

-His own first special interest in food combinations several years ago followed a simple observation of my own digestion. He noticed that on some picnics I would suffer considerable discomfort tow or three hours after eating the sandwiches which were always our lunch. The discomfort was of the unpleasant type most layman call sour stomach. On other picnics he would feel fine all day. Checking soon showed that on the one hand the sandwiches which gave him trouble were those made with meat, eggs, or cheese. When the sandwiches were made of tomato, lettuce, jam, or preserves, he got no unpleasant sour stomach after effect. He felt fine.

-Yet he was aware that meat, eggs, and cheese agreed with him. He ate them frequently with no bad effects whatever. The answer was obvious, they only gave him trouble when eaten in sandwiches.

-Then a man who conducts many picnics came to me for advice on how he could plan picnic lunches and avoid the indigestion so many of his people complained of. GJG told him to not give them meat, cheese, or eggs in their sandwiches.

-He followed GJG's suggestion and was delighted with the result. He reported that his people came home in a happier frame of mind,

that remarks about indigestion had ceased, and that people were going on more picnics.

-Bread is a high carbohydrate. Eating high carbohydrates with proteins interferes with proper digestion. But that isn't all.

-As GJG has stated earlier, many of our illnesses and infirmities are due to deficiencies of certain essential food factors, vitamins and minerals. These deficiencies produce degeneration of certain tissues, and this degeneration results in loss of resistance. Then infections invade us and produce disease.

-These deficiencies are dangerous and must be avoided. It is not enough to have the essential elements in the foods we eat, they must actually be utilized by our bodies, they must be available to our tissues.

-It is a fact that we may eat large quantities of these food elements and get no benefit at all from them. If at the same time we eat other foods which interfere with the proper chemical digestion of the vitamin and mineral bearing foods, then we fail to absorb the essential elements into our circulation.

-If we eat some cheese, rich in calcium, and at the time it reaches our small intestine, an alkaline digestive process is going on there, then very little, if any, of that calcium will be available to us. The calcium will make a chemical combination with the alkali and become non-absorbable, it will pass through and out of our body unused. No matter how much cheese we eat, we may still suffer from calcium deficiency, if the calcium is not absorbed. But if this food reaches our small intestine when an acid condition is present, then much of the calcium will be utilized.

-Obviously then, we must be certain that when we eat cheese, our small intestine will be acid and not alkaline. But how? The answer is clear and incontrovertible, by not eating any high carbohydrates at the same time.

-When we eat carbohydrates, starches and sugars, our small intestine becomes alkaline and a condition is created by which essential factors in other foods cannot be used.

-But that isn't all. These same carbohydrates may interfere with the digestion of certain proteins in the stomach itself, and partially digested protein food actually becomes toxic material. Research has found that proteins may be split up by imperfect digestion into large protein molecules that may be absorbed into the circulation and prove toxic (poisonous) to the body. Instead of being split up into smaller molecules (the amino acids that are end products of normal digestion), proteins eaten with carbohydrates may actually become agents of evil to our tissues, such as the allergy producing and poisonous amines.

-The physiology of digestion: There are two distinctly different types of digestion, an acid digestion for proteins (meat, fish, eggs, and cheese) and an alkaline digestion for carbohydrates (sugars and starches).

-All physiologists agree that proteins are digested largely in the stomach, by the gastric juice, which is acid in reaction. One of the most important constituents of the gastric juice is free hydrochloric acid. Another important ingredient of gastric juice is

pepsin, the ferment that splits the protein; and pepsin acts only in an acid medium. In other words, the stomach contents are always acid, and the stomach must be acid in order to do its job of digesting protein foods.

-Carbohydrate foods, on the other hand, are not digested in the stomach, but are digested largely in the small intestine, principally by the pancreas secretions, which are alkaline. One of the most important constituents of this process is amylopsin, which splits the starch and the amylopsin acts only in an alkaline medium. On their way through the stomach to the small intestine, the carbohydrates inhibit the secretion of the hydrochloric acid in the stomach, but at the same time combine with some of the free hydrochloric acid which is already there. They pick it up and take it with them.

-Fats follow a still different course. Pure fats leave the stomach as fats, and entering the small intestine, cause the gall bladder to empty a quantity of bile into the intestine. This bile saponifies the fat (the first step in digesting fat), but in doing so it liberates fatty acids. These fatty acids, of course, work to neutralize whatever alkaline secretions are present in the small intestine.

-Obviously, therefore, if these fatty acids are produced in the intestine while some carbohydrates are being digested there, its just too bad. The alkaline secretions that are part of that digestive process will be neutralized, and the action of the amylopsin will be stopped. The digestion of the carbohydrates will be interfered with, and they will be left free to ferment and produce gas.

-Hence the following rule, which is not only logical and physiologically sound, but has been proved highly valuable by clinical observation. Rule 1: Do not combine pure fats (butter, cream, bacon fat) with high starches (potatoes, bread, cereal, sweets) at any one meal.

-If you're having high carbohydrates at a meal, don't eat any fats, and if you're having fats, don't eat any carbohydrates. Simple, isn't it? If you're having bacon for breakfast, don't eat cereal or bread. If you're having potatoes for lunch and a sweet dessert, don't put butter on your potatoes or cream in your coffee.

-In the past many physicians have practiced this rule unconsciously, by advising patients to cut out all fats and high starches, or greatly restrict them. Obviously this produced good results, because patients who ate neither could not combine pure fats and high starches. But, with a restriction of fats there was always the serious danger of running into a deficiency of the fat soluble vitamins and certain fatty acids (such as vitamin F) which are necessary to preserve life. Now that the light of science has illuminated the field, neither fats nor starches will have to be eliminated or restricted in most cases. The patient will simply be told to eat them at a different times.

-Interesting evidence that the high starches and pure fats are incompatible came as a sidelight from the observations of Dr. Joslin in his famous diabetic clinic in Boston. He established the fact that if you cut down in high fats in diabetic diets, you can add more carbohydrates without getting any increase of sugar in the urine,

without increasing insulin, if it is an insullin case.

-What does this indicate? Remember that diabetes is a disease in which the carbohydrate food is not split up into its end products, the sugars get into the blood stream and accumulate there, and that is why intake of carbohydrates has always been cut down to the minimum. But when fats are decreased, then the carbohydrates digest better, or split up more readily. Perhaps the presence of pure fats in the intestine while starches were being digested there was a considerable factor in the production of diabetes in the first place. Possible, in that way, as fatty acids were liberated, the pancreas depleted itself in a continued effort to produce secretions to neutralize these acids and became unable to manufacture its own insulin.

-Primitive man did not eat fats with carbohydrates. Eating his food as he found it, he ate lots of fat with his meat when he killed an animal; but in millions of years he never found lumps of pure fat attached to any vegetable (carbohydrate) foods. As he developed he never had need for a digestive mechanism that would digest fats and starches at the same meal, he never developed such mechanism, and today we still haven't any.

-Eat fats with meats, or with any other proteins, fish, eggs, or cheese. In fact you must be sure to eat fats with meat, they not only can be combined but they must be combined.

-One of the most important studies ever made on an exclusive high protein and fat diet, was conducted through the cooperation of the explorers Vilhjalmur Stefansson and Karsten Andersen. The purpose of the test was to demonstrate that man could live on a purely animal diet in our climate for an indefinite time, and in this case it was extended over a period of one full year.

-The complete report of this experiment has been published and the general findings are reported here. It is mentioned only to emphasize the importance of proteins and fats in the diet.

-The conclusions reached by this test were: a. That it is possible for man to live for long periods on meat alone. b. That no ill effects whatever were recorded. c. That the diet, in order to be adequate had to contain large quantities of fat, some liver, and that lean meat alone was not tolerated. d. That the tissues of one animal contain everything which is essential for another animal, in this case, man.

-Important clinical observations in this test support GJG's thesis that there is greater absorption of foodstuffs when eaten in the proper combinations. There was much greater absorption, no gas and a distinct simplification of putrefactive organisms in the intestine. There was no constipation. A further important observation was that both men showed no increase in blood pressure throughout the year, and one of them actually showed a decrease of 20 mm in his systolic pressure.

-This experience and its conclusion, that fats and proteins are an excellent combination, checks with our knowlege of the physiology of digestion. Since protein is digested largely in the stomach by acids, and since the pepsin which helps digest it works only in an acid medium, then when it gets into the small intestine, if fats are

being digested there at the same time and they have liberated enough fatty acid to make the intestine acid, then the action of the pepsin would be prolonged and the digestion, of the protein would be carried further. It all works out beautifully, fats, proteins, acids, they all go together and help each other. Remember that, associate them in your mind: fats, proteins, acids.

-But remember that it's a different story with carbohydrates. Carbohydrates (starches and sugars) are digested by alkalies. Naturally, if any acid is combined with carbohydrates it will tend to neutralize the alkaline digestive juices they need. The more acid present, the more alkaline secretion will be required to neutralize the acid before it can begin to digest carbohydrates. That is simple, too, isn't it, and obvious?

-From this we get Rule 2: Don't combine acids and carbohydrates. Don't take buttermilk, orange juice, lemon juice, grapefruit juice, or vinegar at any meal which also includes high starches and sugar.

-Here again is an interesting corollary that will be good news to many people. Often patients have told GJG "they cannot take orange juice, which they love, because it causes acid stomach". On questioning them, it was invariable that they had taken it at breakfast with cereals, toast, or other carbohydrates. Invariably, when I told them to take it alone or with protein foods only, they did so with great satisfaction and no bad effects. If you have had trouble with orange juice, just try it with bacon and eggs.

-It should be remembered, of course, that most healthy people can combine orange juice and starches without feeling any distress or evidence of impaired digestion. But the impairment goes on just the same. Every time a healthy person combines acids and starches he is making trouble for his digestion, he is getting less value from his foods, and he is hurting himself. The body has remarkable ability to adjust itself to the most terrible treatment. You have heard many people exclaim, "I have the digestion of a horse", or, "I could eat nails and it wouldn't hurt me". Fifteen years later some of these people are wrecks.

-The third rule is, perhaps, the most important of all. Rule 3: Do not combine high proteins (meat, fish, eggs, or cheese) with high starches (potatoes, cereals, breads, sweets) at the same meal.

-This prohibition is based not only on extensive clinical findings but on sound physiology. It has been vigorously disputed in the past but there has not yet been any logical reason or proof against this rule. GJG is certain it will not be disputed in the future.

-Review the evidence. It may seem a little tedious but you should understand this subject for your future full health.

-We know that proteins require acid for their digestion in the stomach. We know that carbohydrates require alkalies for their digestion in the small intestine.

-Some years ago, in a study on sugars which came out of Mayo Clinic, two things were made clear: 1. Sugars inhibit the secretion of the hydrochloric acid in the stomach. 2. Sugars combine with the free hydrochloric acid in the stomach. Both of these actions, by lessening the amount of hydrochloric acid in the stomach, interfere

with the digestion of proteins, which must have that acid.

-Conversely, if proteins are being digested in the stomach and there is more acid there for the sugar to combine with (pick up and take along to the small intestine), then it will require just so much more alkaline secretion from the pancreas to neutralize the extra acid before it goes to work on the sugar. And the same is true of starches which are potential sugars. Not only do the sugars interfere with the digestion of the proteins, but the proteins make more difficult the digestion of the sugars.

-What happens in the stomach when protein is eaten, when starch is eaten, and when proteins and starches are eaten together; and in what condition the food passes from the stomach to the intestine.

-A study by three Philadelphia investigators appeared in the June 1936 issue of the American Journal of Digestive Diseases and Nutrition in which a graph showed the degree of acidity of samples of stomach contents withdrawn at varying times from five subjects; first after protein meals, then after starch, then after combined protein and starch. One and three quarter hours after the ingestion of the meals the record showed that the stomach contents of the protein meal were most acid, the starch least acid, and the mixed meal half way between.

-100 c.c. of the protein meal stomach contents required 60 c.c. of the deci-normal alkaline solution to neutralize the free acid, and the graph was going up sharply. 100 c.c. of the starch meal contents required only 20 c.c. of the alkaline solution to neutralize the free acid and the graph was falling rapidly. 100 c.c. of the mixed meal stomach contents required 40 c.c. of the alkaline solution to neutralize its acid, and the graph was coming down very slowly.

-This means that when the starch meal entered the small intestine comparatively little alkali would be required to neutralize the acid it had picked up in the stomach, but when the mixed meal reached the small intestine just twice as much alkaline pancreatic secretion would be needed to neutralize its acid before starch digestion could begin.

-It is also clear that when the mixed meal was eaten, the proteins in it were being digested under difficult conditions. Instead of the normal acidity required, as shown by the all-protein meal, the acidity was far lower. The presence of the starches had cut the acidity to one-third less. Just such conditions are most likely to produce imperfectly split up proteins, the large toxic protein molecule.

-When high proteins amd high carbohydrates are mixed, this investigation proves, there is not enough acid to digest the protein part readily, and too much acid to digest the starch part readily.

-Now the bad effects of this abuse are not always immediately apparent. The digestion of youth expecially has abundant juices to spare; but if by habits of food combination we impose this extra burden, we deplete that abundance, dip into our reserve power of accommodation, and by the time the age of thirty is reached there is more or less impaired digestion. This may not make itself known by distressing symptoms, but the digestion is nevertheless chemically impaired. This contributes to an increasing deficiency of essential

food elements, and that, in turn, leads to more tissue degenerations. Minor disturbances are directly created, serious diseases are made more probable, and one more obstacle is raised to our being able to live a full, long life of glowing health. Those with perfect health at present, please take note of this.

-High proteins and high starch foods should not be mixed. Now it is true that nearly all foods contain some starch elements and some protein elements. This fact was misleading when GJG first studied mixtures fifteen years ago, and it has misled many other investigators since. Superficially it would seem to indicate that the mixture is natural and therefore presumably healthful, but let us look at the details.

-Meat, leading protein food, does contain carbohydrate, but what kind? It contains a small amount of glycogen, or glucose (muscle sugar). This is a carbohydrate that has been originally eaten by the animal from which the meat came, converted by the digestion of that animal and stored in its muscles as muscle sugar. Little digestion, if any, on our part is required to make this sugar ready to be absorbed, it is ready to be absorbed as soon as it is liberated from the protein part of the meat.

-Similarly, the amount of protein that is in starchy vegetables is small indeed in proportion, and becuase of its negligible quantity presents none of the difficulties in digestion that result from combining large quantities of high protein with high starches. With this point disposed of, GJG believes that his colleagues will agree that the physiological case against eating carbohydrates with proteins is complete and incontrovertible.

-While man, as he evolved, developed two types of digestion for the types of food he ate, other animals confined themselves to one type of food and correspondingly one type of digestion. What do they show us?

-Herbivorous animals, such as the cow or sheep, eating only vegetable food, have specialized on alkaline digestion. They are equipped to eat large quantities of food in proportion to their size, compared to humans. They all first alkalinize their food by much chewing (their saliva being alkali), and they all rechew their food for a long time (chewing the cud). They all have a large sack or pouch where man has his tiny appendix.

-Carnivorous animals, such as lions or wild dogs, have specialized in acid digestion. They bolt their food in large pieces and chew it as little as possible, if at all. Actually the less they chew it, the better it is for them. An experimental study was carried out at the Mayo Clinic in which dogs were fed different articles of diet, and the contents of the small intestine examined for the results of digestion. One comparison was made between meat fed in large chunks and meat ground up. The big pieces were digested far better than the ground meat.

-It is highly significant that meat-eating animals have no appendix or a very small one. Man, with his small appendix, seems plainly in the class of the meat-eating animals, rather than herbivorous animals with their large pouches. Our inability to handle starches and sugars advantageously seems to stem from fundamental

physical sources.

-With the atrophy of our appendix, we lost our ability to get enough protein from vegetable sources to produce the best possible physical man. We cannot chew our cud.

-Sound physiology dictates that when we, like carnivorous animals, eat meat, we should, like them, chew it as little as possible; but when we, like herbivorous animals, eat vegetable foods, we should, like them, chew well and thoroughly. Careful clinical observation corroborates this. Tests show overwhelmingly that the fact proves the theory.

-Americans are notoriously calcium-deficienct. Not because we don't eat foods rich in calcium, but largely because we don't eat them in combination or form in which the calcium can be assimilated. Animals nerver eat high proteins and high carbohydrates at the same meal. They have excellent teeth.

-Proteins made toxic histamine and histaminase. The danger of improperly digested proteins is instead of splitting up into their proper end-products, they split up into intermediate or large protein molecules that are actually toxic.

-Some of these molecules are the substance called histamine; a toxic protein known to pathologists and pharmacologists. (Histamine is used by physicians as an irritant or capillary-dilating element in functions and in external treatments for sprain, arthritis, etc.) The histamine we manufacture within ourselves is a direct cause of many common troubles, especially allergic reactions such as hay fever, asthma, eczema, coryza, migraine headaches, and general malaise.

-A careful and extended series of observations which GJG recently gave unmistakable indications that mixed diets (combinations of fats with starches or high proteins with high carbohydrates), produce more histamine in the system than the combinations he has recommended.

-Histaminase is a substance developed from the intestines of certain food animals, it has the property of splitting up histamine and thus destroying its toxic effect. This put into our hands an excellent means of testing for the presence of histamine, and for finding with a considerable degree of accuracy to what extent bad food combinations produce toxic results.

-GJG has made careful observations on many allergic patients with this substance. When these patients eat a mixed meal, they require more units of histaminase to control their symptoms than when they eat proteins only or carbohydrates only. The mixed diet produced more histamine. Many allergic patients, in fact, lost their symptoms entirely by simply avoiding bad food combinations; they actually lost their hay fever or headaches by eating the kind of meals GJG recommends. But as soon as they slipped and ate an unwise meal, back came the symptoms.

-Clinical observations, careful studies of what happens to people like you and me, sick people, well people, and people almost well, superbly happy people, and people who are just on the verge of physical collapse, have given GJG a complete and confident certainty of the great benefits to be derived and retained by avoiding the bad food combinations described in the foregoing. Theoretical physiology,

laboratory tests and other research confirm it, but to GJG, his clinical experience over a period of fifteen years is the most important evidence of all. GJG believes that your own experience will be equally convincing to you.

-Experiences in curing diseases, experiences in building up weak people, experiences in restoring full vitality to sub-par men and women, experiences in bringing immediate benefits and long-range benefits to all types of cases, have made sure that no other conclusion is possible.

-We have long known that extracts from adrenal gland will control allergic reactions. Undoubtedly the adrenal gland takes care of the normal amount of histamine produced in the body; but when years of improper food habits have given us certain deficiencies and degenerations, the combination of excess histamine and food deficiencies depletes our adrenal glands, the control is lost, allergic reactions appear more readily and we are well on our way to serious bodily degeneration.

-The evidence on the matter of histamine production alone is sufficient to justify all of GJG's recommendations on the food combinations.

-The amino acids: Not all proteins are of equal value in nutrition. The different proteins vary widely in chemical composition and in their ability to satisfy the body's requirement of nitrogen; they vary in the degree to which they supply the amino acids essential for tissue building and tissue repair. There are some ten amino acids which have been isolated from proteins and have been shown to be essential to human nutrition.

-The value of any protein is measured by its ability to supply some or all of these amino acids. A complete protein would be one which would supply all of them, but unfortunately few proteins ever approach this ideal. However, a properly varied diet containing proteins from not only the muscle tissue of animals but also the connective tissues and tissues from their organs, plus eggs, will usually supply all the essential amino acids in sufficient quantity.

-Dietary Rules for Health: The general rule is, be sure you eat enough of the vital food elements, and be sure you eat them in the right combinations.

1. Eat all kinds of meat, fish, eggs, leafy vegetables, citrus fruits (and carbohydrates only if you must) as the safest way to avoid deficiencies.
2. Do not combine pure fats (butter, cream, bacon) with high starches (potatoes, cereals, breads, cakes, sweets) in any one meal.
3. Do not combine acids (citrus juice, vinegar, buttermilk) with high starches at any one meal.
4. Do not combine high proteins (meats, fish, eggs, cheese) with high starches at any one meal.
5. Eat fats freely with proteins and acid solutions.
6. Be sure you get enough of each essential nutritional element as follows:

   a. Meat and eggs: One serving of each, or two servings of one per day, with butter or other fat. Fish or fowl may be substituted for meat or eggs.

b. Milk, buttermilk, or cheese: Two glasses of milk or buttermilk, or two and one-half ounces of cheese a day (or one glass of milk or buttermilk plus an ounce or more of cheese).

c. Raw, low starch fruits and raw green and yellow vegetables: Two servings a day or one large salad bowl a day.

d. Supplement the above daily, with one or two tablespoons of a plain cod liver oil, or its equivalent in other fish liver oils, or their concentrates in capsules. But if you use capsules, then be sure to take plenty butter fats and cream; your liver must have fats, if it is going to make bile for you. If you are a carbohydrate eater, then you should supplement the diet with yeast or other equivalent form of the vitamin B complex.

-These are radical changes in your present eating habits. This will not be easy and there's always a hardship in shaking off old habits and forming new ones. You are certainly entitled to ask the question, "If I undertake this change of habit for one month, what effect can I expect?" A straight question deserves an honest answer. How quickly you feel noticeable improvement depends largely on how good your health is to begin with, and how bad your eating habits have been in the past.

1. If you are now in fine health, have been eating plenty of protein, have no digestive troubles, no marked deficiencies, you may experience no detectable effects of this diet in one month's time. But you will later. If you come around in ten years' time, GJG can tell if you have been following perfect eating habits by just looking at you.
2. If you now suffer from occasional flatulence, indigestion, "acidity", and gas, a month on this regimen with no cheating will work wonders. Your ailments will probably disappear.
3. If you feel "all right" but sluggish and under par, if you have been eating unwisely, too much carbohydrates and not enough of the other food factors, you will experience a new feeling of well being and full health which perhaps you did not believe possible.

-We must be certain to eat high proteins, we need have no fear of eating too much, except those who have no control over their appetites. While man may continue to exist on a relatively low protein intake, there is ample evidence that a more liberal favors the development of a better physique and improvement of general health. Any excess of proteins, above the body's requirement for growth and repair of tissue waste, is efficiently utilized as a source of bodily heat and energy.

-The types of recommended diet is given here generally. They follow the rules that he has been so earnestly urging, but do make some concessions to present habits and permit some low starch fruits and vegetables with high protein meals. While such minor infractions are not serious, it is recommended that even these be avoided after you have acquired the habit of eating proper combinations. For good medical reasons, as well as psychological ones, it is important to make meals planned within these rules as varied, as appetizing and as much in conformance to preferences and previous habits as possible.

DR. GOODHEART'S RESEARCH TAPES
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-Resume of the PAT (Primary Atlas Technique) and the work of Raymond Dart, anatomy professor, Witwaterstrand University, Johansburg, South Africa. Dr. Mungo Douglas of Bolton (Lancashire) wrote "Reorientation of the Viewpoint upon the Study of Anatomy", initially published in the British Journal of Physical Medicine in December 1950, and also in the Universal Constant in Living. There he claimed (1) that the primary function of muscle is the "relationing" of the various parts of the body to one anther; (2) that their function as movers of body-parts upon body-parts is secondary; (3) that, of all such "relationing" in the body, the head-neck relation, brought about by the suboccipital group of muscles (atlas-occipital, axis-occipital, atlas-axis), is of paramount importance, and that it is worthy of the distinction of being recognized as the "primary relation upon which all more ultimate relations depend".

-What Mungo Douglas calls the "relationing of parts" is a neat way of describing the customary postural positions which adjacent body-parts assume as the result of the groups of muscles moving them. His purpose was to demonstrate the muscular anatomical background of "primary control", that is to say the head-neck relation. A further significant anatomical fact is that this important suboccipital group of muscles controlling the head-neck relationship is supplied by a single (sub-occipital) nerve from a single (first cervical) segment of the spinal cord.

-Dart: "This single somite, or body segment, doubtless for a special reason, is the only segment of the entire series of postcranial segments the nerve supply of which is purely motor in character. In other words, this segment of the body musculature is the only one that has been deprived of a segmental skin area proper to itself, and to the stimulation of which it would, therefore, have been too susceptible of reflex response. If its corresponding neural segment has any indigenous sensory receptors, they can only be those lying in the muscles and joints of that segment, and thus are therefore purely proprioceptive. In other words, reflex responses of these muscles and joints as end organs are immune from skin receptor interference by the segmental area of skin. Responses of a reflex sort, evoked by touch and leading to postural contraction of the suboccipital musculature, can only be elicited in this muscle group by stimuli coming in from receptors of skin segments other than its own. The nearest tactual receptor segments are the trigeminal skin area anteriorly and the second cervical skin area posteriorly. Doubtless it was primarily to determine in as direct a segmental reflex manner as possible such tactually evoked postural head-neck relationships that the trigeminal nerve, which receives all the tactual stimuli entering the body from the entire anterior end of the vertebrate body, invaded centrally the posterior column of grey matter, or substantia gelatinosa Rolandi, by a downward or spinal

extension of its descending root: this root proceeds in man as far posteriorly as the fourth cervical segment."

-There are receptors other than tactual, however, which profoundly affect this head-neck relatinoship. There is a brain tract of such high importance in every creature with a head articulated to the trunk (that is to say, every vertebrate), that it forms one of the largest bundles in the brain stem of fishes, amphibians and reptiles. This tract becomes medullated between sixth and seventh months of intrauterine life in man, simultaneously with the anterior intersegmental tract of the spinal cord, of which it is the proximal extension. This tract, the medial longitudinal bundle (fasciculus), binds together anteriorly the three motor nuclei (occulomotor, trochlear, and abducens) supplying the eyeball muscles, and posteriorly it connects the anterior horn cells supplying musculature that links the head to the trunk.

-The most important sensory element, however, in determining the postural adjustments evoked by this bundle, is formed by the intersegmental fibres running from the vestibular nucleus to the eye muscle nuclei anteriorly, and to the anterior cervical segments and posteriorly co-extensive spinal nucleus of the accesory nerve. The intersegmental fibers coming from the brain segment supplied by the eighth (or vestibular) nerve cause those simultaneous modifications in tension in the musculature that controls the position of the eye relative to the head, and of the head relative to the trunk and forelimbs, which are occasioned reflexly by the everchanging positions of a mobile head (supplied by a single pair of tactual nerves).

-We all have experience in palpation and adjustment of the upper cervical spine, whether it be a subluxation or a fixation. One of the ways to diagnose is to use therapy localization. There was an accidental observation on a particular patient that had difficulty therapy localizing by placing the hands behind the neck and placing the fifth finger at the junction of the occiput and atlas, and the other fingers reaching down to the third or fourth cervical. If the patient's hands were held in that position by an assistant, then he could TL. If the patient TLed the atlas with just the index fingers, there was no positive TL. However, by using the patient's thumbs for TL (which made it easier for the patient to TL), then the atlas TL was positive. This was found by accident.

-Parallel: a deaf person must be yelled at in order to hear a sound. This is similar to the atlas. C1 has no sensory root, therefore it is difficult to get TL in the usual fashion. The square millimeter measurement of the thumb is larger than the index finger.

-The sensory root for C1 is the temporomandibular joint.

-Dorsal column stimulator placed into the spinal cord is not effective above C2 because the dorsal column stimulator depends upon the substantia gelatinosa (spinal gate). Sheely used a TENS unit to identify the level and then placed the dorsal column stimulator in order to help intractible pain.

-"Low Back and Leg Pain From Herniated Cervical Disc", Kabat, published by Warren Green Co. St. Louis, MO. He uses a specific method for identifying failed lumbar disc surgery syndromes. He looks

at the patient's history. If the patient fell so that the force was cephalad, from the side, from the front, or caudal, there were different methods of evaluating. He found by trial and error that there was a high percentage of a hidden cervical disc at C6-7 (the layman's term is slipped disc), in the presence of low back pain and sciatic radiation. Often this was found in patient's who had already had lumbar disc surgery. He found the wrist extensors as a useful diagnostic tool. He utilizes manual muscle testing to identify potential spinal problems. If the blow was from above, he taps briskly on the top of the skull (it has nothing to do with governor vessel acupuncture points); if the blow was from the side (as in a car accident), he taps on the side of the shoulder; if the blow was from above on the shoulder, he taps on the top of the shoulder in a caudal direction; if the blow was from below, he has the patient stand on one foot and test the wrist extensors on the same side; if the blow was from the front, he taps vigorously on the forehead from front to back; or if the blow was from the back, he taps the back of the head from back to front. He then tests the wrist extensors after the blow, observing for muscle weakness. Usually one sided muscle weakness, the same side as the sciatic radiation, but necessarily so. If you find the wrist extensor weakness on the side of the sciatic pain, you can reapply the blow that weakened the wrist extensor and then test the anterior tibial, and the anterior tibial would test weak. The proof that it is a cervical involvement and not a reflex from the lumbar spine, he administers a cervical traction with his hands (or from an assistant), repeat the blow to the head/shoulder, and now the wrist extensor or anterior tibial will not test weak. You will probably find a positive indication for challenge and adjustment of the hidden cervical disc (commonly C5 or C6), and adjustment is down the facet line (from anterior to posterior).

-GJG found that the patient had a negative TL to the cervical spine in the usual manner with the fingertips. When the patient TLed C1 with the thumbs, it now showed a positive TL. The tap/blow to the head/shoulder that previously weakened the wrist extensors, would then negate the positive TL of C1 with the thumbs. When the thumbs were removed, the tap/blow test was again positive. C1 is similar to the neck traction in Kabat's test in negating the wrist extensor weakness.

-The dura of the skull is firmly attached to the foramen magnum, and then is firmly attached to the upper anterior portion of the atlas, and then the dura of the skull becomes the endosteum of the spinal canal. The dura of the brain enters the foramen magnum where it is attached, some authorities say it is attached to C1 and some say it is not, but it is then definitely attached to the posterior portion of C2 and C3, the loosely held all the way down to the anterior portion of S2 where then the non-neural filum terminale is attached to the first posterior coccygeal segment. The atlas acts as an adapter in that it can change the tension that exists in the dura.

-"Low Back and Leg Pain From 'Slipped Disc' in the Neck", Kabat. An instruction book for patients.

-Quote from "Low Back and Leg Pain From Herniated Cervical Disc": "This book is the first report of a major scientific and

practical breakthrough in the common, difficult problem of pain in the low back and leg. Low back and leg pain is attributed to a variety of disorders of the lumbosacral region, frequently to herniated lumbar disc, but medical and surgical treatment have often been unsatisfactory. Original clinical research on the pathological physiology of herniated cervical disc left to develop a new method of diagnosis and conservative treatment which is effective at eliminating herniation of the nucleus pulposus and preventing its reoccurance with disappearance of the complaint such as cervical radicular syndrome. In the course of this investigation, it was conclusively shown that compression of the cervical spinal cord by the herniated nucleus pulposus of the cervical disc is the most common cause of pain in the lower back and leg. Conservative treatment exclusively of the herniated cervical disc has achieved complete and lasting relief of low back and leg pain in a large series of cases. In many cases the only complaint caused by the herniated cervical disc is pain in the low back and leg which is indistinguishable from the characteristic symptoms of a herniated lumbar disc without pain in the neck or arms that would call attention to a disorder of the cervical spine. In patients who an immediate lumbar laminectomy was recommended elsewhere for a herniated lumbar disc confirmed by a recent positive lumbar myelogram, the low back and leg pain proved to be solely to herniation of the cervical disc. Pain in the low back and leg has been reported from compression of the cervical spinal cord by tumors, cervical spondylosis, cervical soft disc protrusion, yet this has been ignored in the management of patient's with this complaint. Herniated cervical disc is caused by trauma and is much more common than previously recognized, occuring at almost any age, including childhood. The frequent intermittent symptoms have been explained and a diverse manifestations can simulate a variety of other disorders".

-Kabat's observations are correct. Dart speaks of the C1 nerve as having no sensory root. The head, TMJ, eyes should all be level. We have previously discussed EID (Eyes Into Distortion), EOD (Eyes Out of Distortion), and BID (Body Into Distortion). These have been tied together; the vestibular segment (eighth nerve vestibular branch), trigeminal-TMJ branch, C1 lack of sensory root, the medial longitudinal fasciculus (bundle) tying together the three eye muscles, and the spinal accesory nerve.

-GJG used Sam Yanuk as a demonstration subject of this principle in San Francisco. He was questioned as to any cervical problems which was negative, but he did have back complaints. A tap on the top of his head weakened the wrist extensors bilaterally, and also standing with the weight on the left leg produced weakness in the left wrist extensor. Traction of the cervical spine as well as TL of C1 would negate this muscle weakness.

-Dart said that the abdominal muscles act as if its a pressure holding device and the viscera causes a strain on the abdominal muscles when the abdominals are weak. That in turn causes the normal tension and allows diaphragmatic activity.

-A muscle should get strong when you stretch it. A stretch weakness occurs when the muscle is strong in the clear, but weakens

after it is stretched. This requires fascial flushing correction.

-In Sam Yanuk's case, his posture revealed poor abdominal tone, even though he is relatively thin. The abdominal muscles (rectus, internal and exteral oblique) were strong, but they weakened after a stretch of the abdominal muscles. Method to stretch the abdominals: GJG placed his chest on the table and had Sam arch his back over GJG's back to stretch the abdominals. Dart states that we are hung by the musculature by the back of the neck and the top of the head. If the sagittal suture is compromised, it will cause a weakness in the abdominal muscles when it is pressed together. This is a fail safe mechanism that allows as the anterior pressure in the abdominal muscles continues to be exerted, the sagittal suture opens up and allows the abdominals to stay strong. Example: pulling downward on the front of a shirt would cause a pressure of the patient forward, and the collar would exert a pressure at the C6-7 area.

-Need to diagnose the abdominal stretch weakness, and see if folic acid-B12 negates the weakness. Correct it via fascial flush technique, NL treatment, and give folic acid-B12 if necessary. Retest after correction for improvement in the abdominal stretch. About 70% of the time, testing for the hidden cervical disc will now be negative (tap on the head weakening the wrist extensor, etc.) because the pressure on the C6-7 area has been removed by correcting the abdominal muscle. You may still have to do the hidden cervical disc technique. But C1 will definitely still be in lesion, C1 will not correct itself following the abdominal muscle correction. You have to clear out the relationship of the medial longitudinal fasciculus; EID relationship (i.e. head level off); must be a negative TMJ (including ligament interlink between the TMJ: palpate the TMJ right and left and determine which is the sorest, shove the index finger to the hyoid towards the least sorest side and stimulate the least sorest side of the TMJ), (find the sore pterygoid side and tap T2, T3, and T4 to take the pterygoid pain out); check active and passive cervical range of motion and do cervical compaction technique if there is restriction of movement (if it is restricted in lateral flexion, you take it out with rotation to the same side, if it is restricted in rotation, you take it out with lateral flexion to the same side, if it is restricted in extension, place the head in gradual flexion, if it is restricted in flexion, place the head in gradual extesion; if you found a problem with EID, have the patient hold their eyes into distortion during correction of the cervical compaction; if you found a problem in the TMJ, have the patient open and close the jaw during correction of the cervical compaction; this realigns the cervical column); then recheck thumb TL of C1, will be there still 90% of the time, so challenge C1 for anterior en masse, posterior en masse, lateral left or lateral right, and adjust per challenge.

-"Blocked Atlantal Nerve Syndrome in Infants and Small Children", G. Gutmann, Manual Medicine, Springer-Verlag, 1987. Citation for upper cervical problems in children.

-C1 is capable by way the spinal accessory relationship with the nucleus ambiguous, along with the cranial root of the spinal accessory (which is really more medullary), and the cervical root (comes off, C2,3,4, and sometimes 5 and ascends through the foramen

magnum and goes laterally and forward to the jugular foramen and then comes down again; this area is sometimes referred to by anatomists as part of the vagus). That is why the atlas along with the spinal accessory seems to have profound effect upon digestion and other vagal neurological patterns.

-Gonstead technique for the occipitoatlantal and atlantoaxial levels state that the atlas is adaptive and not primary.

-Muscle Meridian technique sometimes responds to direct manipulation of the muscle meridian and/or the use of B and E Technique, but sometimes patients would require a repitition of it and there would be some recidivism.

-Dale Anderson, D.C. presented Muscle Meridians at a past ICAK meeting. Anderson described Ho points as taught in a course by Dr. Amaro. Ho points are basically on the elbows and knees, and they affect the muscle meridians. BL54, Ho point for the bladder meridian, if therapy localized and is involved, all of the muscles will test weak on the same side.

-There are 12 meridians and 12 months of the year. Each month the body audits itself and a patient may fail the audit/s which compounds problems.

-GJG had a patient who played tennis and had a complaint of a cold hand. GJG was unable to warm the patient's hand until he instituted use of the Ho points. GJG found the Ho point for the month of March, which is the stomach meridian, and tapping the convergent point was the treatment.

-There are four convergent points for all 12 meridians with one convergent point for 3 meridians. TLing the Ho point weakens all the muscles on the same side of the body. There is a spinal relationship for each of the 12 meridians (associated points), but there is no spinal associated point for the muscle meridians, except for C1. If all the muscles are weak on one side of the body with TL of a Ho point, TL of C1 with the thumb will negate the muscle weaknesses. You still have to stimulate the convergent point and then adjust C1.

-C1 is adaptive, most patients have a hidden C1 subluxation which ordinarily does not palpate or does not show up on x-ray (minimal deviation). The postural pattern is strong abdominal muscles that weaken upon a stretch of the muscle. The corollary pattern is ordinarily a hidden cervical disc that requires adjusting anterior to posterior down the facet line at C6 to C7, usually bilateral; and then TL of C1 with the thumbs; using Cervical Compaction. Due to the cerebellar relationship to C1 by way of the vestibular nucleus and by way of the medial longitudinal bundle, it is a good idea to interupt the TL to C1 to be sure that there is no cerebellar adaptation. The cerebellum is an error comparer and an error compensator. Parallel: catcher instructs pitcher to throw a strike, pitcher throws a strike, but it turns out to be a ball, and the catcher catches the ball and moves the glove to the center of the plate and calls it a strike.

-Dart: "Such splitting as occurs has been principally confined to the production of the three-sheeted layering (1) inner or transverse, (2) intermediate or internal oblique and (3) outer or external oblique characteristic of the trunk flexors, (thoracic and abdominal). In like fashion, the longitudinally-split arrangement

characteristic of the double-sheeted limb flexors and extensors was produced, as were the various subdivisions of the sacrospinalis mass. This splitting into sheaths, however, has given origin to a simple double-spiral mechanism of great importance to bodily economy, but the essential simplicity of which is frequently forgotten amidst anatomical detail. For example, let us follow the oblique direction of the fibers of the external oblique muscle, from the midline of the body, pubic symphysis and iliac crest upwards through the single morphological sheet formed by the external intercostalis, ribs and scalene musculature to the transverse processes of the cervical vertebrae, and thence through the deeper lying sheet, formed by the semispinalis musculature, to the cervical spine and occiput. Thus we get a picture, or a bird's eye view, of the manner in which the single superficial sheet, formed by these two opposed diagonally running flexor muscles in front, is continued, through a deeper lying extensor sheet on each side of the spine behind, to suspend the pelvis from the occiput and neck vertebrae. This diagonal suspensional arrangement becomes the more impressive when we recognize that the diagonal direction of pull excercised by each external oblique sheet (intercostal muscles and levatores costarum) is continued across the midline through the deeper lying internal oblique sheet to the perimeter of the pelvis on the opposite side of the body. Thus, any postural twist of the body (and the customary twist in a right handed person is a twisting of the trunk to the left) results in a postural rotation of the thorax, shoulder (right) and head, together with the vertebral column towards the opposite (left) iliac crest; there is also a relative inability to rotate the opposite or heterolateral (left) shoulder towards the homolateral (right) iliac crest. These diagonally disposed sheets, when followed in their continuity around the body, constitute two interwoven spiral layers. The pull excercised on the circumference of the pelvic basin, through the deeper lying (internal oblique) sheet from the ribs and the transverse processes of the spinal vertebrae of the contralateral side, by the superficial layer of muscles (external oblique, quadratus lumborum, external intercostal, levatores costarum and scalene), is a plane of traction that is being simultaneously exerted along the depper lying plane of pull by the deep (multifidus-semispinalis) sheet of the sacrospinalis from the spines of the vertebrae and the occiput. Thus in a very real sense, the occiput and spines of the vertebrae suspend the body by means of two spiral sheets of muscle encircling the trunk. This arrangement of the trunk musculature, in the form of interwoven double spiral sheets, is continued across the dorsal midline just as it is carried over the ventral midline. The superficial layer of the sacrospinalis sheet (iliocostalis, longissimus, and splenius) continues on to the posterior aspect of the ribs, cervical transverse processes and mastoid process the same oblique line of traction as is being exercised on the spines by the deep (or multifidus) sheet of the opposite side of the back. The whole trunk repeats, in its own fashion, the muscular story of the intestinal tract and of the heart, by becoming enwrapped by spiral coils of muscle, which are only prevented by the bony framework of the thorax and pelvis from

completely emptying its contents when they are contracted forcibly".

-What this means is when those muscles are weak after a stretch, and when the muscles pull, instead of pulling the pelvis up so that the patient stands up straight, they pull the body forward. That is why patients are ahead of the plumbline. That is why when patients get older, they move forward. We are trying to take the pull off of the cervical segment.

-Nutritional support may be necessary for the abdominal weakness in the form of ionic manganese and superoxide dismutase.

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-Right and left brain have different elements that each side is related to. Water-soluble are related to the left brain and fat-soluble are related to the right brain. Vitamin C, manganese, thyroid, and the acid ash minerals all relate to the left brain activity. Vitamin E, zinc, steroids, estrogen, alkaline ash minerals all relate to right brain activity. Based on the electron poising concepts of James P. Isaacs, M.D., vitamin A, iron, and copper were all mid-line, which means that they would not bother the right or left brain, or correct it, but sometimes were necessary to maintain function. Practical way to use the electrom poising concepts.

-In 1982 Wally Schmitt observed that tryptophane should be tested when left brain activity weakened right sided muscles, and right brain activity weakened left sided muscles; and tyrosine should be tested when left brain activity weakened left sided muscles, and right brain activity weakened right sided muscles. This is related to a pineal-pituitary alteration due to melatonin. Pyridoxine is needed to transform tyrosine into nor-epinephrine.

-In 1988 Frank Bahr and the Melanin Hypothesis was discussed in the Research Manual. If testing tryptophane weakens a patient, give them tyrosine, and if testing tyrosine weakens a patient, give them tryptophane. This is based on the concept that if the patient has too much tryptophane or tyrosine and you give it to them, it will weaken them. This is helpful in patient's with cyclic problems, i.e. pineal. We found this in individuals who had trouble with rythmic responses, seasonal, diurnal, circadian. Test these patients against tyrosine or tryptophane by placing a small amount of the tip of the tongue and have the patient insalivate it, and then test an appropriate muscle. If tyrosine weakened them, you give them tryptophane, and vice versa, about 500 milligrams. GJG would also measure variations in ranges of motion. If you give the opposite substance, it will reduce the range of motion, i.e. leg abduction.

-Leg turn-in in patients who are already taking sufficient sources of natural B and G would continue to show an imbalanced leg turn-in. Addition of the transformed form of vitamin B would equalize the leg turn-in. Some patients have trouble transforming vitamin B into the active form (i.e. pyridoxine to pyridoxal-5-phosphate).

-This is a preamble to the next discussion on ion resins. This was discussed at the 6th Annual Trace Mineral Conference at the University of Missouri where Isaacs talked about the use of ions on resins. Myers, a founding member of the Internation Academy of Applied Nutrition, also talked about ions, preparation of metal ions of trace minerals.

-"Trace Minerals, Vitamins, and Hormones In Long Term Treatment of Coronary Atherosclerotic Heart Disease", Isaacs, Lamb, et al., Johns Hopkins School of Medicine, Baltimore, Maryland, Piedmont School of Medicine, Atlanta, Georgia. Abstract: "25 patients with severe coronary atherosclerosis or documented myocardial infarction

had been followed for six years on a special regimen of oral zinc, copper, manganese on ion exchange resins...After five years of therapy, blood and urine levels for trace minerals were within normal ranges. No adverse effects of the trace minerals were detected clinically except zinc had significantly altered insulin requirements of diabetic patients, manganese had a sedative and muscle relaxing effect, copper and zinc improved assorted skin, arthritic, and ophthalmic disorders. Skin appearance and texture improved. Finger and toe nail pliability and growth rate and cuticle development were augmented. Hair density and growth rate, and scalp health seemed improved. Gum color, texture, healing of retractions, resistant to trauma, strength attachment to teeth were improved. Comments concerning improvement in epithelia were frequently volunteered by patients. The effects were not difficult to grade. Most of the effects seemed to be related to the trace minerals. Copper improved acuity of visual perception for red and green colors. Manganese had a sedative effect on some patients on the psyche. Along with copper, manganese also had a relaxing effect on skeletal muscle contracture, and fasciculation associated with the arthritis. Zinc and copper were particulary helpful for a variety of skin disorders, including staphyloccocal pyoderma. Zinc on exchange resin had such a remarkable effect on altering insulin requirements in diabetes mellitus that it should be marked for prime investigation in the future. The amount of minerals to produce these various effects in indeed small, and the minerals are much more active when given in the resin form than in the salt form".

-Another article from the 6th Annual Trace Mineral Conference: "Effects of Essential Trace Minerals to Thyrouricil and Lugol Solution on Human Tumors Transplanted Into Hamster Cheek Pouches", Isaacs, Lamb, Department of Pediatric Surgery and Anaesthesiology, Johns Hopkins University School of Medicine, Baltimore, MD. "We have found that trace metal ion, particularly copper and manganese, prevent the epithelial enlargement of the thyroid gland caused by thyroureal compounds..." Thyrouricil or propylthyrouricil has an effect that makes the thyroid enlarge and this was prevented by small amounts of ionic trace minerals.

-The frequent involvement of the piriformis and psoas muscles in dural torque and spinal biomechanical problems led to the accidental but practical use of bilateral "leg turn-in" technique. This early observation allowed the utilization of the transformed form of the B complex with much success utilizing simple leg turn in as indicator of general distortion. The correct nutrient on the tongue immediately balanced unequal leg turn in and in the case of equal leg turn in, the wrong nutrient on the tongue immediately produced unequal leg turn in, or in rare cases, leg turn out. This same method is useful along with regular diagnostic techniques to analyze for the presence of trace mineral deficiencies. For example, copper imbalances can produce zinc deficiencies. In the presence of equal leg turn in, or turn out, copper ions on the tongue will immediately cause unequal leg turn in, or in rare cases, leg turn out. The use of zinc ions on the tongue will immediately correct the evidence of leg turn in produced by the copper, for example, in the presence of a copper

elevation which frequently causes spinal problems. See Spine 5, 1977 "Elevated Copper In Idiopathic Scoliosis", Pratt and Phippen, and also by Schmitt amd Tolen, "The Cranial-Spnal Torque Pattern", ICAK Collected Papers, Summer 1983.

-Excess copper needs, as you can see per the chart from the Mulder's Interaction Chart, neutralization by zinc, manganese, molybdenum, and/or iron. Updated high tech hair analysis has now been found to be useful in diagnosing copper abnormalities, as well as blood cell analysis and 24 hour urinalysis. Folic acid and pantothenic acid are also needed when there is excess copper, as you learned from Schmitt's paper. The ionic form of the trace mineral resin based allows quick, immediate alteration in leg turn in and verification of the need for the appropriate nutrient trace mineral, or its antagonist, as the case may be. The immediate correlation and correction of a Vitamin A Deficiency Syndrome, which has not responded to appropriate and natural sources of vitamin A from the animal, vegetable, and fish origin, by the use of the silver ion is a frequently encountered pattern. The muscle testing response on ingestion of food nutrients, such as grains, dairy, and protein sources can be analyzed by the muscle weakness response and with the offending substance still in the mouth the correction of the muscle weakness by the appropriate therapy localization of the neurolymphatic for the thymus, B and E meridian points (usually for stomach as ST3), or the neurolymphatic for the liver. In the event of failure of correction of weakness induced by the offending food, the use of appropriate trace mineral ion therapy is indicated. Zinc and copper are frequently involved, but each case requires separate and thorough identification of the need for the appropriate ion or ions.

-Standard reference texts such as "The Biology of the Trace Elements", Shutte, Lippincott and Co., Philadelphia, and "Metabolic Aspects of Health", Myers and Schutte, Discovery Press, 1978, are good sources of information on trace minerals. The published material by Myers from the Internation Academy of Nutrition, 1976, and later, are also good sources. Sources from reputable nutrition companies servicing the healing arts professions are also valuable reference material. The unique capacity of the ion source resin based allows a quick response and with the appropriate muscle test indications offer a valuable guide for trace mineral therapy. Many times the use of an appropriate nutrient as a trace mineral does not produce the desired result because of a failure of absorption or utilization. Many times the source of the trace mineral of the non-ionic form can be likened to "putting a bowling ball through a keyhole". The ionic form seems to help in a failure of regular and indicated nutritional sources of the indicated trace minerals, this also includes the chelated form.

-In the article by Myers, "Biological Transmutation of Cobalt and Magnesium in the Support of Good Teeth and Good Health", he says in his conclusion that "Cobalt along with potassium, magnesium, copper, zinc, molybdenum, vanadium, silver, manganese, and iodine aid in the development of the brain, pituitary, thryoid, and other controlled devices of the endocrine and sympathetic nervous systems. These systems control the size, shape, and growth of the bony skeleton, and the size and shape of the dental arches. It is my

suggestion that cobalt is lost by transmutation in the food by cooking even when an adequate diet is consumed. Processing, cooking, sterilizing, and staleness cause a loss of valuble nutrients. This leads to degeneration of the skeleton and produces a face that is narrow and a dental arch too small for the teeth. Supplementation of the diet of the mother, and especially the baby with vitamins and minerals, especially minerals, will serve to correct many of these deficiency patterns which we see so rampant in our population".

-The original ions used by Dr. Isaacs in the Electron Poising Concepts were resin preparations for ionic trace minerals. Dr. Myers worked with Dr. Isaacs and used the resins obtained from the well known laboratory of Rohm and Haas for the source of the ionic resins. Incredibly small amounts of the resin based trace mineral ions seem to produce quick and observable results along with the appropriate structural correction.

-The use of the D.S.O. motif is appropriate here, Diagnose the need, Supply the need, Observe the result, act accordingly.

-Preparation of the metal ion resins: some of the resins are negatively charged which are used for the cations of zinc, cobalt, copper, iron, manganese, magnesium, and silver; and some of the resins are positively charged and are used for anions molybdenum and vanadium. GJG has not yet used any vanadium.

-It would be tempting to make the overly simplistic statement that anions would go on one side of the body and cations on the other side, but this is not the case. GJG does find that zinc works best on the right side and manganese works better on the left.

-Patient with difficulty seeing at night, requires more illumination when reading in the dark, left sided back pain that extends to the shoulder/neck, left upper extremity pain and paresthesias, pain may follow the facial nerve and give pain in the left upper molar teeth, sometimes into the lower molars as well. This can be made temporarily worse by the use of cysteine or methionine. When you test for leg turn in, you will very often find that one leg will turn in better than the other. This symptom pattern listed is often a need for ionic silver. Myers disclosed that silver is a component of natural vitamin A and represents the intrinsic factor that is needed in pernicious anemia. The silver was identified spectroscopically for Myers in two different instances and the remarkable specificity that the ionic silver has in operating on the stomach, facial nerve, and vagus nerve. It may be the factor that we have been looking for now to turn the intrinsic factor of pernicious anemia, the extrinsic factor being cobalt. Silver also has the remarkable effect on the stability of color vision, it makes colors much more vivid and aided in light sensitivity control in the retina, so that one can go into very dim light and have the retina remain sensitive, in other words, to see the menu in a restaurant, and then go into the bright sunlight and have the retina sensitivity greatly reduced again. An adequate amount of tyrosine and tryptophane must also be supplied as well as vitamin A. 6-8 granules of the ionic silver on the tongue will make an immediate change in the leg turn in, and in a week's time you will see a change in the vision.

-The action of cobalt is excellent, it has a tendency to

normalize the pulse and stops irregular ectopic beats. There is a pressure discomfort in the precordium just above the nipple, patients keep saying that they have pain in that area, and it is not related to the lack of iodine that you sometimes see in costochondritis, but the cobalt ions will relieve that chest pain and the chest feels much more open and easier to breathe, "can take a deep breath".

-Cobalt and copper are constantly complementary. If one gives too much cobalt, a tightness occurs in between the shoulder blades and a dryness of the mouth that is relieved by copper and molybdenum. If there is too much cobalt, they may need a natural source of thiamine to neutralize.

-Myers: When there is trouble with the skin, hair, and nails, the skin becomes stronger and tougher, and the fargility and thinness of the aged skin disappears, you see those black and blue spots on the back of the hand that you often see in older patients, the skin appears a lot more youthful, it does not get the blemishes of old age. In some remarkable cases, large black moles were simply washed away leaving normal skin on the face and arms. In many of his cases, warts cleared up in several weeks, and in one case, a hard, needle pointed, wood-like wart behind the right thumb began to soften within 15 minutes of giving cobalt and disappeared completely in two weeks. Another coral-brain type wart disappeared in another month with treatment with no return in 13 years. The basal cell of the growing nail needs both copper and cobalt. Cobalt need can produce a black disintegration of the nails and their base, especially if there has been some injury to it. The fungus that grows underneath them is the usual diagnosis, but not the cause, it is usually the after-result. When the nail is normal, the fungus just doesn't grow. Hair has a tendency to grow more rapidly. The strands are thicker and smoother, the ends do not split and fray. The hair is more easily dressed and lays in place. Several patients remarked that the natural curl returned to their hair which may be due to the copper. Copper effects on wool is to change it from a steely undesirable hay-like pattern into a normal quality crimpy organized pattern in 24 hours after supplying the sheep with the the right type of mineral. Before he knew the beneficial effects of copper on precordial distress he had treated a woman with vitamin A which did fine, but when he gave her vitamin E, the contact lenses she had been wearing for years suddenly caused the corneas to become very sensitive. She also had very wrinkled edges of the lips that looked like the edge of a pie crust decorated with the prongs of a fork. The lips were tender and sore as was the vaginal lips. The addition of cobalt ions in vert small amounts relieved the distress in all these areas as well as the precordial distress. Cobalt makes the eyes feel at ease and makes the eyeball nucleus much more lubricating. Copper plays a role here and makes the tears flow more freely and to relieve dryness. I have found that iodine, cobalt, and copper improve light sensitivity in the retina and reduces irritation from light glare and also makes vision sharper and colors much more vivid. In general, women do not get color blindness because of the difference in the x and y chromosomes. You will still see color blindness or color vision faults in women. Many times the ability to see red color returns in a remarkable way.

An artist/designer from the Maryland Institute was told by her artist husband that she could never see colors properly. At 44 years of age, following hysterectomy she suddenly realized that her ability to see color completely changed. After she had been on some cobalt and copper she could now see all the colors much more vividly, especially the color red. She could see red tints that evidently had been absent. The light intensity of her office increased 10 foot candles to her. Several artist patients had to repaint their pictures because they looked so dark and shadowy that they were dark and dismal. Styes disappear with the aid of zinc, iodine, and cobalt. Red, irritated corneas and lid margins clear up. The cholesterol accumulations on the lid will disappear with the use of cobalt showing us the direct effect of cobalt on cholesterol metabolism. Here you might need a little chromium. A lot of people have herpes simplex. Later sometimes, these patients might get herpes simplex with the menstrual period, and they would also sometimes get it in the vaginal lips. Cobalt is good for that. Molybdenum will precipitate a fever blister. Copper and molybdenum are in close balance. The excess in one produces a deficiency in the other. Shingles and herpes simplex are greatly aided in and completely relieved by magnesium (GJG finds magnesium by mouth to be very helpful, whereas Myers gives it intravenously in the form of magnesium chloride). Athlete's foot is a similar breakdown of the sympathetic nerves between the toes. Zinc and cobalt ions are mostly involved, zinc being the greatest requirement. Once the lesion appears, as in a fever blister, the fungus grows in the damaged tissue. The fungus is the usual diagnosis, but it doesn't start until there is a primary lesion. Spontaneous and profuse bleeding of the nose is difficult to stop. Cobalt stops the bleeding and improves the turgency of the turbinates removing the puffiness and bogginess of the nose, (GJG: along with C5 adjustment). Patient may need magnesium, A, E, and tryptophane and sometimes manganese. Some of these patients have pain in the upper left quadrant of the abdomen, extends up the left back, into the shoulder and arm, something similar to the silver as previously discussed. Often the entire andomen was so tender to the touch that in several cases the gall bladder had been removed to find the cause of the generalized abdominal discomfort, they had a lot of GI series, etc. With magnesium, zinc, copper, and cobalt by mouth, pain and discomfort was eliminated from the abdominal area. (GJG: Sometimes they need liquid iodine, 5 drops in 5 ounces of water, placed on a tampon and placed in every night for a couple of weeks). The cobalt had the greatest effect in the area of the pancreas and completely relieved the pain in the left back and side.

-Stambol, cardiologist with the southern division of the Albert Einstein Medical Center. Atherosclerosis was the result of cholesterol being deposited on the lining of the arteries, particularly in the coronary arteries. This is much more prevalent in men, a ratio of 6 to 1 to women. It is an established fact that the average age of women is greater than men. Note the number of women and men in retirement homes. This difference has been ascribed to estrogen and sporadic attempts have been made to give estrogen to men to correct that difference. Stambol has shown that it is more likely

due to the presence of another hormone-like substance produced in the ovary. This is called protein-bound iodine or diiodotyrosine.

-Breast tissue is produced from about 15 sweat glands in the skin which form the nipple and then grow backwards towards the chest wall. What were embryologically tortuous sweat glands develop into the globules of the breast like a bunch of grapes and each grape-like structure is called an alveolus. The alveoli secrete the cholesterol-type materials that contribute to the formation of milk and it appears that diiodotyrosine is a special hormone secreted by the ovary for the purpose of keeping the cholesterol substance in a liquid form. In the female, this is nature's method of keeping the wax-like cholesterol in solution. It is well known that the breasts are prone to develop cysts and abscesses which are due to improper functioning of this mechanism. Long before Myers knew of Stambol, he was using diiodotyrosine to soften breasts for nursing. In one patient, the left breast was involved in severe pain and induration, and required 200 grams of diiodotyrosine to bring the breast to normal. In another case the mother was nursing the baby with only fair results. The breasts were hard and very painful and having difficulty producing enough milk to satisfy the infant's hunger. The mother was given 10 grams of diiodotyrosine (GJG gives a single drop of iodine in water three times a day and 100-500 milligrams of tyrosine). The milk came out of the breasts within minutes under pressure and could be seen to spurt from the nipple for a distance of about 2 centimeters. This subsided when the pressure in the breast was relieved. The mother had no difficulty nursing the baby after this initial help. Breast soreness and heaviness has been repeated many times. There is a condition called Shimulbushes disease which is described as a bag of worms. The ducts more often feel like strand of spaghetti with nodules along them rather than worms as Shimulbush first described it. They are hard and sometimes form masses which are then described as cystic fibrosis and are quite easily visualized by xerography. The use of diiodotyrosine along with trace elements of magnesium, copper, cobalt, manganese, and silver has a remarkable effect on this syndrome with relieving the condition where the breast almost feels like liquid.

-GJG experience with hypothyroidism treatment with thyroid protomorphogen and iodine, especially iodine intravaginally, produced a remarkable imrpovement in these women. Not only did they have an imprvement in the systemic condition, but in the vaginal mucous. In the beginning, the mucous was thick, white, and paste-like; sometimes the patient would describe it as looking like cottage cheese. Just as iodine helps the flow of tears, so also does it help the mucous switch to a clear liquid fluid flowing from the cervix. Sometimes you spray the iodine on with an atomizer or place it in the vagina and the strand of mucous would be up to 4 centimeters in 15 minutes. This strand of mucous is present normally in women with sufficient iodine in their body. This secretion is used to lubricate the vaginal lining. Premarin ointment is used to lubricate the vagina. It may cause trouble with the mucous membranes and may make it difficult to have intercourse. Iodine also gets rid of infective organisms in the vagina. The carrier of the iodine is the unsaturated fatty acid

linoleic acid. In the case of severe vaginitis it is a good idea to use safflower oil or black currant seed oil as much as 10 capsules a day to get the iodine to get the mucous membrane back to normal. The same is true with Bartholian's cysts. The iodine makes thesecretions of the gland fluid so that the material will flow out without the liquification where the orifice is blocked, enlarged and a painful cyst appears. These are always present in hypothyroid patients. Need to check the circulation sex and triple warmer meridians. May find the circulation sex will be over and the triple warmer will be under. Balance these. Two other remarkable things occur with the application of iodine to the vaginal lining. First, a remarkable softening of the breasts, light and soft. Patients were aware of this change within several minutes of application the iodine. The second change is in the abdomen. Many of the patients complained of abdominal discomfort and after application of iodine, they comment on how comfortable they felt with their abdomen. If you cannot touch the abdomen of the patient, GJG uses 5 drops of iodine in 5 ounces of water. Insert into the vagina with a cotton (have the woman perform this herself). GJG notes that many times the abdominal discomfort is eased. There are cases of women where they had their gall bladder removed but this did not remove the source of the abdominal pain.

-Perkin and Brown, Boston, Mass, 1938: Remarkable insight into the functional differences between the male and the female, and the probable reason why the female lives so much longer without coronary artery disease from arteriosclerosis. Experiments on male dogs show that when the thyroid is removed, its protein-bound iodine drops to one-tenth of the normal value by the next day. When the thyroid is removed from the female, nothing happens until the ovary is removed also, at which time the protein-bound iodine level drops to the same level as the male after thyroidectomy. It is interesting to note that at the estrus period in the female dog which occurs twice a year (March and October), the protein-bound iodine doubles for a few days of her heat period. When she became pregnant, the protein-bound iodine levels dropped to very low levels, a little higher than after removal of the thyroid and ovary. It is important to remember that all these changes occurred in both the male and female dogs were being given an adequate amount of iodine every day (72 milligrams iodine daily in the form of a lugal solution into a stomach tube to assure that it was completely ingested. From these data and the proof by Stambol that the ovary manufactures diiodotyrosine, it can be inferred that the female is endowed with this ovarian function to make it possible for her to feed her offspring. In softening the cholesterol material in the glands of her breasts, she also keeps cholesterol in other parts of her body in solution, thus preventing it from precipitating in the ateries, heart, brain, etc. When one views the remarkable improvement in the woman from the higher intake of iodine protecting her against vaginal infections, cystic fibrosis, and breast pain, one has to rely on that she needs much more iodine that she is getting from the dietary intake, even with iodized salt.

-There are two other symptoms that appear both in the male and female from an additional supply of iodine. One is a loss of stiffness of the neck. Many complain that they cannot turn their head

freely and there is a constant soreness in the muscles of the neck. Frequently, even in younger women, the muscles feel more like steel wires than flexible muscles. They should be very pliable and soft to the touch. For some reason the left side of the neck is more involved than the right both in the male and the female. Why that is, GJG does not know. Although iodine plays a great role in relieving the stiffness, it is not in itself totally effective. Sometimes you have to add other trace minerals to get complete relief, which is also true of the abdomen and breast. Sometimes, you use the ionic form of magnesium, cobalt, copper, zinc, silver, and molybdenum; copper is the ion that has the most to do with the thyroid and catalyzes the formation of diiodotyrosine. A lot of people think that most people have too much copper. GJG finds just the opposite, many people require it. As in nature, copper is almost always associated with silver. It requires silver ions to completely relieve the pain in the left side of the neck and the back. Much of the shoulder/arm syndrome that was discussed earlier, both on the left and right, is relieved by silver, copper, and iodine. It is difficult to say how much one must give to see the changes, but you have to feel your way with these exchange resins (cation and anion resins). Relief comes within minutes, and you can palpate the neck muscles and feel the tension and knots in the muscles practically disappear. Silver seems to have the most effect on the stomach and esophagus which produces pain in the left back about the level of the fifth interspace. GJG has had patients where he was desparate to relieve that and nothing was working. Sometimes the pain there is so severe that the patient cannot press his back against a chair. Pain in this area is such a common occurance that it is difficult to find a patient that is completely free of it. When you place these ions on the tongue of patient, within a few minutes changes occur both in the breast, neck/back, and also the vision. Many times patients will remark that the lights have become brighter in the room. Tyrosine plays a role in the sympathetic nervous system, the visual apparatus. Apparently the activation of the tyrosine by copper produces higher sensitivity of the retina to light. It also improves color sensitivity, especially red is much more vivid and brilliant. There is hardly a patient that Dr. Meyers has seen that had normal red sensitivity, who is always improved by the addition of tyrosine, iodine, and trace elements.

-Much of this information has been abstracted by Meyers' work. When you give the right substance, you maintain the easy leg turn-in, if you give the wrong sustance, you immediately change the leg turn-in or turn-out. You have a standard by which you can apply single or combinations of the elements.

-The new packaging has a tip that allows easy dispensing of single granules, similar to that used in homeopathic doses. If you check the label, the dosage is 5-6 granules, once a day or as needed. In the past, GJG had the patient wet the pad of the little finger, turn the bottle up-side-down, take the proper dosage, and then scrape off the remainder. GJG recommends that you stay within the dosage limits on the package.

-Ion resins are available from DSD International, 640 East Purdue, Suite 106, Phoenix, AZ 85020, 1-800-232-3183 (west of the

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Mississippi). Viotron 1-800-437-1298 (east of the Mississippi).

-An ion is an electrically charged atom or group of atoms that may be positively or negatively charged, attacted to the anion or cation (the cathode or anode source), but it must become disassociated from its parent mass. In the human body, it's the water content that enables ioneization to occur. To get good results with ion therpay, you want to be sure that the patient is hydrated. When the patient places their finger on the tongue, you should be able to see a small sheen of saliva on the pad of the finger. Water is the universal solvent that provides that ideal atmosphere for ionization. You can demonstrate that very well. Distilled water, which is free of minerals, is a very poor conductor of electricity. When you add ordinary salt to it, the conductivity is improved because when the sodium chloride is placed into solution, certain sodium and chloride ions disassociate from the combined chemicals and become ionized or free atoms. These ions act like electrical hands to pass the current along. Ionized minerals help to facilitate varied reactions of chemoelectric effects. An ion in the body can consist of any electrically charged mineral. In the main, those minerals were thought to be calcium, potassium, phosphorous, and sodium, and then various tracce minerals were also concerned, especially iodine and iron. These mineral ions enable reactions to occur in special ways. Ionization is an imporant feature in explaining nerve conductivity. Enzymes systems, hormonal influences, even digestion and assimilation may depend upon the integrity of ionic equilibrium. For example, 90% of the zinc in the body is hooked up with carbonic acid anhydrase. That is very essential for the production of hydrochloric acid. There are all sorts of instances. The ions also act as ionic pumps, or bioelectric generators. The best known is the sodium and potassium pump. Pump pertains to an attraction and repulsion of the positively charged ions which then push or pull themselves across a cell membrane which theoretically acts like a grid to control the flow of current. That is the way it may simulate the action of a radio tube, which is in effect an electronic ionizer. It is probable that all the tissues rely upon ionic pumps or biochemical-electrical generators for their excitability. The nutitrion of the cell is dependent upon those influences. It is well known that too much or too little of electrolytes can cause cells to shrink or swell, which may cause problems with the cell. There is a very delicate balance of ionic equilibrium that influences our nutritional well being. Each tissue compartment, such as that within the cell, the fluid surrounding it, has its own mineral environment. This is what Dr. Isaac speaks of. One may be highly concentrated in potassium, as is the cell, whereas another is in sodium, as in the blood. Yet there is constant interchange of the small amounts between two compartments so that the elements are never static. Thus ions act as a pressure valve to avoid overloads. Potassium has the ability to make a lot of changes in cell metabolism, it is the principle alkaline mineral in the cell. Potassium something like an iceburg with only the tip showing to the diagnostician. It is a mystery mineral that requires a high degree of suspicion before an early diagnosis of its deficiency is likely to occur. Everyone can measure potassium on a SMAC, and you would think

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that the lab test would reveal a potassium deficiency, but such is not the case. You cannot tell how much water is left in a sealed can by looking at the size of the leak. The water may drip out right up to the last drop. Then and only then do you know that it is empty. It is the same way with potassium in the body, except, that the can that holds the potassium is in the cells. We can only see the drips of potassium that show up in the bloodstream, therefore there is not any real good method for accurately measuring the amount of reserve potassium in the cellular storehouse areas. That is why you have to have a high index of suspicion of deficiency. Because potassium is necessary for electrical phenomenon to occur in the body, it is necessary for growth, detoxification (that is why vegetable juices have an enviable reputation). Potassium deficiencies show up in diabetes, kidney disorders, heart disease, and many other conditions. The need for potassium is increased where any rapid cell building/proliferation goes on, especially in the case of healing or rapid growth. Potassium deficiency may be the aggravating factor in acne, no matter what else is being done. Any excess in sugar other than the natural source in fruits is utilized by the uptake of potassium. This causes an increase potassium need in the diet to make up for the extra demands. Nature combines natural sugar with large amounts of potassium. Man extracts this form of potassium in molasses, this upsets the balance, and a price must be paid. Discarding normal body potassium tissue sources is a bitter price to pay for sugar refining.

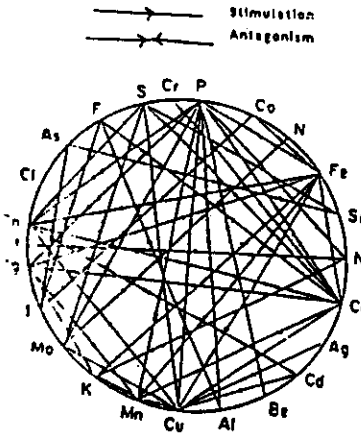
-When the adrenals are depleted, they cannot get rid of excess potassium.

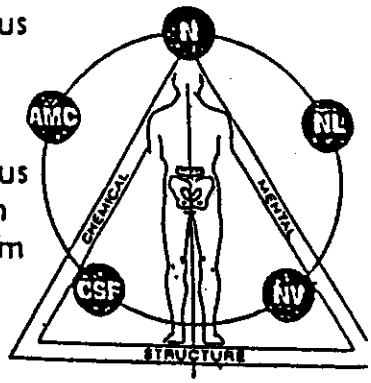
-Good signs of potassium deficiency are: sensitivity to light, lump in throat aggravated by emotion, heart pounds with fright but then does not calm down, acne/blemishes/boils, weight loss/failure to gain, slow healing, gas pains especially those connected to intestinal movement. Dietary sources of potassium are: vegetable juices, molasses, beet extracts, olives, broccoli, potatos, tomato juice. Sometimes you need to prime the pump with the ions.

-Ionic calcium is useful in excess secretions like "runny nose", watery eyes, drooling, sinus drainage, muscle soreness and cramps, menstrual cramps. May also need magnesium. Use the leg turn-in to diagnose whether the patient needs it or not.

MINERALS - HOW THEY RELATE TO EACH OTHER

CALCIUM	Depresses Manganese Depresses Magnesium Depresses Phosphorus Depresses Zinc
SODIUM	Depresses Potassium
ZINC	Depresses Iron Depresses Copper Depresses Phosphorus Depresses Cadmium
COBALT	Depresses Iron
CADMIUM	Depresses Copper
PHOSPHORUS	Depresses Zinc Depresses Iron Depresses Calcium Depresses Magnesium
POTASSIUM	Depresses Sodium Depresses Iron Depresses Manganese
MAGNESIUM	Depresses Phosphorus Depresses Calcium
MANGANESE	Depresses Iron Depresses Phosphorus Depresses Potassium Depresses Magnesium
COPPER	Depresses Iron Depresses Zinc Depresses Phosphorus
IRON	Depresses Potassium Depresses Phosphorus Depresses Copper





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DR. GOODHEART'S RESEARCH TAPES
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-Human skeletal muscles are composed of thousands of individual muscle fibers. One single muscle fiber is about the thickness of one hair and can reach a length of 10-15 centimeters, depending upon the length of the muscle. There are two types of muscle fibers, the slow twitch fibers (Type 1) and the fast twitch (Type II). In adition to these, there are intermediate types, IIa and IIb. Histochemical examination with ATPase stains makes it possible to discriminate slow and fast twitch fibers of an individual muscle. The postural muscles are mainly composed of slow twitch fibers in contrast to phasic muscles that are composed primarily of fast twitch. The slow twitch fibers contract more slowly, approximately 100 milliseconds, in contrast to the fast twitch fibers which contract in about 7 milliseconds. Slow twitch fibers obtain their energy primarily from glycogen and fat with high oxygen consumption and minimal lactic acid production. The fast twitch fibers obtain their energy from glucose (aerobic cycle) with a rapid production of lactic acid. The capillary supply is higher to the slow twitch as compared to the fast twitch, 4.8-5.0 capillaries for slow fibers and 2.9-3.0 capillaries per fast fibers. The slow twitch fibers fatigue after several hundred contractions in comparison to the fast twitch fibers which fatigue after just a few contractions. The slow twitch fibers are primarily innervated by the alpha 2 motorneurons with a great supply of muscle spindles. The fast twitch fibers are innervated by the alpha 1 motorneurons and have only a few muscle spindles. The fact that the muscle spindles are not randomly distributed seems to have significance in the development of functional pathologies of muscles. There are important differences between slow and fast twitch fibers.

-Examination of athletes show that the ratio between slow and fast twitch fibers is not fixed in any one muscle and can be changed by excercise. The quadricep muscle of a marathon runner consists of up to 93% slow twitch fibers while the musculature of an untrained person who does very little running consists of about 48% slow twitch. The biochemical studies done as far back as 1975 indicates that hypomobility of the lumbar spine and compression of the nerve roots can lead to changes of the fast twitch multifidus muscle by decreasing the fast fibers. The same study shows that patients with idiopathic scoliosis have more slow twitch fibers on the side of the convexity than on the concavity.

-Receptors found in voluntary muscles: muscle spindles, golgi tendon organs, paccinian corpuscles, free nerve endings (Type 4 nociceptors), synovial joint capsule receptors (Type 3 mechanoreceptors). The Type 4 and 3 are according to Barry Wycke. Distribution is quite variable of muscle spindles in voluntary muscle. Muscles which are responsible for very delicate and precise movement show a high number of muscle spindles than those that deal

with gross movements. For example, 50 muscle spindles are found in one gram of the rectus femoris muscle, in contrast to the small suboccipital muscles which have 150 muscle spindles per gram of muscle tissue. The paraspinal muscles such as the intertransverse muscles in the cervical region have as many as 200-300 muscle spindles per gram of muscle tissue. The muscle spindles are usually found in proximity to the slow twitch fibers.

-The muscle spindles contain 3-8 muscle fibers which are also known as intrafusal muscle fibers. These intrafusal muscle fibers are parallel to the normal skeletal musculature (extrafusal muscle fibers). When the extrafusal muscle fibers contract, tension in that part of the muscle spindle that does not contract decreases. When the muscle is stretched, tension in both intrafusal and extrafusal muscle fibers is increased. The center of the muscle spindle is sensory, not contractile, and harbors many nuclei.

-Golgi tendon organs consist of a large myelinated nerve fiber (12-18 micrometers in diameter) which terminate with a spray of fine endings between bundles of the collagenous fibers of tendons usually near the musculotendinous junction. To understand their function, it is essential to recognize the fact that they are arranged in series with the extrafusal muscle fibers. Whether the muscle contracts or is stretched, the tendon organ will be stimulated, since in both cases the tension of the tendon organ will increase. In other words the tendon organs are tension recorders, while the muscle spindles give information about the length of the muscle. The muscle spindles also give information about the speed of stretch. For example, if you hold a melon in your hand at the market, and they cost $0.39, but one looks bigger, you test the weight by the tension on the spindle cells in the biceps. If you pick up something that looks like a melon, but it is actually a steel ball painted to look like a melon and you misjudge the weight, you will stretch the biceps so fast that you cannot accomodate to it. When that happens the biceps muscle will contract and it will look like you want to stretch it because the elbow is flexed. If that is a problem and you cannot straighten the arm, if you bring the muscle back to its original position (shorten the muscle by flexing the elbow), then spread your fingers on the biceps belly on the spindles, with inspiration, 30-40 seconds (strain-counterstrain). After that, you should be able to stretch your elbow out in normal fashion.

-The myotonic reflex arc of the phasic reflex depends upon a muscle spindle with intrafusal fibers which contract due to stimulation by the gamma-1-neurons. In this fashion, the central portion of the muscle spindle (the non-contractile, central "sensory" portion) is stretched which in turn stimulates the spiral endings of the Ia-fibers which surround this central "sensory" portion. This stimulation id responsible for the elicitation of a specific automatic myotonic reflex, in which the phasic motorneurons of the alpha I fibers are stimulated via afferent I1-fibers along with their direct reflex collaterals thus causing contraction of the

muscles involved. The gamma-1 firing then eventually causes a decrease in the length of the highly sensitive receptors in the central portion of the muscles spindle. The length of the muscle spindle and therefore the entire muscle tends to be kept constant automatically via the phasic myotonic reflex. The strength of this reflex depends on the strength of the external force as well as the firing rate of the gamma-1 fibers. The intrafusal muscle fibers of the slender muscle spindle contract as a result of the influence of the tonic gamma-2 fibers. When the secondary sensory endings located on the intrafusal muscle fibers change their length due to external stretch these secondary endings send impulses to the ventral horn cells of type alpha-2 via afferent type II fibers and associated multisynaptic pathways. These alpha-2 motorneurons cause the slow postural musculature to contract; the length of these postural muscles is maintained as long as the spindle is kept in a certain contraction state via the gamma-2 neurons. In addition to this tonic stretch reflex, the stretch receptors of the golgi tendon organs take part in the regulation of muscular tension. These golgi organs are located at the junction between muscles and tendons. Rapidly conducting I-b fibers lead from these golgi tendon tension receptors to the spinal cord causing inhibition via several interneurons. They act on both the alpha-2 neurons thereby inhibiting of postural musculature which results from the tonic stretch reflex and also the motorneurons of the alpha-1 fibers and their respective fibers for the phasic extensor motions. This crossed inhibition reflex together with the extensor reflex (which originates from secondary endings) regulates the muscular tension even when it is under the influence of external stretching forces. In addition to impulses from the tendon organs, there is another mechanism that tends to limit the activity of the excited motorneurons, the so-called recurrent or Renshaw inhibition. When a motorneuron fires impulses, these will pass via its recurrent collaterals to Renshaw cells; these are neurons, short axons situated in the ventral horn, having an inhibitory effect on the motorneurons. The Renshaw cells, as with many other interneurons in the cord, are subjected to supraspinal control, for example from the the cerebellum and the mesencephalic reticular formation.

-Schmitt developed gamma 1 and gamma 2 muscle testing. Most of the time you are doing doctor initiated muscle testing, but sometimes there is patient initiated muscle testing response. Doctor initiated produces effects that are influenced by dural involvement. Patient initiated muscle testing produces effects that are supraspinal in character, indicating some type of cranial fault or possibly response from the cerebrum and cerebellum.

-"Empirical Approaches to the Validation of Spinal Manipulation", from the Michigan State College of Human Medicine in Lansing, Michigan. Gamma 2 neurons reflect the normal regulation of the muscle tension. Gamma 1 reflects the influence on muscle length.

-The golgi tendon organs are located near the musculotendinous junction. To understand their function it is important to recognize that they are arranged in a series with the extrafusal muscle fibers so that whether the muscle is stretched or contracts, the tendon organ will be stimulated. In contraction, the extrafusal muscle fibers have tension put on them, but the intrafusal muscle fibers have reduced tension, whereas when the muscle is stretched, both the extrafusal and intrafusal muscle fibers will be conscious of the increased tension.

-Muscle testing can be doctor initiated or patient initiated. Work from the Los Angeles College of Chiropractic: used doctor and patient initiated muscle testing. They used a mechanical device and there was increased statistical evidence that there was more uniformity with patient initiated muscle testing than with doctor initiated muscle testing, but both yielded fairly good results.

-The discussion to follow will focus on patient initiated muscle testing. RMA = repeated muscle activation. This is performed by the patient only.

-Testing of the abdominal muscles in the standard fashion, either seated or standing, will often test strong. A stretch of the muscle may weaken the abdominal, a contraction may weaken the abdominal (Travell vs. Jones). RMA is neither of these conditions.

-RMA is where the patient does his own muscle contraction. For example, in the case of the abdominal, have the patient bend forward 10 times, then the abdominal muscle may weaken. GJG notes a high percentage of this finding, along with left and right rotation weakening that was previously intact (strong).

-This came about from one particular patient. The patient injured himself moving and experienced double vision. He did not respond to other conservative treatment, so he saw GJG. After cranial technique, his double vision had improved. 2-3 months after that problem cleared up, he began having trouble with his right knee with an increasing genu valgum on the right which again did not respond to other conservative treatment, so he again saw GJG. GJG expected to find increased tension on the lateral side of the knee and decreased tension on the medial side of the knee due to the positioning, but this did not show. The patient did not respond and was getting worse to the point of needing a cane. It was getting difficult to muscle test due to the pain. X-rays were negative. There was no pain when he stood or sat, only when he moved. GJG had the patient move his own muscles and then tested to see what would happen with continued movement of the muscles. To the surprise of GJG, the only muscle that weakened was the tensor fascia lata, which was exactly opposite of the scenario that he had created in his mind (the genu valgum), one would have expected the opposite to be true (normal or hypertonic tensor fascia lata). He theorized that the body knows its weak, but when he had the minor fall (initial injury while moving), maybe he injured the tensor fascia lata. Flexion and extension weakened only the tensor fascia lata, not the quadricep,

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piriformis, popliteus, sartorius, gracilis. GJG performed origin-insertion on the tensor fascia lata with immediate improvement in normal gait and lack of pain. This required a couple of repititions, on another treatment, he showed the pattern on the sartorius.

-The origin-insertion treatment on the tensor fascia lata was effective, but had a tendency to recur. GJG tried various nutrients, a non-heat processed veal bone, choline. The patient responded, but then would deteriorate. GJG felt there was a nutritional need that he couldn't identify. The only thing that seemed to help was the veal bone, but it did not last.

-The golgi tendon organ and muscle spindle cells block nociceptor input into the spinal cord, directly and indirectly. Pain (nociceptor input) is blocked by mechanoreceptor input. Therefore, if there is abnormal joint motion, normal joint proprioception is lost and this results in a decrease in the background mechanoreceptor activity which is necessary to maintain a minimal threshold of pain. When the pain threshold is decreased the patient may perceive a sensation that is normally not painful as painful. This perpetuates the problem.

-90% of the patients who showed the RMA pattern had a cerebellar involvement (simple thumb TL to C1 or the occiput) and many showed evidence for an occipital or spinal fixation. Many showed weakness of the extensor muscles which are basically cerebellar involved. Most of them showed a change with activation of the TMJ.

-Muscle testing of the gluteus medius can be accomplished side-lying (after Kendall and Kendall), in the supine position with straight leg abduction and a neutral toe (after Beardall), or in the standing position with leg abduction. If strong, have the patient activate the gluteus medius (RMA, repeated muscle activation) 10 times by abducting the leg in the standing position, then retest the gluteus medius in the standing position, it will weaken in the RMA pattern. This responds to origin-insertion technique of the gluteus medius. This is true for any muscles.

-Possible explanation for this is the relationship between the intrafusal and extrafusal muscle fibers. There is a definite difference between a regular muscle test and the RMA. Duke University has done some work that leads to the conclusion that a lot of athletic injuries are due to microavulsion. Microavulsion represents the random effect of trauma (often unremembered) and the need for origin-insertion. The usual treatment was pressure on the spindle cell towards the belly of the muscle and on the golgi tendon organ towards its insertion, and the addition of non-heat processed veal bone did not suffice. The addition of choline was "spotty" until GJG figured out that it was the golgi tendon avulsion changed the logistics of the availability of acetylcholine.

-The Journal of Nutrition, 1954, Holve and Copeland, Alabama PolyTechnical Institute: "One function of vitamin E is to bring about the synthesis of acetylcholine from choline and acetate.

Pantothenic acid is also involved. A deficiency of choline as well as a deficiency of vitamin E leads to muscular dystrophy. Rabbits given choline deficient diets developed muscular dystrophy between the 7th and 100th day. Symptoms were identical to dystrophy produced by vitamin E deficient diet. When choline was fed in the diet, all signs of muscle weakness subsided in 4 days. The kind of disorder produced by choline deficiency is more like that seen in humans with muscular dystrophy. In human muscular dystrophy there is usually no evidence of severe liver damage."

-The Archives of Pediatrics, Vol. 49, 1949: "25 muscular dystrophy children treated with fresh wheat germ oil, B complex, and C complex all improved, one completely.

-The Internal Record of Medicine and General Practice Clinics, February 1954, Martin: "Complete cure of muscular dystrophy with all natural food substances. Whole grain cereals, raw certified milk, fresh raw fruits, vegetables, eggs, fish, meat, and cheese. Cheese preferably from raw milk and raw cream as well as the use of vitamin E."

-The oil form and the dry form of vitamin E are equally effective GJG's experience. The presence or absence of selenium doesn't seem to be a factor, but GJG thinks that since selenium is a co-factor for vitamin E, he uses that form.

-Scientific American, April 1982: "Uptake and conversion of choline is important. Choline in the extracellular fluid of the brain is taken up by the terminal of the cholinergic neuron and is converted into acetylcholine by the action of vitamin E. As acetylcholine is released into the synaptic cleft when the neuron fires, the acetylcholine may interact with a receptor and thereby transmit a signal to the postsynaptic cells. Alternately, the neurotransmitter may be converted back into choline which may be then again taken up by the presynaptic terminal and may enter the extracellular fluid and the bloodstream."

-1984 Applied Kinesiology Research Manual: "If you pick up a newspaper you'll see many advertisements for Dr. so and so's quick weight loss clinic or other rapid weight loss establishments. You seldom see Dr. so and so's quick weight gain clinic. In general there are more lazy people than there are energenic people, although all of us know people who are the exception to this rule. So it would seem reasonable that people in our culture and population have nervous systems that are prejudiced towards the eat and get fat parasympathetic system as opposed to the fight or flee, slim and trim sympathetic nervous system. This is a general observation. We all know people who have trouble gaining weight despite adequate caloric intake, but this is the exception. The preponderance of the population are in the other area where weight loss is a desirable goal and certainly there are more books on how to lose weight than there are texts that describe how to gain weight. Certainly, the evidence is apparent that there are more people who are lazy than are energetic. Since these facts are self evident, there must be a

reason for this evidence. The chemical nature of the transmitter at the neural junction outside the central nervous system is well known. At the neuromuscular junction, release of acetylcholine by motor neurons stimulate the endplate of the membrane of the muscle fiber. Acetylcholine is release to all and at all autonomic ganglia by the preganglionic neurons and is the transmitter of all parasympathetic cholinergic and some sympathetic adrenergic neural effector junctions. For the rest of the sympathetic junctions, the neurotransmitter is noradrenalin. Therefore, all preganglionic neurons of both adrenergic and cholinergic systems require acetylcholine and all postganglionic neurons of the cholinergic system requires acetylcholine, and even some postganglionic adrenergic neurons use acetylcholine. Therefore the body is prejudiced towards the eat and get fat side of the nervous system and three times as much choline is needed as is adrenalin. If the adrenergic nervous system is deficient in neurotransmitters at the preganglionic site, sources of adrenal material will produce profound weakness of these muscles if they are found strong in the clear, instead of producing strength in the muscles, or if stengthened by TL of any of the 5 factors, adrenal material placed on the lingual receptors produced weakness of the sartorius/gracilis/posterior tibial. The sartorius and gracilis muscles in these patients were therefore weakened by sources of Drenamin, Drenatrophin, Stereotrophic Adrenal Extracts, and other adrenal support supplements available, by greatly aided by choline."

-The key to this discussion is not the choline that is necessarily needed, what is needed is acetylcholine and many times it is the lack of availability of vitamin E (one of the best antioxidants) to synthesize the acetylcholine combination. Recall the Journal of Nutrition reference above, that "the function of vitamin E is to bring about the synthesis of acetylcholine from acetate and choline, pantothenic acid is sometimes involved." Sometimes the patient needs a source of vitamin B. The dry form of E or wheat germ oil works well.

-The next time you see a patient whose head is not level and you've tested the anterior neck flexors, and against the 5 IVF factors, and you've done everything you know of to examine occipital lesions, cervical fixations, EID, BID, aerobic, anaerobic, etc., simply test the anterior neck flexors which are strong, have the patient turn their head to the left to activate the right SCM 10 times may weaken the right SCM. May find this right SCM weak with RMA with the head LOW on the right, even though this seems paradoxical. When the body thinks that the muscle is weak, the body tightens the muscle and that's why the head goes down on the weak side, a kind of stupid body wisdom.

-RMA indicates that there is not enough available acetylcholine im the presence of a microavulsion.

-It is the acetylcholine that is in short supply and this can be shown the following way. Have the patient turn their head to the

left and then you bring it back to neutral. This will not weaken the right SCM. Then have the patient flex and extend the wrist 10 times, then have the patient turn their head to the left and you bring it back to neutral, and now that right SCM will test weak. This shows that it is the use of acetylcholine by the wrist motion and then the acetylcholine is in short supply for the SCM test. This is induced by the microavulsion and requires the strong heavy pressure at the origin-insertion, just as GJG did back in 1964. GJG thinks this is a function of the cerebellum.

-Quote from the paper that GJG presented at the Winter 1994 ICAK meeting: "The neuronal circuitry that is found in the cerebellar cortex is consistent with the notion that it may function as a recognition machine. Preliminary recognition processes are also carried out at the points of convergence of and onto the neurons containing the fibers input to the cerebellum. The descending fibers contributing to the cortico-ponto-cerebellar pathways are intermingled with the descending fibers of the corticospinal system. Presumably, therefore, the cells of origin of these two pathways in the cerebellar cortex also lie close together. Accordingly, while we don't know the precise nature of the signals passing in the cortico-ponto-cerebellar pathways, it seems reasonable to suppose that this pathway responds and reports to the cerebellum something of the "intentions" that are formulated in the cerebral cortex and are thus conveyed to the motor system in the corticospinal tract. The cerebellum is thus in a position to invite a comparison between the state of affairs as reported by the peripheral receptors and a desired state as formulated in the cerebral cortex. It receives information from golgi tendon organs telling it what forces are being made by various muscles and the information from other receptors that may tell it about the consequences in terms of interaction between the body and its environment that follow any particular pattern of motor command. In other words, GJG feels that there is a failure of feedback that comes from the microavulsion and that has to be mechanically corrected in the presence of adequate amounts of natural sources of vitamin E."

-GJG explored a relationship between the calcium sodium potassium pump and the nernst potentials that Guyton talks about in his phsiology sections on neuromuscular transmission and acetylcholine. There does not seem to be a relationship between the RMA and the calcium sodium potassium pump. It is more related to vitamin E and the synthesis of acetylcholine.

-Walking and Limping, Ducroquet, Lippincot Book: Section where the beginning of walking is discussed. "It is the gluteus medius that maintains the relative horizontality of the pelvis. The lateral abdominal muscles on the opposite side act with the gluteus medius in close synergy. It's this action performed by these two muscular groups that permits the harmonious transfer at the thoracic center of gravity in the frontal view. There are instances of weakness of the gluteus medius where the opposite abdominal will preserve in

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part the pelvic horizontality in the body action of suspension. In normal walking, the lateral inclination has two purposes, transfer of the thoracic center of gravity laterally, and reinforcement of the action of the opposite lateral abdominal muscle by the separation of the pelvic and thoracic insertions."

-Sometimes the most common thing you'll see will be in people who have differences in blood pressure sitting, standing, and lying, in addition to the adrenal effect. Sometimes you'll see a patient after they have had the "flu" where they say they are so tired that they don't want to get up, but you don't find the normal adrenal situation with the blood pressure dropping, pupils dilating, etc., yet one of the reasons they feel so badly is that the normal action of the gluteus medius and opposite lateral abdominal is not working and the patient feels as though they are not getting around well and they have to work too hard just to stand up and walk. They need the RMA pattern checked, origin-insertion technique, and a source of vitamin E (wheat germ oil or dry E). Temporary value comes from veal bone and choline, but E is for the long term. In our present society with all the sources of the wrong types of fats and the emphasis on reducing fats, the few sources of vitamin E now become even more meager and more wide spread in its deficiency.

-Try to show the RMA pattern in the abdominal muscle of a patient that does abdominal crunches for excercise. When the activate their abdominal muscles, they then test weak.

-European Chiropractic Union, Fred Lee, 1971: "Look at this walker, he is not lifting his body with extension of the foot, but now look at the same walker from behind. With each step his pelvis makes an excursion towards the side of the carrying leg. This sideways movement of the pelvis changes the trochanteric angle of incidence on the side on which the carrying step is made. The angle becomes more acute, therefore, the pelvis is lifted up on the respective side. The muscle, gluteus medius, stabilizes the entire ilium at this second at the level of the carrying leg, so now the inclined pelvis should come back to the horizontal and higher level. Thus the other leg, the swinging leg is automatically lifted over the ground. The effect of gravity is displaced with each step. The oscillation of the body upward and downward while walking is therefore the result of the alteration of the trochanteric angle of incidence and the work of the gluteus medius. (GJG adds to this the opposite lateral abdominals.) The normal stepping forward, the sacrum is pulled upward by the sacrospinalis on the forward stepping side, so that the scissor-like sacroiliac articulation closes at the top. If the torsion is blocked on this forward stepping side, the sacrospinalis nevertheless contracts rhythmically with each step. its contraction reduces its own length and the spine must give way on the other side, in this instance, the left side. This is the reason for the painful contracted psoas, it's partly antagonistic on the left side. The gluteus medius on the right side must provide help by contracting more than normally and also becomes painful.

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When the gluteus medius fails, for example on the right side, the stabilization of the pelvis in the horizontal plane also fails when the left foot swings forward. The oscillations of the pelvis for the purpose of rhythmic change of the trochanteric angle of incidence continues to exist when movement of the inward rotators at the time of right step forward pulls the pelvis sideways at the same movement time towards that side. When the right gluteus medius is paralyzed, the pelvis is not lifted according to the trochanteric angle of incidence. The left leg has no freedom to swing forward. When the gluteus medius is paralyzed, the whole pelvis is subjected to incorrect rhythmical movements, the spine bends in the direction of the paralyzed gluteus medius, or the weakened one."

-It is interesting to note how many gluteus medius weaknesses you will find using the RMA pattern.

-Hiatal hernia diagnosis: TL with the fingers together the same way that you do for an adhesion complex, using the neck flexors as the indicator muscle. Challenge by pressing upward and testing the pectoralis major clavicular. Treatment is a pulling down coincident with expiration, balance the crura of the diaphragm by balancing the psoas muscles, and they generally need hydrochloric acid and okra pepsin. Check the psoas against the the RMA pattern.

-Illi: "We have muscles which rotate the leg inward (inward rotators) and we have muscles which rotate the leg outward (outward rotators). The relation of the strength of the outward and inward rotators is 155:50. When a person steps forward, the inward rotators normally rotate the leg medially as a muscle bundle acting in antagonism. Therefore the ilium rotates backwards on this side. At the same time the weight bearing leg is rotated outward on the left by the outward rotators, but now the foot is stabilized on the ground and therefore acts as an axis so the ilium rotates forward in its turn. Throughout this you are looking at x-ray motion pictures of the spine while walking. Now you see the consequences correctly. With each step forward the ilium makes a countertorsion backward and vice versa when the leg carrying the body is behind and the ilium turns forward on its side. This reduces to a minimun the torsion made rhythmically by the pelvis. Note how the trochanter leads with each step and the projection becomes smaller and smaller. That lead becomes smaller and smaller following the rhythmical movement forward and backward. The sacroiliac articulation naturally becomes involved. It lies between the immobile spine and the mobile femur heads. The point of the triangle of the sacrum is below and the base above. Certainly there remains only two more movements that have to be damped down. One is the rhythmic movements of the pelvis to and fro, the complication of which is the fifth lumbar vertebra which are not suitable. It's clear thanks to the perfect work of the inward rotators and gluteus medius, the pelvis remains horizontal for the major portion of time in its rhythm with spine will not be

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involved in the false movement. This means that the compensation of walking is affected in full by the sacroiliac articulations."

-RMA (repeated muscle activation) involves testing the muscle after the patient has contracted it 10 times. The stupid body wisdom reveals itself in that the muscle is injured and actually weak, but the body tightens up the muscle. You will often find the sternocleidomastoid weak with RMA on the low occiput side. Treatment is origin-insertion and a form of vitamin E to stabilize the acetylcholine.

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