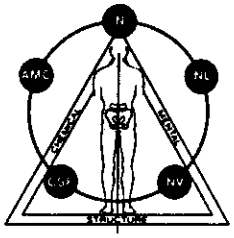


PROCEEDINGS
OF THE
SUMMER MEETING OF THE
INTERNATIONAL COLLEGE OF APPLIED KINESIOLOGY - U.S.A.

Volume I, 1992 - 93

PRESENTED JUNE 17 THROUGH JUNE 20, 1992
DEARBORN, MICHIGAN

A MESSAGE FROM THE CHAIRMAN

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

In thinking about a focus for this volume, I felt the need to look back at the past three issues and was not too surprised that what was felt then is as obvious now. I'd like to emphasize the feelings of those past messages.

These papers are "the sharing of creativity" and "a cornerstone of the ICAK." This principle has endured throughout our history and will be part of our future. And those who "constructively critique" and provide "positive feedback" at the meetings are just as important for our organization.

But let's not forget the bottom line: "one small piece of information from one doctor may be developed by another, and expanded upon by a third, providing dozens of doctors and thousands of patients untold benefits."

Finally, "to everyone, congratulations for being part of a most unique health care organization."

I look forward to seeing you at our Dearborn meeting, June 17-20, 1992.

Introduction

This thirty-third collection of papers from members of the International College of Applied Kinesiology-U.S.A. contains 43 papers by 31 authors. The papers will be presented by the authors to the general membership at the Summer Meeting of ICAK-U.S.A. in Dearborn, MI, June 17-20, 1992. 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 of Proceedings" 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. Dr. George J. Goodheart's Research Report is also included with this edition. This contribution by Dr. Goodheart is made so that all ICAK-U.S.A. members will have up-to-date material regarding his research.

Neither the International College of Applied Kinesiology-U.S.A., its Executive Board or 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.

The Proceedings of the ICAK-U.S.A. are published twice annually, prior to the summer and winter meetings. 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).

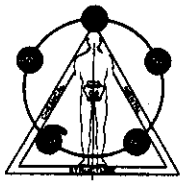
10) The body of the article should be single-spaced on plain paper. No papers typed on office letterhead will be accepted. The manuscript must be an original with dark print, on one side of the paper only, to ensure adequate reproduction in *The Proceedings of the ICAK-U.S.A.* The margins on both sides o 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 25276, Shawnee Mission, KS 66225, (913) 648-2828.

INTERNATIONAL COLLEGE OF APPLIED KINESIOLOGY STATUS STATEMENT



The use of manual muscle testing to evaluate body function as expressed through neuromuscular pathways was introduced by George J. Goodheart, Jr., D.C. in 1964. Applied kinesiology knowledge has continued to expand to provide an additional dimension to the diagnosis of human dysfunction.

Early in AK's development, it became obvious that many treatment methods used in chiropractic and other healing arts disciplines improved neuromuscular function as perceived by manual muscle testing. Standard therapeutic approches comprise the majority of treatment procedures used by applied kinesiologists. Amplification and modification of some of the treatment procedures have occurred as improved approaches have been developed. Some treatment techniques have also been developed which are unique to applied kinesiology.

The most important value of applied kinesiology is its ability as a system to evaluate function via the neuromuscular system to give added dimension to diagnosis. The manual muscle test evaluates the ability of the body's controlling system - the nervous system - to adapt the muscle to meet the changing pressure of the examiner's test. This requires that the examiner be well-trained in the anatomy, physiology, and neurology of muscle function. The action of the muscle being tested, as well as how the body recruits synergistic muscles, must be known. Manual muscle testing is a science and an art, with emphasis on the science.

Many unique observations have been made in applied kinesiology which have given a better insight to body function. It is the International College of Applied Kinesiology's (ICAK) position that the applied kinesiology examination should be combined with approved standard physical diagnosis, laboratory, X-ray, history, and any other special examination procedures of the physician using applied kinesiology as an adjunct to diagnosis. AK examination should enhance standard diagnosis and be enhanced by standard diagnosis.

Applied kinesiology methods add information to an examination, but they should always be used as a part of a multi-faceted investigative endeavor. These procedures - such as therapy localization, nutritional testing, establishing maxillo-mandibular relationships, the muscle-organ association, etc. - can help the physician determine the major cause of a patient's health problem. They should be used with other supporting evidence from standard techniques in diagnosis. A limited approach, whatever the method, can lead to error.

Therapy localization is a phenomenon which is a reproducible clinical tool. Efforts have been and are being made to better understand the mechanism. When positive therapy localization is present, other examination findings should be used to determine, and finally confirm - the diagnosis. For example, positive therapy localization to a vertebral area indicates further examination by palpation of the intrinsic muscles and the structures innervated by the area. Finally, when all factors are considered and a subluxation or fixation is diagnosed and adjusted, therapy localization (as well as other findings) provides the physician with neuromuscular biofeedback as to whether the corrective effort was successful.

Nutritional and chemical evaluation should only be done with the substance stimulating the subject's olfactory or gustatory receptors. It is also necessary to evaluate other factors which may influence the perceived muscle strength. Confirming diagnostic criteria for the need of any nutrition should be present from the patient's other diagnostic work-up, which may include history, type of dysfunction, laboratory tests, physical diagnosis, and dietary inadequacies. Research sponsored by the ICAK (1) revealed a random response to blind testing of nutrition when the latissimus dorsi muscle was tested. Further research is underway to put into perspective the change perceived in manual muscle testing when nutrition is tested. An adequate educational background is needed in evaluating nutritional needs and manual muscle testing. The use of manual muscle testing by lay salespeople has created problems due to their untrained nature and enthusiasm to sell their products.

The muscle-organ/gland association used in applied kinesiology is referred to as part of "body language." A close clinical association has been observed between specific muscle dysfunction and related organ or gland dysfunction. This viscerosomatic relationship is but one of many sources of muscle weakness. Placed into proper 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. An actual diagnosis of anemia is only justified by laboratory analysis of the patient's blood. Body language indications of AK come from the muscle-organ/gland association and other considerations in applied kinesiology. Further examinations confirm or rule out an association in the particular case being studied. It is the physician's total diagnostic work-up which determines the final diagnosis.

There are both lay persons and professionals who use a form of manual muscle testing without the necessary expertise. There are others who fail to coordinate the muscle testing findings with other standard diagnostic procedures. These are sources of error which may lead to misinterpretation of the condition present and thus to improper treatment, or failure to treat the appropriate condition.

When put into proper perspective, applied kinesiology is a tool for evaluating the impact on the nervous system of a multiplicity of endogenous and exogenous stimuli. It is indeed adding a new dimension to diagnosis. Its greatest value is in functional problems. It helps the physician understand functional symptomatic complexes. Along with the usual diagnostic procedures, it helps differentiate functional from pathological factors when pathology has developed.

The proper use of applied kinesiology requires an appreciation and understanding of anatomy, physiology, and functional neurological relationships. In addition, the physician must have an excellent understanding of muscular synergism to be able to properly administer manual muscle testing.

REFERENCE

1. Triano, John J., "Muscle Strength Testing as a Diagnostic Screen for Supplemental Nutrition Therapy: A Blind Study," *Journal of Manipulative and Physiological Therapeutics, Vol. 5, No. 4 (December 1982).*

Approved by the Executive Board of the ICAK January, 1988.

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DR. GEORGE J. GOODHEART - RESEARCH REPORT

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

DIVISION I - INFORMATIVE PAPERS

BUCCINATOR STRETCH CHALLENGE

Richard M. Burger, D.C.

Abstract:

The buccinator muscle may be a significant factor in cranial and temporomandibular joint involvement with a reactive muscle type of relationship with the masseter. Frequently there is an associated hypertonic temporalis muscle. The buccinator may be challenged with stretch by filling both cheeks as full as possible with air. Lateralizing the full cheek gives indication of the side of involvement; rarely bilateral. Confirmation by therapy localization is possible. Treatment is by approximating the muscle belly. This procedure should be a screening test in the evaluation of the stomatognathic system. For patient management in difficult cases the patient can be instructed to maximally expand the cheeks with air and hold the expansion for 30 seconds to two minutes, resulting in reduction of jaw muscle tension and symptoms.

Introduction:

The buccinator muscle has long been recognized as a significant factor in cranial and temporomandibular joint involvement, and was reported by Goodheart[1] early on in the development of TMJ related Applied Kinesiology procedures. The buccinator muscle also known as the "bugler's muscle" lies above the risorius, and is covered in part by the fat pad that lies in the cheek. Its origins are from both the maxilla and the mandible, and posteriorly, from the tendinous bundle stretching from the medial pterygoid plate to the mandible known as the pterygomandibular raphe[2]. Forming the lateral walls of the oral vestibule, it is pierced by the duct of the parotid gland on its path inside the oral cavity. Some of the fibers insert into the upper and lower lips both directly and in a criss-crossing pattern, becoming continuous with the orbicularis oris.

The buccinator allows air to be blown out of the mouth, pulls laterally on the angles of the mouth, and prevents folds in the mucous membranes of the cheeks. It is involved in laughing and crying, and when contracted produces a facial expression of satisfaction.[3] It is proposed that the buccinator acts to hold food between the teeth during mastication[4]. Goodheart has also proposed that it may have a role in mouth closing[5]. It has also been suggested that there may be a reactive muscle type of relationship between the masseter muscle and the buccinator.

Standard testing of the buccinator as reported by Walther in Applied Kinesiology, Vol. II[6], is to palpate the buccal pressure exerted when the patient attempts to contract the muscle. A

second approach is to have the patient purse the lips and apply maximum air pressure within the mouth while the examiner palpates externally for buccinator contraction and bulging.

Discussion:

Based on Goodheart's observation of the relationship between the buccinator and the masseter, I was in the habit of evaluating the buccinator whenever treating a patient with temporomandibular joint disorders, or in the presence of recurring cranial respiratory dysfunction. For many years, my evaluation would be the testing method cited by Walther with good results. Reasoning that the muscle might respond to a stretching challenge, on one occasion I tested a patient's pectoralis clavicular after having asked the patient to "puff the cheeks." To my delight and surprise, the indicator muscle went weak. Therapy localization to the buccinator belly was found to be positive, and muscle spindle challenge was also observed. After successful treatment of the muscle spindle, retesting of the indicator muscle with "puffed cheeks" remained negative. Since that initial observation many years ago, I have used this procedure on hundreds of patients as a screening tool to elicit buccinator involvement.

A frequent finding has been the finding of a hypertonic temporalis muscle with its attendant positive muscle spindle therapy localization and challenge, together with the "puffed cheek" challenge for the ipsilateral buccinator. Treatment of the buccinator muscle spindle has relieved the hypertonic temporalis and negated the positive therapy localization a high percentage of time, with no further treatment being given.

A further development in understanding this procedure came when I began asking patients to puff their cheeks on their own, away from the office any time that they began to experience jaw muscle tension or related head pain. This has been effective at relieving the tense masseter and temporalis muscles and helping patients cope in a pain free manner with the tension that they would otherwise carry in these muscles. It appears that the buccinator stretch inhibits the jaw closing muscles. For example, it can be reasoned that if the mouth is full of food or liquid to the point where the buccinator is stretched, there would be a need to relax the closing muscles to make room, just as contraction of the respiratory diaphragm inhibits the abdominal muscles to allow expansion for the displaced abdominal contents. Once the stretch is taken off of the buccinator by the increased opening dimension, the buccinator could resume its normal function in pushing the food bolus toward the teeth and tongue for normal mastication or swallowing.

Dr. Walther reported on the work of Blanton and co-workers[7] showing that the activity of the buccinator was asynchronous with

the contractions of the masseter and temporalis during normal chewing measured by electromyography. This would support the buccinators activity in opening rather than closing as suggested by Goodheart.

A side benefit of this "cheek puffing" homework has been that patients occasionally will self-correct cranial faults, presumably due to the increased intra-oral pressure exerted on the palate inducing cranial flexion, as well as the closing muscle inhibition which occurs. Further research on this aspect would be beneficial.

Procedure:

Buccinator muscle involvement may be suspected any time that there is a cranial or TMJ related dysfunction. Signs and symptoms which might be expected are sunken cheeks, tendency to bite the inner cheek with attendant scarring on the buccal oral mucosa, diminished interincissal opening, speaking with lips close together and barely moving.

Ask the patient to purse the lips and puff, or fill both cheeks as full as possible with air. When the stretch challenge is present, a strong indicator will weaken, or a weak associated muscle will strengthen, as is typical with challenge procedures. Then ask the patient to lateralize the full cheek, first to one side, then to the other, with testing of the indicator muscle. This will give indication of the side of involvement. It will rarely be bilateral. The buccinator involvement can be confirmed by therapy localization over the muscle's belly as is typically done for muscle spindle evaluation.

Treatment is done by approximating the muscle belly toward the middle with a pinching type of motion to "turn down" the muscle spindle servo-mechanism. Dr. Goodheart reported that he observed the buccinator muscle spindle to respond to transverse rather than parallel stimulation[8], however I have had good results with the parallel approach.

This procedure should be done as a screening test in the total evaluation of the stomatognathic system or as history, symptoms or other findings may dictate. Detailed examination procedures go beyond the scope of this paper, and are well documented elsewhere in the Applied Kinesiology literature, however, attention should be paid to the masseter, pterygoids, temporalis, and the hyoid musculature, as well as the cranio-sacral respiratory system.

In difficult cases, or those where there is a significant recurrence of symptoms due to jaw muscle tension, the patient can be instructed to expand the cheeks full of air as much as possible, and hold the expansion for a minimum of 30 seconds up

to a minute or two. This will result in a marked reduction of the jaw muscle tension and associated symptoms when the patient is away from the office.

Summary:

The buccinator has been recognized to have a role in TMJ dysfunction with a reactive muscle type of response theorized between it and the masseter. This author has also recognized a frequent relationship with the temporalis muscle. The buccinator stretch challenge has been found to be an effective screening tool to elicit buccinator involvement.

In the course of stomatognathic system evaluation, have the patient purse the lips and forcefully expand the cheeks with air as much as possible ("puff the cheeks"). Testing of an indicator muscle will yield a positive response if buccinator hypertonicity is present. Ask the patient to lateralize the expanded cheek, first to one side, then the other, with simultaneous testing of the indicator muscle. This will give an indication of which buccinator muscle is involved. This may be verified with therapy localization to the buccinator muscle belly. Perform a muscle spindle technic on the involved buccinator with approximation of the ends toward the middle of the muscle. Retest the indicator with maximum cheek expansion to verify correction.

In difficult or recurring cases, the patient can be asked to perform the buccinator stretch ("puffed cheek" exercise) for 30 seconds to two minutes away from the office, any time they begin to experience associated symptoms. This will generally result in relaxation of the jaw closing musculature and significant relief of the associated symptoms.

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2. Hollinshead W H. *Textbook of Anatomy*. 3rd ed. Hagerstown, Maryland: Harper & Row, Publishers; 1974:816.

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THE HIATAL HERNIA/ILEOCECAL VALVE CONNECTION: THE VAGUS NERVE

Richard M. Burger, D.C.

Abstract:

Due to its effect on the vagus nerve, a sliding hiatal hernia can have a profound effect on the entire digestive system, including but not limited to the ileocecal valve. When concurrent hiatal hernia and ileocecal valve problems are found, this author's clinical experience has shown that by thoroughly correcting the hiatal hernia, there will frequently be no need to attend to the ileocecal valve once its normal nerve supply has been re-established.

Introduction:

The sliding hiatal hernia is a condition which has been described evaluated and treated using the techniques of applied kinesiology since the early 1970's, with articles in the AK literature by a number of authors. The "etiology is usually unknown, but...may be a congenital abnormality or secondary to trauma."[1] Anatomically, the gastroesophageal junction and a portion of the stomach are above the diaphragm. Some sources suggest that there may be a congenital shortness of the esophagus (brachyesophagus), but also state that the shortening may be spurious since "operative experience shows...the elasticity and recoiling of the esophageal tube may permit the stomach to be drawn and fixed in its normal position."[2] It is also proposed that the acquired shortening may develop as a result of chronic esophagitis and reflux and subsequent shrinkage (presumably reflexive in nature).[3]

Passing through the diaphragm at the esophageal hiatus are the two branches of the vagus nerves with their many anastomoses in the anterior and posterior esophageal plexuses. The vagus nerves provide the parasympathetic afferent and efferent supply to the esophagus and the remainder of the digestive system with the exception of the distal third of the large intestine. The vagi intercommunicate with filaments from the paravertebral sympathetic trunks and their branches "so that, from the neck downward, they are really mixed parasympathetic-sympathetic nerves."[4] The sympathetic supply for the esophagus is from the fourth to sixth thoracic spinal segments primarily. The afferent impulses do not relay in the sympathetic trunks, but rather enter the cord through the posterior spinal nerve roots with other sensory nerves.

The ileocecal valve is a sphincter-type valve between the distal ileum and the colon, and its level of entry is the line of

demarcation between the cecum and the ascending colon. Its parasympathetic nerve supply comes via the vagus to the celiac ganglia, the superior mesenteric ganglia and plexus, and finally via the ileocolic nerves. The sympathetic innervation is derived from the T11 to L1 spinal levels and passes via the splanchnic nerves and the superior mesenteric plexus and ganglia.

Applied kinesiology testing and evaluation has defined two types of dysfunction with the ileocecal valve, namely inappropriate opening, or inappropriate closing. Many previous authors have written on and expanded upon the original work of Goodheart with respect to the ileocecal valve, and thorough review goes beyond the scope of this paper. The basic mechanisms of evaluation and treatment are well known to all applied kinesiologists.

Discussion:

Early in my career, I noticed that many patients would show concurrent positive applied kinesiology findings for a hiatal hernia and an ileocecal valve problem on initial examination. According to my training, I would go about correcting the problems I found and retest to verify correction. On one occasion after I had corrected the hiatal hernia on a patient, I retested for the ileocecal valve therapy localization to find that it no longer was positive, nor was there a positive challenge.

Following this observation, it became my routine to correct the hiatal hernia first on all patients who would show concurrent problems with an ileocecal valve, and in the great majority of cases, the positive ileocecal valve findings were no longer present. It should be noted that I would generally focus my attention on the visceral problems and reflexes after I had already done a thorough evaluation of the spine with appropriate adjustive procedures.

I have noted with great frequency that there has been a thoracolumbar fixation in most of the patients who show both ileocecal and hiatal hernia problems, and generally when there is a hiatal hernia, there will be problems present in the mid-cervical spine which is the source of the phrenic nerve to the diaphragm.

It is my hypothesis that as a result of the hiatal hernia, there is subsequent irritation or impingement of the vagus sympathetic-parasympathetic afferent and efferent pathways as they traverse the esophageal hiatus, resulting in altered nerve tone to the distal digestive system including the ileocecal valve. This altered afferent and efferent activity can also affect higher levels of the vagal trunk as is seen when patients with a hiatal hernia exhibit symptoms which mimic coronary

disease. While it is certainly true that each of these problems may exist in a stand-alone situation, I would like to suggest that the hiatal hernia and the ileocecal valve syndrome be viewed as part of a greater complex which involves the spine, phrenic nerves, diaphragm and vagus nerves as well.

It can become a chicken or egg type of argument with regard to which problem creates what other subsequent problem, however, there is a certain vicious cycle to its perpetuation. One scenario might be the following:

A patient who has developed poor breathing habits, using the chest instead of the diaphragm for primary respiratory activity (paradoxical respiration)5, therefore locks the diaphragm. This in turn leads to a thoracolumbar fixation due to tension in the diaphragmatic crura which insert in that region of the spine.

The diaphragm, like any other muscle which doesn't get enough exercise, becomes tight and weak which results in weakness of the esophageal hiatus. Then as a result of trauma such as a whiplash type injury where the head (with the attached esophagus) is suddenly jerked into flexion or extension, the stomach is pulled up into the diaphragm. To compound this, sudden momentary contraction of the diaphragm is the typical response to sudden injury which further pulls it downward in relation to the ascending esophagus and stomach.

Even in the absence of trauma, the esophagus may, in the course of its normal swallowing peristaltic contractions, pull the stomach upward. This then leads to pressure on the vagus nerve as it transits the hiatus and further results in potential dysfunction of any or all of the end organs and target tissues it supplies, including of course the ileocecal valve. Altered parasympathetic activity is known to change the ability of the stomach to produce hydrochloric acid, which will in turn disrupt all further digestive function down the line, affecting protein breakdown and acid-alkaline balance. This in turn can have an effect on the humoral control of digestive function via altered gastrin and enterogastrone secretion. The putrefaction products of poor protein digestion can then result in autointoxication and irritation to the small intestine, and of course the ileocecal valve.

A common finding in ileocecal valve problems is altered tone of the underlying right iliopsoas muscle, presumably due to the altered lymphatics and blood flow in the region, or perhaps kidney meridian imbalance[6]. It is also well known that there is a frequent association between a reactive right psoas and the diaphragm.[7]

We also have the potential for viscerosomatic involvement in the thoracolumbar region due to ileocecal valve dysfunction which

may perpetuate the fixation there, since its afferent and efferent nerve supply is from splanchnic nerves at those levels. Add to this the fact that a person who is breathing with the chest in favor of diaphragmatic breathing is using the accessory breathing muscles which include the scalenii, the cranial continuation of the intercostal muscles. The scalene muscles (especially the scalene anticus) have their greatest number of attachments at the third through the fifth cervical levels which are of course the source of the phrenic nerves.

Chronic breathing stress to the mid-cervical spine may then result in subluxation or fixation in that region, affecting the phrenic nerve, and leading to further diaphragm dysfunction, thus completing the cycle.

To restate, it may not be particularly important how the cycle gets started, but what is important is that it be interrupted. As seen above, there actually may be cycles within cycles, but it would seem that the common denominator is the diaphragm. It has been my experience, and is therefore my suggestion that correction of the hiatal hernia as the entry point in patients who show concurrent problems with the ileocecal valve is the most efficient approach.

Procedure:

Because of the great potential for the presence of a subclinical hiatal hernia (over 40% of the population)[8], all patients should be evaluated for this problem during routine intake examination using the standard applied kinesiology approach. When found, evaluation for thoracolumbar and mid-cervical spinal subluxations or fixations should be done. Also during examination, observation of the patient's resting respiratory pattern should be made to determine whether or not they are using the diaphragm, or the chest primarily. Observe whether or not the abdomen protrudes during inspiration as it should, or as in the case of chest breathing, does not move or actually is sucked inward. Observe the neck flexors for increased tone during inspiration. With normal at-rest diaphragmatic breathing they should remain relaxed on inspiration. Other diaphragmatic evaluation procedures such as Snyder's test, spirometry, chest excursion, etc. which have been well outlined in the AK literature should also be done as appropriate.

Evaluation and testing for ileocecal valve dysfunction should be performed routinely on all new patients as well, since this syndrome can cause such a varied symptomatic picture.

The time honored rule of "fix what you find" should be adhered to here, however I would suggest initial focus on all of the diaphragm related problems first: thoracolumbar fixation;

mid-cervical corrections; manual reduction of the hiatal hernia; diaphragm neurolymphatic stimulation; reactive psoas correction; instructing the patient in the appropriate breathing pattern; etc. as indicated.

For many years, I have successfully used a breathing exercise with patients which I refer to as "book breathing," and is a modification of the exercise described by Janet Travell[9]. The patient is instructed to perform the exercise for ten minutes each morning as follows: First thing before arising, they are to place a telephone book, or other book or object which has similar weight (large dictionary, etc.), over their navel. They are then to breath in while pushing out with their abdomen as far as possible and not moving the chest to the point of maximum ability where they feel the tension in the diaphragm, just past the point of discomfort with each breath. This is followed by expiration and relaxation of the diaphragm while feeling the book descend. I caution the patient that they may feel pulling or pressure under the clavicle, or in the neck area, and that this is simply referred from the diaphragm as it is being stretched and strengthened. It is quite common to see patients perform a "glottal stop" at the height of inspiration, and this will simply hold tension in the diaphragm, so they are also instructed to not stop the respiratory cycle between inspiration and expiration, but rather to maintain a smooth rhythmic cycle from one to the other. I have the patient perform the exercise in the office while using my hand over their umbilicus to simulate the weight of the "phone book" while at the same time I am able to palpate the tension which they are generating against the diaphragm. I then ask them to become aware of their breathing throughout the day so that they continue with diaphragmatic breathing, even though not in such an exaggerated form. The entire instruction process requires no more than one to two minutes generally. Another time-saver is the fact that frequently when there is a positive therapy localization to the diaphragm neurolymphatic reflex, before the exercise is performed, it will be absent afterwards. The diaphragm is one of the primary lymphatic pumps in the body, and restoring its normal motion can accomplish many benefits.

At the point when they are able to perform a full, deep inspiration with the diaphragm during this exercise at home, with no discomfort, then they are finished with the exercise. I have found with using this exercise, the likelihood of recidivism for the hiatal hernia is quite small, and if there is a recurrence, they are asked to resume the exercise.

After appropriate correction of the hiatal hernia, diaphragm, and the "book breathing" exercise prescription, retest the patient for the presence of the ileocecal valve. It will rarely be found. Obviously, if it is present, the appropriate procedures for correction should be performed.

In the presence of the significant digestive dysfunction that can result from the combined hiatal hernia-ileocecal valve syndrome, it is quite often necessary to supplement the patient on a short term basis with appropriate digestive aids such as HCl, pancreatic enzymes, etc. The patient should be tested for the appropriate supplementation using standard AK protocol, and again the "fix what you find" adage holds true.

Summary:

Correction of a sliding hiatal hernia using the standard manual manipulative approach, as well as correction of any diaphragm related problems including thoracolumbar subluxations or fixations, mid-cervical subluxations or fixations, reactive right psoas muscle, neurolymphatic stimulation, and instruction in proper breathing, etc. will often supersede the need to do corrective procedures for the ileocecal valve.

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APPLIED KINESIOLOGICAL MANAGEMENT OF HUMERAL HEAD ASEPTIC NECROSIS: A CASE HISTORY

Cecilia A. Duffy, D.C.

ABSTRACT: Successful management of humeral head aseptic necrosis utilizing applied kinesiology technique is presented.

INTRODUCTION: Aseptic necrosis is an interuption of the blood supply resulting in tissue death, commonly in the femoral head and less common in the humeral head. It is associated with trauma, corticosteroid use, SLE, pancreatitis, alcoholism, gout, sickle cell anemia, rheumatoid arthritis, diabetes mellitus, and others. (1) (2) It produces subcortical erosion, sclerotic changes, cystic and psuedocystic changes on plain radiographs. (2)

CASE HISTORY: A 62 year old female presented with a complaint of left shoulder and left upper arm pain with loss of motion. She described an episode approximately five months earlier in which she was placing her left arm into her coat and felt a "snap" in the left shoulder. There was progressive pain and loss of motion in the following months.

Standard examination including vitals, three position blood pressure (3) (4), salivary pH (5), lingual ascorbic acid time (6), urinalysis with Koensberg and Sulkawich (7), photomotogram (8), and hematocrit were unremarkable. Petechiometer (9) was slightly positive.

Orthopedic examination of the left shoulder revealed restricted flexion to 110/150 degrees and abduction to 70/150 degrees. Internal and external rotation were of normal motion but produced pain in the shoulder joint. Testing was negative for dislocation, bicep's tendon dysfunction, thoracic outlet syndromes, and bursitis.

Radiographic examination revealed the typical changes of aspetic necrosis of the left glenoid fossa and humeral head.

Orthopedic consult was obtained and recommendations were to continue with conservative therapy, obtain an arthritic profile to rule out rheumatoid arthritis, and if treatment failed to improve the pain then a total shoulder replacement could be considered, but restoration of full motion was not probable with shoulder replacement.

Laboratory testing included an arthritic profile, general chemistry, and complete blood count with differential. All were unremarkable, therefore, many of the associated diseases were ruled out as a cause of the aseptic necrosis. The cause of the aseptic necrosis remains unknown in this case; it can only be speculated that the "snap" sensation was indicative of a traumatic etiology.

General applied kinesiology examination and treatment was utilized. A "fix what you find" approach was taken (as per Dr. Goodheart). She was examined regularly for spinal and pelvic subluxations (10) and fixations (11), cranial faults (11), acupuncture meridian imbalances (12), and postural (13) and T.S. Line (13) muscle weaknesses.

Specific to the left shoulder problem was a recurring left pectoralis minor (14) and pectoralis major clavicular (15) weakness. Nutritional supplementation with Cataplex A and Betaine Hydrochloric Acid (Standard Process Labs) stabilized these muscles.

Standard Process Ostogen (Biost) was used short term after initial diagnosis of aseptic necrosis was made. Approximately one year after initial presentation, she complained of difficulty falling asleep for which she was given Calcium Lactate (Standard Process) (16).

Approximately one and a half years later, she developed functional hypoadrenia (3) due primarily to emotional stress for which she was given Drenamin (Standard Process) and treated appropriately via applied kinesiology.

She was treated every two to three weeks for one year and four months. During this treatment period, she was instructed to perform shoulder excercises by "walking" her fingers up a wall into shoulder flexion and abduction to gradually assist in the return of motion. Over this time period, she had gradual return to full range of motion in the left shoulder with slight pain occurring only intermittently and a complete return to normal activities that were restricted prior to treatment. At this point, I opted to place the patient on a self schedule basis, however, she preferred to make regular appointments every four to six weeks.

I have continued to see this patient for a total of three years and four months. The left shoulder remains normal in motion with very mild intermittent pain that subsides within hours. Repeat radiographs of the left shoulder at three years and four months from the initial radiographs revealed no change in the appearance of the humeral head and glenoid fossa, most importantly, there has been no further degeneration in the joint or bony surfaces.

CONCLUSION: Persistence and patience by both doctor and patient in the treatment of humeral head aseptic necrosis resulted in a very favorable outcome to this potentially surgical problem.

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THE INTERRELATIONSHIPS OF MUSCLES, MYOMERES, VERTEBRAL LEVELS AND SPECIFIC NUTRIENTS

by René Espy, D.C. and Nancy McBride, D.C.

ABSTRACT

Muscles are a storehouse of information and are supreme indicators of various interrelated functions in the body. This paper discusses the interrelationship of a muscle with its myomere, vertebral level and nutrient and offers a specific procedure to determine the positive muscle component that needs to be treated.

During the time span of 1980 through 1985 Alan G. Beardall, D.C. presented to the ICAK and others his research on many of the muscles of the body. These manuals include:

Vol. I - Low Back and Abdomen
Vol. II - Pelvis and Thigh
Vol. III - TMJ, Hyoid and Cervical Muscles
Vol. IV - Upper Extremities, Shoulder, Forearm and Hand
Vol. V - Lower Extremities, Calf and Foot

At the time of his death in 1987, much of the research remained unpublished although he and I, as his associate, used the information in our practices. Some of the nutritional data became obsolete with the introduction of new products and new methods for determining the specific nutrient needed for the specific portion of a muscle. However, the texts of the manuals were never updated. In 1988, Dr. Nancy McBride and I took on the project of researching the exact nutrient for each division of each muscle. We felt, as Kinesiologists, that it was necessary to have as much understanding as possible about each muscle and be able to have many options available to help our patients get well as efficiently as possible.

We feel that this information in no way invalidates the research of Dr. George Goodheart and others, rather it gives us information from a different level in the body and is therefore "another piece of the jigsaw puzzle."

We believe that what we are dealing with is a multi-phasic holographic bio-computer which has more than one level or option for adaptation to take place. One possible concept for a muscle continually testing weak, office visit after office visit, is that the specific segment that is holding the adaptive phase of information does not have the bio-chemistry or the reflex arc information from the myomere or vertebral level it needs to solve its problem. Obviously there are more phases of adaptation then bio-chemical, myomere level or vertebral level relationships, however due to time and space we are presenting only these three.

Body Integration is a system of measurement that has grown out of the fine work of George Goodheart, D.C., Alan Beardall, D.C., and many other outstanding practitioners who have preceded our work. New ideas come about only to refine that which precedes it, and to bring further clarity to already wonderful work. It is not our purpose to change what preceded us, but rather to develop a system of measurement that allows for greater consistency between practitioners.

DEFINITIONS

In order to be able to explain the procedure necessary for applying this research some definitions are necessary to give a working language.

Leg Lock:
The leg lock is the mechanism by which the energetics of the area being evaluated can be held by the biocomputer. This enables the Doctor to evaluate a series of related patterns to find the proper correction for specific problems. The legs are held in this position until it is no longer necessary to hold the energetic pattern on display.

Neutral Leg Lock:
The neutral leg lock consists of abducting the legs from a midline position to one that is 20 degrees lateral to the midline with two eye positions:

1 - Eyes closed - Abduct legs
2 - Eyes open - Adduct to midline and then abduct 20 degrees.

At times it may be necessary to test one leg for muscle tone. The leg lock mechanism is still valid, as long as one leg remains in the abducted position and is not moved.

BASIC EXAMINATION: FIVE SECTOR CHECK

Muscle Integrity

The Five muscle sectors should test strong in the clear.
Five Sectors:

1 - Right Shoulder Flexors
2 - Left Shoulder Flexors
3 - Neck Flexors
4 - Right Hip Abductors
5 - Left Hip Abductors

DISTORTION LOCALIZATION (DL)
THERAPY LOCALIZATION (TL)

Distortion Localization can be defined as the same abnormal bio-computer response to a stimulus that we refer to in Therapy Localization such as:

1 - Using touch to localize an area of complaint or suspect area.
2 - Body position.
3 - Verbalization of a traumatic incident.
4 - Concentration on a repressed emotion.

The TL concept denotes that some therapy is being performed, while in fact what we are doing generally is displaying that the body is in distortion. Therapy usually follows this display.

Procedure: As you DL the body, all five sectors must react by giving the same response if this would be a choice for point of entry.

At this level of entry into the body, the DL is always done with the TIPS of the four fingers, not the thumb.

When therapy is to be applied, the DL should always be done bilaterally, with the hands side by side, not overlapping.

ARM POSITIONS

Leonardo Da Vinci was a prophet in his time. He knew of body frequencies and developed a great deal of precision artwork. Alan Beardall, D.C. studied DaVinci's Canon of Man and noticed that there were very specific angles that were measurable in the two arm positions displayed. We drew a third arm position using the same angle as the other two and drew a perfect circle. We later noticed that there were specific foot and leg positions that again were at specific angles.

Clinical research has shown that the first arm position allows the body to display chemical needs,

the second displays structural needs and the third displays electromagnetic needs. With the use of the Electroacuscope we were able to measure the predominant brain frequency when the arms were placed in the three specific positions:

Yin - 26 Hz. with a carrier wave of 336
1 - Chemistry - 20 degrees arm abduction
Yang - 26 Hz. with a carrier wave of 333

Yin - 10 Hz.
2 - Structure - 90 degrees arm abduction
Yang - 8 Hz.

These positions brought about a whole new era in diagnosis and therapeutics. The body could now display ("talk") and ask for specific corrections. With the development of diagnostic equipment such as the electroacuscope, the Biorhythmic Stimulator and other sophisticated EEG instrumentation, we are now able to display what the body is expressing with specific frequency positions. Instrumentation is necessary in a research setting and when no other assistance can be provided. It is an exciting thought that the body can produce the same frequencies under normal conditions without the need of an instrument. When the proper frequencies are used, correction is accurate, specific and permanent.

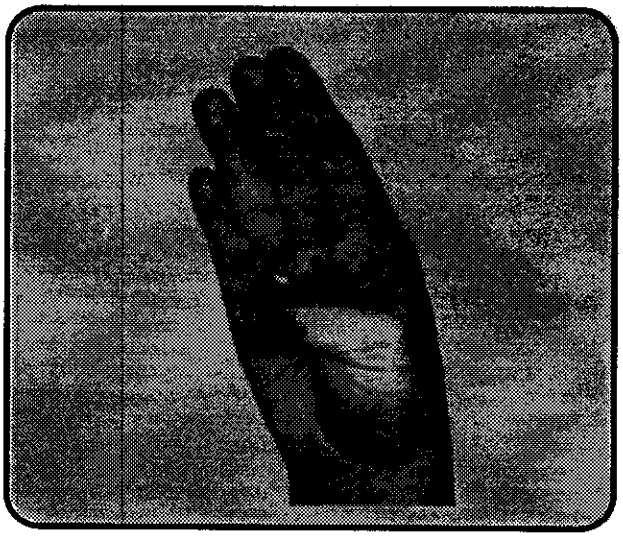
YANG CBR: Consists of the following:
The five group muscle sectors should test strong.

YIN CBR: Consists of the following:
The five group muscle sectors should test weak.

If you need to turn the patient over, use the Body Lock Mode. This locks and holds the pattern you have just diagnosed and enables you to treat the body at the level of energetic distortion.

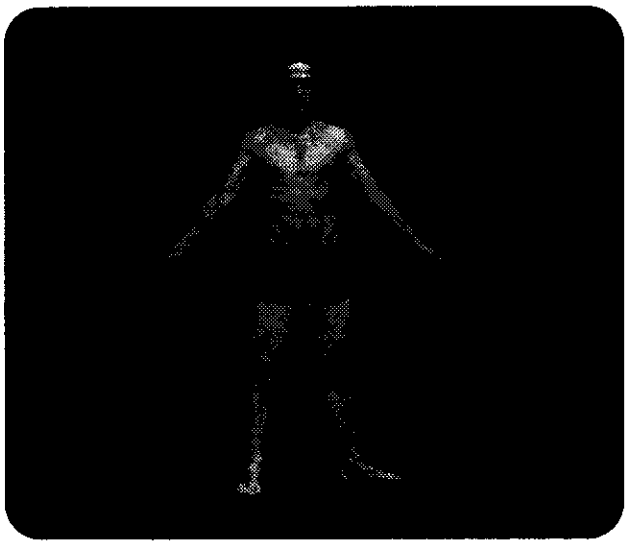
MODE:

Body Lock Mode: Tip of thumb to medial metacarpal phalangeal junction little finger. The mode is always held bilaterally.



CHEMISTRY FREQUENCY LOCK:

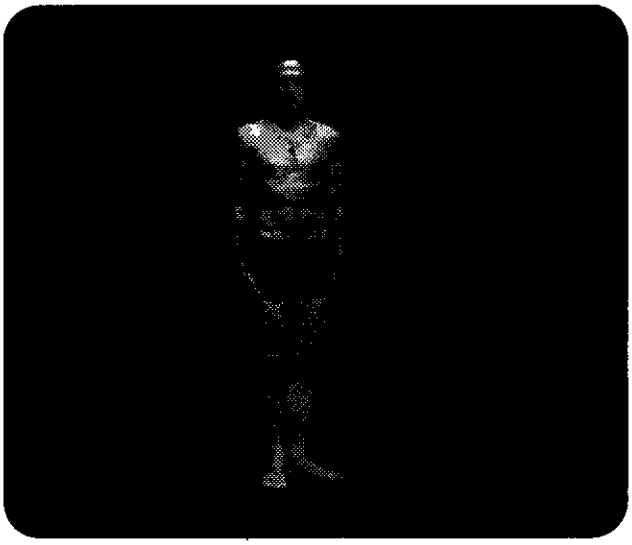
The chemistry frequency lock is as follows:



Procedure:

1 – Abduct Arms 45 .

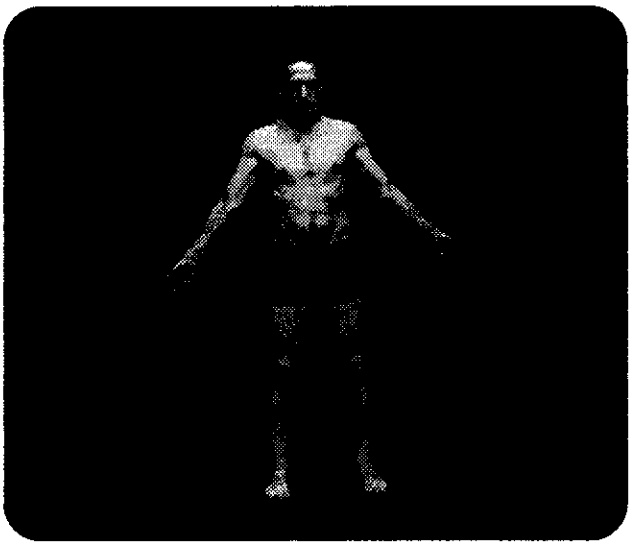
2 – Abduct Legs 20 with Left Foot externally rotated.



3 – Eyes Open:

4 – Eye Motion: Continuous Up-Down, Down-Up

5 – Adduct Legs and Arms



6 – Eyes Closed:

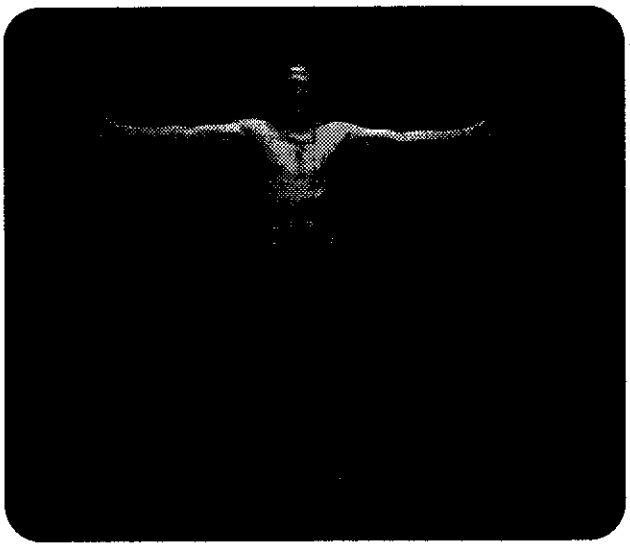
7 – Eye Motion: Continuous Up-Down, Down-Up

8 – Abduct Legs and Arms

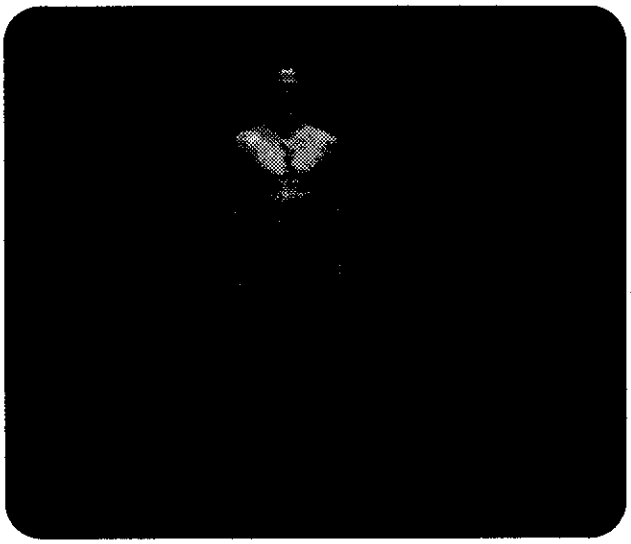
9 – Left Foot Neutral

STRUCTURE FREQUENCY LOCK:

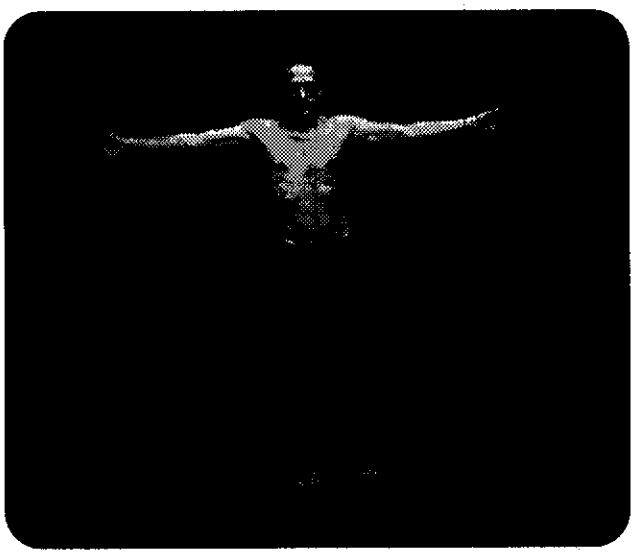
The structure frequency lock is as follows:



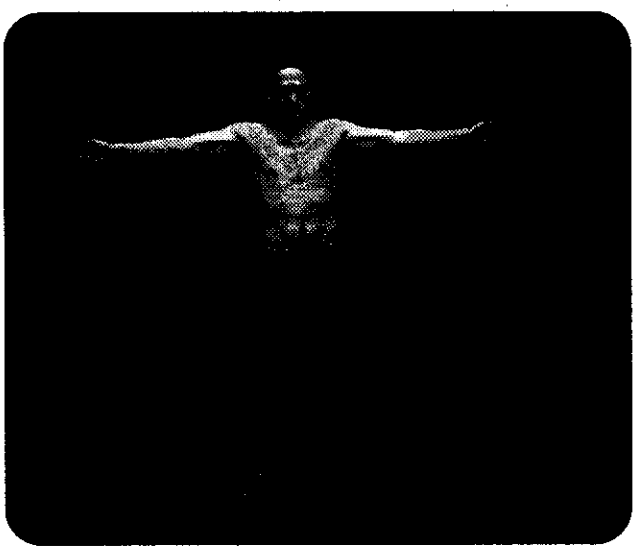
Procedure:
1 – Abduct Arms 90 .
2 – Adduct Legs, externally rotate the left foot.



3 – Eyes Closed.
4 – Eye motion: Continuous Left-Right, Right-Left.
5 – Adduct Arms. Abduct Legs.



6 – Eyes Open.
7 – Eye motion: Continuous Left-Right, Right-Left.
8 – Abduct Arms
9 – Adduct Legs



10 – Abduct Legs. Left foot neutral.

PROCEDURE

1 - Test the specific muscle to be treated. If weak, retest and perform a Neutral Lock.

2 - Retest the muscle again. It should now respond in strength. This allows the body to concentrate on the specific area being treated.

3 - Determine if the therapy will be structure or chemistry:

1 - **Chemistry:**
To determine if specific Chemistry for the muscle is necessary, put the left arm of the patient in the Chemistry Frequency Position. If a strong indicator muscle tests weak, Chemistry is necessary.

Enter the Chemistry Frequency position. The patient will display a YIN CBR. Determine the dosage by whatever means you presently use.

Refer to the following paper to determine the specific nutrient.

2 - **Structure:**
To determine if specific Structure for the muscle is necessary, put the left arm of the patient in the Structure Frequency Position. If a strong indicator muscle tests weak, Structure is necessary.

Enter the Structure Frequency position. The patient will display a Yang CBR.

DL (TL) the specific vertebral level or myomere level for the muscle you have tested. If the patient responds with a Yang CBR then adjust the segment.

Refer to the following paper to determine the specific vertebral level or myomere level.

A PRELIMINARY STUDY IN THE CORRECTION OF PATHOLOGICALLY WEAK MUSCLES WITH THE APPLICATION OF SPECIFIC MUSCLE FREQUENCY

by
Dr. René Espy and Dr. Nancy McBride

ABSTRACT

The purpose of this paper is to examine the possibilities of frequency definition as a potential correction for certain pathological deterioration. Numerous patients have been subjects of this study yet the discussion will be of two who have so called incurable and irretractable pathologies. Until the final study has been completed, these two cases will suffice to describe the process. It was the intent of the authors to determine if muscles that appear to have lost connection with any type of controlled behavior could be regenerated. To our surprise, they were not degenerated, they had merely lost recognition of the frequency that is necessary to make connection with their intent and purpose. The cases studied consist of a patient with a ten year history of Multiple Sclerosis and a patient who was considered a paraplegic due to Supranuclear Palsy.

The instrument used at this time was the Electroacuscope. We are in the process of developing a specific instrument that will digitally give a very specific frequency along with a frequency determined goniometer and a pressure gauge to test the pounds per square inch that the patient pushes and the pounds per square inch that the Doctor pulls.

The frequency used was 10 Hz. plus the carrier wave of the specific muscle found in brackets ( ) after each muscle found in the Muscle testing manuals.

INTRODUCTION

Our universe is bathed in a sea of electromagnetic energy. All that is around us and all that is , is frequency. As frequency takes form, the reality of who we are is able to be expressed.

Body Integration is the study of the holographic display of the body as it expresses as a bio-computer system. This system was not developed to replace any other system. It was developed to give a measurable approach to all systems and to incorporate them in an organized fashion. It is our hope that with this approach one will have the confidence and knowledge to begin to unravel the complex problems that patients display in this day and age.

Physicists have been doing tremendous amounts of research that show that particles can be waves at the same time they are particles because they are probability waves that represent probabilities of interconnections. They are saying that what we used to call "things" are really "events" or paths that might become events in the future.

Our universe is a dynamic interconnection of inseparable energy patterns all part of a greater whole. We are not segmented parts - we are parts of a whole.

Dr. David Bohm wrote in his book Wholeness And The Implicate Order that "parts are seen to be in immediate connection, in which their dynamical relationships depend in an irreducible way on the state of the whole system. Thus, one is led to a new notion of unbroken wholeness which denies the classical idea of analyzability of the world into separately and independently existent parts."

Dr. Karl Pribram has shown that the brain's deep structure is essentially holographic and information is distributed throughout the system, so that each fragment can produce information of the whole.

The above concepts are the basis for Body Integration. Each cell should know at all times what every other cell is doing. Breakdown in communication between cells is the beginning basis for disease.

The body displays an array of interconnecting points of which all must contribute to solve its innermost problems. Once the proper entry point is made the body will lead us to its areas of breakdown - showing its tremendous intelligence and holographic capabilities. Where we once thought we had to determine the body's needs, we began to realize that we need to listen to its innermost desires. The displays that we once thought was the problem, was merely the adaptations that filtered through its many walls and barriers.

PROCEDURE

Due the fact that the cases being presented are so unique we will explain each individually instead of following the normal scientific protocol.

Case 1

Richard was diagnosed as having Multiple Sclerosis ten years ago. He has a history of double vision, significant loss of bladder control, walks with a cane and frequently falls due to lack of muscle control.

Case 2

Cynthia was diagnosed two years ago as having Supranuclear Palsy, a rare form of palsy that exhibits as total loss of vertical eye movement, moderate dementia, and inability to use the lower limbs therefore the status of a paraplegic. The damage is said to be in the brain and exhibits symptoms likened to Parkinson's disease.

The significant areas to check according to the parameters of the study are the muscles of the abdomen. low back, thigh and calf and ankle. These muscle are the following:

MUSCLES OF THE ABDOMEN

1 - Obliquus Externus Abdominis
    1 - Obliquus Externus Abdominis, Anterior Div. (692)
    2 - Obliquus Externus Abdominis, Lateral Division (694)
2 - Transverse Abdominis
    1 - Transverse Abdominis, Upper Division (714)
    2 - Transverse Abdominis, Lower Division (718)
3 - Rectus Abdominus, Fourth Division
    1 - Rectus Abdominus Medialis, Fourth Division (706)
    2 - Rectus Abdominus Lateralis, Fourth Division (708)
4 - Obliquus Internus Abdominis
    1 - Obliquus Internus Abdominis, Lateral Division (698)
    2 - Obliquus Internus Abdominis, Anterior Division (696)
5 - Rectus Abdominis, Second Division (702)
6 - Rectus Abdominis, First Division (700)
7 - Pyramidalis (690)
8 - Rectus Abdominis, Third Division (704)

MUSCLES OF THE ILIUM & LOW BACK

1 - Psoas Major
    1 - Psoas Major, Diaphragmatic Division (726)
    2 - Psoas Major, Lumbar Division (722)
    3 - Psoas Major, Thoracic Division (724)
2 - Iliacus Minor (712)
3 - Iliacus (710)
4 - Psoas Minor (728)

MUSCLES OF THE LOW BACK

1 - Multifidus, Lumbosacral Division (734)
2 - Quadratus Lumborum
    1 - Quadratus Lumborum, Lumbar Division (732)
    2 - Quadratus Lumborum, Costal Division (730)
3 - Longissimus Lumborum (738)
4 - Iliocostalis Lumborum (736)

MUSCLES OF THE PELVIS

1 - Gluteus Medius
    1 - Gluteus Medius, Middle Division (782)
    2 - Gluteus Medius, Anterior Division (784)
    3 - Gluteus Medius, Posterior Division (780)
2 - Gemellus Inferior (862)
3 - Obturator Internus (822)
4 - Coccygeus
    1 - Coccygeus, Coccyx Division (742)
    2 - Coccygeus, Sacral Division (740)
5 - Gluteus Minimus
    1 - Gluteus Minimus, Posterior Division (788)
    2 - Gluteus Minimus, Anterior Division (786)
6 - Gemellus Superior (864)
7 - Gluteus Maximus
    1 - Gluteus Maximus, Sacral Division (850)
    2 - Gluteus Maximus, Iliac Division (848)
    3 - Gluteus Maximus, Coccygeal Division (852)
8 - Obturator Externus (812)
9 - Pubococcygeus (744)
10 - Quadratus Femoris (814)
11 - Ileococcygeus (746)
12 - Piriformis (860)
13 - Pectineus (798)

MUSCLES OF THE THIGH

1 - Tensor Fascia Lata
    1 - Tensor Fascia Lata (790)
    2 - Tensor Fascia Lata, Posterior Division (792)
2 - Rectus Femoris
    1 - Rectus Femoris, Reflected Head (794)
    2 - Rectus Femoris, Straight Head (796)
3 - Semitendinosus (854)
4 - Semimembranosus
    1 - Semimembranosus, Popliteal Division (858)
    2 - Semimembranosus, Tibial Division (856)
5 - Vastus Intermedius
    1 - Vastus Intermedius, Lateral Division (838)
    2 - Vastus Intermedius, Medial Division (836)
6 - Biceps Femoris Longhead
    1 - Biceps Femoris Longhead, Tibial Division (828)
    2 - Biceps Femoris, Longhead, Fibular Division (826)
7 - Adductor Longus
    1 - Adductor Longus, Inferior Division (804)
    2 - Adductor Longus, Superior Division (806)

8 - Vastus Medialis
    1 - Vastus Medialis, Lower Division (820)
    2 - Vastus Medialis, Middle Division (818)
    3 - Vastus Medialis, Upper Division (816)
9 - Adductor Brevis
    1 - Adductor Brevis, Left (800L)
    2 - Adductor Brevis, Right (800R)
10 - Adductor Magnus, Vertical Fibers (842)
11 - Gracilis (808)
12 - Sartorius (810)
13 - Adductor Minimus, Transverse Fibers of Adductor Magnus (846)
14 - Vastus Lateralis
    1 - Vastus Lateralis, Superior Division (830)
    2 - Vastus Lateralis, Middle Division (832)
    3 - Vastus Lateralis, Lower Division (834)
15 - Biceps Femoris Shorthead (824)
16 - Adductor Magnus, Oblique Fibers (844)
17 - Articularis Genu (840)

MUSCLES OF THE CALF AND ANKLE

1 - Soleus
    1 - Soleus, Medial Head (878)
    2 - Soleus, Lateral Head (880)
2 - Popliteus (870)
3 - Tibialis Anterior
    1 - Tibialis Anterior, Supinator Division (896)
    2 - Tibialis Anterior, Dorsiflexor Division (898)
4 - Peroneus Tertius (894)
5 - Peroneus Longus
    1 - Peroneus Longus, Cuneiform Division (886)
    2 - Peroneus Longus, Metatarsal Division (888)
6 - Tibialis Posterior
    1 - Tibialis Posterior, Tibial Division (882)
    2 - Tibialis Posterior, Fibular Division (884)
7 - Plantaris (876)
8 - Peroneus Brevis
    1 - Peroneus Brevis, Fibular Division (890)
    2 - Peroneus Brevis, Septal Division (892)
9 - Gastrocnemius
    1 - Gastrocnemius, Medial Head (872)
    2 - Gastrocnemius, Lateral Head (874)

EXAMINATION RESULTS

Upon examination, Richard was found to have the following weak muscles :
All the muscles of the abdomen, bilaterally, all the thigh muscles on the left weak, all the muscles of the low back weak bilaterally, all the muscles of the ilium weak on the left, all the muscles of the pelvis weak bilaterally, all the muscles of the calf and ankle weak on the left. The most noticeable observation about the muscle testing with Richard was that he had no awareness of his body and therefore the testing was very time consuming as we had to have him concentrate continually on the area to be tested to even begin to be able to perform some semblance of a test.

On the other hand, Cynthia had no awareness of her legs, was extremely tight, and complained of stiffness as she tried to make any type of motion with her entire body.

Testing of the above listed muscle was attempted however the entire test was not performed due to total inability to make any connection with her body from the waist down.

TREATMENT PHASE

The patients were lying supine on the table and electrodes were placed on the forehead with a electrolyte gel. The muscles chosen for the initial test were the Gluteus Medius, Middle Division and the Rectus Femoris, Straight Head. The muscle was tested and found to be weak. The instrument was set at 10 Hz plus the carrier wave for the respective muscle and the muscle was retested. This was done for each muscle bilaterally.

FINDINGS

Not only were the chosen muscles weak but there was no semblance of muscle strength in either patient as both had difficulty making any connection with the possibility of a muscle test. Upon applying the 10 Hz. and the carrier wave the respective muscles tested strong with both patients. After applying the frequency to the patient with MS, he was then able to lift his left leg with normal flexion motion from the hip instead of the pathological movement of his entire body trying to lift his leg. The movement has been maintained from the first day of the treatment and is still holding three months later. The paraplegic is gaining strength each day and is now able to use her legs with greater stability and is able to walk with a walker and with one person holding her hands. As the findings were so astounding further testing was done and each muscle responded in the same fashion.

CONCLUSION

Each muscle in the body has a specific frequency that is the connection between the brain message and the bodily function. Trauma such as injury or pathological disease can inhibit proper communication between body parts and thereby inhibit major necessary physiological responses. This testing and treatment procedure are not proclaimed to be the panacea for all spinal cord injuries or pathologies. However it does show tremendous promise in the treatment of many so called irreversible problems. It is the feeling of the authors that what is being accomplished is opening the lines of communication for disrupted circuits thereby allowing proper treatment to follow. If an injured or diseased area is unable to call for help to the areas that should be available, treatment will not be possible. As is known by the work of Dr. George Goodheart and Dr. Alan Beardall, the muscle is a storehouse of communication channels. These include a neurovascular point, a neurolymphatic point, a muscle acupuncture point, a cranial bone, a foot bone, a vertebral level, a myomere level and two organs or tissues. If a muscle cannot activate the necessary reflex area than numerous physiological areas of the body become inhibited and major physiological and biochemical processes necessary for survival become dormant. It is hoped that with further research and the new instrumentation being developed that we as Kinesiologists will be able to effectively treat a wider scope of muscle problems with an extremely high degree of accuracy and greater efficiency and be able to help more of humanity enjoy life to a higher potential.

Case 1

Richard is now able to walk across a large sized room without the use of a cane. He has regained muscle strength in two thirds of the above muscles and is now gaining confidence in the use of his body.

Case 2

Cynthia has regained full movement of her eye muscles, is able to walk with her walker and is gaining strength each day as treatment progresses.

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UPDATED AND NEWLY RESEARCHED DATA FOR THE MUSCLE TESTING MATERIAL RESEARCHED BY DR. ALAN BEARDALL

by
René Espy, D.C. and Nancy McBride, D.C.

ABSTRACT

One of the many great contributions to the health profession and to the field of Kinesiology was the untiring study of muscle function and action by Dr. Alan Beardall. He enabled us to be able to test with extreme accuracy over 512 muscles of the body and then be able to treat the positive muscle with specific points that were directly correlated to the specific muscle. Alan spent hours studying cadavers to determine the direction of fibers of each muscle division many of which were overlooked by most anatomists due to lack of knowledge that the divisions are significantly important. Alan published his muscle manuals over the span of about five years.

As time went on Alan began to research many other areas of the body and while the muscle research was not ignored, certain areas were left undeveloped and many areas were not updated with the advent of greater understanding of physiology.

In 1988 the authors began the finishing touches on a ten volume series that correlates all the components of the muscles of the body into an integrated composite of body function and physiological interchange. In our research we realized that there were many areas of incomplete muscle research and also that with new understanding of the body some of the earlier research needed to be greatly updated. The following information will update the myomere, vertebral level, and nutrient data of the earlier muscle manuals.

Realizing the amount of data that needed updating, the authors took on the job of completely updating the muscle manuals and they are now available. However for those in the ICAK with the earlier manuals, the following information is presented.
(The numbers in the parentheses refer to the computer numbers in the Muscle Manuals.)

UPDATED MYOMERE INDEX/SEGMENT

(The numbers in the parentheses refer to the computer numbers in the Muscle Manuals of Dr. Beardall.)

CRANIAL III

Levator Palpebrae, Superior Division (058)
Obliquus Inferior Bulbi (140)
Rectus Inferior Bulbi (132)
Rectus Medialis Bulbi (134)
Rectus Superior Bulbi (130)

CRANIAL IV

Obliquus Superior Bulbi (138)

CRANIAL V

Masseter, Deep Division (118)
Masseter, Superficial Division (114)
Pterygoid External Lateralis, Lower Division (126)
Pterygoid External Lateralis, Upper Division Disc (124)
Pterygoid Internal Medialis, Palatine Division (122)
Pterygoid Internal Medialis, Sphenoid Division (120)
Temporalis, Occipital Division (112)
Temporalis, Parietal Division (110)

CRANIAL VI

Rectus Lateralis Bulbi (136)

CRANIAL VII

Buccinator (102)
Platysma, Posterior Division (288)
Depressor Septi (070)
Occipitalis (052)
Orbicularis Oculi, Inferior Division (062)
Orbicularis Oculi, Superior Division (060)
Orbicularis Oris, Inferior Division (100)
Orbicularis Oris, Upper Division (098)
Platysma, Anterior Division (286)

C1 MYOMERE

Longus Capitis (314)
Stylohyoid (294)

C2 MYOMERE

Constrictor Pharyngeus Medius (380)
Cricoarytenoideus Lateralis (362)
Digastric, Anterior Belly (290)
Digastric, Posterior Belly (292)
Geniohyoid (298)
Mylohyoid (296)
Obliquus Capitis Superior (346)
Splenius Capitis, Mastoid Division (322)

Sternocleidomastoid, Sternal Division (274)
Upper Trapezius, Scapular Division (270)

C3 MYOMERE

Longus Coli, Vertical Division (308)
Omohyoid (306)
Semispinalis Capitis (332)
Sternocleidomastoid, Clavicular Division (276)
Sternohyoid (300)
Sternothyroid (302)
Thyroarytenoideus (370)
Thyrohyoid (304)
Trapezius, Middle Division (390)
Upper Trapezius, Clavicular Division (272)

C4 MYOMERE

Diaphragm, Right Lumbar Division (656)
Levator Scapula, Inferior Division (396)
Levator Scapula, Superior Division (394)
Scalenus Anterior (278)
Scalenus Medius (282)
Trapezius, Lower Division (392)

C5 MYOMERE

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

C6 MYOMERE

Biceps Brachii Shorthead (468)
Brachialis (478)
Brachioradialis, Septal Division (482)
Deltoid, Anterior, Clavicular Division (458)
Deltoid, Middle, Anterior Division (454)
Deltoid, Middle, Posterior Division (452)
Deltoid, Posterior, Lateral Division (450)
Deltoid, Posterior, Medial Division (448)
Extensor Carpi Radialis Longus, Abductor Division (518)
Extensor Carpi Ulnaris, Adductor Division (510)
Extensor Digitorum Communis Manus, Lateral Division (550)
Extensor Pollicis Longus, Septal Division (522)
Infraspinatus, Inferior Division (428)
Infraspinatus, Middle Division (426)
Infraspinatus, Superior Division (424)
Interspinalis (Cervical) (350)
Opponens Pollicis, Abductor Division (540)

Opponens Pollicis, Flexor Division (538)
Subclavius, Clavicular Division (410)
Subclavius, Scapular Division (412)
Subscapularis, Inferior Division (442)
Subscapularis, Second Division (440)
Teres Minor (430)

C7 MYOMERE

Abductor Digiti Minimi Manus, Flexor Division (582)
Abductor Pollicis Brevis (542)
Abductor Pollicis Longus, Radial Division (532)
Brachioradialis, Humeral Division (480)
Coracobrachialis, Coracoid Division (444)
Coracobrachialis, Septal Division (446)
Extensor Carpi Radialis Brevis (514)
Extensor Carpi Radialis Longus, Ext Division (516)
Extensor Carpi Ulnaris, Extensor Division (512)
Extensor Digitorum Communis Manus, Medial Division (548)
Extensor Indicis Proprius (554)
Extensor Pollicis Brevis, Septal Division (526)
Extensor Pollicis Longus, Ulnar Division (520)
Flexor Carpi Radialis, Abductor Division (502)
Flexor Pollicis Longus (528)
Latissimus Dorsi, Thoracic Division (414)
Levator Costorum, Inferior Division (642)
Pectoralis Major, Clavicular Division (460)
Pectoralis Major, Costal Division (464)
Pronator Quadratus, Proximal Division (496)
Pronator Teres, Humeral Division (484)
Pronator Teres, Ulnar Division (486)
Serratus Anterior, Inferior Division (404)
Subscapularis, Superior Division (436)
Subscapularis, Third Division (438)
Supinator, Radial Division (492)
Supinator, Ulnar Division (494)
Teres Major, Inferior Division (434)
Teres Major, Superior Division (432)

T1 L MYOMERE

Abductor Digiti Minimi Manus, Abductor Division (584)
Adductor Pollicis Transversus (544)
Anconeus, Olecranon Division (488)
Anconeus, Ulnar Division (490)
Extensor Digiti Minimi Manus (552)
Extensor Pollicis Brevis, Radial Division (524)
Flexor Carpi Radialis, Flexor Division (504)
Flexor Carpi Ulnaris, Flexor Division (506)
Flexor Digiti Minimi Brevis, Manus (580)
Flexor Digitorum Profundus Manus, Lateral Division (562)
Flexor Digitorum Superficialis, Medial Division (556)
Interossei Dorsales Manus, Second (568)
Interossei Dorsales Manus, Third (566)
Interossei Palmaris, Fourth (590)

Interossei Palmaris, Second (594)
Interossei Pollicis (Palmaris First) (536)
Latissimus Dorsi, Lumbar Division (416)
Opponens Digiti Minimi Manus, Abductor Division (586)
Palmaris Longus (500)
Pectoralis Major, Sternal Division (462)
Pectoralis Minor, Superior Division (406)
Triceps, Lateral Head (472)
Triceps, Longhead (470)
Triceps, Medial Head (474)

T1 R MYOMERE

Articularis Cubiti (476)
Flexor Carpi Ulnaris, Adductor Division (508)
Flexor Digitorum Superficialis, Lateral Division (558)
Interossei Dorsales Manus, First (570)
Latissimus Dorsi, Iliac Division (418)
Opponens Digiti Minimi Manus, Flexor Division (588)
Pectoralis Minor, Inferior Division (408)
Pronator Quadratus, Distal Division (498)
Splenius Cervicis (326)

T2 MYOMERE

Abductor Pollicis Longus, Ulnar Division (530)
Flexor Digitorum Profundus Manus, Medial Division (560)
Interossei Palmaris, Third (592)
Lumbricales Manus, First (578)
Lumbricales Manus, Second (576)
Lumbricales Manus, Third (574)
Semispinalis Cervicis (334)

T3 MYOMERE

Serratus Posterior, Superior Division (648)

T4 MYOMERE

Adductor Pollicis Obliquus (546)
Flexor Pollicis Brevis (534)
Lumbricales Manus, Fourth Division (572)
Palmaris Brevis (596)

T5 MYOMERE

Rectus Abdominis, First Division (700)
Spinalis Thoracis, Thoracic Division (612)

T6 MYOMERE

Spinalis Thoracis, Lumbar Division (610)

T7 MYOMERE

Longissimus Thoracis, Superior Division (614)
Rectus Abdominis, Fourth Div., Lateralis (708)
Rectus Abdominis, Second Division (702)

T8 MYOMERE

Interossei Dorsales Manus, Fourth (564)
Rectus Abdominis, Fourth Div., Medialis (706)

T9 MYOMERE

Obliquus Externus Abdominis, Anterior Division (692)
Obliquus Externus Abdominis, Lateral Division (694)
Rectus Abdominis, Third Division (704)
Transverse Abdominis, Lower Division (718)

T10 MYOMERE

Longissimus Thoracis, Inferior Division (618)
Obliquus Internus Abdominis, Anterior Division (696)
Serratus Posterior, Inferior Division (652)
Transverse Abdominis, Upper Division (714)

T11 MYOMERE

Obliquus Internus Abdominis, Lateral Division (698)

T12 MYOMERE

Pyramidalis (690)

L2 MYOMERE

Adductor Magnus, Transverse Division (846)
Longissimus Lumborum (738)
Psoas Major, Diaphragmatic Division (726)
Psoas Minor (728)
Quadratus Lumborum, Lumbar Division (732)
Vastus Lateralis, Lower Division (834)

L3 MYOMERE

Adductor Longus, Inferior Division (804)
Adductor Magnus, Oblique Division (844)
Articularis Genu (840)
Cremaster (752)
Iliacus Minor (712)
Iliacus (710)
Multifidus, Lumbosacral Division (734)
Pectineus (798)
Psoas Major, Thoracic Division (724)
Quadratus Lumborum, Costal Division (730)
Rectus Femoris, Reflected Head (794)
Sartorius (810)
Vastus Lateralis, Middle Division (832)
Vastus Lateralis, Superior Division (830)
Vastus Medialis, Lower Division (820)
Vastus Medialis, Middle Division (818)

L4 MYOMERE

Adductor Brevis (Left) (800)L
Adductor Brevis (Right) (800)R
Adductor Longus, Superior Division (806)

Adductor Magnus, Vertical Division (842)
Gracilis (808)
Iliocostalis Lumborum (736)
Obturator Externus (812)
Psoas Major, Lumbar Division (722)
Rectus Femoris, Straight Head (796)
Tibialis Anterior, Supinator Division (896)
Vastus Intermedius, Lateral Division (838)
Vastus Intermedius, Medial Division (836)
Vastus Medialis, Upper Division (816)

L5 MYOMERE

Extensor Digitorum Longus, Medial Division (912)
Flexor Digitorum Brevis, Lateral Division (948)
Gemellus Inferior (862)
Gemellus Superior (864)
Gluteus Medius, Anterior Division (784)
Gluteus Medius, Middle Division (782)
Gluteus Medius, Posterior Division (780)
Gluteus Minimus, Posterior Division (788)
Peroneus Brevis, Fibular Division (890)
Peroneus Longus, Cuneiform Division (886)
Peroneus Tertius (894)
Piriformis (860)
Popliteus (870)
Quadratus Femoris (814)
Semitendinosus (854)
Tensor Fascia Lata, Anterior Division (790)
Tibialis Anterior, Dorsiflexor Division (898)
Tibialis Posterior, Fibular Division (884)
Tibialis Posterior, Tibial Division (882)

S1 MYOMERE

Adductor Hallucis, Inferior Division (922)
Adductor Hallucis, Superior Division (920)
Biceps Femoris, Shorthead (824)
Extensor Digitorum Longus, Lateral Division (914)
Extensor Hallucis Longus, Fibular Division (906)
Extensor Hallucis Longus, Interosseous Division (904)
Flexor Digitorum Brevis, Medial Division (946)
Flexor Digitorum Longus, Medial Division (908)
Flexor Hallucis Longus, Fibular Division (902)
Gluteus Maximus, Coccygeal Division (852)
Gluteus Minimus, Anterior Division (786)
Peroneus Brevis, Septal Division (892)
Peroneus Longus, Metatarsal Division (888)
Plantaris (876)
Semimembranosus, Popliteal Division (858)
Semimembranosus, Tibial Division (856)
Tensor Fascia Lata, Posterior Division (792)

S2 MYOMERE

Abductor Digiti Minimi Pedis (974)
Abductor Digitus Pedis, Fifth (972)
Abductor Digitus Pedis, Fourth (970)
Abductor Hallucis Transverse Head, Medial Division (936)
Adductor Digitus Pedis, Second (958)
Biceps Femoris, Longhead, Fibular Division (826)
Biceps Femoris, Longhead, Tibial Division (828)
Coccygeus, Coccyx Division (742)
Extensor Digitorum Brevis (976)
Extensor Hallucis Brevis (940)
Flexor Digitorum Longus, Lateral Division (910)
Flexor Digitus Pedis, Second (950)
Flexor Digitus Pedis, Third (952)
Flexor Hallucis Brevis, Cuboid Division (930)
Flexor Hallucis Brevis, First Cuneiform Division (924)
Flexor Hallucis Brevis, Tendonal Division (926)
Flexor Hallucis Brevis, Third Cuneiform Division (928)
Flexor Hallucis Longus, Tibial Division (900)
Gastrocnemius, Lateral Division (874)
Gastrocnemius, Medial Division (872)
Gluteus Maximus, Iliac Division (848)
Iliococcygeus (746)
Obturator Internus (822)
Pubococcygeus (744)
Quadratus Plantae, Lateral Division (944)
Soleus Medial Division (878)
Soleus, Lateral Division (880)

S3 MYOMERE

Abductor Digitus Pedis, Second (966)
Abductor Digitus Pedis, Third (968)
Abductor Hallucis Oblique Head, Metatarsal Division (934)
Abductor Hallucis Oblique Head, Peroneus Division (932)
Abductor Hallucis Transverse Head, Lateral Division (938)
Adductor Digitus Pedis, Fifth (964)
Adductor Digitus Pedis, Fourth (962)
Adductor Digitus Pedis, Third (960)
Coccygeus, Sacral Division (740)
Flexor Digitus Pedis, Fifth (956)
Flexor Digitus Pedis, Fourth (954)
Gluteus Maximus, Sacral Division (850)
Quadratus Plantae, Medial Division (942)

UPDATED MYOMERE INDEX/NUMERICAL

(052) MM Cranial VII	Occipitalis
(058) MM Cranial III	Levator Palpebrae, Superior Division
(060) MM Cranial VII	Orbicularis Oculi, Superior Division
(062) MM Cranial VII	Orbicularis Oculi, Inferior Division
(070) MM Cranial VII	Depressor Septi
(098) MM Cranial VII	Orbicularis Oris, Upper Division
(100) MM Cranial VII	Orbicularis Oris, Inferior Division
(102) MM Cranial VII	Buccinator
(110) MM Cranial V	Temporalis, Parietal Division
(112) MM Cranial V	Temporalis, Occipital Division
(114) MM Cranial V	Masseter, Superficial Division
(118) MM Cranial V	Masseter, Deep Division
(120) MM Cranial V	Pterygoid Internal Medialis, Sphenoid Division
(122) MM Cranial V	Pterygoid Internal Medialis, Palatine Division
(124) MM Cranial V	Pterygoid External Lateralis, Upper Div.-Disc
(126) MM Cranial V	Pterygoid External Lateralis, Lower Division
(130) MM Cranial III	Rectus Superior Bulbi
(132) MM Cranial III	Rectus Inferior Bulbi
(134) MM Cranial III	Rectus Medialis Bulbi
(136) MM Cranial VI	Rectus Lateralis Bulbi
(138) MM Cranial IV	Obliquus Superior Bulbi
(140) MM Cranial III	Obliquus Inferior Bulbi
(270) MM C2	Upper Trapezius, Scapular Division
(272) MM C3	Upper Trapezius, Clavicular Division
(274) MM C2	Sternocleidomastoid, Sternal Division
(276) MM C3	Sternocleidomastoid, Clavicular Division
(278) MM C4	Scalenus Anterior
(282) MM C4	Scalenus Medius
(284) MM C5	Scalenus Posterior
(286) MM Cranial VII	Platysma, Anterior Division
(288) MM Cranial VII	Platysma, Posterior Division
(290) MM C2	Digastric, Anterior Belly
(292) MM C2	Digastric, Posterior Belly
(294) MM C1	Stylohyoid
(296) MM C2	Mylohyoid
(298) MM C2	Geniohyoid
(300) MM C3	Sternohyoid
(302) MM C3	Sternothyroid
(304) MM C3	Thyrohyoid
(306) MM C3	Omohyoid
(308) MM C3	Longus Coli, Vertical Division
(314) MM C1	Longus Capitis
(322) MM C2	Splenius Capitis, Mastoid Division
(326) MM T1 R	Splenius Cervicis
(332) MM C3	Semispinalis Capitis
(334) MM T2	Semispinalis Cervicis
(346) MM C2	Obliquus Capitis Superior
(350) MM C6	Interspinalis (Cervical)
(362) MM C2	Cricoarytenoideus Lateralis
(370) MM C3	Thyroarytenoideus
(380) MM C2	Constrictor Pharyngeus Medius

(390) MM C3	Trapezius, Middle Division
(392) MM C4	Trapezius, Lower Division
(394) MM C4	Levator Scapula, Superior Division
(396) MM C4	Levator Scapula, Inferior Division
(398) MM C5	Rhomboid Minor
(400) MM C5	Rhomboid Major
(402) MM C5	Serratus Anterior, Superior Division
(404) MM C7	Serratus Anterior, Inferior Division
(406) MM T1 L	Pectoralis Minor, Superior Division
(408) MM T1 R	Pectoralis Minor, Inferior Division
(410) MM C6	Subclavius, Clavicular Division
(412) MM C6	Subclavius, Scapular Division
(414) MM C7	Latissimus Dorsi, Thoracic Division
(416) MM T1 L	Latissimus Dorsi, Lumbar Division
(418) MM T1 R	Latissimus Dorsi, Iliac Division
(420) MM C5	Supraspinatus, Spine Division
(422) MM C5	Supraspinatus, Fossa Division
(424) MM C6	Infraspinatus, Superior Division
(426) MM C6	Infraspinatus, Middle Division
(428) MM C6	Infraspinatus, Inferior Division
(430) MM C6	Teres Minor
(432) MM C7	Teres Major, Superior Division
(434) MM C7	Teres Major, Inferior Division
(436) MM C7	Subscapularis, Superior Division
(438) MM C7	Subscapularis, Third Division
(440) MM C6	Subscapularis, Second Division
(442) MM C6	Subscapularis, Inferior Division
(444) MM C7	Coracobrachialis, Coracoid Division
(446) MM C7	Coracobrachialis, Septal Division
(448) MM C6	Deltoid, Posterior, Medial Division
(450) MM C6	Deltoid, Posterior, Lateral Division
(452) MM C6	Deltoid, Middle, Posterior Division
(454) MM C6	Deltoid, Middle, Anterior Division
(456) MM C5	Deltoid, Anterior, Scapular Division
(458) MM C6	Deltoid, Anterior, Clavicular Division
(460) MM C7	Pectoralis Major, Clavicular Division
(462) MM T1 L	Pectoralis Major, Sternal Division
(464) MM C7	Pectoralis Major, Costal Division
(466) MM C5	Biceps Brachii Longhead
(468) MM C6	Biceps Brachii Shorthead
(470) MM T1 L	Triceps, Longhead
(472) MM T1 L	Triceps, Lateral Head
(474) MM T1 L	Triceps, Medial Head
(476) MM T1 R	Articularis Cubiti
(478) MM C6	Brachialis
(480) MM C7	Brachioradialis, Humeral Division
(482) MM C6	Brachioradialis, Septal Division
(484) MM C7	Pronator Teres, Humeral Division
(486) MM C7	Pronator Teres, Ulnar Division
(488) MM T1 L	Anconeus, Olecranon Division
(490) MM T1 L	Anconeus, Ulnar Division
(492) MM C7	Supinator, Radial Division
(494) MM C7	Supinator, Ulnar Division
(496) MM C7	Pronator Quadratus, Proximal Division

(498) MM T1 R	Pronator Quadratus, Distal Division
(500) MM T1 L	Palmaris Longus
(502) MM C7	Flexor Carpi Radialis, Abductor Division
(504) MM T1 L	Flexor Carpi Radialis, Flexor Division
(506) MM T1 L	Flexor Carpi Ulnaris, Flexor Division
(508) MM T1 R	Flexor Carpi Ulnaris, Adductor Division
(510) MM C6	Extensor Carpi Ulnaris, Adductor Division
(512) MM C7	Extensor Carpi Ulnaris, Extensor Division
(514) MM C7	Extensor Carpi Radialis Brevis
(516) MM C7	Extensor Carpi Radialis Longus, Ext Division
(518) MM C6	Extensor Carpi Radialis Longus, Abductor Division
(520) MM C7	Extensor Pollicis Longus, Ulnar Division
(522) MM C6	Extensor Pollicis Longus, Septal Division
(524) MM T1 L	Extensor Pollicis Brevis, Radial Division
(526) MM C7	Extensor Pollicis Brevis, Septal Division
(528) MM C7	Flexor Pollicis Longus
(530) MM T2	Abductor Pollicis Longus, Ulnar Division
(532) MM C7	Abductor Pollicis Longus, Radial Division
(534) MM T4	Flexor Pollicis Brevis
(536) MM T1 L	Interossei Pollicis (Palmaris First)
(538) MM C6	Opponens Pollicis, Flexor Division
(540) MM C6	Opponens Pollicis, Abductor Division
(542) MM C7	Abductor Pollicis Brevis
(544) MM T1 L	Adductor Pollicis Transversus
(546) MM T4	Adductor Pollicis Obliquus
(548) MM C7	Extensor Digitorum Communis Manus, Medial Division
(550) MM C6	Extensor Digitorum Communis Manus, Lateral Division
(552) MM T1 L	Extensor Digiti Minimi Manus
(554) MM C7	Extensor Indicis Proprius
(556) MM T1 L	Flexor Digitorum Superficialis, Medial Division
(558) MM T1 R	Flexor Digitorum Superficialis, Lateral Division
(560) MM T2	Flexor Digitorum Profundus Manus, Medial Division
(562) MM T1 L	Flexor Digitorum Profundus Manus, Lateral Division
(564) MM T8	Interossei Dorsales Manus, Fourth
(566) MM T1 L	Interossei Dorsales Manus, Third
(568) MM T1 L	Interossei Dorsales Manus, Second
(570) MM T1 R	Interossei Dorsales Manus, First
(572) MM T4	Lumbricales Manus, Fourth Division
(574) MM T2	Lumbricales Manus, Third
(576) MM T2	Lumbricales Manus, Second
(578) MM T2	Lumbricales Manus, First
(580) MM T1 L	Flexor Digiti Minimi Brevis, Manus
(582) MM C7	Abductor Digiti Minimi Manus, Flexor Division
(584) MM T1 L	Abductor Digiti Minimi Manus, Abductor Division
(586) MM T1 L	Opponens Digiti Minimi Manus, Abductor Division
(588) MM T1 R	Opponens Digiti Minimi Manus, Flexor Division
(590) MM T1 L	Interossei Palmaris, Fourth
(592) MM T2	Interossei Palmaris, Third
(594) MM T1 L	Interossei Palmaris, Second
(596) MM T4	Palmaris Brevis
(610) MM T6	Spinalis Thoracis, Lumbar Division
(612) MM T5	Spinalis Thoracis, Thoracic Division
(614) MM T7	Longissimus Thoracis, Superior Division
(618) MM T10	Longissimus Thoracis, Inferior Division

(642) MM C7	Levator Costorum, Inferior Division
(648) MM T3	Serratus Posterior, Superior Division
(652) MM T10	Serratus Posterior, Inferior Division
(656) MM C4	Diaphragm, Right Lumbar Division
(662) MM C5	Diaphragm, Left Lumbar Division
(690) MM T12	Pyramidalis
(692) MM T9	Obliquus Externus Abdominis, Anterior Division
(694) MM T9	Obliquus Externus Abdominis, Lateral Division
(696) MM T10	Obliquus Internus Abdominis, Anterior Division
(698) MM T11	Obliquus Internus Abdominis, Lateral Division
(700) MM T5	Rectus Abdominis, First Division
(702) MM T7	Rectus Abdominis, Second Division
(704) MM T9	Rectus Abdominis, Third Division
(706) MM T8	Rectus Abdominis, Fourth Div., Medialis
(708) MM T7	Rectus Abdominis, Fourth Div., Lateralis
(710) MM L3	Iliacus
(712) MM L3	Iliacus Minor
(714) MM T10	Transverse Abdominis, Upper Division
(718) MM T9	Transverse Abdominis, Lower Division
(722) MM L4	Psoas Major, Lumbar Division
(724) MM L3	Psoas Major, Thoracic Division
(726) MM L2	Psoas Major, Diaphragmatic Division
(728) MM L2	Psoas Minor
(730) MM L3	Quadratus Lumborum, Costal Division
(732) MM L2	Quadratus Lumborum, Lumbar Division
(734) MM L3	Multifidus, Lumbosacral Division
(736) MM L4	Iliocostalis Lumborum
(738) MM L2	Longissimus Lumborum
(740) MM S3	Coccygeus, Sacral Division
(742) MM S2	Coccygeus, Coccyx Division
(744) MM S2	Pubococcygeus
(746) MM S2	Iliococcygeus
(752) MM L3	Cremaster
(780) MM L5	Gluteus Medius, Posterior Division
(782) MM L5	Gluteus Medius, Middle Division
(784) MM L5	Gluteus Medius, Anterior Division
(786) MM S1	Gluteus Minimus, Anterior Division
(788) MM L5	Gluteus Minimus, Posterior Division
(790) MM L5	Tensor Fascia Lata, Anterior Division
(792) MM S1	Tensor Fascia Lata, Posterior Division
(794) MM L3	Rectus Femoris, Reflected Head
(796) MM L4	Rectus Femoris, Straight Head
(798) MM L3	Pectineus
(800)L MM L4	Adductor Brevis (Left)
(800)R MM L4	Adductor Brevis (Right)
(804) MM L3	Adductor Longus, Inferior Division
(806) MM L4	Adductor Longus, Superior Division
(808) MM L4	Gracilis
(810) MM L3	Sartorius
(812) MM L4	Obturator Externus
(814) MM L5	Quadratus Femoris
(816) MM L4	Vastus Medialis, Upper Division
(818) MM L3	Vastus Medialis, Middle Division
(820) MM L3	Vastus Medialis, Lower Division

(822) MM S2	Obturator Internus
(824) MM S1	Biceps Femoris, Shorthead
(826) MM S2	Biceps Femoris, Longhead, Fibular Division
(828) MM S2	Biceps Femoris, Longhead, Tibial Division
(830) MM L3	Vastus Lateralis, Superior Division
(832) MM L3	Vastus Lateralis, Middle Division
(834) MM L2	Vastus Lateralis, Lower Division
(836) MM L4	Vastus Intermedius, Medial Division
(838) MM L4	Vastus Intermedius, Lateral Division
(840) MM L3	Articularis Genu
(842) MM L4	Adductor Magnus, Vertical Division
(844) MM L3	Adductor Magnus, Oblique Division
(846) MM L2	Adductor Magnus, Transverse Division
(848) MM S2	Gluteus Maximus, Iliac Division
(850) MM S3	Gluteus Maximus, Sacral Division
(852) MM S1	Gluteus Maximus, Coccygeal Division
(854) MM L5	Semitendinosus
(856) MM S1	Semimembranosus, Tibial Division
(858) MM S1	Semimembranosus, Popliteal Division
(860) MM L5	Piriformis
(862) MM L5	Gemellus Inferior
(864) MM L5	Gemellus Superior
(870) MM L5	Popliteus
(872) MM S2	Gastrocnemius, Medial Division
(874) MM S2	Gastrocnemius, Lateral Division
(876) MM S1	Plantaris
(878) MM S2	Soleus Medial Division
(880) MM S2	Soleus, Lateral Division
(882) MM L5	Tibialis Posterior, Tibial Division
(884) MM L5	Tibialis Posterior, Fibular Division
(886) MM L5	Peroneus Longus, Cuneiform Division
(888) MM S1	Peroneus Longus, Metatarsal Division
(890) MM L5	Peroneus Brevis, Fibular Division
(892) MM S1	Peroneus Brevis, Septal Division
(894) MM L5	Peroneus Tertius
(896) MM L4	Tibialis Anterior, Supinator Division
(898) MM L5	Tibialis Anterior, Dorsiflexor Division
(900) MM S2	Flexor Hallucis Longus, Tibial Division
(902) MM S1	Flexor Hallucis Longus, Fibular Division
(904) MM S1	Extensor Hallucis Longus, Interosseous Division
(906) MM S1	Extensor Hallucis Longus, Fibular Division
(908) MM S1	Flexor Digitorum Longus, Medial Division
(910) MM S2	Flexor Digitorum Longus, Lateral Division
(912) MM L5	Extensor Digitorum Longus, Medial Division
(914) MM S1	Extensor Digitorum Longus, Lateral Division
(920) MM S1	Adductor Hallucis, Superior Division
(922) MM S1	Adductor Hallucis, Inferior Division
(924) MM S2	Flexor Hallucis Brevis, First Cuneiform Division
(926) MM S2	Flexor Hallucis Brevis, Tendonal Division
(928) MM S2	Flexor Hallucis Brevis, Third Cuneiform Division
(930) MM S2	Flexor Hallucis Brevis, Cuboid Division
(932) MM S3	Abductor Hallucis Oblique Head, Peroneus Division
(934) MM S3	Abductor Hallucis Oblique Head, Metatarsal Division
(936) MM S2	Abductor Hallucis Transverse Head, Medial Division

(938) MM S3	Abductor Hallucis Transverse Head, Lateral Division
(940) MM S2	Extensor Hallucis Brevis
(942) MM S3	Quadratus Plantae, Medial Division
(944) MM S2	Quadratus Plantae, Lateral Division
(946) MM S1	Flexor Digitorum Brevis, Medial Division
(948) MM L5	Flexor Digitorum Brevis, Lateral Division
(950) MM S2	Flexor Digitus Pedis, Second
(952) MM S2	Flexor Digitus Pedis, Third
(954) MM S3	Flexor Digitus Pedis, Fourth
(956) MM S3	Flexor Digitus Pedis, Fifth
(958) MM S2	Adductor Digitus Pedis, Second
(960) MM S3	Adductor Digitus Pedis, Third
(962) MM S3	Adductor Digitus Pedis, Fourth
(964) MM S3	Adductor Digitus Pedis, Fifth
(966) MM S3	Abductor Digitus Pedis, Second
(968) MM S3	Abductor Digitus Pedis, Third
(970) MM S2	Abductor Digitus Pedis, Fourth
(972) MM S2	Abductor Digitus Pedis, Fifth
(974) MM S2	Abductor Digiti Minimi Pedis
(976) MM S2	Extensor Digitorum Brevis

UPDATED NUTRIENT/MUSCLE LIST

(The numbers in the parentheses refer to the computer numbers in the Muscle Manuals)

BLACK CURRANT SEED OIL
Flexor Digitorum Profundus Manus, Lateral Division (562)
Orbicularis Oris, Inferior Division (100)

CORE ADRENAL
Masseter, Superficial Division (114)
Psoas Major, Diaphragmatic Division (726)
Sartorius (810)
Subclavius, Scapular Division (412)

CORE BILE
Geniohyoid (298)
Gluteus Medius, Posterior Division (780)
Opponens Digiti Minimi Manus, Abductor Division (586)

CORE BONE MATRIX
Opponens Digiti Minimi Manus, Flexor Division (588)
Digastric, Posterior Belly (292)
Interossei Palmaris, Third (592)

CORE BRAIN/SPINAL
Extensor Digiti Minimi Manus (552)

CORE CALCIUM
Rectus Femoris, Reflected Head (794)
Stylohyoid (294)
Tibialis Anterior, Supinator Division (896)
Upper Trapezius, Clavicular Division (272)

CORE CARBO GEST
Abductor Hallucis Oblique Head, Metatarsal Div.(934)
Abductor Hallucis Transverse Head, Lateral Division (938)
Adductor Longus, Superior Division (806)
Articularis Genu (840)
Deltoid, Anterior, Scapular Division (456)
Extensor Pollicis Longus, Septal Division (522)
Flexor Digitorum Brevis, Lateral Division (948)
Interossei Dorsales Manus, Second (568)
Palmaris Longus (500)
Pectoralis Major, Sternal Division (462)
Platysma, Anterior Division (286)
Rectus Lateralis Bulbi (136)
Scalenus Medius (282)

CORE D-TOX
Extensor Hallucis Brevis (940)
Longus Capitis (314)
Rectus Abdominis, First Division (700)
Tensor Fascia Lata, Posterior Division (792)

CORE DENT MATRIX

Flexor Digiti Minimi Brevis, Manus (580)
Obliquus Superior Bulbi (138)
Opponens Pollicis, Abductor Division (540)

CORE FOLIC ACID

Biceps Brachii Longhead (466)
Flexor Hallucis Brevis, First Cuneiform Division (924)
Interossei Pollicis (Palmaris First) (536)
Longus Coli, Vertical Division (308)
Mylohyoid (296)
Obliquus Internus Abdominis, Anterior Division (696)
Obliquus Internus Abdominis, Lateral Division (698)
Omohyoid (306)
Pectoralis Major, Clavicular Division (460)
Pronator Teres, Humeral Division (484)
Rectus Abdominis, Fourth Div., Medialis (706)
Splenius Cervicis (326)
Thyroarytenoideus (370)

CORE HEALTH RESERVE

Adductor Brevis (Left) (800L)
Adductor Brevis (Right) (800R)
Biceps Brachii Shorthead (468)
Biceps Femoris, Longhead, Fibular Division (826)
Extensor Digitorum Longus, Medial Division (912)
Longissimus Thoracis, Superior Division (614)
Peroneus Brevis, Septal Division (892)
Pterygoid Internal Medialis, Sphenoid Division (120)
Teres Major, Superior Division (432)
Triceps, Longhead (470)

CORE HEART

Abductor Digiti Minimi Manus, Flexor Division (582)
Interossei Dorsales Manus, Fourth (564)
Pyramidalis (690)

CORE ILEODUODENAL

Abductor Digiti Minimi Manus, Abductor Division (584)
Deltoid, Posterior, Lateral Division (450)
Extensor Carpi Ulnaris, Adductor Division (510)
Gastrocnemius, Medial Division (872)
Peroneus Longus, Cuneiform Division (886)
Semimembranosus, Popliteal Division (858)

CORE INOSITOL

Coracobrachialis, Coracoid Division (444)
Depressor Septi (070)
Extensor Pollicis Brevis, Radial Division (524)
Opponens Pollicis, Flexor Division (538)
Popliteus (870)

CORE IRON

Abductor Digitus Pedis, Second (966)
Adductor Magnus, Transverse Division (846)
Buccinator (102)
Cremaster (752)
Extensor Carpi Radialis Longus, Abductor Division (518)
Flexor Hallucis Longus, Tibial Division (900)
Gluteus Medius, Middle Division (782)
Iliocostalis Lumborum (736)
Longissimus Lumborum (738)
Peroneus Brevis, Fibular Division (890)
Serratus Posterior, Superior Division (648)

CORE KIDNEY

Flexor Digitus Pedis, Third (952)
Pronator Quadratus, Distal Division (498)
Psoas Major, Lumbar Division (722)
Psoas Major, Thoracic Division (724)
Rectus Abdominis, Fourth Div., Lateralis (708)
Scalenus Anterior (278)

CORE LEVEL OXIDATE

Flexor Hallucis Brevis, Third Cuneiform Division (928)
Semitendinosus (854)
Transverse Abdominis, Upper Division (714)

CORE LIVER

Deltoid, Anterior, Clavicular Division (458)
Lumbricales Manus, First (578)
Rhomboid Major (400)
Rhomboid Minor (398)
Soleus, Lateral Division (880)

CORE LUNG

Deltoid, Middle, Posterior Division (452)
Obturator Internus (822)
Pectoralis Minor, Superior Division (406)

CORE MAGNESIUM

Adductor Magnus, Vertical Division (842)
Extensor Digitorum Longus, Lateral Division (914)
Extensor Hallucis Longus, Fibular Division (906)
Flexor Digitorum Profundus Manus, Medial Division (560)
Gemellus Inferior (862)
Multifidus, Lumbosacral Division (734)
Obliquus Inferior Bulbi (140)
Pectineus (798)
Plantaris (876)
Pterygoid Internal Medialis, Palatine Division (122)
Semimembranosus, Tibial Division (856)
Serratus Posterior, Inferior Division (652)
Spinalis Thoracis, Thoracic Division (612)
Vastus Medialis, Lower Division (820)

CORE MANGANESE

Abductor Digitus Pedis, Fourth (970)
Anconeus, Olecranon Division (488)
Flexor Pollicis Brevis (534)
Gluteus Minimus, Anterior Division (786)
Iliacus (710)
Orbicularis Oris, Upper Division (098)
Peroneus Longus, Metatarsal Division (888)
Peroneus Tertius (894)
Sternothyroid (302)
Thyrohyoid (304)
Trapezius, Lower Division (392)

CORE METHIONINE

Iliacus Minor (712)
Levator Scapula, Superior Division (394)
Vastus Lateralis, Middle Division (832)

CORE NIACIN

Adductor Pollicis Obliquus (546)
Articularis Cubiti (476)
Biceps Femoris, Longhead, Tibial Division (828)
Extensor Carpi Radialis Longus, Ext Division (516)
Quadratus Plantae, Lateral Division (944)
Rectus Femoris, Straight Head (796)
Sternocleidomastoid, Clavicular Division (276)
Temporalis, Parietal Division (110)

CORE OVARY/ORCHIC

Pterygoid External Lateralis, Upper Div.-Disc (124)

CORE PANCREAS

Gluteus Maximus, Iliac Division (848)
Lumbricales Manus, Fourth Div.(572)
Subscapularis, Superior Division (436)
Vastus Medialis, Middle Division (818)

CORE PANTOTHENIC ACID

Abductor Digitus Pedis, Third (968)
Adductor Magnus, Oblique Division (844)
Flexor Carpi Radialis, Abductor Division (502)
Pectoralis Major, Costal Division (464)
Platysma, Posterior Division (288)
Sternohyoid (300)

CORE PARATHYROID

Lumbricales Manus, Third (574)
Quadratus Femoris (814)
Vastus Lateralis, Lower Division (834)

CORE PEPSIN

Pubococcygeus (744)

CORE PITUITARY

Flexor Digitorum Superficialis, Lateral Division (558)
Masseter, Deep Division (118)
Pronator Teres, Ulnar Division (486)
Trapezius, Middle Division (390)

CORE POTASSIUM

Adductor Digitus Pedis, Fourth (962)
Adductor Hallucis, Inferior Division (922)
Adductor Hallucis, Superior Division (920)
Cricoarytenoideus Lateralis (362)
Flexor Hallucis Brevis, Cuboid Division (930)
Gluteus Maximus, Coccygeal Division (852)
Latissimus Dorsi, Iliac Division (418)
Levator Palpebrae, Superior Division (058)
Quadratus Lumborum, Costal Division (730)
Semispinalis Capitis (332)
Splenius Capitis, Mastoid Division (322)
Subscapularis, Second Division (440)
Tibialis Posterior, Fibular Division (884)
Triceps, Medial Head (474)

CORE RNA

Adductor Pollicis Transversus (544)
Subclavius, Clavicular Division (410)

CORE RUTIN

Abductor Hallucis Transverse Head, Medial Division (936)
Abductor Pollicis Longus, Radial Division (532)
Biceps Femoris, Shorthead (824)
Extensor Pollicis Brevis, Septal Division (526)
Flexor Digitorum Longus, Lateral Division (910)
Interossei Palmaris, Fourth (590)
Obliquus Capitis Superior (346)
Rectus Inferior Bulbi (132)
Soleus Medial Division (878)

CORE SELENIUM

Adductor Longus, Inferior Division (804)
Flexor Digitorum Brevis, Medial Division (946)
Flexor Hallucis Longus, Fibular Division (902)
Obliquus Externus Abdominis, Anterior Division (692)
Spinalis Thoracis, Lumbar Division (610)
Sternocleidomastoid, Sternal Division (274)
Upper Trapezius, Scapular Division (270)

CORE THIAMINE

Gemellus Superior (864)
Interossei Dorsales Manus, First (570)
Interspinalis (Cervical) (350)

CORE THYMUS

Coccygeus, Coccyx Division (742)
Extensor Digitorum Brevis (976)

CORE THYRO

Abductor Hallucis Oblique Head, Peroneus Division (932)
Adductor Digitus Pedis, Third (960)
Brachialis (478)
Constrictor Pharyngeus Medius (380)
Extensor Carpi Radialis Brevis (514)
Flexor Carpi Radialis, Flexor Division (504)
Flexor Hallucis Brevis, Tendonal Division (926)
Gastrocnemius, Lateral Division (874)
Occipitalis (052)
Psoas Minor (728)
Rectus Abdominis, Second Division (702)
Semispinalis Cervicis (334)
Serratus Anterior, Superior Division (402)
Teres Minor (430)
Vastus Lateralis, Superior Division (830)

CORE UTERUS/PROSTATE

Gracilis (808)

CORE VITAMIN A

Abductor Digiti Minimi Pedis (974)
Adductor Digitus Pedis, Fifth (964)
Flexor Digitorum Superficialis, Medial Division (556)
Rectus Medialis Bulbi (134)
Vastus Medialis, Upper Division (816)

CORE VITAMIN B 6

Abductor Pollicis Longus, Ulnar Division (530)
Extensor Digitorum Communis Manus, Medial Division (548)
Flexor Carpi Ulnaris, Flexor Division (506)
Lumbricales Manus, Second (576)
Supinator, Radial Division (492)
Levator Costorum, Inferior Division (642)

CORE VITAMIN C

Abductor Digitus Pedis, Fifth (972)
Brachioradialis, Septal Division (482)
Coccygeus, Sacral Division (740)
Iliococcygeus (746)
Rectus Superior Bulbi (130)

CORE VITAMIN E

Digastric, Anterior Belly (290)
Flexor Digitus Pedis, Fourth (954)
Gluteus Maximus, Sacral Division (850)
Orbicularis Oculi, Superior Division (060)
Palmaris Brevis (596)
Pectoralis Minor, Inferior Division (408)
Piriformis (860)

CORE ZINC

Abductor Pollicis Brevis (542)
Adductor Digitus Pedis, Second (958)
Flexor Digitorum Longus, Medial Division (908)
Gluteus Medius, Anterior Division (784)
Latissimus Dorsi, Thoracic Division (414)
Obturator Externus (812)
Tibialis Posterior, Tibial Division (882)

PARE-X

Anconeus, Ulnar Division (490)
Brachioradialis, Humeral Division (480)
Coracobrachialis, Septal Division (446)
Flexor Digitus Pedis, Second (950)
Gluteus Minimus, Posterior Division (788)
Levator Scapula, Inferior Division (396)
Quadratus Plantae, Medial Division (942)
Temporalis, Occipital Division (112)
Tensor Fascia Lata, Anterior Division (790)
Triceps, Lateral Head (472)
Vastus Intermedius, Medial Division (836)

SPLEN-X

Infraspinatus, Middle Division (426)
Infraspinatus, Superior Division (424)
Rectus Abdominis, Third Division (704)

SPORE-X

Diaphragm, Left Lumbar Division (662)
Extensor Digitorum Communis Manus, Lateral Division (550)
Extensor Indicis Proprius (554)
Extensor Pollicis Longus, Ulnar Division (520)
Flexor Pollicis Longus (528)
Interossei Palmaris, Second (594)
Latissimus Dorsi, Lumbar Division (416)
Pronator Quadratus, Proximal Division (496)
Quadratus Lumborum, Lumbar Division (732)
Scalenus Posterior (284)
Subscapularis, Third Division (438)
Supinator, Ulnar Division (494)
Teres Major, Inferior Division (434)
Tibialis Anterior, Dorsiflexor Division (898)
Vastus Intermedius, Lateral Division (838)

SUPER EPA

Flexor Digitus Pedis, Fifth (956)
Interossei Dorsales Manus, Third (566)

THYM-X

Deltoid, Middle, Anterior Division (454)
Deltoid, Posterior, Medial Division (448)
Diaphragm, Right Lumbar Division (656)
Extensor Hallucis Longus, Interosseous Division (904)
Infraspinatus, Inferior Division (428)

Obliquus Externus Abdominis, Lateral Division (694)
Serratus Anterior, Inferior Division (404)
Supraspinatus, Fossa Division (422)
Supraspinatus, Spine Division (420)
Transverse Abdominis, Lower Division (718)

UTER-X/PROSTA-X

Extensor Carpi Ulnaris, Extensor Division (512)
Flexor Carpi Ulnaris, Adductor Division (508)
Longissimus Thoracis, Inferior Division (618)
Orbicularis Oculi, Inferior Division (062)
Pterygoid External Lateralis, Lower Division (126)
Subscapularis, Inferior Division (442)

UPDATED NUTRIENT INDEX/NUMERICAL

(The numbers in the parentheses refer to the computer numbers in the Muscle Manuals.)

(052) Core Thyro
(058) Core Potassium
(060) Core Vitamin E
(062) Uter-X/Prosta-X
(070) Core Inositol
(098) Core Manganese
(100) Black Currant Seed
(102) Core Iron
(110) Core Niacin
(112) Pare-X
(114) Core Adrenal
(118) Core Pituitary
(120) Core Health Reserve
(122) Core Magnesium
(124) Core Ovary/Orchic
(126) Uter-X/Prosta-X
(130) Core C-TR
(132) Core Rutin
(134) Core Vitamin A
(136) Core Carbo Gest
(138) Core Dent Matrix
(140) Core Magnesium
(270) Core Selenium
(272) Core Calcium
(274) Core Selenium
(276) Core Niacin
(278) Core Kidney
(282) Core Carbo Gest
(284) Spore-X
(286) Core Carbo Gest
(288) Core Pantothenic Acid
(290) Core Vitamin E
(292) Core Bone Matrix
(294) Core Calcium
(296) Core Folic Acid
(298) Core Bile
(300) Core Pantothenic Acid
(302) Core Manganese
(304) Core Manganese
(306) Core Folic Acid
(308) Core Folic Acid
(314) Core D-Tox
(322) Core Potassium
(326) Core Folic Acid
(332) Core Potassium
(334) Core Thyro
(346) Core Rutin
(350) Core Thiamine
(362) Core Potassium

Occipitalis
Levator Palpebrae, Superior Division
Orbicularis Oculi, Superior Division
Orbicularis Oculi, Inferior Division
Depressor Septi
Orbicularis Oris, Upper Division
Orbicularis Oris, Inferior Division
Buccinator
Temporalis, Parietal Division
Temporalis, Occipital Division
Masseter, Superficial Division
Masseter, Deep Division
Pterygoid Internal Medialis, Sphenoid Division
Pterygoid Internal Medialis, Palatine Division
Pterygoid External Lateralis, Upper Div.-Disc
Pterygoid External Lateralis, Lower Division
Rectus Superior Bulbi
Rectus Inferior Bulbi
Rectus Medialis Bulbi
Rectus Lateralis Bulbi
Obliquus Superior Bulbi
Obliquus Inferior Bulbi
Upper Trapezius, Scapular Division
Upper Trapezius, Clavicular Division
Sternocleidomastoid, Sternal Division
Sternocleidomastoid, Clavicular Division
Scalenus Anterior
Scalenus Medius
Scalenus Posterior
Platysma, Anterior Division
Platysma, Posterior Division
Digastric, Anterior Belly
Digastric, Posterior Belly
Stylohyoid
Mylohyoid
Geniohyoid
Sternohyoid
Sternothyroid
Thyrohyoid
Omohyoid
Longus Coli, Vertical Division
Longus Capitis
Splenius Capitis, Mastoid Division
Splenius Cervicis
Semispinalis Capitis
Semispinalis Cervicis
Obliquus Capitis Superior
Interspinalis (Cervical)
Cricoarytenoideus Lateralis

(370) Core Folic Acid	Thyroarytenoideus
(380) Core Thyro	Constrictor Pharyngeus Medius
(390) Core Pituitary	Trapezius, Middle Division
(392) Core Manganese	Trapezius, Lower Division
(394) Core Methionine	Levator Scapula, Superior Division
(396) Pare-X	Levator Scapula, Inferior Division
(398) Core Liver	Rhomboid Minor
(400) Core Liver	Rhomboid Major
(402) Core Thyro	Serratus Anterior, Superior Division
(404) Thym-X	Serratus Anterior, Inferior Division
(406) Core Lung	Pectoralis Minor, Superior Division
(408) Core Vitamin E	Pectoralis Minor, Inferior Division
(410) Core RNA	Subclavius, Clavicular Division
(412) Core Adrenal	Subclavius, Scapular Division
(414) Core Zinc	Latissimus Dorsi, Thoracic Division
(416) Spore-X	Latissimus Dorsi, Lumbar Division
(418) Core Potassium	Latissimus Dorsi, Iliac Division
(420) Thym-X	Supraspinatus, Spine Division
(422) Thym-X	Supraspinatus, Fossa Division
(424) Splen-X	Infraspinatus, Superior Division
(426) Splen-X	Infraspinatus, Middle Division
(428) Thym-X	Infraspinatus, Inferior Division
(430) Core Thyro	Teres Minor
(432) Core Health Reserve	Teres Major, Superior Division
(434) Spore-X	Teres Major, Inferior Division
(436) Core Pancreas	Subscapularis, Superior Division
(438) Spore-X	Subscapularis, Third Division
(440) Core Potassium	Subscapularis, Second Division
(442) Uter X/Prosta-X	Subscapularis, Inferior Division
(444) Core Inositol	Coracobrachialis, Coracoid Division
(446) Pare-X	Coracobrachialis, Septal Division
(448) Thym-X	Deltoid, Posterior, Medial Division
(450) Core Ileoduodenal	Deltoid, Posterior, Lateral Division
(452) Core Lung	Deltoid, Middle, Posterior Division
(454) Thym-X	Deltoid, Middle, Anterior Division
(456) Core Carbo Gest	Deltoid, Anterior, Scapular Division
(458) Core Liver	Deltoid, Anterior, Clavicular Division
(460) Core Folic Acid	Pectoralis Major, Clavicular Division
(462) Core Carbo Gest	Pectoralis Major, Sternal Division
(464) Core Pantothenic Acid	Pectoralis Major, Costal Division
(466) Core Folic Acid	Biceps Brachii Longhead
(468) Core Health Reserve	Biceps Brachii Shorthead
(470) Core Health Reserve	Triceps, Longhead
(472) Pare-X	Triceps, Lateral Head
(474) Core Potassium	Triceps, Medial Head
(476) Core Niacin	Articularis Cubiti
(478) Core Thyro	Brachialis
(480) Pare-X	Brachioradialis, Humeral Division
(482) Core C-TR	Brachioradialis, Septal Division
(484) Core Folic Acid	Pronator Teres, Humeral Division
(486) Core Pituitary	Pronator Teres, Ulnar Division
(488) Core Manganese	Anconeus, Olecranon Division
(490) Pare-X	Anconeus, Ulnar Division
(492) Core B6	Supinator, Radial Division

(494) Spore-X	Supinator, Ulnar Division
(496) Spore-X	Pronator Quadratus, Proximal Division
(498) Core Kidney	Pronator Quadratus, Distal Division
(500) Core Carbo Gest	Palmaris Longus
(502) Core Pantothenic Acid	Flexor Carpi Radialis, Abductor Division
(504) Core Thyro	Flexor Carpi Radialis, Flexor Division
(506) Core B6	Flexor Carpi Ulnaris, Flexor Division
(508) Uter-X/Prosta-X	Flexor Carpi Ulnaris, Adductor Division
(510) Core Ileoduodenal	Extensor Carpi Ulnaris, Adductor Division
(512) Uter-X/Prosta-X	Extensor Carpi Ulnaris, Extensor Division
(514) Core Thyro	Extensor Carpi Radialis Brevis
(516) Core Niacin	Extensor Carpi Radialis Longus, Ext Division
(518) Core Iron	Extensor Carpi Radialis Longus, Abductor Division
(520) Spore-X	Extensor Pollicis Longus, Ulnar Division
(522) Core Carbo Gest	Extensor Pollicis Longus, Septal Division
(524) Core Inositol	Extensor Pollicis Brevis, Radial Division
(526) Core Rutin	Extensor Pollicis Brevis, Septal Division
(528) Spore-X	Flexor Pollicis Longus
(530) Core B6	Abductor Pollicis Longus, Ulnar Division
(532) Core Rutin	Abductor Pollicis Longus, Radial Division
(534) Core Manganese	Flexor Pollicis Brevis
(536) Core Folic Acid	Interossei Pollicis (Palmaris First)
(538) Core Inositol	Opponens Pollicis, Flexor Division
(540) Core Dent Matrix	Opponens Pollicis, Abductor Division
(542) Core Zinc	Abductor Pollicis Brevis
(544) Core RNA	Adductor Pollicis Transversus
(546) Core Niacin	Adductor Pollicis Obliquus
(548) Core B6	Extensor Digitorum Communis Manus, Medial Division
(550) Spore-X	Extensor Digitorum Communis Manus, Lateral Division
(552) Core Brain and Spinal	Extensor Digiti Minimi Manus
(554) Spore-X	Extensor Indicis Proprius
(556) Core Vitamin A	Flexor Digitorum Superficialis, Medial Division
(558) Core Pituitary	Flexor Digitorum Superficialis, Lateral Division
(560) Core Magnesium	Flexor Digitorum Profundus Manus, Medial Division
(562) Black Currant Seed Oil	Flexor Digitorum Profundus Manus, Lateral Division
(564) Core Heart	Interossei Dorsales Manus, Fourth
(566) Super EPA	Interossei Dorsales Manus, Third
(568) Core Carbo Gest	Interossei Dorsales Manus, Second
(570) Core Thiamine	Interossei Dorsales Manus, First
(572) Core Pancreas	Lumbricales Manus, Fourth Division
(574) Core Parathyroid	Lumbricales Manus, Third
(576) Core B6	Lumbricales Manus, Second
(578) Core Liver	Lumbricales Manus, First
(580) Core Dent Matrix	Flexor Digiti Minimi Brevis, Manus
(582) Core Heart	Abductor Digiti Minimi Manus, Flexor Division
(584) Core Ileoduodenal	Abductor Digiti Minimi Manus, Abductor Division
(586) Core Bile	Opponens Digiti Minimi Manus, Abductor Division
(588) Core Bone Matrix	Opponens Digiti Minimi Manus, Flexor Division
(590) Core Rutin	Interossei Palmaris, Fourth
(592) Core Bone Matrix	Interossei Palmaris, Third
(594) Spore-X	Interossei Palmaris, Second
(596) Core Vitamin E	Palmaris Brevis
(610) Core Selenium	Spinalis Thoracis, Lumbar Division
(612) Core Magnesium	Spinalis Thoracis, Thoracic Division

(614) Core Health Reserve	Longissimus Thoracis, Superior Division
(618) Uter-X/Prosta-X	Longissimus Thoracis, Inferior Division
(642) Core B6	Levator Costorum, Inferior Division
(648) Core Iron	Serratus Posterior, Superior Division
(652) Core Magnesium	Serratus Posterior, Inferior Division
(656) Thym-X	Diaphragm, Right Lumbar Division
(662) Spore-X	Diaphragm, Left Lumbar Division
(690) Core Heart	Pyramidalis
(692) Core Selenium	Obliquus Externus Abdominis, Anterior Division
(694) Thym-X	Obliquus Externus Abdominis, Lateral Division
(696) Core Folic Acid	Obliquus Internus Abdominis, Anterior Division
(698) Core Folic Acid	Obliquus Internus Abdominis, Lateral Division
(700) Core D-Tox	Rectus Abdominis, First Division
(702) Core Thyro	Rectus Abdominis, Second Division
(704) Splen-X	Rectus Abdominis, Third Division
(706) Core Folic Acid	Rectus Abdominis, Fourth Div., Medialis
(708) Core Kidney	Rectus Abdominis, Fourth Div., Lateralis
(710) Core Manganese	Iliacus
(712) Core Methionine	Iliacus Minor
(714) Core Oxidate	Transverse Abdominis, Upper Division
(718) Thym-X	Transverse Abdominis, Lower Division
(722) Core Kidney	Psoas Major, Lumbar Division
(724) Core Kidney	Psoas Major, Thoracic Division
(726) Core Adrenal	Psoas Major, Diaphragmatic Division
(728) Core Thyro	Psoas Minor
(730) Core Potassium	Quadratus Lumborum, Costal Division
(732) Spore-X	Quadratus Lumborum, Lumbar Division
(734) Core Magnesium	Multifidus, Lumbosacral Division
(736) Core Iron	Iliocostalis Lumborum
(738) Core Iron	Longissimus Lumborum
(740) Core C-TR	Coccygeus, Sacral Division
(742) Core Thymus	Coccygeus, Coccyx Division
(744) Core Pepsin	Pubococcygeus
(746) Core C-TR	Iliococcygeus
(752) Core Iron	Cremaster
(780) Core Bile	Gluteus Medius, Posterior Division
(782) Core Iron	Gluteus Medius, Middle Division
(784) Core Zinc	Gluteus Medius, Anterior Division
(786) Core Manganese	Gluteus Minimus, Anterior Division
(788) Pare-X	Gluteus Minimus, Posterior Division
(790) Pare-X	Tensor Fascia Lata, Anterior Division
(792) Core D-Tox	Tensor Fascia Lata, Posterior Division
(794) Core Calcium	Rectus Femoris, Reflected Head
(796) Core Niacin	Rectus Femoris, Straight Head
(798) Core Magnesium	Pectineus
(800)L Core Health Reserve	Adductor Brevis (Left)
(800)R Core Health Reserve	Adductor Brevis (Right)
(804) Core Selenium	Adductor Longus, Inferior Division
(806) Core Carbo Gest	Adductor Longus, Superior Division
(808) Core Uterus/Prostate	Gracilis
(810) Core Adrenal	Sartorius
(812) Core Zinc	Obturator Externus
(814) Core Parathyroid	Quadratus Femoris
(816) Core Vitamin A	Vastus Medialis, Upper Division

(818) Core Pancreas	Vastus Medialis, Middle Division
(820) Core Magnesium	Vastus Medialis, Lower Division
(822) Core Lung	Obturator Internus
(824) Core Rutin	Biceps Femoris, Shorthead
(826) Core Health Reserve	Biceps Femoris, Longhead, Fibular Division
(828) Core Niacin	Biceps Femoris, Longhead, Tibial Division
(830) Core Thyro	Vastus Lateralis, Superior Division
(832) Core Methionine	Vastus Lateralis, Middle Division
(834) Core Parathyroid	Vastus Lateralis, Lower Division
(836) Pare-X	Vastus Intermedius, Medial Division
(838) Spore-X	Vastus Intermedius, Lateral Division
(840) Core Carbo Gest	Articularis Genu
(842) Core Magnesium	Adductor Magnus, Vertical Division
(844) Core Pantothenic Acid	Adductor Magnus, Oblique Division
(846) Core Iron	Adductor Magnus, Transverse Division
(848) Core Pancreas	Gluteus Maximus, Iliac Division
(850) Core Vitamin E	Gluteus Maximus, Sacral Division
(852) Core Potassium	Gluteus Maximus, Coccygeal Division
(854) Core Oxidate	Semitendinosus
(856) Core Magnesium	Semimembranosus, Tibial Division
(858) Core Ileoduodenal	Semimembranosus, Popliteal Division
(860) Core Vitamin E	Piriformis
(862) Core Magnesium	Gemellus Inferior
(864) Core Thiamine	Gemellus Superior
(870) Core Inositol	Popliteus
(872) Core Ileoduodenal	Gastrocnemius, Medial Division
(874) Core Thyro	Gastrocnemius, Lateral Division
(876) Core Magnesium	Plantaris
(878) Core Rutin	Soleus Medial Division
(880) Core Liver	Soleus, Lateral Division
(882) Core Zinc	Tibialis Posterior, Tibial Division
(884) Core Potassium	Tibialis Posterior, Fibular Division
(886) Core Ileoduodenal	Peroneus Longus, Cuneiform Division
(888) Core Manganese	Peroneus Longus, Metatarsal Division
(890) Core Iron	Peroneus Brevis, Fibular Division
(892) Core Health Reserve	Peroneus Brevis, Septal Division
(894) Core Manganese	Peroneus Tertius
(896) Core Calcium	Tibialis Anterior, Supinator Division
(898) Spore-X	Tibialis Anterior, Dorsiflexor Division
(900) Core Iron	Flexor Hallucis Longus, Tibial Division
(902) Core Selenium	Flexor Hallucis Longus, Fibular Division
(904) Thym-X	Extensor Hallucis Longus, Interosseous Division
(906) Core Magnesium	Extensor Hallucis Longus, Fibular Division
(908) Core Zinc	Flexor Digitorum Longus, Medial Division
(910) Core Rutin	Flexor Digitorum Longus, Lateral Division
(912) Core Health Reserve	Extensor Digitorum Longus, Medial Division
(914) Core Magnesium	Extensor Digitorum Longus, Lateral Division
(920) Core Potassium	Adductor Hallucis, Superior Division
(922) Core Potassium	Adductor Hallucis, Inferior Division
(924) Core Folic Acid	Flexor Hallucis Brevis, First Cuneiform Division
(926) Core Thyro	Flexor Hallucis Brevis, Tendonal Division
(928) Core Oxidate	Flexor Hallucis Brevis, Third Cuneiform Division
(930) Core Potassium	Flexor Hallucis Brevis, Cuboid Division
(932) Core Thyro	Abductor Hallucis Oblique Head, Peroneus Division

(934) Core Carbo Gest	Abductor Hallucis Oblique Head, Metatarsal Division
(936) Core Rutin	Abductor Hallucis Transverse Head, Medial Division
(938) Core Carbo Gest	Abductor Hallucis Transverse Head, Lateral Division
(940) Core D-Tox	Extensor Hallucis Brevis
(942) Pare-X	Quadratus Plantae, Medial Division
(944) Core Niacin	Quadratus Plantae, Lateral Division
(946) Core Selenium	Flexor Digitorum Brevis, Medial Division
(948) Core Carbo Gest	Flexor Digitorum Brevis, Lateral Division
(950) Pare-X	Flexor Digitus Pedis, Second
(952) Core Kidney	Flexor Digitus Pedis, Third
(954) Core Vitamin E	Flexor Digitus Pedis, Fourth
(956) Super EPA	Flexor Digitus Pedis, Fifth
(958) Core Zinc	Adductor Digitus Pedis, Second
(960) Core Thyro	Adductor Digitus Pedis, Third
(962) Core Potassium	Adductor Digitus Pedis, Fourth
(964) Core Vitamin A	Adductor Digitus Pedis, Fifth
(966) Core Iron	Abductor Digitus Pedis, Second
(968) Core Pantothenic Acid	Abductor Digitus Pedis, Third
(970) Core Manganese	Abductor Digitus Pedis, Fourth
(972) Core C-TR	Abductor Digitus Pedis, Fifth
(974) Core Vitamin A	Abductor Digiti Minimi Pedis
(976) Core Thymus	Extensor Digitorum Brevis

UPDATED VERTEBRAL LEVEL INDEX/SEGMENT

(The numbers in the parentheses refer to the computer numbers in the Muscle Manuals of Dr. Beardall.)

VL C2

Adductor Magnus, Oblique Division (844)
Extensor Pollicis Brevis, Septal Division (526)
Lumbricales Manus, First (578)
Pectoralis Major, Costal Division (464)
Piriformis (860)
Semimembranosus, Popliteal Division (858)

VL C3

Abductor Pollicis Brevis (542)
Digastric, Anterior Belly (290)
Levator Palpebrae, Superior Division (058)
Obliquus Internus Abdominis, Lateral Division (698)
Orbicularis Oris, Upper Division (098)
Pterygoid Internal Medialis, Palatine Division (122)
Splenius Capitis, Mastoid Division (322)
Tibialis Posterior, Tibial Division (882)
Vastus Lateralis, Middle Division (832)

VL C4

Abductor Digitus Pedis, Fourth (970)
Abductor Hallucis Oblique Head, Metatarsal Division (934)
Articularis Genu (840)
Coccygeus, Coccyx Division (742)
Extensor Indicis Proprius (554)
Gluteus Maximus, Iliac Division (848)
Peroneus Tertius (894)
Quadratus Plantae, Medial Division (942)
Subscapularis, Inferior Division (442)

VL C5

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

VL C6

Adductor Pollicis Obliquus (546)
Deltoid, Posterior, Medial Division (448)
Extensor Pollicis Brevis, Radial Division (524)
Gluteus Maximus, Coccygeal Division (852)
Gluteus Minimus, Posterior Division (788)

Latissimus Dorsi, Lumbar Division (416)
Longissimus Thoracis, Superior Division (614)
Lumbricales Manus, Fourth Division (572)

VL C7

Splenius Cervicis (326)
Trapezius, Lower Division (392)
Trapezius, Middle Division (390)

VL T1

Abductor Digitus Pedis, Fifth (972)
Abductor Pollicis Longus, Radial Division (532)
Adductor Longus, Superior Division (806)
Coccygeus, Sacral Division (740)
Extensor Carpi Radialis Brevis(514)
Flexor Digitus Pedis, Fourth (954)
Flexor Hallucis Brevis, First Cuneiform Division (924)
Interossei Dorsales Manus, Fourth (564)
Orbicularis Oris, Inferior Division (100)
Platysma, Anterior Division (286)

VL T2

Abductor Digiti Minimi Manus, Flexor Division (582)
Adductor Magnus, Vertical Division (842)
Extensor Carpi Ulnaris, Extensor Division (512)
Gemellus Superior (864)
Gracilis (808)
Interossei Palmaris, Fourth (590)
Opponens Digiti Minimi Manus, Flexor Division (588)
Pronator Teres, Ulnar Division (486)
Rectus Superior Bulbi (130)
Sternohyoid (300)
Subscapularis, Second Division (440)
Supinator, Ulnar Division (494)

VL T3

Levator Costorum, Inferior Division (642)
Obliquus Inferior Bulbi (140)
Obliquus Internus Abdominis, Anterior Division (696)
Rectus Femoris, Straight Head(796)
Rectus Inferior Bulbi (132)
Rectus Medialis Bulbi (134)
Soleus Medial Division (878)
Spinalis Thoracis, Thoracic Division (612)
Sternothyroid (302)
Subscapularis, Third Division (438)
Teres Minor (430)
Triceps, Medial Head(474)
Vastus Intermedius, Lateral Division (838)

VL T4

Adductor Brevis (Left) (800)
Cricoarytenoideus Lateralis (362)
Flexor Digitorum Longus, Lateral Division (910)
Gluteus Medius, Posterior Division (780)
Infraspinatus, Middle Division (426)
Longus Coli, Vertical Division (308)
Quadratus Lumborum, Lumbar Division (732)
Rectus Abdominis, Third Division (704)
Rectus Femoris, Reflected Head(794)
Semispinalis Capitis (332)
Subscapularis, Superior Division (436)

VL T5

Coracobrachialis, Septal Division (446)
Flexor Digitorum Brevis, Lateral Division (948)
Flexor Digitorum Superficialis, Medial Division (556)
Iliocostalis Lumborum(736)
Opponens Pollicis, Flexor Division (538)
Pectoralis Minor, Inferior Division (408)
Peroneus Brevis, Septal Division (892)
Pronator Quadratus, Proximal Division (496)
Quadratus Femoris(814)
Serratus Anterior, Inferior Division (404)
Supinator, Radial Division (492)
Temporalis, Parietal Division (110)

VL T6

Adductor Digitus Pedis, Third (960)
Depressor Septi (070)
Extensor Carpi Ulnaris, Adductor Division (510)
Extensor Digitorum Communis Manus, Medial Division (548)
Extensor Pollicis Longus, Ulnar Division (520)
Flexor Digitorum Brevis, Medial Division (946)
Flexor Digitorum Profundus Manus, Lateral Division (562)
Interossei Dorsales Manus, Second (568)
Longus Capitis (314)
Rectus Abdominis, Fourth Div., Lateralis(708)
Spinalis Thoracis, Lumbar Division (610)
Sternocleidomastoid, Clavicular Division (276)
Vastus Lateralis, Superior Division (830)

VL T7

Abductor Digitus Pedis, Second (966)
Abductor Pollicis Longus, Ulnar Division (530)
Adductor Longus, Inferior Division (804)
Anconeus, Ulnar Division (490)
Flexor Pollicis Longus(528)
Interossei Dorsales Manus, Third (566)
Latissimus Dorsi, Thoracic Division (414)
Lumbricales Manus, Second (576)
Rectus Lateralis Bulbi (136)

Rhomboid Minor (398)
Supraspinatus, Spine Division (420)
Teres Major, Inferior Division (434)
Tibialis Posterior, Fibular Division (884)
Transverse Abdominis, Upper Division (714)
Vastus Lateralis, Lower Division (834)

VL T8

Adductor Digitus Pedis, Fifth (964)
Brachioradialis, Septal Division (482)
Cremaster (752)
Deltoid, Middle, Anterior Division (454)
Extensor Digitorum Communis Manus, Lateral Division (550)
Flexor Hallucis Brevis, Tendonal Division (926)
Longissimus Thoracis, Inferior Division (618)
Orbicularis Oculi, Inferior Division (062)
Pectoralis Major, Sternal Division (462)
Peroneus Longus, Metatarsal Division (888)
Rhomboid Major (400)
Sternocleidomastoid, Sternal Division (274)
Supraspinatus, Fossa Division (422)
Vastus Medialis, Lower Division (820)

VL T9

Abductor Hallucis Transverse Head, Medial Division (936)
Adductor Digitus Pedis, Fourth (962)
Buccinator (102)
Extensor Hallucis Brevis(940)
Flexor Carpi Ulnaris, Flexor Division (506)
Gastrocnemius, Lateral Division (874)
Geniohyoid (298)
Iliacus Minor (712)
Obturator Internus (822)
Occipitalis (052)
Opponens Pollicis, Abductor Division (540)
Palmaris Brevis (596)
Palmaris Longus (500)
Pronator Teres, Humeral Division (484)
Rectus Abdominis, First Division (700)
Semitendinosus (854)
Soleus, Lateral Division (880)
Vastus Medialis, Middle Division (818)

VL T10

Abductor Hallucis Oblique Head, Peroneus Division (932)
Adductor Brevis (Right) (800)
Adductor Magnus, Transverse Division (846)
Extensor Digitorum Longus, Lateral Division (914)
Extensor Digitorum Longus, Medial Division (912)
Flexor Digitus Pedis, Second (950)
Flexor Pollicis Brevis(534)
Interossei Dorsales Manus, First (570)
Interossei Palmaris, Third (592)

Obliquus Capitis Superior (346)
Opponens Digiti Minimi Manus, Abductor Division (586)
Sartorius (810)
Triceps, Lateral Head (472)
Upper Trapezius, Clavicular Division (272)

VL T11

Adductor Pollicis Transversus (544)
Platysma, Posterior Division (288)
Extensor Carpi Radialis Longus, Abductor Division (518)
Flexor Digitus Pedis, Third (952)
Gastrocnemius, Medial Division (872)
Obliquus Externus Abdominis, Anterior Division (692)
Peroneus Brevis, Fibular Division (890)
Peroneus Longus, Cuneiform Division (886)
Quadratus Plantae, Lateral Division (944)
Subclavius, Clavicular Division (410)

VL T12

Abductor Hallucis Transverse Head, Lateral Division (938)
Biceps Brachii Longhead(466)
Diaphragm, Left Lumbar Division (662)
Diaphragm, Right Lumbar Division (656)
Flexor Digitus Pedis, Fifth (956)
Mylohyoid (296)
Obliquus Superior Bulbi (138)
Pronator Quadratus, Distal Division (498)
Psoas Major, Diaphragmatic Division (726)
Psoas Major, Thoracic Division (724)
Scalenus Posterior (284)
Semispinalis Cervicis (334)
Tensor Fascia Lata, Posterior Division (792)

VL L1

Extensor Digiti Minimi Manus (552)
Flexor Digiti Minimi Brevis, Manus (580)
Gemellus Inferior (862)
Infraspinatus, Inferior Division (428)
Multifidus, Lumbosacral Division (734)
Obliquus Externus Abdominis, Lateral Division (694)
Popliteus (870)
Psoas Major, Lumbar Division (722)
Pterygoid External Lateralis, Lower Division (126)
Scalenus Anterior (278)
Transverse Abdominis, Lower Division (718)
Vastus Medialis, Upper Division (816)

VL L2

Abductor Digitus Pedis, Third (968)
Deltoid, Anterior, Scapular Division (456)
Deltoid, Posterior, Lateral Division (450)
Extensor Digitorum Brevis (976)

Extensor Hallucis Longus, Fibular Division (906)
Extensor Pollicis Longus, Septal Division (522)
Flexor Digitorum Superficialis, Lateral Division (558)
Flexor Hallucis Longus, Tibial Division (900)
Gluteus Medius, Anterior Division (784)
Iliococcygeus (746)
Interossei Pollicis (Palmaris First) (536)
Levator Scapula, Inferior Division (396)
Longissimus Lumborum (738)
Pectoralis Minor, Superior Division (406)
Serratus Posterior, Superior Division (648)
Subclavius, Scapular Division (412)
Tensor Fascia Lata, Anterior Division (790)
Tibialis Anterior, Supinator Division (896)
Upper Trapezius, Scapular Division (270)

VL L3

Abductor Digiti Minimi Manus, Abductor Division (584)
Adductor Hallucis, Inferior Division (922)
Adductor Hallucis, Superior Division (920)
Constrictor Pharyngeus Medius (380)
Extensor Hallucis Longus, Interosseous Division (904)
Flexor Carpi Radialis, Abductor Division (502)
Flexor Carpi Radialis, Flexor Division (504)
Flexor Carpi Ulnaris, Adductor Division (508)
Flexor Hallucis Brevis, Third Cuneiform Division (928)
Levator Scapula, Superior Division (394)
Lumbricales Manus, Third (574)
Masseter, Superficial Division (114)
Omohyoid (306)
Orbicularis Oculi, Superior Division (060)
Pyramidalis (690)
Temporalis, Occipital Division (112)
Teres Major, Superior Division (432)
Thyroarytenoideus (370)
Thyrohyoid (304)

VL L4

Adductor Digitus Pedis, Second (958)
Anconeus, Olecranon Division (488)
Biceps Femoris, Longhead, Tibial Division (828)
Deltoid, Anterior, Clavicular Division (458)
Digastric, Posterior Belly (292)
Flexor Digitorum Longus, Medial Division (908)
Flexor Digitorum Profundus Manus, Medial Division (560)
Gluteus Maximus, Sacral Division (850)
Gluteus Medius, Middle Division (782)
Iliacus (710)
Masseter, Deep Division (118)
Obturator Externus (812)
Pectineus (798)
Plantaris (876)
Pterygoid External Lateralis, Upper Div.-Disc (124)

Quadratus Lumborum, Costal Division (730)
Rectus Abdominis, Fourth Div., Medialis (706)
Rectus Abdominis, Second Division (702)
Scalenus Medius (282)
Triceps, Longhead (470)

VL L5

Articularis Cubiti (476)
Biceps Brachii Shorthead (468)
Biceps Femoris, Longhead, Fibular Division (826)
Biceps Femoris, Shorthead (824)
Brachialis (478)
Brachioradialis, Humeral Division (480)
Flexor Hallucis Brevis, Cuboid Division (930)
Gluteus Minimus, Anterior Division (786)
Interossei Palmaris, Second (594)
Interspinalis (Cervical) (350)
Latissimus Dorsi, Iliac Division (418)
Psoas Minor (728)
Pterygoid Internal Medialis, Sphenoid Division (120)
Pubococcygeus (744)
Semimembranosus, Tibial Division (856)
Stylohyoid (294)
Vastus Intermedius, Medial Division (836)

UPDATED VERTEBRAL LEVEL INDEX/NUMERICAL

(The numbers in the parentheses refer to the computer numbers in the Muscle Manuals of Dr. Beardall.)

(052) VL T9	Occipitalis
(058) VL C3	Levator Palpebrae, Superior Division
(060) VL L3	Orbicularis Oculi, Superior Division
(062) VL T8	Orbicularis Oculi, Inferior Division
(070) VL T6	Depressor Septi
(098) VL C3	Orbicularis Oris, Upper Division
(100) VL T1	Orbicularis Oris, Inferior Division
(102) VL T9	Buccinator
(110) VL T5	Temporalis, Parietal Division
(112) VL L3	Temporalis, Occipital Division
(114) VL L3	Masseter, Superficial Division
(118) VL L4	Masseter, Deep Division
(120) VL L5	Pterygoid Internal Medialis, Sphenoid Division
(122) VL C3	Pterygoid Internal Medialis, Palatine Division
(124) VL L4	Pterygoid External Lateralis, Upper Div.-Disc
(126) VL L1	Pterygoid External Lateralis, Lower Division
(130) VL T2	Rectus Superior Bulbi
(132) VL T3	Rectus Inferior Bulbi
(134) VL T3	Rectus Medialis Bulbi
(136) VL T7	Rectus Lateralis Bulbi
(138) VL T12	Obliquus Superior Bulbi
(140) VL T3	Obliquus Inferior Bulbi
(270) VL L2	Upper Trapezius, Scapular Division
(272) VL T10	Upper Trapezius, Clavicular Division
(274) VL T8	Sternocleidomastoid, Sternal Division
(276) VL T6	Sternocleidomastoid, Clavicular Division
(278) VL L1	Scalenus Anterior
(282) VL L4	Scalenus Medius
(284) VL T12	Scalenus Posterior
(286) VL T1	Platysma, Anterior Division
(288) VL T11	Platysma, Posterior Division
(290) VL C3	Digastric, Anterior Belly
(292) VL L4	Digastric, Posterior Belly
(294) VL L5	Stylohyoid
(296) VL T12	Mylohyoid
(298) VL T9	Geniohyoid
(300) VL T2	Sternohyoid
(302) VL T3	Sternothyroid
(304) VL L3	Thyrohyoid
(306) VL L3	Omohyoid
(308) VL T4	Longus Coli, Vertical Division
(314) VL T6	Longus Capitis
(322) VL C3	Splenius Capitis, Mastoid Division
(326) VL C7	Splenius Cervicis
(332) VL T4	Semispinalis Capitis
(334) VL T12	Semispinalis Cervicis
(346) VL T10	Obliquus Capitis Superior
(350) VL L5	Interspinalis (Cervical)
(362) VL T4	Cricoarytenoideus Lateralis

(370) VL L3 Thyroarytenoideus
(380) VL L3 Constrictor Pharyngeus Medius
(390) VL C7 Trapezius, Middle Division
(392) VL C7 Trapezius, Lower Division
(394) VL L3 Levator Scapula, Superior Division
(396) VL L2 Levator Scapula, Inferior Division
(398) VL T7 Rhomboid Minor
(400) VL T8 Rhomboid Major
(402) VL C5 Serratus Anterior, Superior Division
(404) VL T5 Serratus Anterior, Inferior Division
(406) VL L2 Pectoralis Minor, Superior Division
(408) VL T5 Pectoralis Minor, Inferior Division
(410) VL T11 Subclavius, Clavicular Division
(412) VL L2 Subclavius, Scapular Division
(414) VL T7 Latissimus Dorsi, Thoracic Division
(416) VL C6 Latissimus Dorsi, Lumbar Division
(418) VL L5 Latissimus Dorsi, Iliac Division
(420) VL T7 Supraspinatus, Spine Division
(422) VL T8 Supraspinatus, Fossa Division
(424) VL C5 Infraspinatus, Superior Division
(426) VL T4 Infraspinatus, Middle Division
(428) VL L1 Infraspinatus, Inferior Division
(430) VL T3 Teres Minor
(432) VL L3 Teres Major, Superior Division
(434) VL T7 Teres Major, Inferior Division
(436) VL T4 Subscapularis, Superior Division
(438) VL T3 Subscapularis, Third Division
(440) VL T2 Subscapularis, Second Division
(442) VL C4 Subscapularis, Inferior Division
(444) VL C5 Coracobrachialis, Coracoid Division
(446) VL T5 Coracobrachialis, Septal Division
(448) VL C6 Deltoid, Posterior, Medial Division
(450) VL L2 Deltoid, Posterior, Lateral Division
(452) VL C5 Deltoid, Middle, Posterior Division
(454) VL T8 Deltoid, Middle, Anterior Division
(456) VL L2 Deltoid, Anterior, Scapular Division
(458) VL L4 Deltoid, Anterior, Clavicular Division
(460) VL C5 Pectoralis Major, Clavicular Division
(462) VL T8 Pectoralis Major, Sternal Division
(464) VL C2 Pectoralis Major, Costal Division
(466) VL T12 Biceps Brachii Longhead
(468) VL L5 Biceps Brachii Shorthead
(470) VL L4 Triceps, Longhead
(472) VL T10 Triceps, Lateral Head
(474) VL T3 Triceps, Medial Head
(476) VL L5 Articularis Cubiti
(478) VL L5 Brachialis
(480) VL L5 Brachioradialis, Humeral Division
(482) VL T8 Brachioradialis, Septal Division
(484) VL T9 Pronator Teres, Humeral Division
(486) VL T2 Pronator Teres, Ulnar Division
(488) VL L4 Anconeus, Olecranon Division
(490) VL T7 Anconeus, Ulnar Division
(492) VL T5 Supinator, Radial Division

(494) VL T2 Supinator, Ulnar Division
(496) VL T5 Pronator Quadratus, Proximal Division
(498) VL T12 Pronator Quadratus, Distal Division
(500) VL T9 Palmaris Longus
(502) VL L3 Flexor Carpi Radialis, Abductor Division
(504) VL L3 Flexor Carpi Radialis, Flexor Division
(506) VL T9 Flexor Carpi Ulnaris, Flexor Division
(508) VL L3 Flexor Carpi Ulnaris, Adductor Division
(510) VL T6 Extensor Carpi Ulnaris, Adductor Division
(512) VL T2 Extensor Carpi Ulnaris, Extensor Division
(514) VL T1 Extensor Carpi Radialis Brevis
(516) VL C5 Extensor Carpi Radialis Longus, Ext Division
(518) VL T11 Extensor Carpi Radialis Longus, Abductor Division
(520) VL T6 Extensor Pollicis Longus, Ulnar Division
(522) VL L2 Extensor Pollicis Longus, Septal Division
(524) VL C6 Extensor Pollicis Brevis, Radial Division
(526) VL C2 Extensor Pollicis Brevis, Septal Division
(528) VL T7 Flexor Pollicis Longus
(530) VL T7 Abductor Pollicis Longus, Ulnar Division
(532) VL T1 Abductor Pollicis Longus, Radial Division
(534) VL T10 Flexor Pollicis Brevis
(536) VL L2 Interossei Pollicis (Palmaris First)
(538) VL T5 Opponens Pollicis, Flexor Division
(540) VL T9 Opponens Pollicis, Abductor Division
(542) VL C3 Abductor Pollicis Brevis
(544) VL T11 Adductor Pollicis Transversus
(546) VL C6 Adductor Pollicis Obliquus
(548) VL T6 Extensor Digitorum Communis Manus, Medial Division
(550) VL T8 Extensor Digitorum Communis Manus, Lateral Division
(552) VL L1 Extensor Digiti Minimi Manus
(554) VL C4 Extensor Indicis Proprius
(556) VL T5 Flexor Digitorum Superficialis, Medial Division
(558) VL L2 Flexor Digitorum Superficialis, Lateral Division
(560) VL L4 Flexor Digitorum Profundus Manus, Medial Division
(562) VL T6 Flexor Digitorum Profundus Manus, Lateral Division
(564) VL T1 Interossei Dorsales Manus, Fourth
(566) VL T7 Interossei Dorsales Manus, Third
(568) VL T6 Interossei Dorsales Manus, Second
(570) VL T10 Interossei Dorsales Manus, First
(572) VL C6 Lumbricales Manus, Fourth Division
(574) VL L3 Lumbricales Manus, Third
(576) VL T7 Lumbricales Manus, Second
(578) VL C2 Lumbricales Manus, First
(580) VL L1 Flexor Digiti Minimi Brevis, Manus
(582) VL T2 Abductor Digiti Minimi Manus, Flexor Division
(584) VL L3 Abductor Digiti Minimi Manus, Abductor Division
(586) VL T10 Opponens Digiti Minimi Manus, Abductor Division
(588) VL T2 Opponens Digiti Minimi Manus, Flexor Division
(590) VL T2 Interossei Palmaris, Fourth
(592) VL T10 Interossei Palmaris, Third
(594) VL L5 Interossei Palmaris, Second
(596) VL T9 Palmaris Brevis
(610) VL T6 Spinalis Thoracis, Lumbar Division
(612) VL T3 Spinalis Thoracis, Thoracic Division

(614) VL C6 Longissimus Thoracis, Superior Division
(618) VL T8 Longissimus Thoracis, Inferior Division
(642) VL T3 Levator Costorum, Inferior Division
(648) VL L2 Serratus Posterior, Superior Division
(652) VL C5 Serratus Posterior, Inferior Division
(656) VL T12 Diaphragm, Right Lumbar Division
(662) VL T12 Diaphragm, Left Lumbar Division
(690) VL L3 Pyramidalis
(692) VL T11 Obliquus Externus Abdominis, Anterior Division
(694) VL L1 Obliquus Externus Abdominis, Lateral Division
(696) VL T3 Obliquus Internus Abdominis, Anterior Division
(698) VL C3 Obliquus Internus Abdominis, Lateral Division
(700) VL T9 Rectus Abdominis, First Division
(702) VL L4 Rectus Abdominis, Second Division
(704) VL T4 Rectus Abdominis, Third Division
(706) VL L4 Rectus Abdominis, Fourth Div., Medialis
(708) VL T6 Rectus Abdominis, Fourth Div., Lateralis
(710) VL L4 Iliacus
(712) VL T9 Iliacus Minor
(714) VL T7 Transverse Abdominis, Upper Division
(718) VL L1 Transverse Abdominis, Lower Division
(722) VL L1 Psoas Major, Lumbar Division
(724) VL T12 Psoas Major, Thoracic Division
(726) VL T12 Psoas Major, Diaphragmatic Division
(728) VL L5 Psoas Minor
(730) VL L4 Quadratus Lumborum, Costal Division
(732) VL T4 Quadratus Lumborum, Lumbar Division
(734) VL L1 Multifidus, Lumbosacral Division
(736) VL T5 Iliocostalis Lumborum
(738) VL L2 Longissimus Lumborum
(740) VL T1 Coccygeus, Sacral Division
(742) VL C4 Coccygeus, Coccyx Division
(744) VL L5 Pubococcygeus
(746) VL L2 Iliococcygeus
(752) VL T8 Cremaster
(780) VL T4 Gluteus Medius, Posterior Division
(782) VL L4 Gluteus Medius, Middle Division
(784) VL L2 Gluteus Medius, Anterior Division
(786) VL L5 Gluteus Minimus, Anterior Division
(788) VL C6 Gluteus Minimus, Posterior Division
(790) VL L2 Tensor Fascia Lata, Anterior Division
(792) VL T12 Tensor Fascia Lata, Posterior Division
(794) VL T4 Rectus Femoris, Reflected Head
(796) VL T3 Rectus Femoris, Straight Head
(798) VL L4 Pectineus
(800 L)VL T4 Adductor Brevis (Left)
(800 R)VL T10 Adductor Brevis (Right)
(804) VL T7 Adductor Longus, Inferior Division
(806) VL T1 Adductor Longus, Superior Division
(808) VL T2 Gracilis
(810) VL T10 Sartorius
(812) VL L4 Obturator Externus
(814) VL T5 Quadratus Femoris
(816) VL L1 Vastus Medialis, Upper Division

(818) VL T9 Vastus Medialis, Middle Division
(820) VL T8 Vastus Medialis, Lower Division
(822) VL T9 Obturator Internus
(824) VL L5 Biceps Femoris, Shorthead
(826) VL L5 Biceps Femoris, Longhead, Fibular Division
(828) VL L4 Biceps Femoris, Longhead, Tibial Division
(830) VL T6 Vastus Lateralis, Superior Division
(832) VL C3 Vastus Lateralis, Middle Division
(834) VL T7 Vastus Lateralis, Lower Division
(836) VL L5 Vastus Intermedius, Medial Division
(838) VL T3 Vastus Intermedius, Lateral Division
(840) VL C4 Articularis Genu
(842) VL T2 Adductor Magnus, Vertical Division
(844) VL C2 Adductor Magnus, Oblique Division
(846) VL T10 Adductor Magnus, Transverse Division
(848) VL C4 Gluteus Maximus, Iliac Division
(850) VL L4 Gluteus Maximus, Sacral Division
(852) VL C6 Gluteus Maximus, Coccygeal Division
(854) VL T9 Semitendinosus
(856) VL L5 Semimembranosus, Tibial Division
(858) VL C2 Semimembranosus, Popliteal Division
(860) VL C2 Piriformis
(862) VL L1 Gemellus Inferior
(864) VL T2 Gemellus Superior
(870) VL L1 Popliteus
(872) VL T11 Gastrocnemius, Medial Division
(874) VL T9 Gastrocnemius, Lateral Division
(876) VL L4 Plantaris
(878) VL T3 Soleus Medial Division
(880) VL T9 Soleus, Lateral Division
(882) VL C3 Tibialis Posterior, Tibial Division
(884) VL T7 Tibialis Posterior, Fibular Division
(886) VL T11 Peroneus Longus, Cuneiform Division
(888) VL T8 Peroneus Longus, Metatarsal Division
(890) VL T11 Peroneus Brevis, Fibular Division
(892) VL T5 Peroneus Brevis, Septal Division
(894) VL C4 Peroneus Tertius
(896) VL L2 Tibialis Anterior, Supinator Division
(898) VL C5 Tibialis Anterior, Dorsiflexor Division
(900) VL L2 Flexor Hallucis Longus, Tibial Division
(902) VL C5 Flexor Hallucis Longus, Fibular Division
(904) VL L3 Extensor Hallucis Longus, Interosseous Division
(906) VL L2 Extensor Hallucis Longus, Fibular Division
(908) VL L4 Flexor Digitorum Longus, Medial Division
(910) VL T4 Flexor Digitorum Longus, Lateral Division
(912) VL T10 Extensor Digitorum Longus, Medial Division
(914) VL T10 Extensor Digitorum Longus, Lateral Division
(920) VL L3 Adductor Hallucis, Superior Division
(922) VL L3 Adductor Hallucis, Inferior Division
(924) VL T1 Flexor Hallucis Brevis, First Cuneiform Division
(926) VL T8 Flexor Hallucis Brevis, Tendonal Division
(928) VL L3 Flexor Hallucis Brevis, Third Cuneiform Division
(930) VL L5 Flexor Hallucis Brevis, Cuboid Division
(932) VL T10 Abductor Hallucis Oblique Head, Peroneus Division

AN IMPROVED SCREENING PROCEDURE FOR NEUROMUSCULR HYPEERSENSITIVITY REACTIONS

Timothy D. Francis, D.C.

ABSTRACT: Many patients suffer recurrent health problems due to food sensitivities. This paper describes a more sensitive and clinically time efficient method for food sensitivities screening.

After studying food and other sensitivity screening procedures from various authors (Schmitt, Lebowitz, Vreeland) and attempting to identify various allergens/antigens which were preventing some of my patients from reaching optimum health and function, I embarked upon an investigation of my own to try to find a more effective means of identifying these troublesome particles.

I found that screening with powdered antigens orally to be only partially accurate. Many times this procedure did not cause universal muscle weakening, but did cause strengthening of a weak Gamma II muscle. In addition, Dr. Bob Blaich made me aware that the suspected offender may only cause special muscle weakening as opposed to universal weakening. This author has also found this to be true and yet I did not find this to be clinically time efficient.

While attending Dr. Schmitt's seminars I became aware of immune circuit neurolymphatics at the upper sternum (thymus), lower sternum (pectoralis minor NL), and spleen neurolymphatic. I found that if the suspected antigen was placed in the mouth and universal muscle weakening and/or strengthening of a weak Gamma II was negative, simultaneous TL to one of the immune circuits would cause a weakening of a strong indicator muscle. However, it soon became apparent that there were more than these three circuits and it could actually be of any circuit, again becoming a cumbersome procedure.

Along about this time, I received some seminar tapes from Michael Lebowitz, D.C. describing testing antigens under the south pole of a magnet over GV 20. Again I experimented with this procedure and cross correlated with oral antigen testing. This procedure also proved to have too many false negatives under therapy localizing one or more lymphatic areas.

Another interesting procedure I observed was Dr. Vreeland's demonstration of negative oral antigen testing, then having the patient swallow with the antigen in the mouth; placing additional antigen on the patient's tongue and upon re-testing demonstrating weakening of a strong indicator muscle.

This posed another question - how many false negatives are occuring with our testing procedures?

Recalling an earlier procedure described in Dr. Lebowitz's and

(934) VL C4 Abductor Hallucis Oblique Head, Metatarsal Division
(936) VL T9 Abductor Hallucis Transverse Head, Medial Division
(938) VL T12 Abductor Hallucis Transverse Head, Lateral Division
(940) VL T9 Extensor Hallucis Brevis
(942) VL C4 Quadratus Plantae, Medial Division
(944) VL T11 Quadratus Plantae, Lateral Division
(946) VL T6 Flexor Digitorum Brevis, Medial Division
(948) VL T5 Flexor Digitorum Brevis, Lateral Division
(950) VL T10 Flexor Digitus Pedis, Second
(952) VL T11 Flexor Digitus Pedis, Third
(954) VL T1 Flexor Digitus Pedis, Fourth
(956) VL T12 Flexor Digitus Pedis, Fifth
(958) VL L4 Adductor Digitus Pedis, Second
(960) VL T6 Adductor Digitus Pedis, Third
(962) VL T9 Adductor Digitus Pedis, Fourth
(964) VL T8 Adductor Digitus Pedis, Fifth
(966) VL T7 Abductor Digitus Pedis, Second
(968) VL L2 Abductor Digitus Pedis, Third
(970) VL C4 Abductor Digitus Pedis, Fourth
(972) VL T1 Abductor Digitus Pedis, Fifth
(974) VL C5 Abductor Digiti Minimi Pedis
(976) VL L2 Extensor Digitorum Brevis

A RELATIONSHIP BETWEEN CHINESE ACUPUNCTURE AND JAPANESE REVIVAL POINTS

HANNES L. HENDRICKSON, D.C., P.E., B. Ch.E.

ABSTRACT: This is a study of the relationship between Chinese acupuncture and Japanese revival points.

DISCUSSION: The Japanese developed jiu-jitsu prior to the nineteen hundreds to a very high degree inflicting severe blows to their opponents. However, they also developed revival methods such as KUATSU (1). It is interesting to note that many of these points of revival were also noted in acupunture, developed by the Chinese, and written about by Dr. Mary Austin (2). Denis & Joyce, Lawson-Wood (3) also pictured the acupuncture points used to revive etc. participants in sports. They also discuss the methods of well being using acupuncture.

Dr. Austin points out on page 249 of her book that a "SPECIAL POINT OF 'KAUTSU'": "between the 12 dorsal spinous and the 1st lumbar spinous "--that point stimulates heart action, stimulates kidneys, aorta, peritoneum, and brain. She points out it would be a serious omission if she had left out that point. Called the point Gov. .5bis- ie between 5 & 6.

Conclusions: No doubt a knowledge of these points would be an asset to members of ICAK since so many are involved with sports. Perhaps these points also play a role in coma arousal--which is so prevalent today as a result, in most cases, from car accidents, diving accidents etc.

REFERENCES: (1)H.L.Hendrickson "The Restoration Of Life-Kuatsu" Collected Papers ICAK ,Winter 1987.
(2) Dr. Mary Austin ,"Acupuncture Therapy" ASI Publishers Inc., New York, N.Y.1972
(3) D. & J. Lawson-Wood, "Judo Revival Points Athletes' Points and Posture(1960)"

AN IMPROVED SCREENING PROCEDURE - Francis

Dr. Steele's book, Correcting Chronic Health Problems, they used homeopathic allergens as a screen for food allergies.

I began testing patients with a 6x dilution of food allergens and cross checking the results with powdered antigen testing. I found that when the homeopathic tested positive (causing weakening of a strong indicator muscle and/or strengthening of a weak Gamma II) the powdered antigen may or may not cause an indicator muscle change unless simultaneously therapy localizing an immune circuit point or some other lymphatic point or testing all the muscles of the body. Furthermore, when the powdered antigen tested positive, the homeopathic antigen always tested positive.

CONCLUSION: I would recommend testing for antigen/allergen reactions using homeopathics versus powdered antigens for two reasons: one is to avoid false negatives, and two, much more testing may be completed in the span of time alloted for an office visit.

Food testing kits are available from Dolisos America, Inc., 3014 Rigel Ave., Las Vegas, Nv. 89102, or you may call them at 1-800-365-4767. The cost is $99 per kit. There are 2 kits covering over 170 food items.

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ATYPICAL FIBULAR SUBLUXATION IN A CASE OF LOWER LEG NEURITIS

By John M. Heidrich, D.C.

Abstract: A case history of radicular leg pain is presented with atypical anterior displacement of the fibular head. Applied kinesiology management is outlined.

Case History:

A 50 year-old, physically active male complained of left leg "toothache" type pain of almost three months duration. This symptom began as a burning sensation that extended from the lateral knee to the dorsum of the foot. The pain had rapidly increased to an "intolerable" level within the previous six weeks. Onset followed a particularly "vigorous" golf swing on a driving range. In desperation he had consulted two different family practitioners, an acupuncturist, an independent orthopedist, followed a trial of physical therapy, and was currently under treatment at the orthopedic department of the Cleveland Clinic. These previous therapies, including a variety of analgesics and anti-inflammatories provided little relief. He was maintaining daily doses of Percocet to control pain. Lumbar magnetic resonance imaging was negative for discopathy. Electromyographic and nerve conduction velocity tests were positive for left deep peroneal radiculopathy. He was scheduled for magnetic resonance imaging of the left knee within two weeks of presenting to our office. Within the last week there was an onset of acute left sacro-iliac pain which "did not feel connected" with the leg pain.

Postural exam revealed a left genu valgus and depression of the left iliac crest. Active and passive knee range of motion was normal and the usual compression and distraction orthopedic tests were unremarkable. Palpatory pain was elicited at the anterosuperior border of the fibular head at a very specific point in the interosseous groove and followed a path into the lateral collateral ligament. This was differentiated between peroneal nerve sensitivity at the lateral fibular neck. There was no pain at the tibial prominences or at the posterior popliteal area. The atypical impression was a comparative lack of pain at the posterior fibular head which is often found in lateral knee problems. Muscle testing revealed weakness of left sartorius, tensor fascia lata, popliteus, anterior tibialis, and peroneus tertius.

Direct posterior-to-anterior challenge (1) for the fibular head revealed an anterior displacement. With the patient supine, I bent the knee slightly and stabilized the calf between my knees. I attempted a quasi-meniscus adjustment while externally rotating the fibular head. This had no effect. With the knee over the side of the table, I then grasped the inside of the foot with my left hand and contacted the fibula with my right thenar. While everting the foot, I brought the knee into slight varus and thrust "A to P". This

resulted in a loud articulation and mutual astonishment, although I think his astonishment may have been for different reasons. Subsequent correction was made of a talus subluxation (1) and a Category II posterior ilium (2) on the left. The patient complained of severe pain in the knee for the next two days. He was seen five days later and fascial flush (3) was performed on the tensor fascia lata. Within nine days there was 90% subjective improvement in pain with mild residual burning in the dorsum of the ankle.

Typical presentation of fibular subuxation is described by DeJarnette (4), Hearon (5), Beatty (6), Walther (7), and others as posterolateral. This typical presentation often follows valgus stress injuries to the knee with internal rotation of the tibia, pronation of the foot, and separation of the proximal tibiofibular joint (8). This also correlates with the typical sartorius/posterior ilium and genu valgus presentation (2). The osteology is such that the facet angle between the tibia and fibula runs obliquely anterolateral and superior (8) and therefore does not predispose anterior movement of the fibular head. Hearon describes a technique for anterolateral subluxation with similar correction while the knee joint is in full extension (9). Although I could not recall performing a deliberate anterior fibula correction in the past, I was surprised to find that my two proximate colleagues, with a combined 26 years of clinical experience, could not recall encountering one either.

Discussion:

It is proposed that the sudden genu varus position on the end phase of the golf swing prompted the anteriority of the fibula during forceful external rotation of the tibia. This apparently allowed the fibula to jam against the tibia and traction the peroneal nerve with concommitant strain of the tensor fascia lata and lateral ligaments of the knee. This case presentation may be helpful to reinforce the importance of proper diagnosis through challenge.

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Foot Orthotics - Redefining a system

David W. Leaf, D.C.

Abstract:
Orthotic Evaluation and casting: This paper describes a new method of creating an orthotic to correct an individuals foot imbalances. New keys for examining the patient for need of an orthotic are also included.

Many doctors utilize orthotics for correction of foot imbalances. Various types and methods of creation are used for manufacturing of the insert. There are three major types of orthotics. They are first those that reduce impact and cushion the foot. Second, those that support structures of the foot, and finally those that resduce abnormal pressure and strains on the foot. Different systems have been developed to try and accomplish one or more of these functions.

Most systems are dependent upon creating a cast or impression that normalizes the forefoot to hindfoot angles. A study comparing casting techniques using either non-weight bearing supine or prone methods or a semiweight-bearing procedure found that the nonweight bearing techniques obtained similiar results. The weight bearing procedure did not conform to the standards. This study measured the casts being prepared in the office setting and are used to prepare an orthotic that the technician/physician is in control of. Alternative methods that depend upon laboratory correction of foot alignment imbalances are also available for in office use. One of the most common and easiest to use involves stepping into a foam box and sending the foot imprint to a lab for manufacturing. This method is completely dependent upon the laboratory to determine the correct correction for the patient's foot. While this is effective for many patients it is dependent upon the laboratory for determining the degree of correction. Other methods have greater doctor input into the final correction provided by the insert. One popular method in the podiatric field is to make a plaster cast of the foot with the talus in a concentric position. This position allows the subtalar joint to be stabilized in this neutral position. The plaster is applied to the foot and the alignment of the calcaneus and talus is shifted between inversion and eversion until equal parts of the talus are palpated on the medial and lateral aspects of the ankle. The orthotic is then fabricated to stabilize this position. Another method is to have the patient stand and then walk across a plate which measures the weight distribution on the foot both statically and kinetically. While this would appear to be the most accurate method, further investigation into one of the facilities using this fabrication procedure disclosed that only five different types of correction were used.

Dr. Pierre DesRouche of Montreal, following a seminar on foot problems, had Foot Levelers create a series of differing supports to correct the longitudinal arch and stabilize the navicular. Foot Levelers now produces over 14 differing sizes of correction. Once a determination has been made that supporting the arch is needed.

The correct amount of support can be determined by using these different size forms under the arch. When the correct amount of correction is inserted under the arch, related pain patterns and muscle integrity will be found.

A discussion of related pain patterns and muscle weakness findings associated with foot pronation is appropriate. When the foot pronates, the talus shifts medial and the calcaneus shifts . The shifting of the calcaneus creates palpable tenderness on the lateral aspect. The shifting of the ankle creates a torsion on the tibia. This torsion creates increased tonus of the popliteus and can be palpated on the postero-medial aspect of the tibia at the insertion of the muscle. The torsion of the tibia creates a rotation of the femur. This can be felt in two areas. First, the muscular attachment of the hamstrings at the ischial tuberosity will be tender, and the muscle fibers superior to the attachment of the gluteus medius on the greater trochanter will be tender to palpation. This last finding is significant in many patients and explain the ubiquitous point pain that many complain off. It is easy to demonstrate the effect of pronation on the patient by having them palpate over the trochanter while pronating and supinating the foot. Continuing superior, the quadratus lumborum and erector spinae muscles will be tender to palpation. In the cervical region, the scaleni muscles will be found to be tender and finally, the muscle in the pterygoid pocket will be tender on the side of foot pronation.

On weightbearing, if the navicular has dropped excessively, this will create the tarsal tunnel syndrome. This finding is many times missed by checking the patient in a non-weightbearing state for weakness of the flexor hallucis brevis (FHB). While difficult on the examiner, the FHB is easily tested in the standing position. If found to be strong, the patient should be instructed to shift their weight forward over the foot. The FHB is then retested. The FHB should remain strong throughout stance including toe off. Weakening of the FHB indicates either a dropping of the navicular and elongation of the longitudinal arch or an imbalance in the metatarsal arch.

Evaluation of the metatarsal arch is easily performed in the standing position after determining if correction of the longitudinal arch is needed and which support needs to be added. While the patient is standing with the correct support under the foot, palpate over the metatarsal heads. If pain is present and or the FHB is weak, slide a tongue depressor under the metatarsals increasing the height of the arch and move it until the pain is gone. If this only decreases she pain, try two. This position will also strengthen the FHB. Note: this step can only be done after the correct arch height has been determined using the forms supplied by Foot Levelers. Use this same procedure to determine the degree of correction needed to correct pain under the first metatarsal due to a sesamoid bone. Correct casting of a patient will allow the orthotic to remove the extra stress creating the pain. A paper published in the American Journal of Sports Medecine reports that 8 of 10 cases of pain due to a sesamoid bone beneath the first metatarsal were relieved by the proper application of an orthotic. It should be noted that pain over the first metatarsal that is commonly related to the beginning stages of a bunion formation can be relieved by

normalizing proper foot mechanics. In this case, posterior movement of the calcaneus with the accompnying dropping of the navicular causes plantar fascitis and the pain pattern over the first metatarsal. Adequate correction of the foot will show an immediate reduction to palpable pain.

After determining the degree of correction needed for the patient, and if metatarsal support is needed, casting the the patient's foot is done using the foam model. The information regarding the degree of support is then added to the order slip and the laboratory is then able to make a support to your orders. Each trial support is numbered so that the record keeping is easily done. Simply order for example a # 9 on the left and a #8 on the right. If metatarsal support is needed, record the height and exact location for it.

This procedure for determing both the need and the degree of correction has been of great benefit to the patient. This procedure is only used after correction of all foot misalignments, balancing of any pelvic involvments, and correction of any underlying foot/ankle muscle involvements.

For information on obtaining the set of graded supports, contact Dwayne Bennett at 1-800-553-4860.

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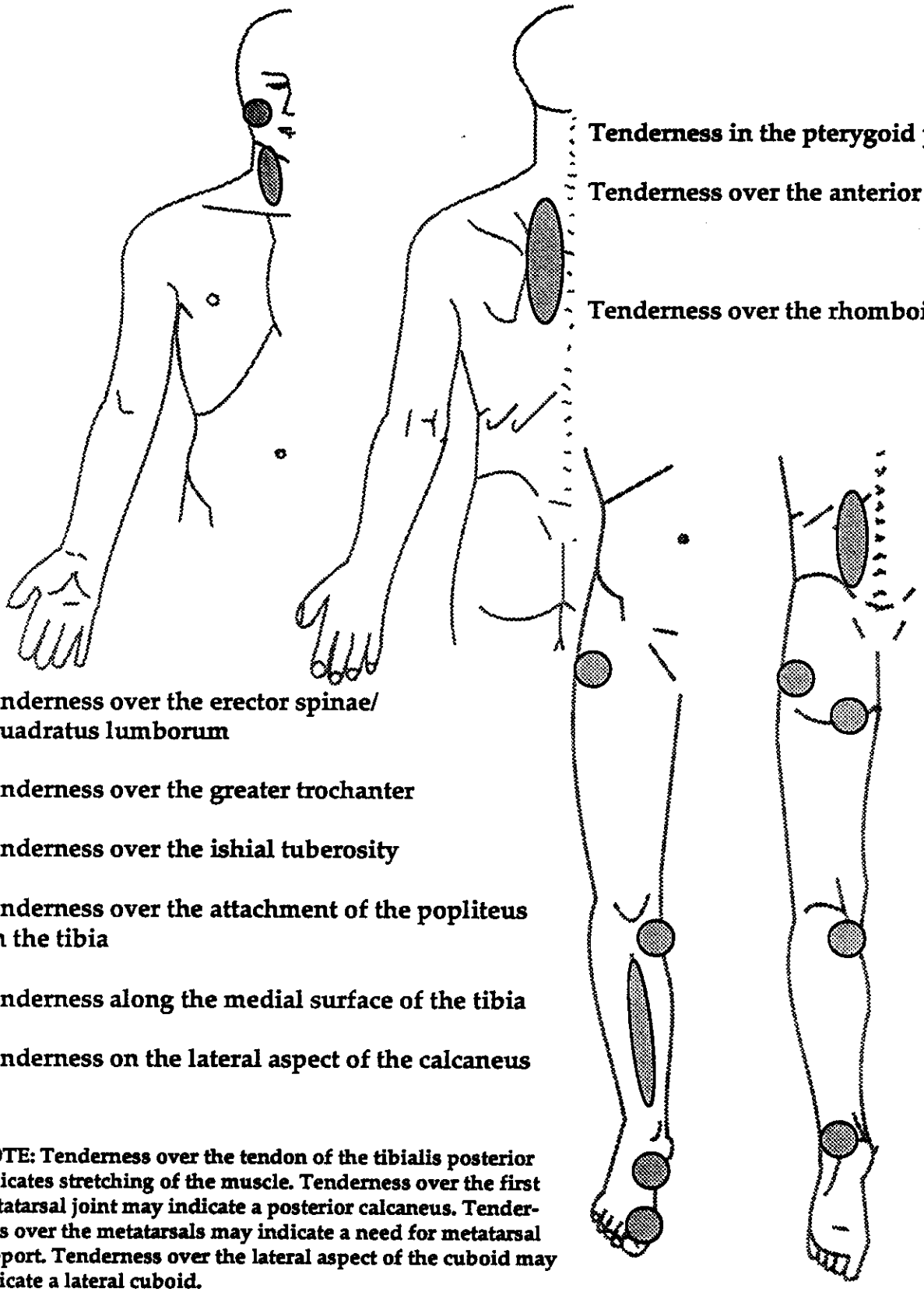
Palpable pain areas associated with foot pronation



Tenderness in the pterygoid pocket

Tenderness over the anterior scalene

Tenderness over the rhomboid

Tenderness over the erector spinae/ quadratus lumborum

Tenderness over the greater trochanter

Tenderness over the ishial tuberosity

Tenderness over the attachment of the popliteus on the tibia

Tenderness along the medial surface of the tibia

Tenderness on the lateral aspect of the calcaneus

NOTE: Tenderness over the tendon of the tibialis posterior indicates stretching of the muscle. Tenderness over the first metatarsal joint may indicate a posterior calcaneus. Tenderness over the metatarsals may indicate a need for metatarsal support. Tenderness over the lateral aspect of the cuboid may indicate a lateral cuboid.

DWL/3.92

The Piriformis Syndrome

David W. Leaf

Abstract:
The Applied Kinesiological approach to the piriformis syndrome:A frequently overlooked condition is the piriformis syndrome. The question of why the piriformis becomes involved, and a quick diagnostic test for this condition are the purposes of this paper.

A frequently overlooked condition is the piriformis syndrome. This condition causes various symptoms from pain to paresthesia throughout the pelvis, thigh, leg and foot. Characteristically, the symptoms are aggravated by prolonged sitting, or motions that cause hip flexion combined with rotation.

One study showed that this syndrome was more prevalent in patient's suffering of low back/leg pain than disc protrusion. Another study demonstrated that the piriformis was one of the most frequent sites of myotonic reflexes in the lumbar/pelvic region.

The question of why the piriformis becomes involved, and a quick diagnostic test for this condition are the purposes of this paper.

The piriformis is a thick muscle that arises from the anterior surface of the sacrum by separate digitations 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 exits the pelvis through the greater sciatic foramen. 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 muscle. 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 pudental 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 gemilli muscles. The inferior gluteal is important as it supplies the gluteus maximus muscle. It should be noted that the nerve may pass through the piriformis muscle with the peroneal portion of the sciatic nerve.

If the piriformis is considered as a pelvic stabilizer, the gluteus maximus must then be considered as its synergistic partner in the stabilization of the sacroiliac joint. The gluteus maximus attaches to the posterolateral surface of the sacrum, the side of the coccyx and to the ilium along the posterior border and the posterior iliac crest. It also attaches to the sacrotuberous ligament, has an aponeurosis with the erector spinae muscles and attaches to the fascia of the gluteus medius muscle. The muscle has two attachments on the femur. One, accounting for the majority of the fibers, attaches to the iliotibial band of the fascia lata; the other, deep inferior fibers attach to the gluteal tuberosity between the attachments of the adductor magnus and vastus lateralis muscles.

It is clear from the attachments of these muscles that coordinated contraction stabilizes the sacroiliac joint. Any weakness of the gluteus maximus will result in increased contraction of the piriformis and potentially create entrapment syndromes at the greater sciatic foramen.

Diagnostically, two different approaches can be made to determine the existence of this condition. First, an understanding of the possible nerve entrapment syndromes can be used as a diagnostic key. Specific muscle weakness patterns will be found depending upon which nerves are entrapped. These findings can be related to weight bearing and non-weightbearing muscle tests. The second approach is the examination of the function of the piriformis as a thigh rotator. With the patient in a prone position, thigh in a neutral position, the piriformis laterally rotates the thigh. Consequently, increased contraction of the piriformis will restrict medial rotation. This restriction of rotation is more difficult to show in the supine position as the effect of the piriformis on thigh rotation changes depending upon the degree of thigh flexion. When the thigh is fully flexed, the piriformis when contracted will medially rotate the thigh and result in restriction of lateral rotation. This change in rotation function of the piriformis becomes important when testing the muscle, as incorrect positioning of the patient will result in different findings.

After determining that a possible hypertonic piriformis is present, examine the strength of the gluteus maximus. Remember that this muscle may be found weak due to entrapment of the inferior gluteal nerve. Approximate the sacroiliac joints using the patient's hands, your hand and hip or a rib or trochanter belt. Retest for the findings relative to a hypertonic piriformis. First test for an increased range of motion of the femur in rotation. If the piriformis no longer needs to stay in a state of increased contraction to stabilize the sacroiliac joint, the range of motion will be increased. Alternatively, test the weak related muscles due to the entrapment syndrome. These should now test strong as the piriformis relaxes when the reason for the contraction has been reduced.

Piriformis. Leaf

The author's experience has been that the integrity of the gluteus maximus is the major initiating factor for increased contraction of the piriformis. The gluteus maximus may be found to be inhibited due to alterations in the alignment of the sacroiliac joint, stretching of the sacroiliac ligaments, entrapment of the inferior gluteal nerve, spinal subluxations, etc.. Consideration must also be given to frank disuse atrophy due to changes in walking gait patterns.

With regard to walking, the piriformis undergoes an inhibition pattern during the gait cycle. However, on heel strike the piriformis contracts to aid in the stabilization of the pelvis. This action occurs only if contact is made with the ground with the heel. If landing is accomplished with a flat foot or with the metatarsals, this stabilizing contraction will not occur.

Successful management of the piriformis syndrome depends upon diagnosis, understanding of the underlying physiological reasons for the hypertonic condition of the muscle, proper treatment of the synergistic and antagonistic muscles, and finally, specific treatment to the piriformis to correct any muscular myotonic reflexes.

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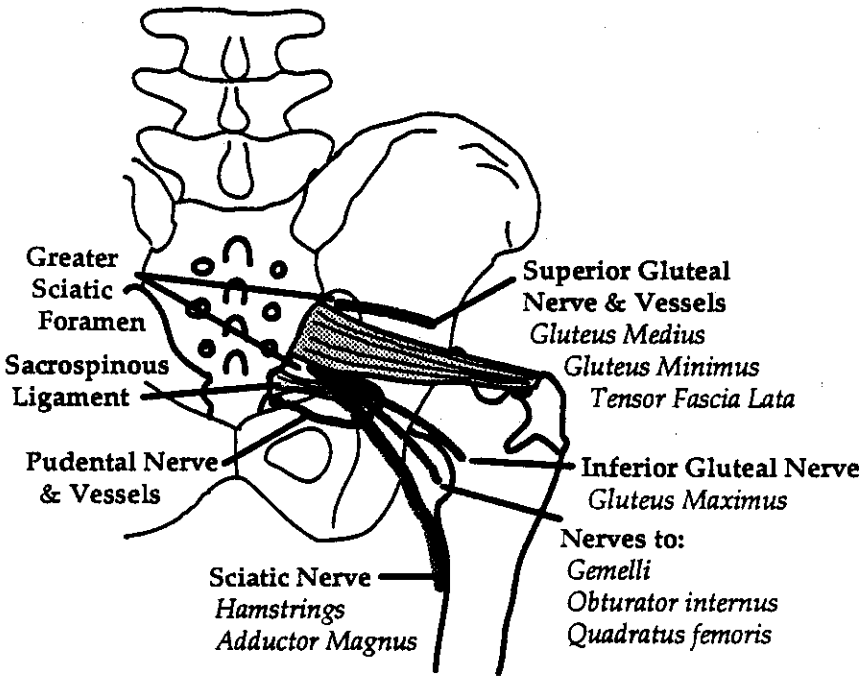

SCAR TISSUE

DYSFUNCTION REFERRED FROM SCAR TISSUE

TONSILLECTOMIES
EPISIOTOMIES

by John E. Longbottom, D.C.

ABSTRACT: Some scars from what may be generally considered 'minor' surgical procedures, cause health problems in varying degrees, from subclinical, to the other extreme of quite severe and debilitating pathophysiology.

DISCUSSION: Further to my original article (1) on scar tissue treatment, and pathologies referred from scar tissues and trauma sites, some definite patterns have become evident from patient records.

In our western society, it has in modern times become accepted as normal practice for a primipara to be subjected to an episiotomy. With many mothers, an episiotomy has been performed with every delivery.

How many females have 'not been the same' since they delivered one of their babies?

By far the great majority (more that 70%) of female patients who have had an episiotomy, need local corrections of the scar. Following local correction of that scar, it has been found, that when the patient T.L's on the scar, one or the other or both sartorius muscles will 'unlock', and the other A. — K. Tests are also positive.

Tonsillectomies have been performed with less frequency in recent years, compared with a generation or two ago.

Patients who report a tonsillectomy, are asked to T.L. just posterior to the ramus of the mandible, first on one side, and then on the opposite side. Local correction is made, first on one side, and then on the other side, using direction, and respiration, and also hyperextension of the cervical spine.

SCAR TISSUE —— LONGBOTTOM

Following local scar tissue correction, the patient is instructed to T.L. bilaterally, with the thumb on one side of the upper cervical spine, just posterior of the ramus of the jaw, and the fingers on the other side.

It has been found that 100% of tonsillectomy patients, when they T.L. on the neck, the subscapularis muscles will 'unlock', the acupuncture pulse test (heart) will be positive, as will the other K. — K. factors for heart.

(Perhaps we can coin the term 'hypocardiosis').

While gathering the statistics on 100+ tonsillectomy cases, 182 patients with tonsills intact were tested. Of these patients, 130 did not indicate positive for a heart problem when they T.L'd on their upper cervical spine. There was however 52 patients who did indicate positive for cardiac deficiency. In nearly all of these patients, their case histories revealed severe trauma to the cervical spine, eg automobile crashes, falls from horses or motor cycles, 'rolled' by a bull, surgery on a sub-lingual abscess, repairs to a cheiloschisis and uranoschisis etc.

CONCLUSION: Episiotomies are performed at the junction of the C.V. and G.V. meridians, and also may disturb the integrity of the coccygous and other perineal muscles, perhaps altering the tension on the filum terminale.

The tonsills are near 'the origins of the Superior Cardiac Branches of the Vagus nerve, which arise from the upper part of the neck, and . . . . . communicate with the cardiac branches of the sympathetic'. (2)

This may explain why all tonsillectomy patients, and many patients with severe upper cervical traumas may indicate positive for cardiac problems (hypocardiosis) when they T. L. bilaterally, just posterior of the ramus of the jaw.

These patients should all be investigated very closely, and their progress monitored

SCAR TISSUE —— LONGBOTTOM

It is believed that there is more neurological disturbing factors still to be found at the site of the scar or trauma.

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Conservative Approach to Herpes Zoster Ophthalmicus

H. Louis Obersteadt, D.C.

ABSTRACT: The case of a 53 year old male with herpes zoster ophthalmicus is reviewed. Herpes zoster virus infections are usually a benign skin disease that is often associated with the trunk of the body. However, its most common target is the VII cranial nerve. The conservative therapy using applied kinesiology technique is discussed.

Mr. C. H., a 53 year old, caucasion, male entered this office on May 1, 1991, reportedly for treatment of loss of vision in the right eye that had been previous diagnosed as resulting from a herpes zoster infection since 1982. The virual infection moved into the right eye in 1983 and as a result he has had to have two corneal transplants. The virus reportedly started in the middle of the right scapula, moved cephalad to the top of the shoulders, medially to the base of the neck, up the lateral neck to the ramus of the jaw, across the face, moving just under the maxilla, up to and across the bridge of the nose and into the right eye. The symptoms have been continuous in the right eye since 1983 with increases and decreases in severity with no apparent cause. The worst exacerbation was after the first transplant in 1983. He had a second transplant in 1990. The visual symptoms were loss of acuity, fogginess, and only able to see vague forms of movement. He could not see my chiropractic assistant that was helping with his examination. There was a constant itchy, irritation of the right eye and a feeling like "something was in the eye". At the time of the first visit, the right eyelid was closed with sutures to the midline. It is my opinion that this was done to decrease the irritation caused by hemorrhagic and necrotic changes and inflammation to the conjunctiva. There appears to be no factors which aggravate or relieve the above complaints. The right iris is much darker in the inner half than on the left. There is more redness of the sclera, the lens was hazy, and the color of the iris is more sharp on the right than the left. There is no facial scaring from the previous episode of infection.

Immediately after the first corneal transplant there was a severe exacerbation of symptoms and he was referred to another medical specialist because his treating physician felt that there was no more he could do with his condition. His current medical doctor was not aware of the patient's decision to seek chiropractic care as the patient chose not to discuss his decision to consider chiropractic care. Therefore, he did not tell him that the 50% improvement in his vision was as a result of chiropractic care and not the sewing shut of the eylid which the medical doctor felt sure was the cause for the change.

The patient is a healthy male with normal developed musculature and has had no prior history of other herpes infection. Interestingly, he did recall his mother had told him that when he had had chicken pox as a child his case was severe and that the rash had taken a long time to heal (in particular, the area between the eyes).1 Mr. C. H.'s condition is unusual

in that the infection crossed the midline in his case and usually it does not.2 Other past medical history was a benign mass that was removed from his breast in 1970 and he had a hernia operation approximately five years ago. There is no prior family history or marital history of the Herpes. He stated that he did have measles and mumps as a child. Recently, he added 2,000 square feet to his house and did most of his own work.

Varicella (chicken pox) and herpes zoster (shingles) are very different in their physical distinction, but are caused by a virus that is morphologically similar. The virus starts as chicken pox and later in life, usually when the immune system becomes suppressed, the virus will reactivate affecting approximately one percent of the population of the United States.2 The virus is also the same for herpes simplex I and II, the Epstein-Barr virus, and the human cytomegalovirus. Man is the only host for this virus and contract varicella when there is no immunity and the zoster when there is partial immunity. It is a viral disease that mainly affects the opposite ends of the age spectrum. Being more prevalent in the 50 year old and above age group.3 Patients that are treated for cancer, taking immune suppresive drugs, transplants and intravenous drug abusers have an increased incidence and severity of this disease.4 After the original infection of varicella the virus is usually found in the posterior nerve root and affects the body dermatones related to that nerve root and the fifth cranial nerve at the trigeminial or geniculate ganglion which is near the apex of the petrous portion of the temporal bone. The trigeminial neve is the most common site of all the nerve roots infected and is called herpes zoster ophthalmicus. When active the vesicles will usually appear with trophic changes in the peripheral nerve branch and the cornea with necrosis in the skin, sclera, and cornea. This usually causes the cornea to become hazy with pitting of the mucous membrane (conjunctiva) of the anterior of the eye. The trigeminial ganglion in the last changes later in life give rise to neuralgia since cutting of the posterior root does not give relief.

Mr. C. H.'s orthopedic and neurological examination findings were within normal limits. There were no obvious posture distortions and his gait was normal. Manual muscle testing revealed a full range of motion, but a lack of resistance of the following muscles. The loss of resistance could be regained by touching different body reflexes or vertebra. Muscles found to have a lack of resistance were the upper trapezius, bilateral latissmus dorsi, right tricep, bilateral medial neck flexors, bilateral gracilus, bilateral psoas, right sartorius, right posterior tibialis.

The body reflex points that changed the loss of resistance in the muscle to strengthen were the vertebra, neurovascular reflex point, neurolymphatic reflex point, acupuncture alarm points, cranial sutures and or bones. Inspirational assist challenges also caused the muscle to

strengthen.5 That is, the patient was asked to inhale and hold his breath and the muscles were tested for resistance. Specific cranial challenges were inspiration assist, sphenoid tilt, and internal frontal. Other techniques that caused changes in the muscle to strengthen was Immuno-challenging technique and V cranial nerve challenge taught by Dr. Walter Schmitt6 and Master Set Point Challenge taught by Dr. Michael Lebowitz.7

Nutrients that reversed the lack of resistance in the muscles were calcium lactate, Cataplex F, and betaine hydrochloride. All of these are from Standard Process. It's been my experience that when there is tissue irritation that these three nutrients work well together at decreasing the lesion. Since the patient had been diagnosed with a viral disease, I also used Virus from Standard Enzyme to help increase his immune system based on the homeopathic theory.

The therapeutic rationale was to treat all indicators above that increased the resistance of the muscles. On the second and third visits my intent was to use nutrients that would support the adrenals because of the specific muscles involved (sartorius, gracilus, and posterior tibialis). The fact that the patient has had eight years of stress with chronic pain, and three major surgeries. I also had intended to support the immune system with different nutrients, but the patient has responded so well to manual treatment that I did not move to that phase of treatment. His second visit was a 50% improvement in his vision, and the third visit was a 50% increase over the previous improvement. This was confirmed by his ophthalmologist and the sutures were removed after the third visit at the request of the patient.

Mr. C. H. has responded very well to conservative chiropractic care, but this does not infer that this would be a standard form of treatment for Herpes Zoster Ophthalmicus.

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APPLIED KINESIOLOGY MANAGEMENT OF COMPETITIVE AEROBICS INJURIES

ROBERT A OZELLO, D C.

Abstract: This paper summarizes the experiences of the author treating athletes at national and regional team aerobics championships.

The author had the privilege of being invited to treat athletes at two team aerobics championships. Each team consisted of ten to twelve members who compete in an intense choreographed presentation. Injuries are numerous and varied. Standard applied kinesiological methods including postural analysis, TS line and muscles testing were employed. Thirty eight women age 14-29 were treated. They were treated before and after the competition.

Most of the patients complained of foot, knee and leg pains. A smaller percentage had low back pain while a few had neck and shoulder problems.

The most outstanding finding was fixations. All had various spinal fixations as their primary causitve finding. Also most had a high incidence of psoas, quadratus lumborum and anterior tibialis strain counterstrain.

There were several cases of dehydration where the ingestion of water immediately strengthed many weak muscles. Ileocecal valve syndrome was conspicuously absent. Evidently the high physical activity level of these individuals helped colon and ICV function considerably. All patients had considerable improvement of symptoms after treatment.

One special case bears mentioning. One woman had severely injured her left foot. She complained of extreme pain in the left foot and couldn't stand on it. A podiatrist at the competiton diagnosed the problem as a stress fracture (without x-rays) and told her to see a sports medicine doctor. The patient then asked me to take a look at the foot. There was no swelling but the foot was extemely tender to the touch. I corrected several fixations which caused a slight improvement. Further examination found a weak vastus medialis on the left which required muscle spindle cell work (extremely tender to palpation), neurolymphatic and neurovascular treatment. The patient reported an immediate 90% improvement in pain and was able to walk normally.

Conclusion: When using applied kinesiological methods the doctor can confidently and effectively treat a wide variety of unusual or severe athletic injuries.

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PSOAS IMBALANCE CAUSING SEVERE HEADACHE

Robert A. Ozello, D.C.

Abstract: A patient with severe headaches was found to have a psoas strain-counterstrain causing these headaches.

A seventeen year old female presented herself complaining of severe right temporal and frontal headaches for several months. She would wake up with a slight headache and as the day progessed the headache would steadily worsen. By 11:00 AM she would have to leave school and go home to sleep. She would awaken the next morning and the cycle would repeat itself. She had previously seen her family doctor, two neurologists and an orthopedic surgeon. She had plain x-rays, an MRI, a Cat scan and an EEG. All tests including blood work were negative.

During the initial consultation she could not recall anything that caused the headaches. They started for no reason. Upon close questioning she revealed that she had slipped but caught herself shortly before the headaches started.

Examination revealed multiple TS line indicators especially on the right. The patient was checked for fixations. An occipital, upper cervical, lumbo-dorsal and sacral fix was found and corrected. The patient then reported that the headache was completely gone. She was told to return the next day.

The next day the patient reported that she felt good the rest of the previous day but the headache returned in its usual fashion and severity the next day. Over the next several visits other problems including TMJ, spheno-basilar fault, anterior C5, and re-ocurring fixations were diagnosed and treated using standard applied kinesiology procedures. The patient always felt much better immediately after the treatment but the headaches always returned the next day. The headaches disappeared permanently when a left psoas strain-counterstrain was corrected. The headaches have not returned in two years.

Evidently the psoas strain-counterstrain was causing a sacral fix which weakened the neck extensors bilaterally. This got worse as the patient moved around during the day. The neck extensor weakness made the cervical spine and cranium unstable and irritated the skull where the neck extensors attached causing the headaches.

Conclusion: This case shows the importance of correcting structural imbalances using standard applied kinesiology methods and keeping a open mind when diagnosing and correcting health problems.

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1. Walther, David S. Applied Kinesiology Synopsis, 1988; Systems DC, Pueblo , Colorado, pp. 191-194

FUNCTIONAL CATEGORY II

William S. Seplow D.C.P.A.

ABSTRACT

We are all familiar with the concepts of Category II interrelationships of the adrenals and their nutritional patterns as well as the relationship of the sartorius, gracilis and tibialis posterior muscles. We too are aware of the momentary postural changes reflecting sluggish adrenal function such as postural hypotension. This paper will review a way to use old knowledge to discover new information.

PROCEDURE

Observing that there was momentary postural hypotension in most of my Category II patient, I began to ask myself why after the Category II symptoms were clear and the patient no longer showed positive therapy localization or challenge for Category II did the postural hypotension still present. Some of the patients still exhibited some Category II symptoms but would not test positive with EID, BID, weight bearing or sitting and were negative for Category I and Category III. Then one day I had a patient assume a standing posture and she immediately therapy localized to the sacroiliac joint and it was positive for Category II. I explained to her that this was a weight bearing test and that's why it was different than when she had been lying prone and supine and while she was standing I tested her again to show her. This time it was negative, so I put her the same sequence of events of first being supine for at least 1 minute, then therapy localized within 10 seconds of her assuming a standing posture and she was again positive. We waited 30 seconds then she therapy localized again and was then negative. I knew we had discovered something so we began to test many patients like this. What we discovered was, that all the Category II adrenal factors would show in this type of patients during the first 10 or 15 seconds of postural stress. We found the need for copper as well as adrenal substance. I felt from listening to Dr. Goodheart's lectures that the copper had something to do with the electrical potential created that allows the muscular walls of the greater arteries to contract under adrenal influences. This approach has provided a more complete restoration of health for our Category II adrenal patients.

SUMMARY OF PROCEDURE

1. Eliminate possible Category I or Category III.
2. Have the patient rest in a supine position for at least one minute.
3. Have the patient therapy localize in prone, supine, sitting, EID, BID, etc., to further eliminate hidden Category I or Category III.
4. Then have the patient assume an upright posture and therapy localize the sacroiliac joint and test the indicator muscle within 10 seconds. (It helps to explain to the patient before you do the test, so that no time is lost).
5. Test any other factors in the same fashion and treat appropriately with five factor approach.

6. I have had success with copper material provided by Biotics Research and Drenamin provided by Standard Process as well as Cyto AD provided by Biotics Research.

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GALLBLADDER PROBLEMS AN OVERVIEW

By: Paul T. Sprieser, B.S.,D.C.

Abstract: A review of physiology and pathology of this organ.

Definition: The gallbladder is a small sac on the inferior surface of the liver.

Bile is secreted by the liver continuously and is stored in the gallbladder and concentrated there.

The gallbladder does not empty till a specific stimulus that is initiated by the presence of fat in the small intestine.

Fat in the small intestine will cause the mucosa to secrete a substance called cholecystokinin, which is absorbed by the blood and goes to the gallbladder causing it to rhythmically contract.

This alone is not sufficient to empty the gallbladder because the sphincter of Oddi which constricts the common bile duct, which enters the duodenum.

This sphincter remains tonically contracted despite the gallbladder contractions. But the peristaltic wave passing over the duodenum causes the sphincter to relax allowing a small squirt of bile to flow into the lumen.

The release of bile is therefore the result of both the production and stimulation of cholecystokinin and the acton of peristalsis on the sphincter of Oddi.

BILE:

All hepatic cells are continually forming bile which reach the hepatic duct from many smaller canals called bile canaliculi.

The hepatic duct join with the cystic duct to form the common bile duct.

The gallbladder is filled by the continuous production of bile that flows down the common bile duct and is blocked by the sphincter of Oddi, which backs up the bile to the cystic duct, which in turn flow back to fill the gallbladder.

The liver produces 600 to 800 ml. of bile a day. The maximum volume of the gallbladder is only 40 to 70 ml.

At least 12 hour production of bile can be stored in the gallbladder. This is done by the mucosa of the gallbladder continually absorbing water, sodium and chlorine.

This action causes the concentration of the bile constituents,
which includes the bile salts, cholesterol, and bilirubin. The bile is normally concentrated about five fold, but it can concentrate up to
ten fold.1

COMPOSITION OF BILE

	Liver bile	Gallbladder bile
Water	97.5 gm%	92.0gm%
Bile Salts	1.1 gm%	3 to 10 gm%
Bilirubin	0.2 gm%	0.6 to 2.0 gm%
Cholesterol	0.1 gm%	0.3 to 1.2 gm%
Fatty Acids	0.12gm%	0.3 to 1.2 gm%
Lecithin	0.04gm%	0.1 to 0.4 gm%
Na	145 m Eq/l	130 m Eq/l
K	5 m Eq/l	9 m Eq/l
Ca	5 m Eq/l	12 m Eq/l
Cl	100 m Eq/l	75 m Eq/l
HCO	28 m Eq/l	10 m Eq/l

FORMATION OF BILE SALTS

The main constituent of bile is cholesterol which may come form diet or produced for the liver through fat metabolism and then converted to cholic acid.

This will combine with glycine to form glycocholic acid and to a lesser extent with taurine to form taurocholic acid. These acid salts are secreted in bile.

ACTION

There are four important actions of bile in the alimentary canal.

1. A detergent action on fat particles. This causes a reduction of the surface tension thereby allowing agitation of the intestinal tract to split the fat globules into smaller size. This is know as emulsification or detergent action.

2. The second action which is even more important then the emulsification action, bile salts help the absorption of fatty acids and monoglycerides known as hydrotropic function.

It is believed that the bile salt ions cause a negatively charge to occur to the fatty acids which increase the solubility of the fatty acid which allow for easier passage through the intestinal mucosa.

Without the bile salts present a great portion of the fatty acid would be lost in the stool causing a soap to be formed and a loss of the fat soluble vitamins.

Since the fat soluble vitamins are A,E,D & K. We can see the general effect the vitamins have on the body in area such as skin

and mucus membrane, bone formation and calcium distribution and utilization. Vitamin K which has it connection to prothrombin can lead to serious blood coagulation problems.

3. Neutralization of acidity of the chyme from the stomach because bile has a pH slightly above seven.

4. Excretion is an important vehicle for removal of cholesterol, bile acids, drugs, toxins, bile pigments, various inorganic substances such as mercury, copper, and zinc.1,2

COMPOSITION OF HEPATIC AND GALLBLADDER BILE

	% Total Bile	% Total Solids	Bile GB
Water	97.0		85.92
Solids	2.52		14.08
Bile Acids	1.93	36.9	9.14
Mucin & Pigments	0.52	21.3	2.98
Cholesterol	0.06	2.4	0.26
Fatty Acid	0.14	5.6	0.32
Inorganic Salts	0.84	33.3	0.65
Specific gravity	1.01		1.04
pH	7.1-7.3		6.9-7.7

The liver synthesizes bile from cholesterol but the exact mechanism is not completely understood.

Cholic and chenodeoxycholic acid are 2 primary bile acids formed in the liver in a ration of 2:1 and make up about 80% of bile.

These bile acid are conjugated in the hepatocytes with glycine and taurine.

In a fasting state about 50% of the bile produced flows to the gallbladder by the cystic and the rest to the common bile duct.

These bile acids are poorly absorbed by passive diffusion in the upper part of the small intestine. However, when the chyme containing the bile acid reach the terminal ilium, about 90% of the bile will be reabsorbed into the portal venous circulation by active transport.

This allows the liver to retrieve these used bile salts and remodify them and excrete them again as bile.

The total body pull is about 3 to 4 gm. of bile acids, that undergo an intrahepatic circulation (retrieval) about 10 to 12 times a day. During these pass through the colon anaerobic bacteria containing 7 gama-hydroxylase form a secondary bile acid. Cholic acid is converted to deoxycholic acid, this acid is mainly reabsorbed and conjugated, and acts as a precursor. Chenodeoxycholic acid is conjugated in the colon to their secondary bile acid form, lithocholic acid. This is a insoluble bile acid that is partially reabsorbed and the rest is lost in the feces.

These bile acid give the feces part of its normal color.2

MALFUNCTION OF THE GALLBLADDER

Most extrahepatic disorders of the biliary tract relate to gallstones, which have a great prevalence in women an also certain ethnic groups (eg. North American Indians) and also have an increased occurrence with age. It is estimated that about 20% of person living in the U.S.A., over the age of 65 have gallstones and about 300,000 will undergo removal of the gallbladder each year. Other facts that seem to increase the likely hood of developing gallstones include, western diet, obesity, and family history of gallstones.

As much as 90% of water in the gallbladder bile is removed an a electrolyte solution though the mucosa of the bladder, making the bile that remains in the gallbladder a concentrated solution of bile acids and sodium.

When a fatty meal enters the duodenum it will cause the release of a large portion of the total body pool of bile acids into the small intestine.3

PATHOPHYSIOLOGY

The major component of most gallstones is cholesterol, which is highly insoluble in water. Cholesterol is usually incorporated into micelle which make it tremendously solubilized and can be excreted in bile.

Note: The micelles of bile salts are constructed of a water soluble (ionic hydorphilic) region of the molecule which faces outward into the aqueous solution, and a insoluble (nonpolar) portion (hydrophobic) steroid nuclei which faces inward. The cholesterol is on the soluble interior of these molecule which forms a spheroid micelles, the cholesterol carrying ability is enhanced by the addition of lecithin, a polar phospholipid (phosphotydalcholine).

Stone formation is believed to be caused by the supersaturation of cholesterol in native bile solution, however, this is not the only cause, since supersaturated bile is frequently found in fasting person that do not have gallstones.

Other factor that must be taken into account in initiating stone formation is cholesterol monohydrate crystal.

In gallbladder bile the lithogenic factors, of supersaturation of cholesterol and a rapid nucleation of cholesterol crystals. There are other forces at play that may prevent nucleation, which includes specific proteins, or apoproteins, gallbladder stasis, and gallbladder mucin.

Almost all stones that are formed occur in the gallbladder, however, there are some rare occurrences with formation in the biliary duct due to strictures that cause stasis or form scar tissue due to cholecystectomy.1,3

SIGNS AND SYMPTOMS OF GALLBLADDER MALFUNCTION

Many patients with stones in the gallbladder may remain asymptomatic for long periods of time, or frequently for life.

Stones can traverse the cystic without pain if they are small enough and these can pass down the common bile duct and enter the small intestine.

Symptoms will occur when a stone migrates and becomes obstructed in the cystic or common bile duct. This does not occur in more then 50% of the cases.

Obstruction of the common duct (choledocholithiasis) occur in 10% to 20% of patients with gallstones, which will cause biliary colic, jaundice, inflammation of the bile duct (cholangitis), or pancreatitis.

Statically only about 20% of persons with asymptomatic gallstones will develop symptoms over a 20 year period.

If symptoms due develop the initial one is usually uncomplicated biliary colic rather than acute cholecystitis or choledocholithiasis.

This fact leads us in Applied Kinesiology to be of great service.4

DIAGNOSIS OF GALL STONES (SYMPTOMS & SIGNS)

Transient cystic duck obstruction results in colicky pain, whereas a persistent obstruction usually cause inflammation with acute cholecystitis.

Most obstructive occurrence are transient, and produce biliary colic lasting up to several hours.

Pain location vary some what, but most often it will be in the epigastrium or upper right quadrant, with radiation to the right lower scapula. The pain is usually constant, with a rising intensity to a plateau with a gradual fall. Nausea and vomiting are common. Fever and chill will not be present if the gallbladder colic is uncomplicated.

Stones that pass out of the cystic duct may pass down the common bile duct with out symptoms into the small intestine. If they lodge in the sphincter of Oddi, there maybe obstruction of both the common bile duct or Wirsung pancreatic duct where they join together at the ampulla of Vater. If this occur much more serious consequences will develop. Not only is pain present, but jaundice, pancreatitis, or infection (cholangitis).

These serious complication are rare on the first episode of colic.3,4

Other common symptoms of (dyspepsia) indigestion, and intolerance of fatty food, belching, bloating, fullness, or nausea may occur but should be differentiated from other digestive malfunctions such as peptic ulcers.

Diagnostic techniques that are available may include ultrasonography which is non invasive and method of choice for gallstones. Another method is a oral cholecystography this technique is about 95% effective. It use a compound that is iodinated called Iopanoic acid with x-ray. Other more invasive test are available but should not be first choice.

Applied Kinesiology techniques that can be helpful in detecting gallbladder pathology are Therapy Localization over the gallbladder region and T.L. to the gallbladder alarm point at the costal cartilage of the ninth rib which is also a common point of clinical pain in gallstone problems. Another method can be

scratching or pinching of the refereed pain area for gallbladder problems and see if the associated muscle weakens. This response is more indicative of actual gallbladder disease.

Muscle weakness patterns that might indicate gallbladder pathology would first be weakness of the popliteus muscle, remembering first to rule out fixation. Weakness of the Pectoralis sternal division if the common bile duct had become obstructed. If pancreatic involvement is present then weakness of the latissimus dorsi and triceps might be expected. However these must be substantiated with other laboratory test or x-ray findings.

Blood chemistry that might be found to be involved would be raised serum levels of bilirubin, alkaline phosphatase and transaminase (SGOT OR SGPT) and (aminotransferase) elevated in 30 to 40 % of patients. If infection of gallbladder is present elevated WBC usually 10,000 to 15,000 with a shift to the left might be expected.

Other physical signs are Murphy's sign pain during palpation of the upper right quadrant and inspiration arrest it. Boas sing is less common which is tenderness in the right scapula region.

Other possible reason for stone formation that we need to consider for the conservative mean of treating gallbladder problems. If these method are successful in having the patient passing the stone or if they are dissolved by medical treatment with chenodeoxycholic acid (chenodial) or ursodeoxycholic acid (ursodial) for noncalcified cholesterol gallstones. We must find a way of preventing further formation and recidivism of this problem.3,4,8

We must consider these factors that stasis of bile in the gallbladder can be caused by a low pH of the acid production in the stomach. This may lead to less responsive release of cholecystikinin (CCK) because of the reduced pH of the chyme entering the duodenum and perhaps less active peristalsis actin of the small intestine, which may well cause less active release of the bile form the common bile duct.

Another reason that possible associates with stone formation may be Dr. Goodheart observation of Isogai gait length that associates with the change of polarity seen on the tongue.

To refresh your memory Dr. Goodheart in 1980 observed that when a nutrient was insalivated it would strengthen a weak muscle if it was associated to it. If the patient had a gait abnormality in length of stride. If the nutrient was placed to the right or left of the mid line of the tongue, one side would cause a strengthening of a weak indicator muscle but when placed on the other side of the mid line, it had no effect.

Dr. Goodheart hypothesized that the tongue was the first part of the alimentary canal this was an indicator that the polarity of the brush cell border of the small intestine may also be changed.

This fact could explain why some patient have to take mega dosages of vitamins to get any absorption. If the intestinal brush cell border polarity had been altered from normal it may repel what should be easily absorbed if this condition did not exist.7

ADDITIONAL FACTORS FOR PREVENTION OF GALLSTONES

1. Over weight person especially women have a great tendency to develop gallstones. So a reduction of at least 20 Kg or 44 lbs., in one to four months is recommended.

2. Changing of drug that may be used by the patient should be considered. The drugs that have been implicated in gallstone formation are estrogen, oral contraceptives. An even new medical procedure was developed at Mayo Clinic using a catheter device to deliver a new compound called Methyl tertiary butyl-ether (MBTE) can dissolve a large stone in 17 hours.

3. Nutritional treatment that can be used both to treat the gallstone or help in passing it are as follows.

a. Betacol (SPL) is used as a liver detoxifier due to a sluggish liver activity. This product helps the liver remove toxin that have accumulated from the bowel such as guanidine. This product also contains the Wulzen Factor (anti-stiffness factor) stigmasterol. This product is also helpful in biliary stasis with thick bile.

b. Other benefits can be derived for A-F Betafood (SPL) along with iodine.

4. Avoidance of fatty food (especially ones with saturated fats present), fried foods as well as chocolate, coffee and refined carbohydrates.

Treatment methods that seem to help this problem was suggest by Dr. Walter Schmitt in a paper he present in the ICAK winter paper, 1981. This paper described the use of Ligament Interlink technique to eliminate a positive Murphy's sign.

The relationship was the fact that the gallbladder was associated with the popliteus muscle, which is a posterior knee muscle.

Ligament Interlink is a shared neurological relationship to contralateral joints, in this case knee to elbow.

Have the patient TL (hold) the elbow and test a strong muscle it should be negative then have the patient TL the opposite knee and test a strong indicator muscle it too should be negative. Now both the elbow and the opposite knee are TL simultaneously and tested against an appropriate muscle it will now be weak.

While the patient holds the positive ligament interlink pattern we challenge the hyoid bone and see what direction neutralizes the weakness. The treat that joint on the side that the hyoid challenged positively. The treatment is a heavy meading pressure to the ligaments of the involved joint. After which, we go back an rechallenge Murphy's sign. Treatment is continued till pain is reduced at least 80% or better.

It is also a good idea to treat the Neurolymphatic reflex for the gallbladder which is it the fifth intercostal space on the right.5,6

DISCUSSION:

As chiropractors we have all heard the term "Gallbladder Flush", this was supposed to be a conservative method of removing gallstones from the gallbladder.

This procedure consisted of having the patient consume a half cup of pure olive oil mixed with half cup of lemon or grapefruit juice about a half hour before bed. This is supposed to cause the gallbladder to reflexly contract emptying it contents along with

any stones that may be present. This method very definitely will do just what its name suggest.

However you should remember this might cause a medical emergency for the patient even requiring surgical intervention to prevent permanent injury or even death. If you are going to suggest a gallbladder flush be sure you know exactly the type of stones present and there size so you can be sure that the will likely be able to pass through the common bile duct and the sphincter of Oddi, without causing an obstruction.

CONCLUSIONS:

If conservative methods can be used to treat gallstones it is necessary for the doctor to know the size and type of stones present, and whether they can be passed safely through the common bile duct and sphincter of Oddi. This would mean that proper diagnostic tests be done before the "Gallbladder Flush" is attempted.

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Bilateral Muscle Inhibition an Alternative Correction

by

John F. Thie, D.C.

Abstract: A discription is given for correction of bilaterally inhibited muscles as an alternative to fixation subluxation adjustments

The generally accepted method for correcting bilateral muscle weakness in an Applied kenesiology type of muscle examination described by Goodheart,Walther, Stoner and others has been to correct a fixation subluxation. The reason for the bilateral weakness has been generally thought to be a fixation in a group of three vertebrae. The the restoring of facilitation has been demonstrated to be a special chiropractic adjustment of the spine by hand. These chiropractic adjustments for the fixation subluxation did indeed correct the bilateral muscle weakness, in most cases. Conable showed in a paper presented to the ICAK that the description of the correction was not the same by different authors.

I have found that another method of correction which has been effective for me and others that have been shown this method.

The method is very simple and can be used safely by the patient at home having a member of the family or friend help him/her by testing the previously found inhibited muscles and applying the reflex correction methods. The bilateral weakness problems which I have found, tend to recur with fatigue of the patient regardless of the original method of correction and strengthening of the muscles, that is fixation adjustment or the method I will describe later in this paper.

The spinal fixation subluxations did not appear to follow the spinal nerve pathways to the muscles that were involved in the pattern of subluxations. The fixation of the upper cervicals were discovered by a bilateral weakness of the gluteus maximus muscle and bilateral hamstrings were associated with fixation of the occiput. My observation also indicate some other pathway of communication seems to be envolved in at least some of the situations where bilateral weakness are discovered by manual muscle testing.

When I was observing Bruce Dewe, M.D. making corrections for a bilateral psoas weakness, I observed that he did not use an osseus thrust but just moved the skin on the occiput with respiration in the direction of the indicated by evaluating the tongue stress procedures. I experienced this correction on myself and found that I had at least as much benefit from this correction as I had from rapid thrust adjustments of the occiput for the correction. In my own personal experience of the bilateral psoas muscle weakness and in patients I have examained this alternative correction is very more effective than the rapid thrust of the occiput. I have since advocated this method other and they also have found that the light pressure moving the skin is very effective and very safe.

I then attempted a similar correction of moving the skin over the spinus processes in a headward to a footward direction for other bilateral weaknesses when I found them in my patients. I have used this procedure now for four years and have found that is is very effective and much easier to correct the problems. The indicators for fixation subluxations are usually gone following the correction and retesting of the bilateral muscles and finding the weakness abolished.

Not every time did I find both muscles of the previously inhibited muscles strengthened by the movements of the skin in a repeated cephalad-caudal motions, sometimes a unilateral muscle weakness would remain and would be correct by neurolympathic reflex massage or other reflexes such as neurovascular, meridian tracing or golgi tendon/spindle cell methods. Occationally I found it necessary to use more than one or more of the other reflexes to complete the corrections.

I felt that my patients and others needed to know how to help themselves and test the muscles so I worked out a mapping

procedure whereby I have been able to map all the muscles and their spinal areas that I have used in my book Touch for Health.

The procedure I found is that a lateral motion over the spinus tips of the appropriate vertebra(e) will inhibit bilateral muscle function and that vertical motion moving the skin will facilitate the same muscles if bilaterally weak. The lateral movement of the skill will not weaken muscles unilaterally in my experience.

I would be interested in any other findings that members of the college have regarding bilateral muscle weaknesses.

The accompanying chart taken from my manual Touch for Health gives the location of the vertebrae that I have found to be associated with the various muscles. These are also listed on the TFH reference chart and the TFH Folios that have been revised early in 1992.

Conclusion: A method which corrects bilateral muscle inhibition, which can be utilized by paraprofessionals and lay persons as well as professionals in the course of their manipulation of patients is an important addition to applied kinesiological therapy. This technic needs to be further studied to determine the neurological, or other mechanisms which would cause this phenomina to occur. The orginal premise of fixation subluxation being the one cause of bilateral muscle weakness is expanded by these observations.

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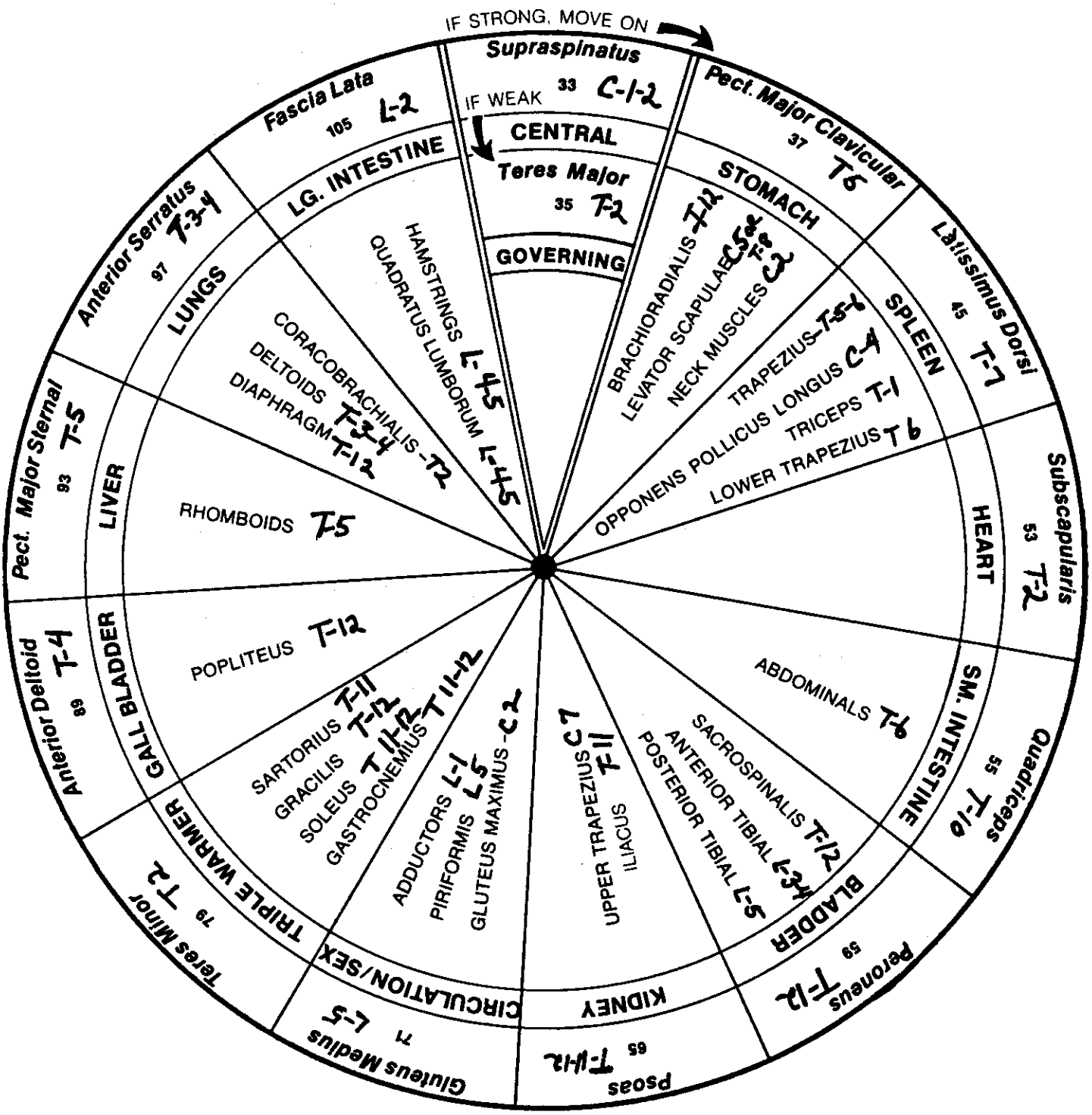
Bilateral Muscle Weakness Correction-
Thie, D.C.



KIM VIEIRA/ALICE VIEIRA

DIVISION II - CRITICAL REVIEW PAPERS

EVALUATION OF COMPLEMENT FACTORS USING APPLIED KINESIOLOGY AS A METHOD OF DIAGNOSIS

Michael D. Allen, DC, NMD

ABSTRACT: The complement system is recognized as a major effector mechanism for many of the biological activities of the humoral immune system. There are two pathways to the complement cascade. The classical pathway is important in amplifying the effects of specific antibodies in host defense against bacteria, viruses, and parasites. The alternative pathway of complement (AKA "APC"; the "properdin" system) activation is less efficient, yet it phylogenetically antedates the specific antibody system. The complement system can interact with certain microorganisms and provide a first line of defense before specific antibody is synthesized. Of all its eleven factors, C3 is the most abundant and important component of the complement system. The APC is the most important avenue of protection against gram negative, gram positive and spirochete infection. The production of the C3 factor is discussed in light of the treatment of Lyme disease (Borrelia burgdorferi).

INTRODUCTION

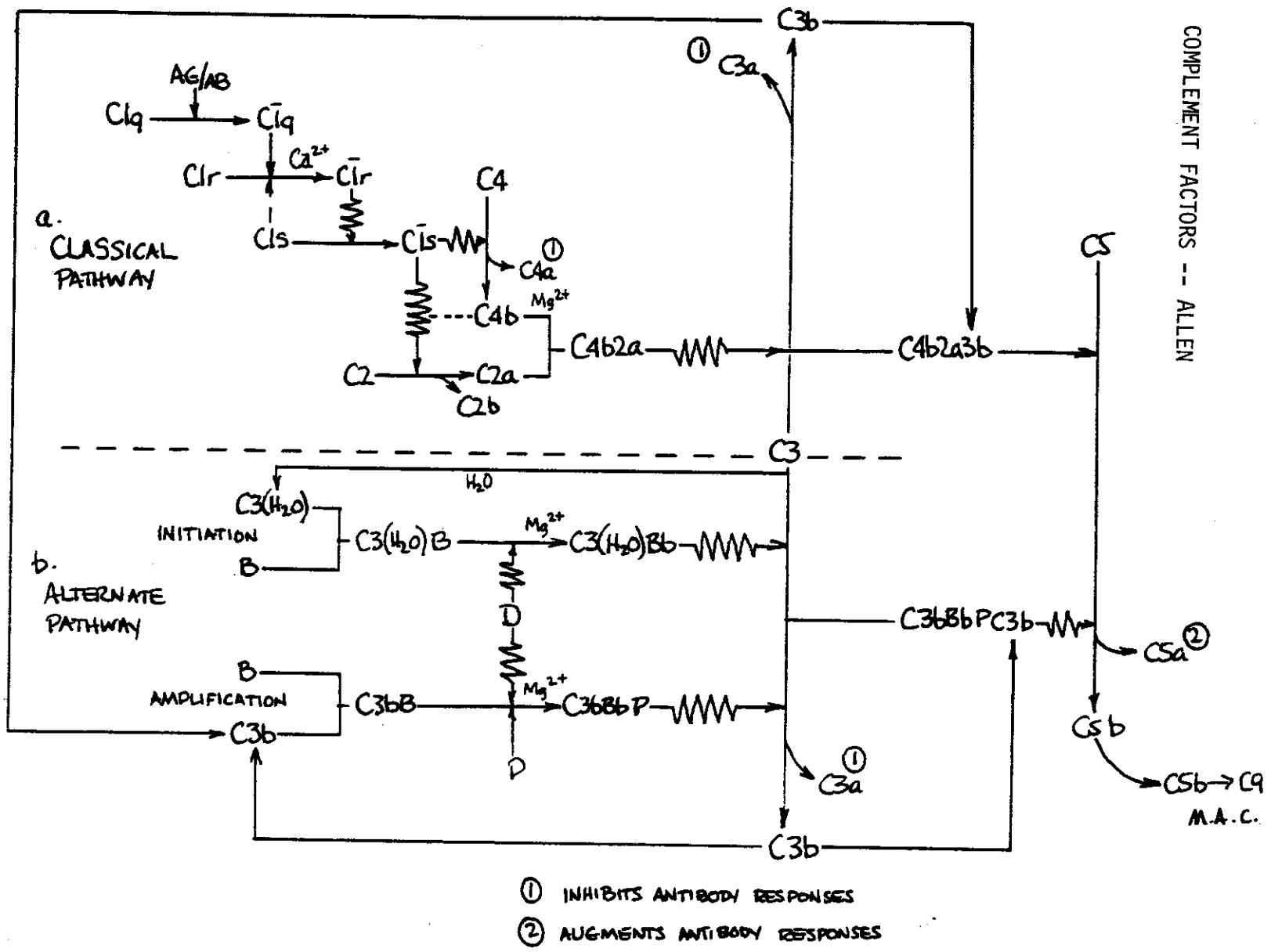
There are four major plasma mediator systems: The coagulation, fibrinolytic, kinin and complement systems. Together, they constitute the contact activation system. The complement system is the focus of this paper.

An assessment of the serum complement system can be useful in the evaluation and management of patients presenting with various clinical manifestations which may be produced by the generation of, or through the deficiency of, the various biologic activities inherent in the complement system.

The complement system plays a major role in host defence. It comprises a group of self-assembling plasma proteins which protect against infection and mediate immunopathologic inflammation.

THE COMPLEMENT SYSTEM

The complement cascade is activated on cell membranes or antigen-antibody complexes. The components are able to increase vascular permeability and attract leukocytes. They immobilize the complexes at sites of inflammation, stimulate phagocytosis and promote the formation of a cytolytic complex.



· PATHWAYS OF COMPLEMENT CASCADE ·

COMPLEMENT FACTORS -- ALLEN

Nomenclature

The constituent proteins are termed "components", and each is given a number prefaced by the symbol "C". The proteins of the classical pathway are generally numbered in their order of interaction, from C1 through C9, with two exceptions. C1 is a calcium-dependent complex of three independent proteins which were subsequently labeled C1q, C1r and C1s. C4 was the fourth component to be discovered but subsequently was found to interact second in the classical pathway sequence; however, its historical enumeration as C4 was retained. Therefore, the reaction sequence in order of activation is C1q, C1r, C1s C4, C2, C3, C5, C6, C7, C8 and C9.

The proteins of the APC are called "factors" and each is represented by a letter, e.g., factor B, factor D, properdin (factor P). These are usually abbreviated to B, D and P, respectively. (1)

DISCUSSION

Classical Pathway Activation

The classical pathway is activated following the binding of antibody to antigen. There are two separate complement pathways, each leading to the cleavage of C3. The first to be recognized was the classical pathway, although it is phylogenetically more recent. It consists of components C1, C4 and C2, (see figure) and are responsible for the formation of the C3 activating enzyme C4b2a (AKA C3 convertase).

The C1q subunit is the recognition unit of C1. It is activated in the presence of antigen-antibody complexes which contain antibodies of the IgM or IgG classes. The reaction proceeds through three glycoprotein molecules C1q, C1r and C1s held together in a calcium-dependent complex.(2)

The mechanism of activation of C1 is only partially understood. From this humble introduction proceeds a snowball effect, the most important part being the process of C3 conversion and the involvement of properdin.

C3 is the most abundant protein of the complement system. It is split into its component parts by C3 convertases (C4b2a of the classical pathway, or C3bBbP of the APC) resulting in the release of C3a and C3b. The C3a is an anaphylatoxin capable of degranulating mast cells and basophils and of contracting smooth muscle. It will not be discussed further.

At this point in the reaction, it is critical for C3b to bind promptly to a suitable acceptor molecule such as certain polysaccharides or certain cell surfaces, which partially protects the C3b from the action of certain inhibitors. Failure to attach to the activator causes the C3b to remain inactive through the function of factor H, but the details of this process are not germane to this discussion. This binding allows the APC to target its biological functions at a variety of biological surfaces, such as basement membranes and notably surfaces of pythogenic micro-organisms.

Alternative Pathway Activation

The second and probably the evolutionarily older and more primitive of the two pathways is the APC. Activation of the APC is concerned with the disruption of large insoluble immune complexes, while the classical pathway prevents the formation of large insoluble antigen-antibody lattices.

Activation of either pathway results in the formation of convertase enzymes which activate C3 and C5, with subsequent assembly of the multimolecular membrane attack complex from the rest of the cascade. Once the C4b2a from the classical pathway has cleaved C3 into C3a and C3b, the C3b binds with factor B to form C3bB -- a molecule which expresses weak C3 convertase activity (3). The binding sites for this reaction are magnesium-dependent. Bound C3b plays a central role in complement activation and in the expression of its biological activities. It is now able to cleave more C3 through its reaction with factors D and P (see figure). Both are important to the formation of the alternative pathway C3 convertase discussed below.

C3 Convertase

Four proteins, C3, B, D and P, are involved in the assembly of alternative pathway C3 convertase. The presence of an antibody is not essential for APC activation. It occurs in the presence of repeating polysaccharides and other polymeric structures such as those found on the surfaces of microorganisms.

In the presence of C3bB, factor D can split factor B into its component parts (Ba and Bb). The Ba fraction does not enter into this discussion. The Bb fraction attaches to the C3 convertase enzymatic site, attaching Bb to C3b. This results in the formation of the alternative pathway C3 convertase, C3bBb.

C3 is quantitatively the most abundant complement protein, and its very important biologic properties place it in a pivotal position

in the complement sequence. C3 is the focal point of the complement system, being a part of both the classical and the alternative-amplification pathways.

Amplification of C3 Convertase

The classical pathway depends on both calcium and magnesium, whereas the APC is only magnesium dependent. Therefore, the classical pathway can be blocked, but the APC can still be activated in the presence of the chelating agent which binds calcium more strongly than magnesium.

The amplification of C3 convertase is the hallmark of the APC. Its formation depends on the availability of C3b which, regardless of its mode of generation, is able to interact reversibly with factor B provided magnesium ions are present.

The positive feedback mechanism of the pathway is dependent on C3b and serves its amplification. Each newly generated C3b molecule has the potential to form a new complex proteinase with factor Bb. C3b molecules may be generated through the APC, the classical pathway, or the action of conventional proteinases which could be released from cells participating in inflammatory reactions. Such reactions may thus be able to recruit the biological activities of the complement system.

The Role of Properdin

Properdin, although not an essential factor for the functioning of the APC, P prolongs the half-life of the alternative pathway convertase C3bBb, in a dose-dependent fashion by retarding the decay of Bb from the complex.(4) Properdin which exists in an inactive form in serum becomes activated upon bonding to C3bB and stabilizes the alternative-pathway C3 and C5 convertases. Apparently, this activation is the result of a conformational change in the molecule.

The properdin-stabilized enzyme (C3bBbP) can cleave another C3 component to release C3a and leave C3b. Thus C3b, a product of the enzymatic action of C3 convertase on C3, is itself a constituent of the alternative pathway C3 convertase. This produces a positive feedback loop which, in the absence of control, could theoretically continue until the supply of C3 or B is completely consumed.

From this pair of factors -- the classical and alternative pathways; C4b2a and C3bBbP respectively -- the remaining and terminal components of the complement cascade can be produced.

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One of the main functions of alternative pathway activation is to protect non-immune individuals against bacterial and perhaps other infections.

SYNTHESIS OF COMPLEMENT PROTEINS

A number of cell types are known to produce complement proteins, although the sites of synthesis of certain components are still far from clear. It appears, however, that the hepatocyte is the liver cell which is responsible for the synthesis of the majority of the complement proteins.(5-11) Epithelial cells of the genito-urinary and gastrointestinal tracts synthesize functionally active C1 and its subcomponents.(12)

C3 Deficiency

This is a rare abnormality. It may be due to reduced production of C3 or increased catabolism. Since C3 is important to the functioning of the classic and the alternative complement pathway, a deficiency leads to severe pyogenic infections.(13)

C3 is the cornerstone of the complement system. It is an essential component of both the classical and alternative activation pathways and is necessary for initiation of the membrane attack pathway. Despite its central role, several individuals have been described who completely lack C3. For reasons which are not clear, C3 deficiency, unlike other autosomally inherited complement deficiencies, is not equally common in males and females: over 80% of the reported cases of C3 deficiency have been in females.

There are more than a dozen reported cases of homozygous C3 deficiency in the world literature. As might be expected, an increased tendency to bacterial infections is a prominent feature of the symptomatology, especially infections with encapsulated organisms. (14-17)

Factor B Deficiency

Complete deficiency of factor B is extremely rare, suggesting that alternative pathway activation of C3 may be of greater biological importance than the classical activation pathway.

Properdin (Factor P) Deficiency

It is interesting to note that of the three proteins specific to the alternative pathway, factor B, factor D and properdin, complete deficiency has been described only for properdin. However, no

genetic variants have been described thus far. It is also possible that factor P gene expression is under some form of hormonal influence.

Unlike all the other autosomally inherited complement deficiencies, properdin deficiency is transmitted as an X-linked recessive trait. Thus all deficient individuals so far reported are male. Until recently properdin deficiency was considered to be very rare. However, recent reports particularly from Scandinavia and Holland suggest that it may not be uncommon. Males with properdin deficiency typically present with meningococcal infections which can be fulminant and life-threatening. These individuals have an intact classical pathway and on second encounter with the organism can mount a normal response.

Factor D and B Deficiencies

Factor D is responsible for cleavage of factor B. Deficiencies of factor D as well as factor B has not been found, suggesting that these two complement components may have a direct influence on viability or some role in embryonic development.

CLINICAL USE OF COMPLEMENT ASSAYS

Elevated levels of complement synthesis can be found in many inflammatory disease states. These observations are no more helpful diagnostically than other measurements of acute-phase reactants, such as sedimentation rate.

Most of the clinical interest in assays of serum complement component levels relates to reductions of these levels in various diseases. Serum levels of complement components can be reduced as a result of decreased synthesis or of increased consumption, usually related to activation.

Decreased or absent synthesis of individual complement components may be due to a genetic deficiency of the synthesis of the component. Increased breakdown of complement proteins can occur through activation of the alternative or classical pathway, most often associated with circulating immune complexes; or increased catabolism of complement proteins may be secondary to a genetic deficiency of a regulatory protein such as C1 esterase inhibitor or C3b inactivator.

For most clinical purposes, a reasonable assessment of the status of the complement system can be achieved by combining a functional total hemolytic complement determination with immunochemical determinations of C3, C4 and factor B. Occasionally, assays for

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C1, C1q, C1 inhibitor and C5 may be useful. As functional assays for alternative pathway activation become more widely available, they will probably be useful in screening for potential host defense deficiencies.

The most common and clinically the most important genetic defect of complement is hereditary angioedema. Genetic deficiencies of each of the individual complement components of the classical pathway have been reported.

It was found that persistent positivity of the C1q binding assay in patients with Lyme arthritis was associated with a higher incidence of neurologic or cardiac complications and that persistent inflammatory arthritis was associated with high titer of C1q binding materials in the synovial fluid. Thus, persistent immune complexes in patients with Lyme arthritis might identify a group of patients for whom more aggressive therapy would be beneficial.(18) However, there is not yet a standard from laboratory to laboratory, and their clinical usefulness is not entirely clear at the present time.

LYME DISEASE

Lyme disease, transmitted through the bite of a tick infected with the spirochete, Borrelia burgdorferi, exhibits a variety of symptoms. Inflammation around the tick bite eventually causing skin lesions, erythema chromicum migrans (ECM), is the first stage of disease. Lyme disease has long been recognized throughout Eastern and Western Europe, and in the Soviet Union, China, Japan, Australia and North America and globally is considered the most common tick-borne illness. It was first recognized in the United States in 1975 in Lyme, Connecticut. B. burgdorferi disease is also associated with neurologic or cardiac symptoms (stage 2) or arthritic symptoms (stage 3). Disease symptoms, however, may not appear for months or even years after initial exposure through the tick bite, so the availability of a definitive diagnostic test is essential for best patient management. In some cases, these secondary symptoms may occur even though the patient does not remember a tick bite or rash.

Diagnosis of the patient based on clinical grounds alone is difficult unless the typical ECM lesions are present. Currently, detection of B. burgdorferi antibodies best identify patient exposure to the agent.

The high prevalence of this disease in the United States has only recently become evident with the availability of clinical laboratory tests and the adoption of widespread screening in the U.S. of populations that are likely to be exposed to the disease.

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Anyone may be exposed to these common ticks and to Borelia by being outdoors in woodland areas where deer, mice, or birds are commonly found.

PROCEDURE

Since the hepatocyte is the area responsible for the synthesis of most of the complement proteins, and functionally active C1q seems to be synthesized in the genitourinary and gastrointestinal tracts, this technique utilizes the liver, small intestine and gonad neurolymphatic reflexes.

First, test the rectus femoris and gluteus medius muscles for strength.

Next, the liver, small intestine and gluteus medius neurolymphatic reflexes should not therapy localize individually when tested with either muscle.

If there is an indication of a deficiency of calcium and magnesium, then the strong rectus femoris muscle should weaken when simultaneously therapy localizing the liver NL with that of the small intestine.

If there is an indication of a deficiency of magnesium only, then the strong gluteus medius muscle should weaken when simultaneously therapy localizing the liver NL with that of the gonads.

If the first pair therapy localize, have the patient insalivate a calcium substance, then a magnesium substance. At this point, you could consider that the classical pathway was deficient.

If the second pair therapy localize, have the patient insalivate a magnesium substance. This finding might indicate that the APC was deficient.

To treat the deficiency, supplement the patient's diet with the proper substance.

SUMMARY

In summary, there is no doubt that complement can influence disease susceptibility, especially those diseases which appear to have some underlying immunological defect. Genetic defects of complement can result in poor health, showing that the complement system is an important natural barrier to infection, and necessary part of humoral immunity. Complement is but one of a number of barriers important in causing inflammation and bacterial opsonization.

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REVIEW OF THE TECHNIQUE

Step 1: Check the rectus femoris muscle for strength bilaterally.

Sept 2: Check the gluteus medius muscle for strength bilaterally.

Step 3: Therapy localize the liver neurolymphatic reflex, and test the rectus femoris and gluteus medius muscles.

Step 4: Therapy localize the small intestine neurolymphatic reflex, and test the rectus femoris and gluteus medius muscles.

Step 5: Therapy localize the gonad neurolymphatic reflex, and test the rectus femoris and gluteus medius muscles.

Step 6: If any weakness is found in steps 3-5, then fix what you find, and continue.

Step 7: Therapy localize the liver and small intestine neurolymphatic reflexes simultaneously and check the rectus femoris and gluteus medius muscles.

Step 8: Therapy localize the liver and gonad neurolymphatic reflexes simultaneously and check the rectus femoris and gluteus medius muscles.

Step 9: If the patient tested weak to step 7, then have them insalivate a calcium and a magnesium substance.

Step 10: Recheck the rectus femoris and gluteus medius muscles.

Step 11: If the patient tested weak to step 8, then have them insalivate a magnesium substance.

Step 12: Recheck the rectus femoris and gluteus medius muscles.

NOTE: If the above procedure indicates a possible problem with complement, it would be wise to run a complement study. If there has been a history of Lyme disease, then a P-39 test would be indicated.

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DIAPHRAGM AND FASCIA - "FRIEND OR FIEND"

John W. Brimhall, B.A.,B.S.,D.C.,F.I.A.C.A.

ABSTRACT:

This is a diaphragm release technique that releases stored up shock in our system. One contacts the abdominal midline with finger tips of both hands. You press with increasing pressure carefully until the tissue softens. The whole diaphragm -fascia complex releases with favorable results throughout the whole body and mind.

INTRODUCTION:

Dr. Robert C. Fulford, D.O., who practiced in Tucson for years was known as a magician in healing. He used a midline diaphragm and fascia release that he felt releases stored up shock in the body. This releases both physical and emotional shock from previous trauma.

Trauma has a tendency to fixate the diaphragm, the rib cage and the surrounding tissues. This can happen at birth or anywhere along the way. This can affect the quantity and quality of life.

Symptoms can vary from epigastric pain, hiatal hernia symptoms, difficult breathing, emotional distress, projectile vomiting, and even panic and anxiety attacks.

ANATOMICAL CONSIDERATION:

The diaphragmatic motion is approximately 24,000 movements a day. It aids respiration and has an action on abdominal and thoracic viscera. With its pulling and pushing of both viscera above and below the diaphragm it becomes a major component of life and function.

Our trunk with both pleural and peritoneal cavities are structurally and physiologically married through the diaphragm and the fascia.

Our diaphragm acts like a piston of an engine, rising and falling with our thorax the cylinder wall. So their rise and

fall, if restricted affects pressures, viscera, air flow, vertebral and rib motion and even emotions because of the long standing and far reaching involvements.

It is felt you can improve the entire lymphatic and cerebral spinal fluid movement by releasing the diaphragm and related structures. Palpation along the left inferior border of the ribs going midline will show a notch to be depressed at the sternum rib junction if there is a problem. It will not be present on the right side.

THE CHALLENGE:

I find the best way to test for its involvement is to challenge: You press with fingertips against the midline of the abdomen between the xyphoid process and the umbilicus. If the area is too tight and reactive, a previously in tact muscle will blow weak. You want to press hard enough in the challenge to feel marked resistance against your pressing hand.

THE CORRECTION:

(1) On a male patient you stand on the right side to correct and on a female you stand on the left side. Dr. Fulford says this is because of proper energy flow and is very important. Reactions can occur if you forget.

(2) You then tell the patient this will be very sore and uncomfortable for about two minutes until the spastic area releases.

(3) Now you press with the fingertips of both hands lined up end to end in the area inferior to the patients xyphoid to just about the umbilicus, so as not to contact the umbilicus.

(4) You press down A to P and go increasingly deeper with respiratory excursions until you hit a wall of restriction that moves no further. You hold that, pressing just slightly through the resistance.

(5) You assure the patient you know this is painful but will last only a minute longer. Then you will feel a release like your finger just slipped through that barrier and the whole area will soften. Usually the patients breathing pattern will change at that point.

(6) New research has shown you need to get a further release on some patients by a double hand (thumb contact) on the inferior borders of the ribs bilateral. The placement is

inferior and lateral to the zyphoid. You put light lateral pressure evenly. You'll feel this are a soften and it will fell like it spreads further and further during the process.

(7) If you use a pucussor (electric hammer), you have the patient lay on their left side. You purcuss the left rib cage just lateral and inferior to the zyphoid again with light pressure and speed. You monitor the release with your other hand at 4th thoracic. With this release, be sure and balance the ceberal spinal fluid, cranium and sacrum.

CONCLUSION:

You can expect better, easier breathing, increased cerebral spinal fluid movement and a general increased feeling of well being. Lymphatic flow is usually improved also.

I have had babies with projectile vomiting quit on the spot with one treatment. I've seen insomnia corrected in one treatment, that was a long time problem.

One case we had a lady in hysteria where she could not get air in and was turning blue and clutching on my arm in desperation for help. The mother was panicking (as was I) and wanted to get her to the hospital emergency room.

I pressed quickly between the xyphoid and umbilicus, stayed there until I felt it release. She started to sigh and get normal oxygenation in about a minute and a half. She went on to complete restoration at that visit.

Many times they will get an emotional release then or commonly later. The patients state its like a ton has been lifted off their chest.

As previously stated this releases shock that has had this area locked up from a previous accident, emotional incident or in some cases even since birth.

It must be remembered you stand on the right of males and the left side of females to correct.

In conditions like asthma, emphysema and other respiratory conditions you must make sure cranial and sacral-coccyxgeal primary respiratory mechanisms are free also. That can be topics for future discussions.

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FOUR MORE SPHICNTORS AS IMPORTANT AS THE ILEOCECAL VALVE

Dr. John W. Brimhall

ABSTRACT:

There are four more sphinctors besides the ileocecal valve that we haven't given proper attention. Their diagnosis and treatment can give us as far reaching results as the ileocecal valve times five. We also have found the treatments can be augmented by a bipolar magnet that helps to solve the electromagnetic pollution problem and strengthen our auric field.

ANOTOMICAL CONSIDERATION:

The sphinctor-like areas of the digestive system are the cardiac, the pyloris, the sphinctor of Oddi, the duodenojejunal junction and the ileocecal valve.

It's hard to imagine practicing health care without knowing how to recognize and treat an ileocecal valve problem. We all have briefcases full of histories of the people with varied problems we have helped with that technique.

Its time to open up your briefcase with an added awareness of other sphinctor-like areas in the digestive system.

The other insights we need to acquire are more about viscera and their motility and mobility.

BACKGROUND:

The purest may wonder if it falls in the scope of chiropractic or applied kinesiology. The viscera affect the vertebrae much as the vertebrae affect the viscera. The nerve, lymphytic, ceberal spinal fluid, vascular and acupuncture doors swing both ways. In fact, with many tie-ins of the body the doors swing side ways, up and down and all around.

The visceral motility can be felt by just laying your hand on the area over the organ very lightly. You can feel it the same as the thorax moving during respiration or the cerberal spinal fluid movement at the cranium or sacrum. Its just much more subtle. Its movement is always three dimentional

but the sphinctors can be felt as clock wise and counter clock wise very well.

DIAGNOSIS:

The movement is felt usually best by the thenar or pisiform part of the hand. Its movement should be mostly clock wise rotation. If you feel no motion or predominantly counter clock wise rotation, then problems are eminent.

To reduce the diagnosis to a A. K. challenge, you can therapy localize in the clear sometimes. I find a torquing counter clockwise or clockwise of the area with your fingers to be most consistent. In other words you should be able to torque the area counter clock wise or clock wise and not have a previous strong muscle go weak on testing, if proper function exists.

TREATMENT: (fig. 1)

Treatment can be similar to an ileocecal valve treatment e.g. manipulate the area carefully. Then treat the lymphatics of the corresponding organ e.g. stomach lymphatics for the cardiac and pyloric area and the small intestine lymphatics for the duodenojejunal (D.J.) junction. For the sphinctor of Oddi you may have to treat the lymphatics for the gallbladder and the small intestine. Of course the vascular points the set points for corresponding meridians all fit the same patterns etc.

Specific lymphatics, vasculars, etc. are still under investigation. If you can feel; the ceberal spinal fluid or rhythm of the body, you should synchronize this area like you would the cranium. I prefer the right hand on top and the left hand on the bottom for proper polarity of doctor to patient.

The Upledger Foundation and Dr. Barral teach a rebound manipulation to free this area. You palpate and feel the motion. You go with it and take it to the direction its going and hold it down through the patients inhale and exhale phase. When they start to breath in, the next cycle you recoil off of the area quickly. Please move off quickly and not into it.

You are taking the soft tissues and viscera to its direction of movement and motility through the exhale phase and recoiling off it part way into inhale.

One or all five or any combination may be involved. Usually one of them is the ring leader and if you free it, they will all improve.

ELECTROMAGNATIC AND FURTHER INSIGHTS:

Of course all of the things that affect digestion can cause problems here. That is food quality and quantity, emotions, and electromagnetic sensitivity, etc. Dr. Becker in "Cross Currents" and many other authors are telling us the power lines, ELF frequencies, microwaves, etc. are killing us.

I find this sphinctor area especially sensitive to currents. To challenge this you put a magnet close to the area and challenge both north and south pole exposure. If either or both blows the body weak, generally I feel the auric field has a weakness. You can put the patient in front of a television or a computer and there weaknesses will show also. We often use a hair dryer to show they weaken if its in their field. Usually the television, computer or hair dryer will cause general muscle weakness all over the body.

I correct this by a bi-polar magnet. That is one that has north/south, north/south, north/south on the same side. I place it over the shock release area or the area between the zyphoid and the umbilicus. This corrects the magnet or electric field induced weakness within two minutes anywhere on the body. It also frees the sphinctors much easier or completely by itself.

These bi-polar magnets are being used by us for many purposes and we will report on them with further papers. You can secure them for $20.00 by calling 1-800-658-9122. They are the size, weight and shape of a credit card.

SUMMARY:

To summarize, the case histories would fill new briefcases. Five sphinctors need to be checked on, challenged and corrected by manual and or bi-polar magnet treatment. It is exciting to feel the clock wise motion restored and hear all of the peristalsis, gurgling and draining that begins almost immediately.

May your life and journey by full of paradigm shifts.

Four More Sphinctors - Brimhall Page 4

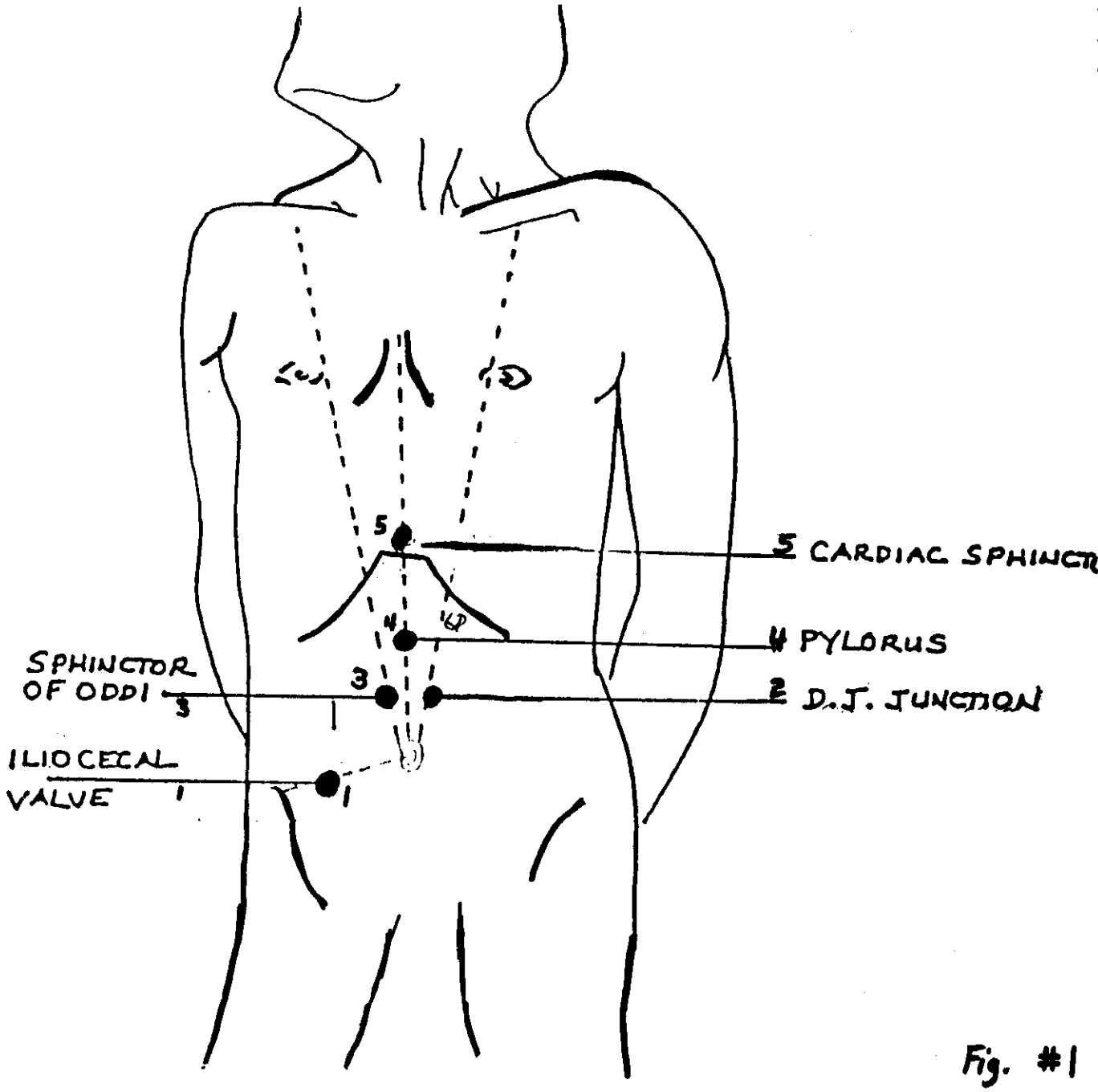

Fig. #1

A "New" World Class Herb for A.K. Practice:

Uncaria tomentosa - a.k.a. Uña de Gato (UDG)

Brent W. Davis, D.C.

Abstract: Background information is provided on one of the Amazon's most valuable medicinal plants. The author describes his original field research and specimen collection of Uncaria tomentosa (UDG) in the Amazon beginning in 1988, and his subsequent A.K. clinical evaluation of that herb continuing to the present. The pharmacology and therapeutics of UDG are presented. Its profound healing ability is outlined. In important therapeutic areas, UDG far surpasses other world class master herbs (such as Astragalus, Echinacea, Ganoderma, Goldenseal, Artemisia annua, Siberian "Ginseng", Panax, et al.), as well as potent over-the-counter products such as undecylenic acid, "citrus seed extract", caprylic and lauric acids.

INTRODUCTION

Toward the middle of 1987 I experienced an unsettling feeling which I suspect other A.K. practitioners have also felt. At the time I was observing good patient response to A.K. structural and nutritional/herbal therapies. Patients' musculoskeletal problems resolved more permanently and more quickly than if only straight manipulation were employed; abnormal visceral symptoms ameliorated such that those under my care could often stop taking harmful drugs; disposition and mental outlook of patients generally improved. Despite these good signs of healing, I sensed in some of the more difficult cases that beneath the level of my detection processes of serious deterioration were likely taking place. At times I also felt that even if I understood what and where certain destructive processes were, I would have no way of stopping them anyway–I might only be equipped with band-aid approaches. I had the frustration of knowing that with a relatively few number of "maintenance" visits, most patients would likely stay feeling well (especially considering A.K.'s constant advancement with new techniques.) But what if patients were feeling well, I mused, and all the while quiet pathology were progressing. Perhaps I had seen one too many "healthy" young women "spontaneously" develop a uterine mass.

The serious disturbances I was sensing in some patients were what European practitioners of biological medicine would call *pathogenicity of terrain* – a core disturbance of constitution. In more conventional terms, one might call it oncogenicity – the prevalent modern tendency to deteriorate into cancer.

Such feelings made me yearn for a way to more fundamentally direct the core of patients

toward health.

As it has often been my habit, I went into Nature to ponder the matter. I would sit in the company of plants, appreciating communion with them, hoping for inspiration and guidance. Months passed, and in the beginning of 1988 I serendipitously received an invitation to a medicinal plant congress in Lima, Peru the coming summer. I resisted the urge to attend because it would take a long period out of my practice. I felt utterly compelled to go, however, so I did. I took six weeks to attend the International Congress on Traditional Medicines in Peru, and to do long awaited herbal research in Brazil.

CONGRESS PROCEEDINGS

Many of the presenters at the congress on traditional medicines were holistic medical physicians who had used herbs in clinical practice for years. UDG was discussed as one of about a dozen herbs which are used consistently to cure cancer and other serious disorders. Since 1988 I have been clinically evaluating many of the great South American herbs mentioned at the congress (and from other avenues as well) in terms of their applications in general practice – not for their oncolytic properties. In some profoundly important areas, Uncaria tomentosa stands out above many others.

BRIEF CHARACTER, PHARMACOLOGY & CLINICAL THERAPEUTICS OF UNCARIA TOMENTOSA

Character

Like all great herbs, UDG has a history of use among indigenous peoples in its native land. It is regarded as a sacred herbal panacea which has a powerful connection with benevolent forces in Nature. Labels on packages of UDG tea in Peru read as follows:

> "Uña de Gato is a medicinal herb native to the central jungles of Peru whose curative properties are almost unlimited. This is attributed to Uña de Gato being a powerful cellular reconstitutor.
>
> It has applications in gastritis, ulcers, cancer, arthritis, rheumatism, irregularities of the female cycle, acne. It is also good for treatment of organic depression.
>
> In external application, it is excellent for the treatment of wounds, fungus, fistulas, hemorrhoids, etc."

Two species of Uncaria (U. guianensis, U. tomentosa) grow in the jungle regions of Peru, all the way from the Amazonian basin to several hundred miles south in the high jungle areas of the province of Junin. Accompanied by native guides, I collected bulk specimens of Uncaria tomentosa off the Amazon about 50 miles out of Iquitos, as well as U. tomentosa & U. guianensis from other regions further south. My subsequent clinical evaluation of the two species of Uncaria agrees with native wisdom and the results of Peruvian physicians, which is to say that Uncaria tomentosa is more valuable than U. guianensis, though they are chemically similar.

UDG is a giant woody vine, often growing up into the trees more than a hundred feet in length. The largest diameter of the vines I collected was about 6 inches across. The root and lower portions of the vine are typically the parts used for medicine.

European and Peruvian scientific studies of UDG so far have been conducted with both the wood (central portion of the vine), and with the bark of the vine. The chemical constituents of each are different and have somewhat different physiological effects.

UDG was brought to the attention of European practitioners in the early 1980's. A few years before, the modern rediscovery of Uña de Gato took place. Apparently in the early 70's an Austrian journalist travelling in Peru happened upon the herb which he gave to an ailing relative who took it and subsequently was cured of cancer. The journalist then invested a great deal of time and effort in researching Uncaria tomentosa, and came to the conclusion that the central woody portion of the plant is the useful part. He developed this opinion on the basis of chemical analyses and clinical evaluation done in Germany and Austria which showed that a very active alkaloid is in the highest concentration in that part of the plant. He and other collaborators filed for international patents on this "active ingredient" which may appear in the marketplace in the near future.

Italian researchers have looked more at the bark of the vine which also has therapeutically active constituents. As is often the case with great herbs, researchers frequently try to "own" a particular part of the plant and prove it to be the part containing the "active ingredient." This phenomenon is a direct outgrowth of the drug model of health intervention in which pharmaceutical firms directly or indirectly support research with the motivation of finding new proprietary items to patent. Now ambitious natural products companies are also following this financially lucrative path. In actual fact, most important herbs have numerous "active ingredients," often from different parts of the plant.

From information I gathered in Peru, the journalist's relatives working there supply about one third to one half of the world's market of Uncaria tomentosa, which goes mostly to Europe. (My Peruvian associates estimate the world market to be 15-20 tons.) This herb (which is of good quality) is collected from delicate forest ecosystems and is **not** being replanted. The rest of the herb is generally unreliable. It is collected rather recklessly by what I call the "chain saw gang." These are individuals in a desperately poor economy who, sadly, subsist by taking contracts on the wild rainforest herb to sell to outside vendors. Unless individuals in the U.S. are informed and given an alternative, the two above-mentioned sources of the herb are what manufacturers here will be using. Uña de Gato from those sources started to be sold on a small scale in the U.S. in approximately 1985.

I was given information about UDG by a very wise native Indian elder in Peru. He told me quite a bit about the herb, suggesting I should investigate the whole plant. I have begun that process. He stated that even though UDG is powerful, it is nevertheless very sensitive, and that if it is utilized with a lack of good motivation, it will lose much of its wide healing ability

and will become only a "chemical shadow" of its real self. He cautioned that perhaps more than any other herb, Uña de Gato has to be treated with reverence.

I promised that in return for his information and trust, I would make every effort to protect and augment the amount of Uña de Gato in its native habitat even as I was working on developing it as a remedy. Due to the scope of the project, and the need for financing well beyond personal resources, I formed a non-profit public benefit organization in 1988 (The Foundation For Herbal Healing & Conservation - FHHC) to raise funding for herbal conservation, organic agricultural development, and research. FHHC is now working on herbal conservation in Peru and other areas as well. If the reader has an interest in supporting this endeavor, I would be very pleased to hear from you.

Pharmacology

The first published chemical analysis of Uncaria tomentosa appeared in 1974 [1]. Two primary alkaloids were chromatographically identified as Rynchophyllin and Isorynchophyllin, as well as 5 secondary alkaloids. In 1985, Dr. B. Kreutzkamp and co-workers in Germany undertook more extensive analysis of UDG and came up with a classification of 6 alkaloids, the most immunologically active one being Isopteropodin [2]. It has been reported in the literature that several color variations of *fresh* Uncaria tomentosa wood correspond with different concentrations of alkaloids, the darker colored wood being the richest in Isopteropodin.

Different of the alkaloids have been experimentally shown[2] to be:

1. Immunostimulating by way of enhancing phagocytosis;
2. Ganglion-blocking with an enhancing effect on parasympathetic tone;
3. Inhibitory to striated muscle contraction;
4. Hypotensive, uterostimulant and antipyretic;
5. Diuretic.

Specially prepared liquid extracts (first alkalized, then treated with ethyl acetate, and then acidified) – *not conventional alcoholic extracts* – had a strong stimulating effect on phagocytosis [2]. For the effect of the alkaloids, powdered whole wood(or tablets of the wood) of Uncaria tomentosa or a simple tea from it should be used – not tinctures or regular alcoholic extracts.

Italian researchers took a different approach from their German and Austrian colleagues. They found chemical and biological activity in the *bark* of the UDG vine. They reported quinovic acid glycosides and triterpenes to be active ingredients with anti-viral and anti-inflammatory activity [3]. Another group of Italian researchers found a steroidal fraction of UDG bark to be anti-inflammatory [4]. Glycosides and triterpenes from the bark are soluble in methanol, which is to say that conventional grain alcohol extracts of the bark are therapeutic.

A very good recent article of Italian researchers' work discusses cytotoxicity, mutagenicity, and anti-mutagenicity tests performed with a special alcoholic extract of the bark of UDG [5]. The researchers found that "All the Uncaria tomentosa extracts exerted a protective action

against photomutagenesis induced by 8-MOP + UVA. The values ranged from a minimum of about 30% to a maximum of about 70%."

In the experimental work, to test the anti-mutagenic effects of UDG, two healthy donors, one smoker and one non-smoker, were requested to drink daily for 15 days an aqueous alcoholic infusion of Uncaria tomentosa *bark*. Urine samples from the two donors collected before, during and 8 days after the last treatment were concentrated and added to the anti-mutagenicity bacterial tester strains. Non-smoker urine didn't show any mutagenic activity before, during and after treatment with U. tomentosa. Smoker urine, with mutagenic activity before treatment, showed a dramatic decrease of mutagenic potential at the end of U. tomentosa treatment, persisting 8 days after the end of treatment.

The Uncaria tomentosa extracts, with all the doses employed, showed no mutagenic effect but a significant anti-mutagenic activity as antioxidants. The test used shows an aspect of anti-mutagenicity consisting of the quenching of singlet oxygen and the scavenging of other oxyradicals generated under UVA irradiation.

The authors concluded, "The anti-mutagenic effect of the extract employed in these series of oxidative reactions is probably attributable to the quinovic acid glycosides from Uncaria tomentosa. This plant shows anti-mutagenic activity *in-vivo* in smokers, confirming its high anti-oxidant potential."

The experimental human studies certainly need to be repeated on a larger scale, but the results seem to confirm the empirical clinical findings of health professionals, and the indications of traditional usage of UDG as a "cellular reconstitutor."

Applications of UDG in U.S. General Practice

If the reader has asked himself or herself, *What relevance does an anti-neoplastic agent have in my structural / nutritionally-based practice?*, that is a good question. It is a question which struck me forcibly as I listened to one Peruvian physician relating his team's good success rate with UDG and other herbs in treating 14 types of accurately diagnosed cancer in 700 patients between 1984 and 1988.

I asked myself: If UDG truly were a life-saving anti-neoplastic natural substance, what good does that do me and my colleagues? I'm not in a position to treat cancer, and for anyone else who might wish to do so, it would legally be impossible in the United States given the current complicity of the FDA with pharmaceutical firms' vested financial interests. Owing to an uneducated and often complacent general public, the forces of organized medicine have been able to effectively place a stranglehold on natural therapeutics. The only allowable treatments for cancer at this time, of course, are surgery, drugs and chemotherapy.

As I pondered this matter, the following thought occurred to me: If an agent can change the course of cancer, then it must be able to favorably influence the abnormal physiology which precedes cancer. Analyze UDG's action on the gut primarily, and also the mind and the

immune system – then you will find the applications for general practice. I have pursued that thought over the last 4 years. My findings are reported after the following summary of European practitioners' experience with UDG therapy from approximately 1980-1990.

European Applications of UDG Therapy

In a presentation to *heilpraktikers* in Europe, German doctor of medicine, Iwan Diehl, summarized well the therapeutic usage of the wood of UDG by German and Austrian physicians:

Effectiveness: Because of the mode of action of Uncaria extract, side-effects do not appear if the recommended dose is taken. In association with individuals who have frequently used laxatives, there may be temporary disorders of intestinal peristalsis, which disappear spontaneously after a few days. For the alkaloids of Uncaria extract, the following effects have been proven:

- Stimulation of the non-specific immune system with activation of macrophages and granulocytes to eliminate non-physiological substances;
- Enhancement of the sensitivity and reactivity of the immune system to seize and to eliminate very weak antigens;
- Inhibition of inflammation by a repairing incorporation of lipids into the lipid matrix of damaged cell membranes;
- Selective inhibition of growth of malignant cells by simultaneous improvement of erythrocyte and macrophage function;
- Enhancement of the growth inhibitory effect of [pharmaceutical] cytostatics by an intact immune system;
- Selective growth inhibition of virustransformed cells.

Indications: A positive influence has been observed on the following disorders:

— Dermatological disorders
— Allergic disorders
— Rheumatic disorders
— Chronic inflammation
— Viral diseases (herpes zoster)
— Malignant diseases (cytostasis and radiotherapy are more efficient under a concomitant therapy with Uncaria.)

Uncaria is contraindicated for transplant carriers, because of possible graft rejection. During pregnancy, Uncaria should not be used. In cases of treatment with H_2–antagonists [e.g. anti–ulcer medications], a potentiation of the H_2–antagonist might be expected.

Before I encountered information from Dr. Diehl, I had already worked empirically with UDG for two years. I had observed several of the effects he noted, and I was pleased with the corroboration.

During the 1988 Peruvian Congress, an Italian pathologist, Francesco Iaccarino, related the case study of a young physician he treated with UDG tea and powdered wood of UDG in tablets.

The presentation was titled: *A case study on the resolution of anti-connective tissue autoantibody syndrome.* For 8 years, the ill young physician had experienced severe muscular and joint pain, fatigue, immune dysfunction, and mental depression. Her very high level of immune complexes and anti-connective tissue autoantibodies diminished to normal after 9 months of treatment with UDG. She became virtually symptom free.

Clinical Therapeutics: UDG Applications in A.K. Practice

A.K. practitioners with a broad scope of practice can appreciate from the previous information that UDG has the possibility of helping many different types of patients. If one ties together several of the therapeutic effects of UDG mentioned – ganglion blocking with an enhancing effect on parasympathetics (a stress-sparing, potential "yin-preserving"effect), immunostimulating, anti-microbial, anti-neoplastic, anti-inflammatory, anti-self antibody-clearing, anti-allergic, anti-depressant – one system, directly or indirectly, could have a relationship to all of them: the gut.

The weight of the human intestinal microflora is nearly equal to the weight of the liver. Like an organ itself, the flora performs several vital metabolic functions. If it is imbalanced/ infected, it "is capable of engaging in reactions that can generate carcinogens, mutagens or tumor promoters in the large bowel [6], and inflammatory agents which contribute to joint and dermatological diseases [7]. When the flora is healthy, it produces necessary vitamins, balances electrolytes and fluid levels, regulates cholesterol levels, and influences unsaturated fatty acid metabolism [8,9]. Certain bacteria of human microflora were found to liberate immunomodulating peptides which reconstituted cellular immune function and lymphatic tissue weight in immunosuppressed mice [10]. That phenomenon very conceivably pertains to humans as well.

As a class, A.K. practitioners have put general awareness of bowel inflammation on the map(ICV Syndrome.) But beneath recurring ICV problems lie many other health disturbances, especially:

- "Leaky Bowel Syndrome," a disturbance of mucous membrane pore size and transport ability often caused by damage from antibiotic overuse or overexposure;
- Chronic parasite infestation and/or intestinal flora imbalance tending toward chronicity due to weak "energy" in the abdomen, frequently associated with exaggerated stress response, low self-esteem, etc.
- Autonomic plexus dysfunction due to: local mechanical injury; cranial lesions; environmental toxin poisoning; or dysfunctional personality with neuroses. Maybe a more compassionate way of describing the latter is: people in spiritual crisis.

In my experience on approximately 150 patients during the last four years (who have received adjunctive monotherapy with UDG), I have seen Uncaria tomentosa break through severe intestinal derangements that no other available products can touch, including the strong & very useful undecylenic acid.

> The right type of preparation of bio-active UDG has a profound ability to get rid of deep-seated infection which has lodged in the bowel and perhaps even in the mesentery, and which can derange the uterus and adnexa, the prostate, the liver, the spleen, the kidneys, the thymus, and the thyroid – for starters.

In some of the most difficult parasite cases, with combined infestation of Blastocystis hominis, Entamoebas, and Giardia for example, UDG can often blast through the "parasitic miasm." It seems to allow an "opening" so that other supplementation (such as homeopathics, Chinese Artemisia/Goldenseal/Ginger and other herbals, undecylenic acid,etc.), alternated or in combination, can complete the cure. I have nicknamed Uncaria tomentosa "The Opener of the Way." It breaks through metabolic log jams and then allows the practitioner to make further progress with other therapies.

Bad bowels... Bad immunity... Cancer. That relationship is certainly no mystery. It is a key concept all the way from folk medicine to advanced gastroenterology. Gastrointestinal function (and intestinal flora composition) in relationship to oncogenicity has been widely studied academically[11,12]. We may know a relationship exists, but we are powerless to change it without the proper tools. *UDG is an indispensable tool. But for heaven's sake use it as a legitimate tool, and do not mention it in relationship to cancer.* Perhaps there is a relationship, provable by *unbiased* science, between consumption of UDG and the prevention of the onset of cancer. Perhaps there is even a relationship to cure, but it has not yet been *proven*. Sadly at this time, due to the pharmaceutical-FDA-medical complex, the United States is not a place where open-minded evaluation could even occur. The domain of UDG is in the realm of herbal nutritional therapeutics.

Administration

• UDG wood is administered as a tea, or as tablets, in the amount of 3 to 25 grams per day, or that equivalent if a concentrate is used. Sensitive individuals may require less than 3 g. – Once a consistent location of harvest for the herb has been obtained, and specimens are monitored for alkaloids, a reasonably constant level of alkaloids can be achieved. The idea that the herb should be standardized for alkaloid content is not a useful one, because in so doing, one would have to mettle with the energetics of the plant, and as my Indian friend mentioned, that's not wise. Due to the fact that Uncaria tomentosa is not toxic, it is not critical if the product is a little weaker or stronger. Dosage has to be adjusted for desired effects anyway.
• Liquid extract of UDG bark can be taken at an equivalent of 3 grams per day, which is roughly 25 drops of fluid extract 3 times daily. This often must be adjusted up or down according to the individual.

I generally do not use over 5 grams per day–more commonly 3 grams/day. One phase of treatment normally lasts 10-14 days. Then other formulations can be used, cycling back to UDG in tough cases in perhaps 7-10 days. Individuals who treat very advanced stages of pathology might routinely use between 10-20 grams per day for several weeks at a time.

Incorporating UDG into practice

Individuals who present with symptoms of:

- Imbalanced autonomic function with diminished intestinal peristalsis, or who have frank G/I problems such as recurring ulcers, regional ileitis (Crohn's disease), diverticulitis, or spastic bowel syndrome(recurring ICV problems), or
- symptoms suggestive of or found in association with diagnosed anti-self antibodies, or
- dysbiosis (intestinal flora imbalance), parasitism, and chronic "candida," or
- organic depression found in association with intestinal toxicity

are prime candidates for UDG therapy. UDG also can be used to advantage semi-annually for a couple of weeks at a time as a general deep cleanse.

A.K. challenge for numerous metabolic faults can be abolished with lingual testing of UDG. Sometimes cranial lesions and torsion patterns will abolish with UDG when viscerosomatic reflexes are the cause. Many reflexes having hypothalamic/pituitary components can be influenced. If pineal hypofunction is suspected, test for the need of UDG as you would test for melatonin precursors. Often essential fatty acid problems are aided by UDG in an adaptogenic manner. UDG is enormously helpful in clearing the way so that specific nutrients the patient lacks can better do their job.

> After a positive pre-administration clinical screening which shows compatibility with Uncaria tomentosa, there is no substitute for trial. As you are learning about UDG, try monitoring its effects as a monotherapy for 7 to 14 days on several well chosen cases. It can be impressive.

Care with UDG

Possible contraindications. Peruvian and European practitioners say there are no side-effects from UDG use. In my experience, that is not entirely true. What I find interesting is that the appearance of what seems to be an undesirable side-effect, is generally an indication that UDG is working well. The undesirable side-effect is diarrhea. In the patients who have benefited most from UDG *wood*, the herb tends to progressively alter bowel consistency from hard or normal to loose. It's kind of like the ascorbic acid advocates' "take it 'til it runs and then back off" phenomenon. In **rare** cases UDG causes what appears to be full blown dysentery. I am hypothesizing that in the latter, actual encystment or some other type of sequestration of parasites has occurred in the patient's past, and that UDG breaks open the encystment, and that is why the previously intractable problem finally moves toward resolution.

In some cases where there is a very positive pre-administration screening, actual trial of UDG paradoxically produces a constipating effect, and the crude single herb cannot be used. I am working on formulations from the standpoint of Ayurvedic and Chinese medicine which hopeful will allow patients to benefit from the "energy" of UDG by putting it into formula. Another possible explanation is that the "energy" of UDG is needed in the homeopathic form and not in a physiological dose. I will soon be receiving fresh plant extracts from the jungle, from which dilutions can be made, and then I will be able to test that line of thought.

In cases where simple loose stool occurs with use, simply modify the dosage until stool consistency is normal to slightly soft. Sometimes the herb needs to be stopped for a few days. In the rare case where dysentery-like symptoms manifest, stop the herb. If the condition does not self-limit within one day, check for the need of homeopathic Mercurius solubilis or Mercurius corrosivus in the decimal or centesimal dilution at 30 or above, or millesimal 6-18. In my practice that has always taken care of the problem in cases of major healing crisis. If there is only a mild loose stool, astringent and antimicrobial herbs will correct the problem [13]

UDG and the mind

There is a burgeoning amount of information over the last decade in the field of psychoneuroimmunology attesting to the relationship between the mind and immunity, and between depressed mental states and organic disease. In our own field, considerably more attention has been paid to mental/emotional conditions than was a decade ago. In the 1991-92 ICAK Collected Papers, Dr. Stephen Kaufman wrote that in his practice, "25-40% of all patients suffer from significant emotional trauma caused by an event they've never fully recovered from.[14] " I would say those figures apply to my practice as well. The proper medicinal plants are very useful in opening up the patient to access their mental states – grief or fear – *before* corrective physical techniques such as "Grief Relief" are applied, as well as being nurturing and helpful in between treatments to sustain corrections. UDG is so highly energized that it can work on the subtle mental level in a similar way to Bach and homeopathic remedies. Uña de Gato translates from Spanish as "claw of the cat." The thorns on the apical shoots of the vine look almost exactly like cat's claws–hence the common explanation for its name. I found in the plant another likeness to cats. In ancient Egypt cats were revered for their vigilance as benevolent psychic protectors. UDG is also a great protector, especially to the mind and "heart" of dysfunctional beings who suffer from chronic neurologic disorganization. UDG powerfully aligns the electric field of the body, bringing with it for human benefit the magnificent vital energies of the Amazon.

Challenges with UDG

The main challenge I have been facing with UDG over the last four years is obtaining consistently good quality plant material produced in an ecologically sound manner–and I've been working hard at it, aided by a dedicated group of Peruvian co-workers! Notwithstanding, the performance of tea, tablets and liquid extracts I am presently using is good [13].

I have employed a forestry engineer, a botanist and a general foreman as part of the Foundation For Herbal Healing & Conservation's operation in Peru to oversee the conscientious chemical free development of Uncaria tomentosa and other herbs. A plan is being implemented whereby the production of UDG will result in reforestation of previously destroyed secondary rainforest. I suspect this project will require assiduous work for the next one to two decades. It's worth it. Hopefully at the end of that time, through FHHC's work, ecologically safe Uncaria tomentosa will be available to all manufacturers worldwide.

Summary

Derangement of human intestinal microflora is a common problem with the potential of fundamentally disrupting numerous metabolic pathways, producing diverse unhealthy symptomatologies. Uncaria tomentosa is a world class herb which has the power to improve intestinal flora balance, enhance immune function, and arrest and reverse deep seated pathology, allowing a more rapid return to health in the context of concomitant A.K. therapies.

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A CHIROPRACTIC PERSPECTIVE ON NEUROLOGICAL INFLUENCES OF THE TEMPOROMANDIBULAR JOINT

By Vincent Esposito, D.C.
Board Certified Craniopath
S.O.R.S.I.

ABSTRACT: The purpose of this paper is to show you how to use standard orthopedic, neurological, physical, chiropractic, and kinesiological examining procedures to show how the temporomandibular joint influences the body systemically.

INTRODUCTION:

One of my first observations in the treatment of TMJ conditions has been the variety of responses to standard orthopedic, neurological, physical, chiropractic, and kinesiological examining procedures. The TMJ patient may affect these tests by altering the position of their jaw.

PROCEDURE:

For example, you can do a dermatome examination on a patient and record your findings with the patient keeping his head and jaw in the neutral position. Then have the patient move their jaw to the right, and on the same dermatome examination you may get a completely different response. This is now done with the jaw moved to the left, and the mouth opened. Again, perform the exam with the mouth opened in the neutral position, and then do the exam with the mouth closed or any combination of these positions which may be involved. This approach which I call **Stress testing** or **Challenging,** which is a variation of the standard testing procedure, which can give different sets of responses from the initial test procedure.

An example of this would be to have the patient do a Romberg Sign (significance - The sign is present in Cerebellar or Labyrinthine disease). If a patient has a negative Romberg sign after doing the test, now try doing the test with the patient moving his jaw into different positions. **If at this point the patient has a positive response or a semi-positive response with jaw movement, the A**

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BY DR. VINCENT ESPOSITO
BOARD CERTIFIED CRANIOPATH
S.O.R.S.I.

patient can being showing signs of a TMJ problem that is not just localized at the temporomandibular joint but is affecting the body systemically as well.

Another good test to do this way is the **finger to finger & finger to nose** tests (significance - ability to accurately hit the mark with eyes open but not with eyes closed indicates posterior column disease or malfunction. Inability to hit the mark normally and in a coordinated manner either with eyes open or closed indicates cerebellar disease or dysfunction). Now if these tests are negative when first done (in the clear without moving the jaw), we now will try the same test with moving the jaw right, left, open, closed, biting down, or any other position that we might think to be positive at times and will bring out a positive and evaluate the response observed.

These 3 tests (Romberg sign, finger to nose, and finger to finger), can make up a quick screening procedure for checking for a TMJ problem. My experience in examining patients with suspected TMJ involvement has been that the more serious the TMJ problem is, the more it might affect the body systemically. It may also affect other reflexes, signs, ranges of motion, gait, posture, the sensory system, proprioceptive system, muscle testing, cranial nerves and tests. For example, I have had patients that it has shown up when doing the following tests: Heel-Toe test, Heel-Walk test, Accommodation reflex, Achilles reflex, Auditory reflex, Babinski sign, Brachioradialis reflex, Bragard test, Dejerine's sign Fajersztajn's test, Heel-Knee test, Jandrassik's maneuver for jaw reflex, Jaw Jerk sign, Lasegue differential sign, Lindner's sign, past pointing test, plantar reflex, quadriceps reflex, Trendelenburg test, Weber test, Bechterew's sitting test, Bragard sign , double leg raise, Fajersztajn's test lasegue test, percussion test, Sicard's sign, Valsava maneuver.

DISCUSSION:

It has been my experience that this stress testing will show up positive in all or some of these tests, when a patient has a TMJ problem. I have seen it where a patient has had a few positive tests or all of them. Some phases of jaw motion may indicate a lesion, or a positive test, while **A**

others may not!

It is important at this point for you to understand the importance of the TMJ and how it affects the body on a systemic level. The TMJ, in my opinion and in the opinion of other's, may very well be the most important joint in the body in terms of its homuncular relationship.

"Homunculus" means "little man" and refers to the Penfield and Rasmussen diagram in Gray's anatomy and other anatomical books. This shows the percentage of nerve cells related to the areas of the body. Penfield and Rasmussen say that 40 -50% of all the nerves in the body are related to the face and the head. **These imbalances in the TMJ can have many far reaching systemic effects and symptoms due to the large neurological importance.** A large number of brain cells are devoted to the oral cavity which include the TMJ, which is what the Penfield and Rasmussen diagram shows. This is true for the sensory and motor homunculus.

"Our aspirations are our possibilities."
- Robert Browning

CONCLUSION:

It is without a doubt the most influential joint in the human body on a motor and sensory functional basis. This is one of the main reasons why the TMJ has such far reaching effects systemically on the body. It has been my experience that evaluating this type of testing procedure for the past decade has demonstrated its **efficacy and consistency! Try it, you'll probably find it very interesting and fun.**

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ILEOFEMORAL LIGAMENT TECHNIQUE

By Kenneth S. Feder, DC

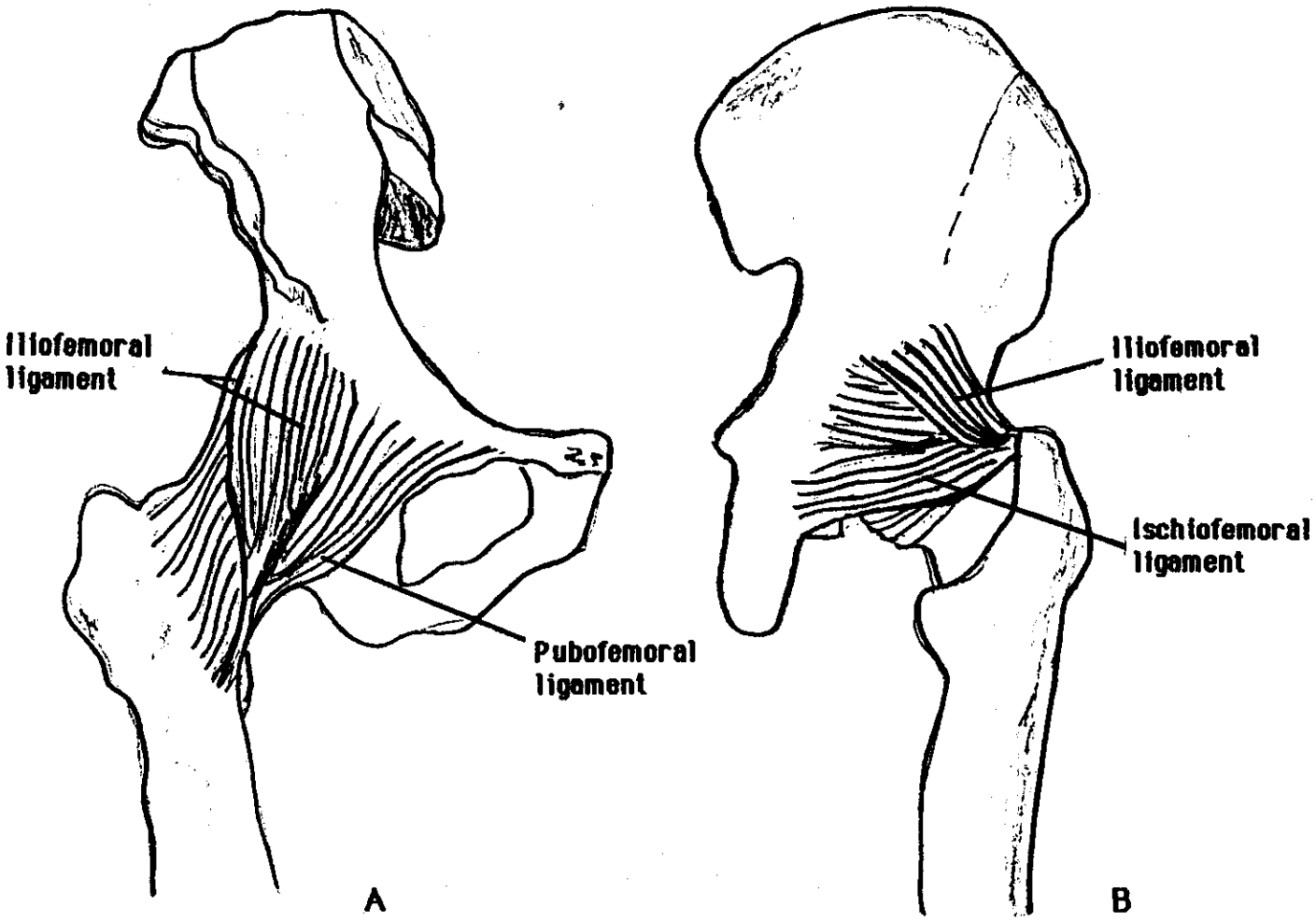
ABSTRACT: Extension injuries to the lumbar spine and pelvis may cause injury to the ileofemoral ligament. This may contribute to pelvic instability and may be causal in the recidivism of Categories I, II, and III. Correcting the involved ileofemoral ligament may prove helpful in assisting in the treatment of difficult pelvic and lumbar problems.

ANATOMY:

The ileofemoral ligament is one of the strongest ligaments in the body. It is sometimes referred to as the "Y Ligament of Bigelow" since it resembles an inverted "Y". It attaches proximally to the lower portion of the anterior inferior iliac spine and to an area on the ilium just proximal to the superior and posterior rim of the acetabulum. The ligament as a whole spirals around to overlie the anterior aspect of the hip joint, attaching to the intertrochanteric line. The more lateral fork of the "Y" attaches to the anterior aspect of greater trochanter, whereas the more medial fibers twist around to attach just anterior to the lesser trochanter.

The ligament primarily checks internal rotation and extension. The ligament is stretched by any attempt to extend the femur beyond a straight line with the trunk. It is the chief agent in maintaining the erect position without muscle fatigue. It allows a person to stand with joint in extension using a minimum of muscle action. By rolling the pelvis backward, a person can hang on the ligaments. The ligament prevents excessive movement in the direction toward the closed-packed position of the hip joint.

ILEOFEMORAL LIGAMENT



Anterior (A) and posterior (B) views of the joint capsule of the hip joint.

PROCEDURE:

The ileofemoral ligament can be therapy localized, and it may show in the clear. The ligament may be involved but may require a more active TL approach. Have the patient attempt the following TL procedures to determine if the ileofemoral ligament is involved:

1. Have the patient TL the ligament while supine and with the lumbar spine in full extension causing posterior movement of the pelvis. Test an indicator muscle for weakness.

2. Have the patient stand and TL the ligament while the patient flexes the thigh opposite the TL side. This will have the patient standing on one leg and placing stress on the ileofemoral ligament. Test an indicator muscle for weakness.

The ileofemoral ligament may be challenged to determine its involvement. Have the patient either supine or standing, and challenge the ends of the ligament toward each other. If a weakness occurs at the indicator muscle, then proceed as outlined below.

CORRECTION:

1. The patient is supine.

2. Have patient flex thigh on involved ligament side toward abdomen.

3. Contact ileofemoral ligament, and press the ends of the ligament toward each other for approximately 45 seconds while the patient maintains the flexed hip position.

4. Retherapy localize and/or challenge to determine the necessity for additional treatment.

5. Raw bone used as nutritional support.

ALTERNATIVE CORRECTION:

1. Patient is in standing position.

2. Contact involved ileofemoral ligament.

3. Press ends of ligament toward each other as patient walks slowly forward and backward.

CONCLUSION:

This procedure has been helpful in cases of hyperextension injury to the lumbar spine and pelvis.

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A CORRELATION OF APPLIED KINESIOLOGICAL PROCEDURES WITH ZINC TASTE TEST

Darrel W. Hestdalen, D.C.

ABSTRACT: Thirty five patients that demonstrated a need for zinc supplementation where given the zinc taste test. The need for zinc supplementation was indicated when oral testing of a zinc supplement would cancel a positive therapy localization to the neurolymphatic for the pancreas and/or a positive indication for Right Thoracic Duct Technique. A positive finding for these indicators show strong correlation with poor ability to taste a zinc solution.

INTRODUCTION

The need for zinc supplementation may be indicated by a positive therapy localization to the neurolymphatic for the pancreas.[1] Dr. Goodheart has discussed the rationale for testing for the need for zinc supplementation when a patient demonstrates the need for Right Thoracic Duct Technique.[2]

The zinc taste test determines relative zinc levels by the timed response to the tasting of a zinc sulfate solution.

DISCUSSION

Patient started testing or gamma-2 (G-2) muscle testing has been described by Dr. Walter H. Schmitt, Jr.[3] Dr. Schmitt stated that G-2 muscle testing is responsive to the taste receptors of the tongue.

Oral insalivation of a zinc supplement was tested when a G-2 muscle weakness was cancelled by therapy localization (TL) to the neurolymphatic for the pancreas. This reflex is located in the left seventh rib interspace at the mid-axillary line. The need for zinc was considered positive if the G-2 muscle weakness was abolished by the oral testing of the zinc supplement. Three different zinc supplements were tested individually. These were Chezyn-Standard Process Labs, Zincate- Nutri-West, and Zn-Zyme from Biotics Research Corporation. In this testing, the patient was considered positive for the need for zinc if one, two, or three of the supplements cancelled the G-2 weakness.

The need for the Right Thoracic Duct Technique was discussed by Dr. Goodheart in the Applied Kinesiology 1987 Workshop Procedure Manual. The patient was considered positive for the RTD Technique if the right PMC muscle weakened with head elevation and inspiration cancelled the right PMC weakness. The patient was then given one of the three zinc supplements to insalivate. If the right PMC weakness was cancelled the patient was considered positive for the need for zinc supplementation.

If a patient demonstrated either of the above needs for zinc supplementation, he/she was then tested with the zinc taste test.

The patient was instructed to give a hand signal as soon as they could detect a dry, mineral, furry, or sweet taste. The patient was then given 10 ml of a zinc sulfate solution an told to gently swish it around in their mouth for 10 seconds. They were timed from the second the container was emptied into their mouth

until they signaled a response or 10 seconds passed.

The response was rated as follows:

1. No specific taste or other sensation is noticed within 10 seconds.
2. No immediate taste is noted, but within 5 to 10 seconds a taste or sensation is noted.
3. A definite taste or sensation is noted in 1 to 5 seconds.
4. An immediate strong and unpleasant taste is noted.

The solution is 1 gm. of zinc sulfate to 1 liter of distilled water. The zinc sulfate was obtained from Nutri-West and is called the "Zinc Taste Test".

TABLE 1

Patient #	Pancreas NL	Oral zinc	RTD	Zinc Taste
1.	+	+	+	1
2.	+	+	+	1
3.	+	+	+	2
4.	+	+	+	1
5.	+	+	+	1
6.	+	+	+	3
7.	+	+	+	2
8.	+	+	+	1
9.	+	+	+	1
10.	+	+	+	2
11.	+	+	+	4
12.	+	+	+	1
13.	+	+	+	1
14.	-	+	+	1
15.	+	+	+	1
16.	+	+	+	1
17.	+	+	+	1
18.	+	+	+	1
19.	+	+	+	1
20.	+	+	+	1
21.	+	+	+	1
22.	+	+	+	1
23.	+	+	+	1
24.	+	+	+	1
25.	+	+	+	1
26.	+	+	+	1
27.	+	+	+	1
28.	+	+	+	1
29.	+	+	+	2
30.	+	+	+	2
31.	+	+	+	1
32.	+	+	+	2
33.	+	+	+	1
34.	+	+	-(LTD+)	1
35.	+	+	+	1
	34	35	34	46-Ave. 1.3

The results are given in table 1. Four categories are reported; a G-2 weakness cancelled by TL to the pancreas NL, G-2 weakness abolished by oral zinc insalivation, a positive need for RTD Technique, and the rating of the zinc taste test.

Only 2 of the 35 (6%) of the patients tested had adequate zinc taste response but demonstrated the need for zinc supplementation by applied kinesiological testing.

31 out of the 35 (89%) subjects demonstrated low zinc taste test response and were positive on both applied kinesiological indicators for the need for zinc supplementation.

Accurate records were not kept as to the number of subjects that responded to the different forms of zinc. I observed that some subjects did not respond to all forms of zinc. It is my opinion that this reflects the individual differences in biochemistry. The possibility that a positive response to oral testing of nutrients may depend on testing with different forms of the same element should be considered in researching this phenomenon.

CONCLUSION

The results of this study support the hypothesis that zinc supplementation may be needed if a patient tests positive for the Right Thoracic Duct Technique and/or G-2 muscle weakness is abolished by TL to the pancreas neurolymphatic.

This would also indicate the need for further testing including larger numbers of subjects and specific research design with statistical evaluation.

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A Comparison of Three Methods of Nutrient Testing

By James D.W. Hogg, D.C.

Abstract: The method of nutrient testing via magnetic exposure through the skin first proposed by Michael Lebowitz[1] is compared to standard oral nutrient testing and skin testing without magnetic exposure.

The method of nutrient testing presented at the Summer 1991 ICAK meeting by Michael Lebowitz[1], while somewhat exotic, suggests several advantages over more conventional oral nutrient testing. Over the years there has existed controversy regarding the most appropriate method of nutrient testing, the two most commonly proposed methods being oral nutrient testing by placing the suspect nutrient on the patient's tongue to stimulate the gustatory receptors and placing the suspect nutrient on the patient's skin. An indicator muscle is then tested to monitor any change in facilitation. More esoteric methods such as placing the nutrient in the patient's "aura" have also been suggested.

This paper reports on a follow up study performed in my office in an attempt to corroborate the findings presented by Lebowitz[1]. In my study three methods of nutrient testing are compared to determine possible correlations. Randomly selected patients were exposed to suspect nutrients by placing the nutrient in the mouth and retesting a muscle that had previously been determined to be weak on a "gamma 2" basis. The same nutrient was then placed on the patient's body, usually on the abdomen and a 5000 gauss magnet was placed over it with the South pole down and the weak "gamma 2" muscle was again evaluated for strengthening. This procedure was repeated a third time placing the nutrient directly on the patient's bare skin without the addition to a magnet and the "gamma 2" weak muscle evaluated yet again. A more limited number of trials was made testing for reaction to various noxious substances (eg. allergens) in which a previously strong muscle was tested for weakening using the above three methods. In trials that showed a change in muscle strength in one or more of the above methods, the results of all three tests were recorded. If none of the three methods of exposure elicited a change no record was made.

Table 1 shows the basic results of these trials. As noted in table 1 both oral and magnet testing showed positive a high percentage of the time whereas simple skin testing demonstrated a much lower percentage of positive tests.

TABLE 1	Positive tests	% of total
Oral testing	86	94.51
Magnetic testing	82	90.11
Skin testing	22	24.18
Total trials	91	100.00

Table two looks at the correlation between the different testing methods a bit more directly.

TABLE 2 - Agreement	# of correlation	% agreement
Magnet and Oral positive	79	91.86
Skin and Oral positive	21	24.42
All three positive	21	24.42

Table three compares these testing methods from yet another angle.

TABLE 3 - Disagreement	# of trials	% of total
Oral positive, magnet negative	7	7.69
Magnet positive, oral negative	3	3.30
Skin positive, oral negative	1	1.10
Skin positive, magnet and oral negative	1	1.10
Oral positive, magnet negative, skin positive	0	0.00
Magnet positive, oral negative, skin positive	0	0.00
Total trials	91	100

One exception to the general findings is in regard to homeopathic remedies. When testing homeopathics and some herbal tinctures there was a much higher correlation between oral, magnetic and skin testing. By the time I noticed the consistency of this finding the project was too far along to start keeping stats based on type of substance being tested and so I have no information on just how consistent this observation was.

Lebowitz suggests that a higher percentage of positive tests are obtained with magnet testing if the substance in question is tested directly over the symptomatic area. I have observed that frequently a negative magnet test will change to positive if tested over a different area, frequently the head.

Likewise, it is likely that a higher percentage of positive results with oral testing might be obtained by testing against specific muscles rather than the more general gamma 2 test performed or against weakness produced by therapy localization to a symptomatic area. For the sake of consistency and reduction of variables, the data in my study was gathered by tests performed using a single muscle and on a specific area when body contact was used.

Discussion

The findings in this study and the one previously reported by Lebowitz[1] present much food for thought. While some questions are answered, others are raised. While there appears to be a high degree of correlation between oral and magnet testing, the correlation is not total. Furthermore, in those cases where there is disagreement, neither method is consistently positive. As our illustrious founder is wont to say, "Why is that?".

Why does magnet testing in some cases seem to show a need for a nutrient that does not show on oral testing. For that matter, why does magnet testing work at all? One obvious advantage of oral nutrient testing is that we at least have some known neurological gustatory pathways that seem to explain how it works. Why do homeopathic and some herbal remedies seem to give more consistent test results than standard nutrients (if, as stated above, actual statistical results would agree with what I <u>think</u> I've observed).

One theory that would answer some of the above questions has it's basis both in modern quantum physics and ancient ayurvedic philosophy. As the nature of matter is examined ever more minutely, the distinction between the smallest subatomic particles and waves of energy become less distinct. Indeed some observations indicate that these smallest particles may cycle back and forth between matter and energy[2]. Since these same particles are the ultimate building blocks of what we call matter, it seems that matter may be just a more stable form of energy. In ayurvedic philosophy all things are considered in terms of their "vibrational" nature. Every herb, for example, is believed to have a specific vibration. Part of the science of ayurvedic healing is to find and provide the herb with vibrational qualities that will create a sympathetic and normalizing vibration in a poorly functioning organ[3].

To carry this line of thought further, it occurs to me that perhaps each nutrient or combination of nutrients has an electromagnetic "signature" as well as a biochemical one. Perhaps running a strong magnetic field through a nutrient is a way of introducing this "signature" into the complex energy patterns of a living organism in a way that may be recognized by the organism. If this were the case it would follow that homeopathic preparations which are energetic rather than biochemical in nature might need less help in the introduction process than other substances more based in "matter". As for the apparent higher correlation between skin testing and oral testing when certain herbal tinctures are being tested, perhaps some of our master herbalists such as Brent Davis may have some insights to share.

While the above line of speculation may be a bit of a "walk on the wild side", I have the feeling that, given the direction of certain types of physical research, this "walk" may be considered tame within the next decade or two. While this discussion is intended more as food for thought than an actual explanation of my observations, the fact remains that the actual observations,

which agree fairly closely with Lebowitz's, suggest a potentially fertile field for further investigation with significant possible payoffs in terms of clinical efficacy.

Summary

The results of this statistical comparison between nutrient/chemical testing via oral, magnetic, and simple skin contact introductions correspond in general with those reported by Lebowitz[1]. There appears to be a high degree of correlation between testing for nutritional and chemical effects when tested via oral insalivation and via magnetic introduction through the skin. When there was disagreement between oral and magnetic testing, neither method was consistently positive, suggesting the possible usefulness of checking with both methods when there are other indicators of nutrient need or chemical/allergen reaction. When these results are compared to testing on the skin without magnetic assistance the degree of correlation is much lower. There appears to be a somewhat higher correlation between simple skin testing and oral testing when homeopathic and certain herbal products are being tested. Lebowitz has suggested several advantages of magnetic testing over oral testing, especially when testing substances to which the patient may have a hypersensitive or toxic reaction.

Regardless of the ultimate explanation regarding pathways or mechanisms for these phenominae, this author feels that the observations and studies to date warrant serious consideration of magnetic nutrient/chemical testing for further studies and clinical application.

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Across the Midline Muscle Testing, Mineral Metabolism, and N-Acetyl Cysteine

Gary N. Klepper, D.C.

ABSTRACT: Across the midline muscle testing and N-acetyl cysteine can be used for non-invasive assessment of mineral status.

INTRODUCTION

Dr. Walter Schmitt, Jr. has described a procedure in which the normal muscle tests for the clavicular division of the pectoralis major and the psoas are modified by placing the distal portion of the arm or leg being tested across the midline of the body and testing with a vector moving away from the midline[1]. A positive test result is considered as a possible functional indication of heavy metal toxicity or hypersensitivity. This is further investigated by seeing if a sample of the herb yellow dock placed on the tongue will negate the weakness[2]. If it does, this is interpreted as further evidence that a heavy metal problem exists, in that yellow dock has been described as a botanical agent for the chelation of heavy metals.

ANOTHER INTERPRETATION OF THE TEST

On checking out this testing procedure in my own practice, I found a high occurrance of positive tests. This led me to be hesitant about the interpretation of the test as indication of heavy metal toxicity, as this would mean a very high incidence of this problem. Also, the herb yellow dock not only can chelate heavy metals, but can favorably influence mineral transport in general.

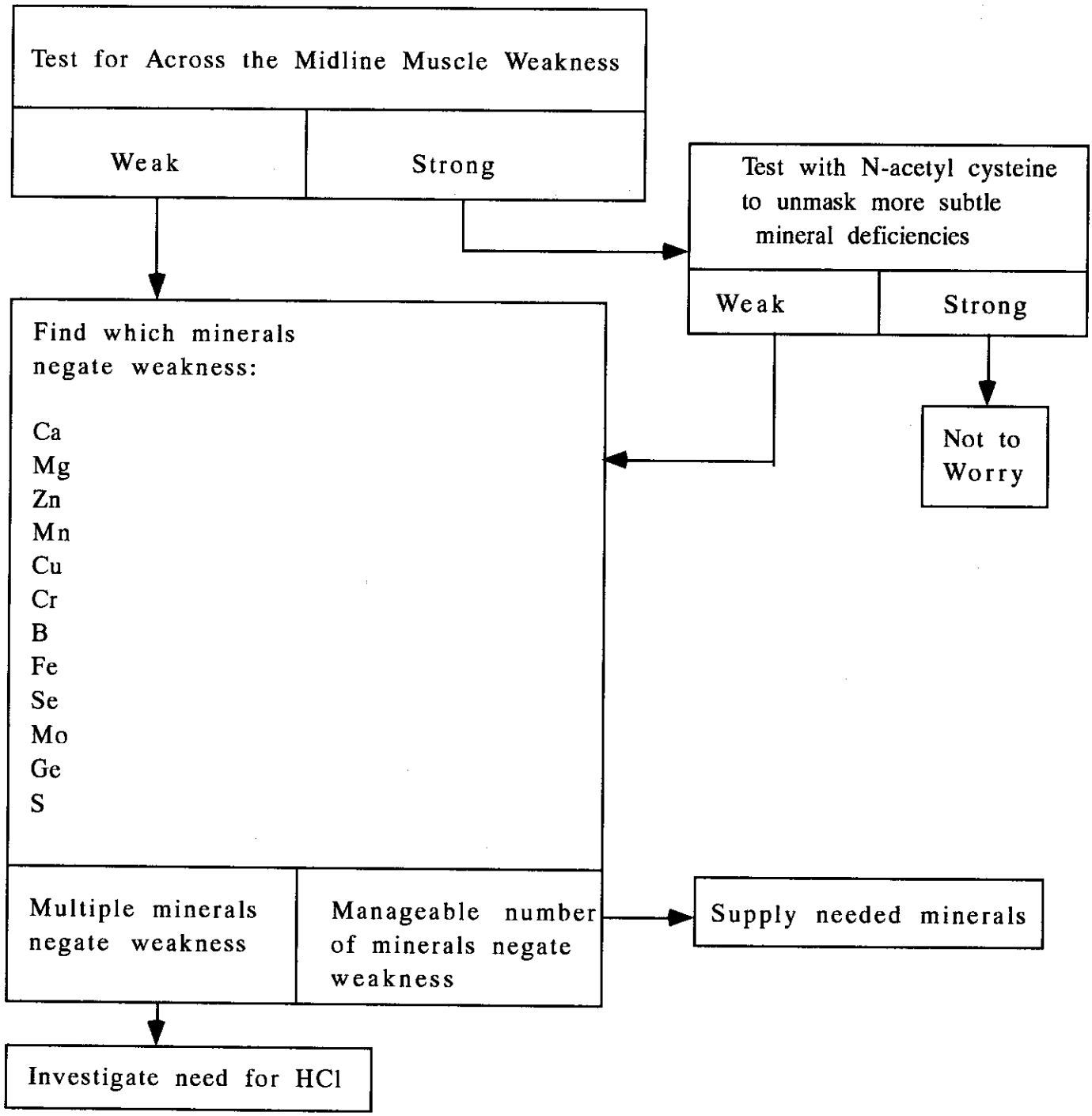
I then selected N-acetyl cysteine as a known agent which does cause mobilization of heavy metals as well as increases mobility of minerals in general (excluding monovalents)[3].

I found a very low incidence of the across the midline test being negated by N-acetyl cysteine. In fact, I found a fair number of positives induced in previously negative patients with this substance. On the other hand, I found it routinely possible to negate the across the midline test with mineral supplements.

CONCLUSION

My conclusion is that the across the midline test is indicative of significant mineral deficiencies in general. This is not mutually exclusive of the interpretation of heavy metal problems, because a very effective method of dealing with low grade heavy metal intoxication is supplementation with optimal levels of the major macrominerals such as Ca, Mg, Zn.

Across the Midline Testing Flowchart



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Neutrophilic Hypersegmentation Index and the Nutrient Activation Indicator

Gary N. Klepper, D.C.

ABSTRACT: The neutrophilic hypersegmentation index is a functional lab assay which will reveal deficiency in folate status and occasionally deficiencies in other nutrients. Findings of this test were compared with a type of therapy localization often interpreted by practitioners of applied kinesiology as being indicative of deficiency in folate status. This particular type of therapy localization was found to involve much more than just folate status, so was renamed by the author the nutrient activation indicator. Using a neutrophilic hypersegmentation index value of 0 as being normal, it was found that restoration of normal lab findings was also much more involved than simply providing supplemental folates. The result has been the development of a simple protocol for assessing the need for nutrients necessary for normal cellular maturity and integrity which includes the evaluation of the liver's ability to activate B vitamins to their coenzyme form.

INTRODUCTION

Dr. Jonathan Wright has suggested the routine use of the neutrophilic hypersegmentation index as a functional evaluation of folate status(1,2). This test involves the counting on a blood smear of the ratio of neutrophils which have hyperlobulated nuclei (4 or more lobes) to the total number of neutrophils. It is conventional wisdom that a certain number of 5 or 6 lobed neutrophils will be present in a normal person (2), so that a value of 0-15% neutrophilic hypersegmentation is often considered to be normal. However, when a patient is supplemented with folates, it is generally seen that this laboratory value tends towards a value of 0, often with a good clinical response in a variety of conditions. After much observation of this phenomenon, my opinion is that any shift from 3 towards 5-lobed neutrophils represents a degree of cellular immaturity and nuclear deformity. Thus it is seen that for folates as for most other nutrients, there is an optimum level of nutrition which may far exceed the average level of dietary intake.

In applied kinesiology practice, a particular type of therapy localization is sometimes observed. This is where an area of the body will therapy localize when one of the patient's hands is placed over the other hand, but this response will not be obtained when the other hand is placed first on the body. For instance, the left hand is placed on an area with the right hand on top of the left hand, and this gives a positive therapy localization response whereas if the left hand is on top of the right hand the response will be negative, or vice versa. This is commonly seen when therapy localizing a very metabolically active part of the body such as the liver. It has been interpreted as being a sort of polarity disturbance that occurs in metabolically active tissue when folic acid deficiency is present.

interpreted as being a sort of polarity disturbance that occurs in metabolically active tissue when folic acid deficiency is present.

On routine evaluation, I find many patients show this finding. I have also found that it does not always respond to challenge with folic acid, but will also often show a response to vitamin B12, iron, and other B vitamins. Of special interest is the finding that when B vitamins are required, it is usually the coenzyme form of the B vitamin needed, and the standard form will not work.

The patients who are most likely to show this finding are those who would by traditional Chinese medicine criteria be categorized as having liver chi and blood stagnation. Common western medicine diagnoses that may correspond include PMS, indigestion, migraines, depression. It is felt that the liver is functionally impaired in its capacity to conjugate folic acid and convert B vitamins to their coenzyme form. Major clinical benefit is then seen when these functions are supported and corrected and the needed nutritional support is given.

In light of these observations, I have named this type of therapy localization the nutrient activation indicator.

PROCEDURE

1- Before performing this procedure, make sure that all basic obvious nutritional needs have been taken care of. These would include water, protein adequacy, or obvious need for general supplementation with a multiple vitamin-mineral product.

2-Test an intact indicator muscle against a liver therapy localization. If the liver TLs in the clear, correct this before proceeding.

3- Test an intact indicator muscle against a liver therapy localization using the particular left over right and right over left hand placements described above. If a discrepancy is noted in the two tests, the patient is a candidate for this procedure.

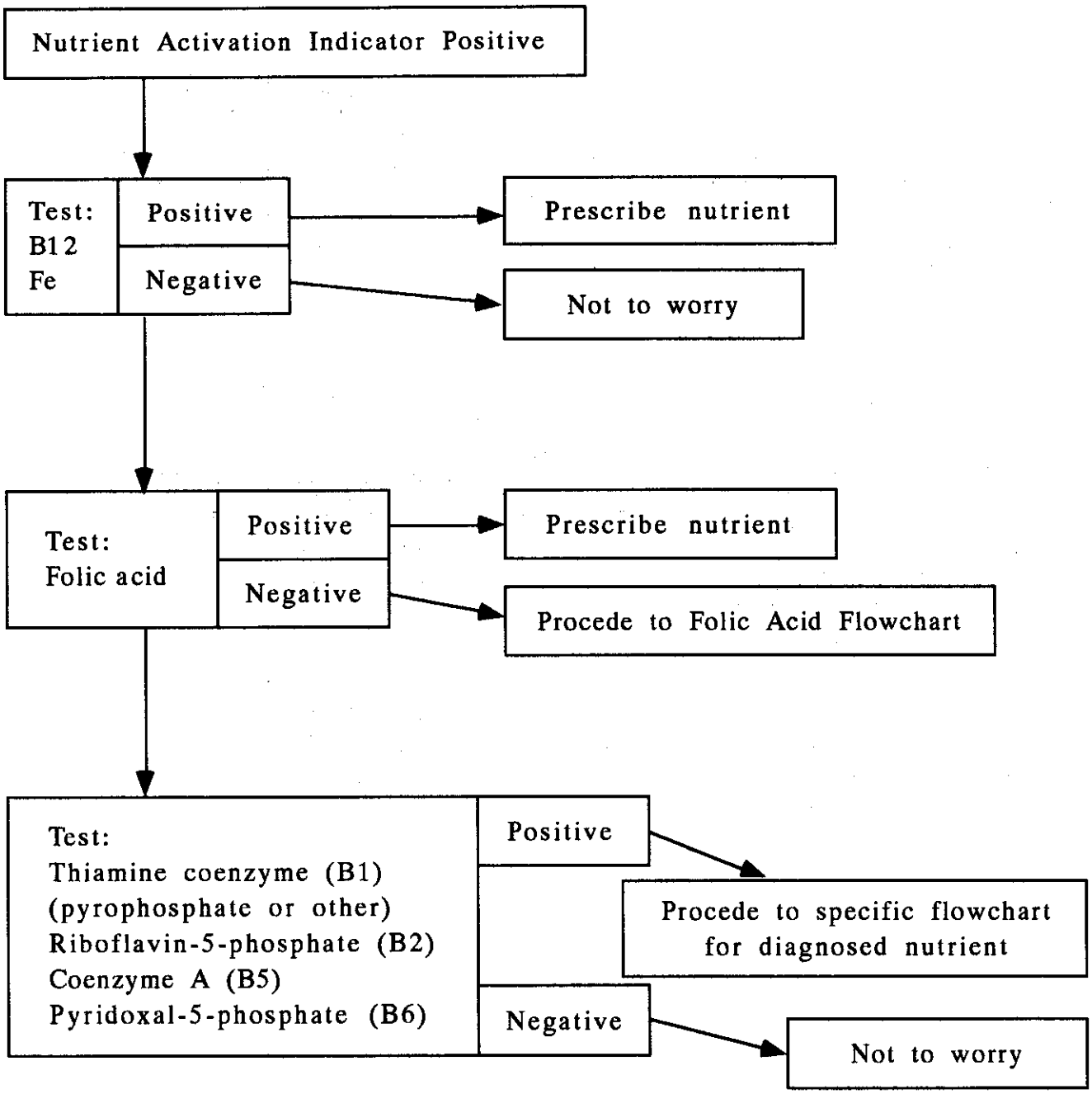
4- Test sources of folic acid, B12, and iron. If positive, prescribe an appropriate level of supplementation. I have sometimes seen as much as 50 mg. of folic acid needed to normalize a neutrophilic hypersegmentation index.

5- Test the co-enzyme forms of B1, B2, B5, B6. If positive, see if the non-coenzyme forms will suffice. If response is only to the co-enzyme form, try testing a combination of the non-coenzyme form and nutrient co-factors needed for activation of the nutrient to its coenzyme form(3). If positive, prescribe these nutrients in appropriate amounts, and also consider short term supplementation with the coenzyme form of the vitamin.

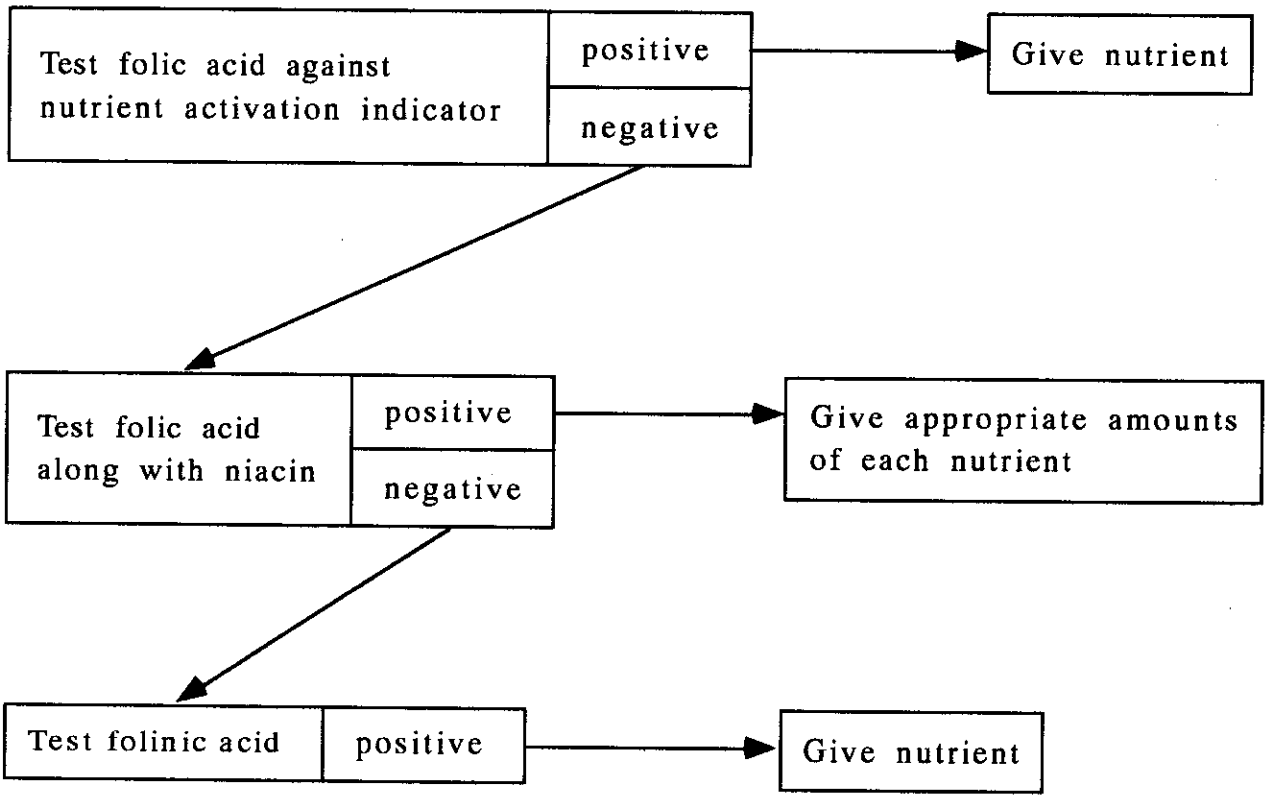
6- If the patient is to be supplemented with the coenzyme form of the vitamin, make sure that no stress is induced in the body by that substance. If stress is noted, it generally means that the coenzyme form of the vitamin is activating metabolic pathways for which the patient has inadequate nutrient reserves of other nutrient cofactors in the same pathway(4). Diagnose and supply those nutrient cofactors along with the vitamin coenzyme.

7- If reading the previous 6 steps is too confusing, then instead refer to the supplied flowcharts.

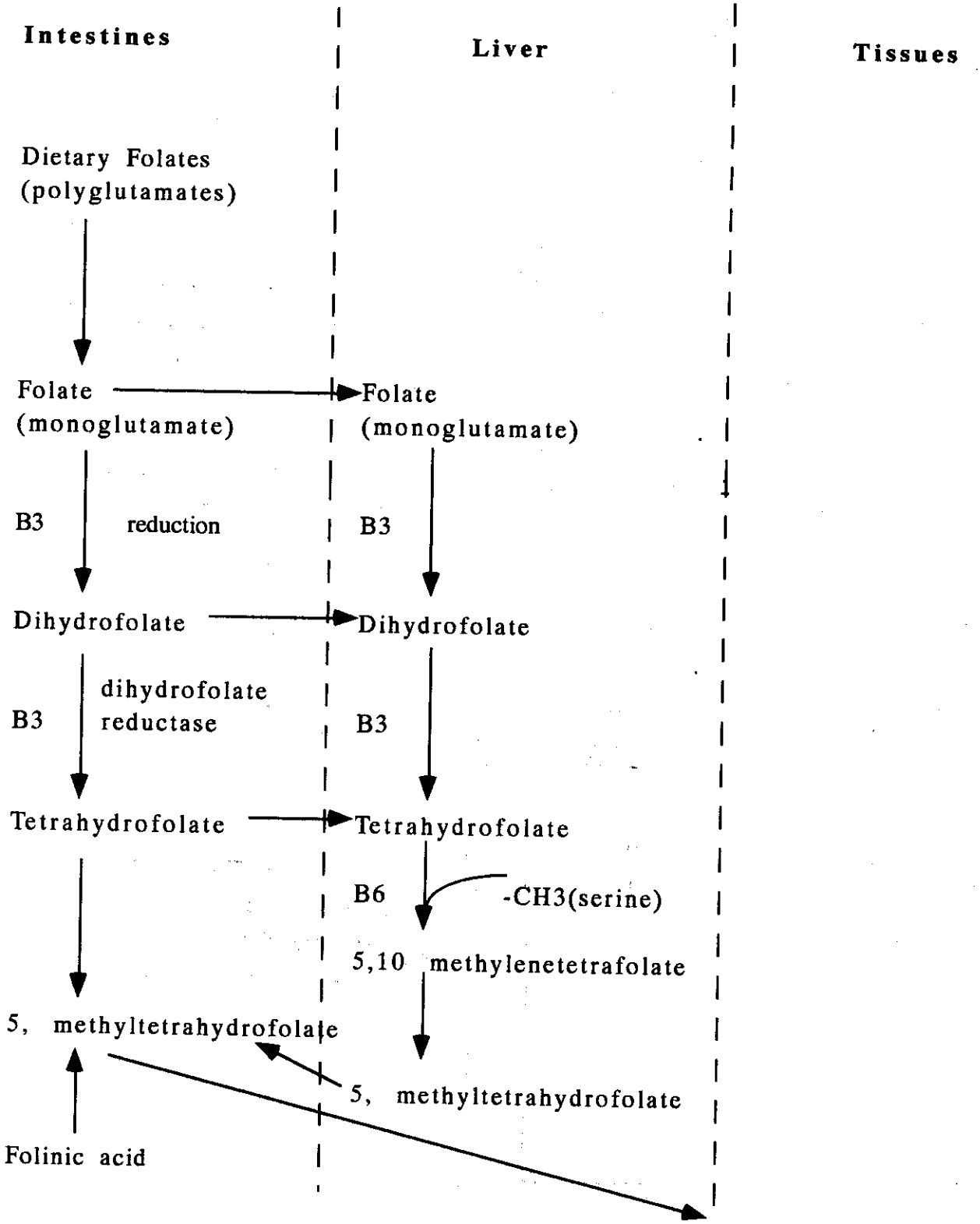
Nutrient Activation Indicator Flowchart



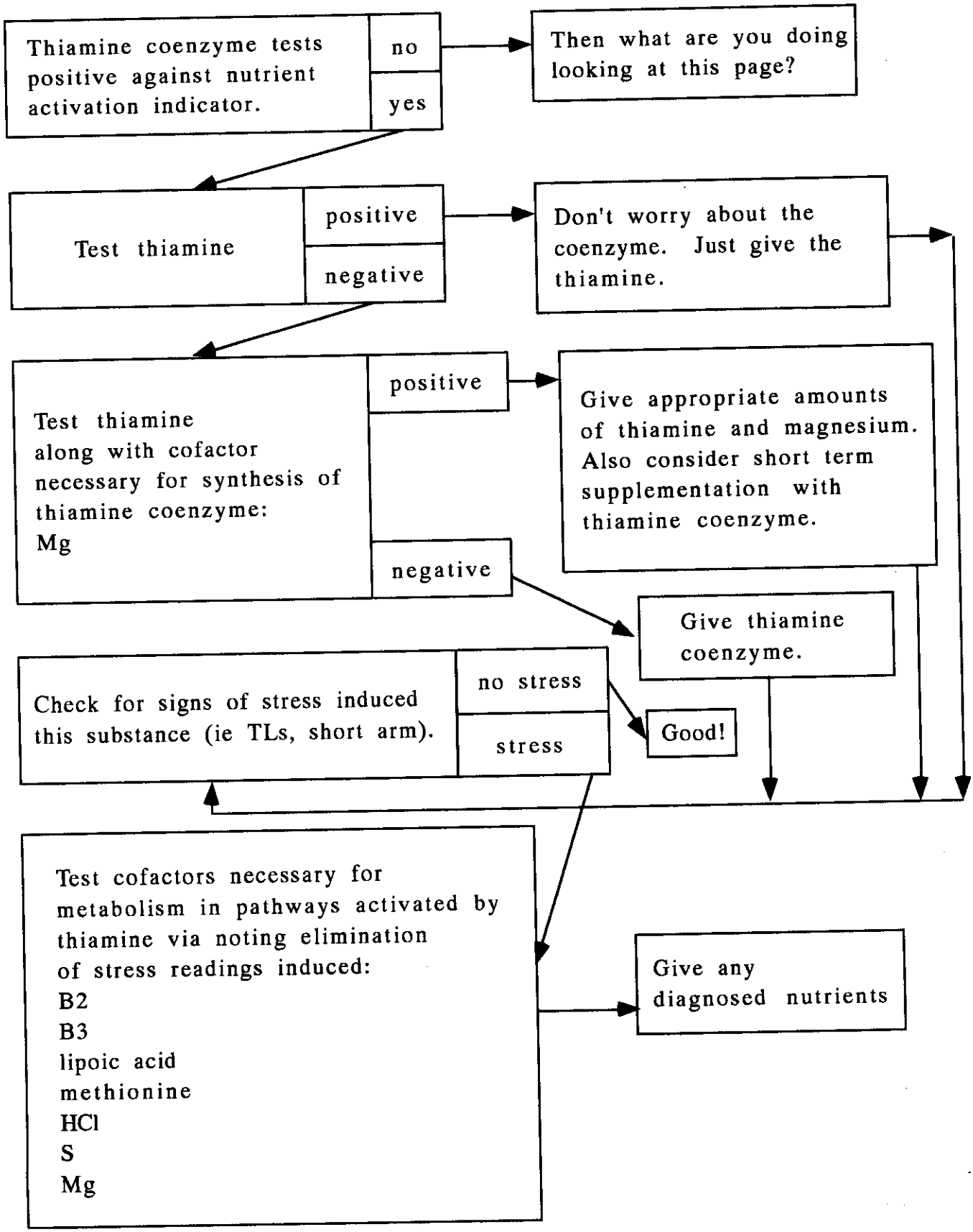
FOLIC ACID FLOWCHART



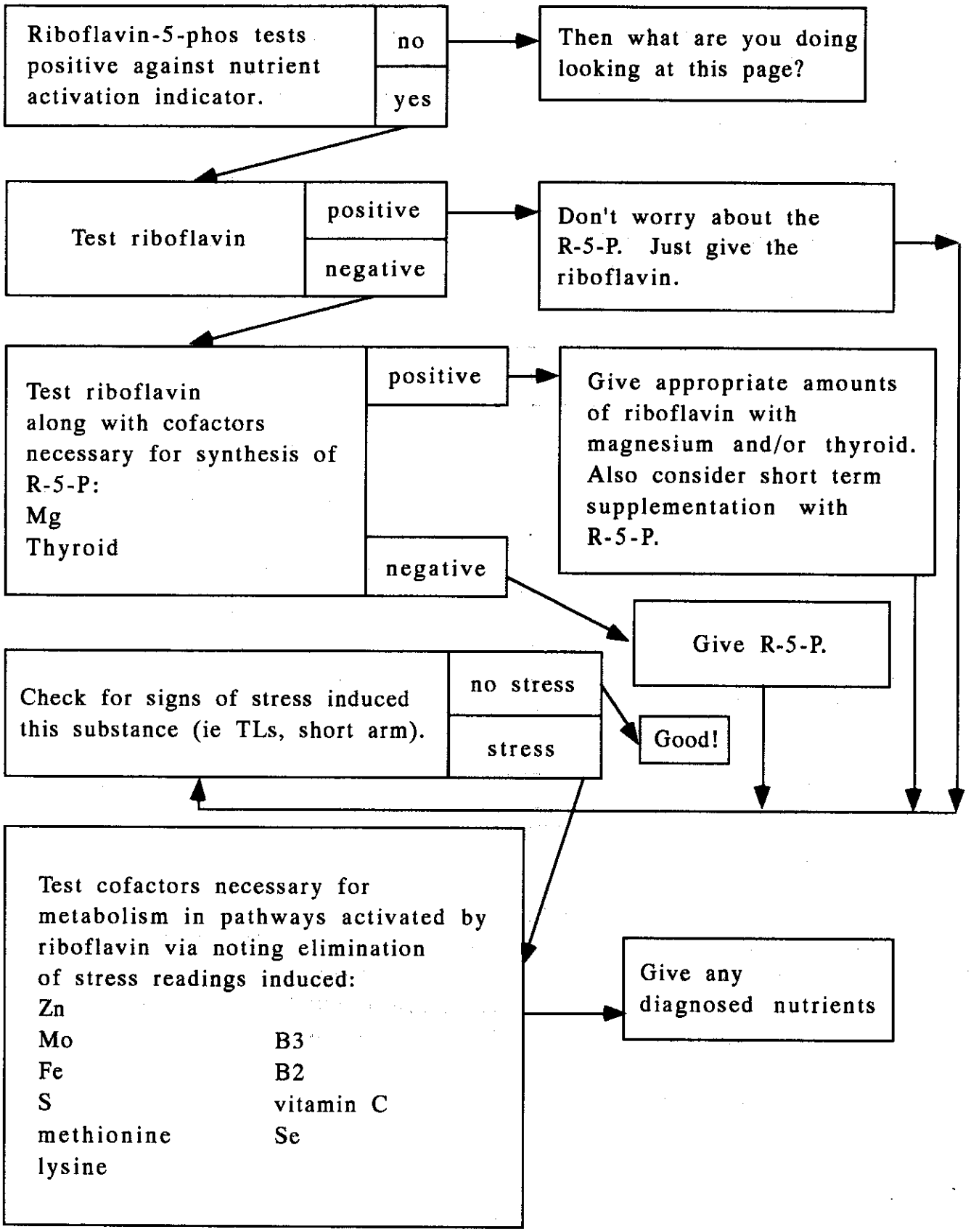
Folate Metabolism



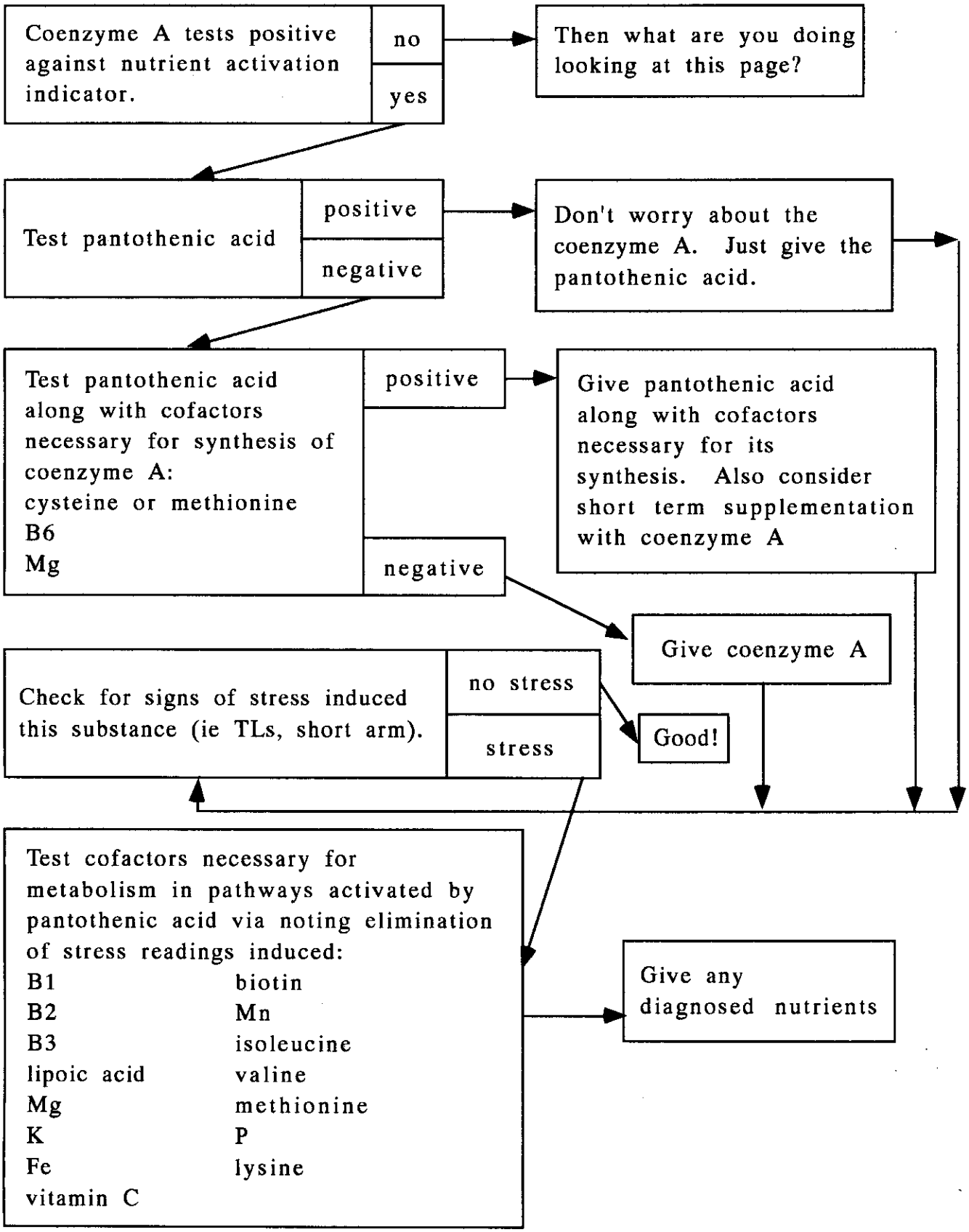
THIAMINE COENZYME FLOWCHART



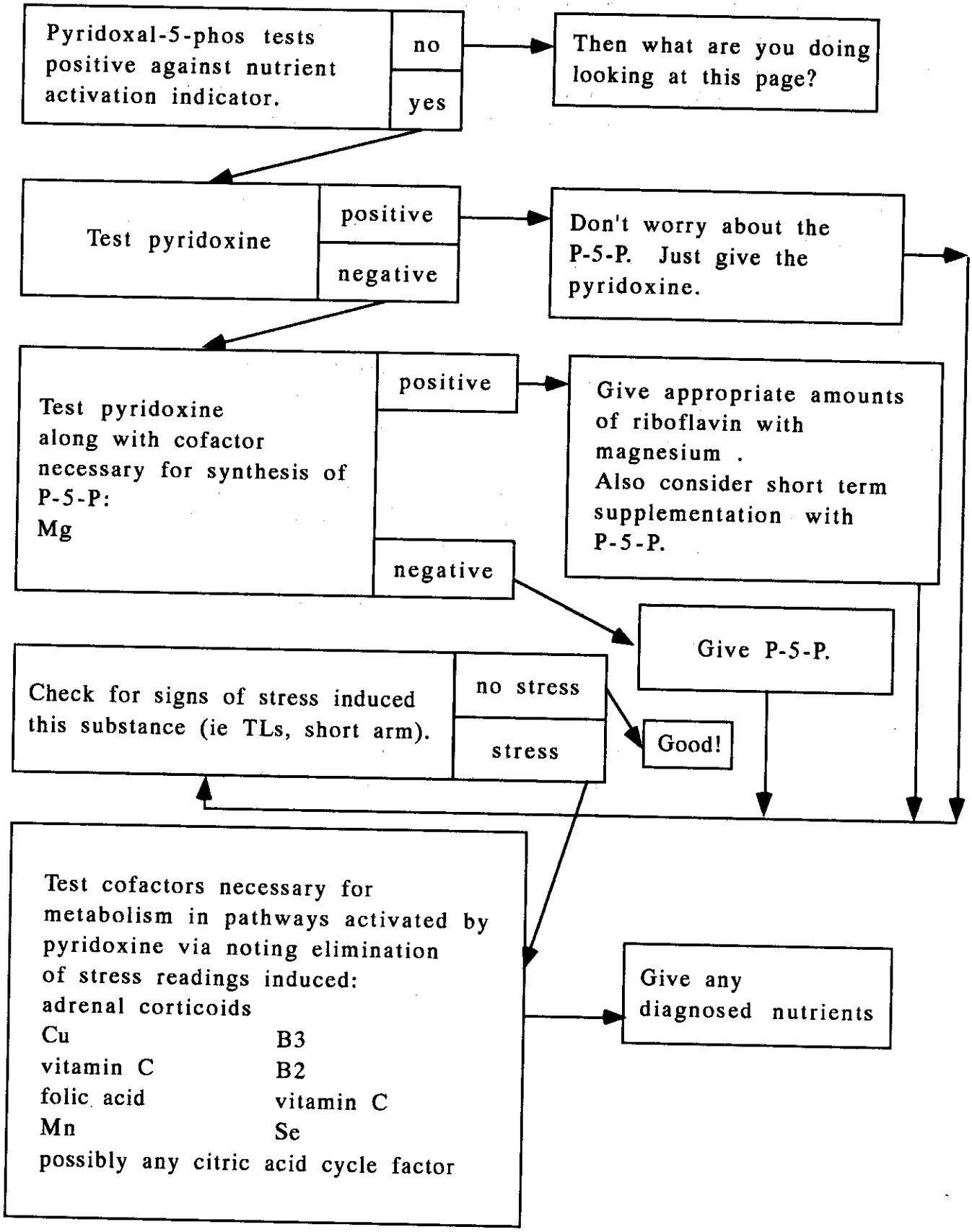
RIBOFLAVIN- 5 - PHOSPHATE FLOWCHART



COENZYME A FLOWCHART



PYRIDOXAL- 5 - PHOSPHATE FLOWCHART



FOLLOWUP

Testing of the nutrient activation indicator will tell you from visit to visit if further nutrient cofactors need to be added to the patient's nutritional program. If you wish to re-evaluate the entire nutrient program, have the patient discontinue supplementation for a few days prior to their office visit.

To monitor long-term progress, recheck the neutrophilic hypersegmentation index at approximately 3 month intervals(5).

CONCLUSION

The nutrient activation indicator if used routinely in a nutrient evaluation procedure will significantly increase the clinical success of nutritional intervention in the applied kinesiology practice.

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1- The Neutrophilic Hypersegmentation Index ("NHI") An Inexpensive, Underutilized Test of Folate Nutrition, Jonathan Wright, M.D. Privately published. Tahoma Clinic, 24030 132nd SE., Kent, WA 98042.

2- Neutrophilic Hypersegmentation as an Indicator of Incipient Folic Acid Deficiency, Terence Bills, F.I.M.L.S. and Lawrence Spatz, Ph.D., American Journal of Clinical Pathology, Vol. 68, No. 2, August 1977.

3- Role of Nutrition in Health and Disease, Cornatzer, W.E., Ph. D., M.D., pp. 259-340, Charles Thomas, Springfield, Illinois, 1989.

4- Ibid.

5- Neutrophilic Hypersegmentation Index can be ordered from Meridian Valley Clinical Laboratory, 24030 132nd Avenue SE., Kent Washington 98042, (800) 234-6825.

ELECTROMAGNETIC FIELD SENSITIVITY SYNDROME

MICHAEL LEBOWITZ D.C.

ABSTRACT:Sensitivity to electromagnetic fields is a fairly common phenomena as we approach the 21st century. As with fungi, foods, and chemicals, the symptoms vary greatly. Diagnosis and treatment is discussed.

There has been alot of press recently about the hazards of EMF's. As with fungi, toxic metals, hazardous chemicals, etc., they pose a threat on both the toxic and hypersensitive level.

Some people claim that all people have indicator muscles weaken when in front of TV's, computers, etc. We have not found that to be the case. We have found that approximately ¼ of our patient load will either 1)have a strong indicator muscle weaken when a source of high EMF's is turned on near the patient, or 2)the field is turned on while GV-20 (which tests negative in the clear) is therapy localized with the south pole of a diagnostic magnet (1). We consider either of the above two to be a positive test. The room you are testing in should not have any other significant EMF's to assure an accurate test. In over 90% of cases testing Pineal Plus (2) simultaneously under the south pole of a magnet will negate the weakness. In rare cases either iron or manganese picolinate, pyridoxal-5-phosphate, or ginkgo biloba extract (2) is used instead.

The usual symptoms we see are fatigue, headaches, irritable disposition, and in many of these cases exposure to high EMF's can undo your work on the patient if they aren't taken into account and treated. The main target organ is the pineal, occasionally the pituitary is involved.

SUMMARY OF PROCEDURES

1. See if a strong indicator muscle weakens when a source of high EMF's is turned on near the patient (TV, hairdryer, etc.). The patient may weaken in the clear or may have to be touching GV-20 with the diagnostic magnet. See if Pineal Plus negates the weakness.
2. Treat the master set points.
3. If appropriate supplement with Pineal Plus (one capsule twice daily, morning and evening). Counsel the patient on decreasing EMF exposure. A Tri-Field Meter (1) is an excellent instrument to locate high EMF sources in your environment. Remember you cannot shield out EMF's.
4. A patient sensitive to a battery operated watch (or heart monitor) usually only shows this by placing the S pole of a diagnostic magnet on the watch while the patient is wearing it. In a sensitive patient a strong indicator muscle will weaken. Pineal Plus again will most often negate the weakness.

Conclusion : Sensitivity to EMF's is another important factor to consider in the chronically ill patient.

Sources

1. Mid American Marketing P.O.Box 124, Eaton, Ohio 45320, 800-922-1744, 219-749-6666
2. Thorne Research P.O.Box 3200, Sandpoint, Idaho 83864, 800-228-1966, 208-263-1337

JUST BECAUSE A SUBSTANCE STRENGTHENS A WEAK MUSCLE, DOES THAT MEAN IT IS GOOD FOR YOU?

MICHAEL LEBOWITZ D.C.

ABSTRACT: Many substances harmful to a patient may strengthen a weak muscle due to its short term biochemical or endocrine stimulation, though in reality it is detrimental.

We are all familiar with the lay muscle tester who does an arm pull down test with a sugar cube in someones mouth and states that everyone weakens on sugar. In reality we know that an accurate muscle tester will find a certain percentage of patients weaken on oral insalivation of white sugar, a certain percentage will have no muscle test change, and a certain percentage will strengthen on white sugar. Do those that strengthen have a white sugar deficiency and should we supplement it three cubes, twice daily? Of course not, but what many of us feel this indicates is that many of these patients may be suffering from low blood sugar at the time of testing. Since white sugar will temporarily elevate the blood sugar level, it is strengthening the weak muscle. In the long run though it prolongs the problem and should be avoided in the hypoglycemic. Here is where proper interpretation of the muscle test result is critical.

A certain percentage of food sensitivities are delayed reactions, where the patient may actually feel more energetic and a sense of well being after ingesting the food, only to crash a few hours later. Many patients through careful detective work have recognized their sensitivities to these foods. Interestingly on muscle testing, Many of these foods do not weaken strong muscles on either oral or biomagnetic testing. What we did find though is that these foods strengthen weak G-2 muscles in many cases.

Kurt Vreeland D.C. discovered that in some of his patients, if they chewed and swallowed a suspect food that didn't weaken a strong muscle and then took a second sample and did oral testing, it would now often cause muscle weakness. He presented this in a seminar we co-taught in 1991. This idea lead me to find that simultaneously testing two samples of a food biomagnetically, one over the skull, the other over the abdomen, will often bring muscle weakness when either food tested separately will not. This can be a valuable tool though very time consuming. Further experimentation showed that foods that weakened on either of the two double sample tests explained above but not on a single sample would actually strengthen a weak G-2 if tested singularly.

This has evolved into our present procedure where we screen for food sensitivities by checking for both a weakening of a strong muscle and a strengthening of a weak G-2- any muscle test change is considered a positive test. Doing this for over a year I have found about 50% of all positive foods cause muscle weakening, the other 50% muscle strengthening. Leaving strengthening foods out of the patients diet (along with weakening foods) while correcting the food sensitivities has brought major clinical improvements in difficult patients that were not making as much progress before. I feel it is an essential part of neuromuscular hypersensitivity screening for foods. The following page is our food screening procedure as taught at our fall 1991 Denver seminar.

CONCLUSION: To accurately access food hypersensitivities, foods must be checked for both strengthening and weakening- either change being considered a positive test.

1. A person is sensitive to a food if the antigen[1] either strengthens a weak gamma-2 muscle * or weakens a strong indicator muscle if placed under the south pole of a 4x6 magnet over GV-21 or the patients most symptomatic area. Check for both. Any muscle strength change is a positive test.
2. If positive see if either Basic Nutrients IV[2], Pyridoxal-5-Phosphate[2], zinc or copper picolinate[2], or B_3 complex[2] negates the weakening or strengthening caused by the positive food. Whatever nutrient does is the proper one to give.
3. Tap the master set points ten times each. The foods should now test negative.
4. Have the patient avoid the positive foods for three weeks.
5. Supplement as follows with whichever nutrients were appropriate:
   Basic Nutrients IV- 3 capsules twice daily with meals
   Pyridoxal-5-Phosphate 1 capsule 3 times daily (twice daily if also on Bas.Nut.)
   Zinc Picolinate 30 mg. daily
   Copper Picolinate 2 Mg. daily
   B_3 Complex- one daily (none if on Basic Nut.)
6. Retest foods in three weeks. If negative put one food back daily to make sure there are no adverse reactions. If any do cause reactions retreat.

* Food antigens that strengthen a weak gamma-2 muscle are usually of an addictive nature biochemically for that patient. They will weaken a strong indicator muscle on a double test (2 samples of the food tested simultaneously, one on GV-21 one on abdomen, both under magnets). Occasionally you will find a food that strengthens a patient because it is beneficial. In these cases it will not weaken on a double test (this is not a common finding).

Sources

1. International Biologicals 1-800-245-5729
2. Thorne Research 1-800-228-1966

THE MASTER SET POINTS

MICHAEL LEBOWITZ D.C.

ABSTRACT: The master set points can reset or correct virtually any "turned off" neurological circuit in the body: NL's, set points, cranials, and desensitize to foods, fungi, toxic metals, chemicals, etc. if done in the proper manner.

Development of the Discovery

Richard Meldener D.C. and I were discussing the fact that the earths magnetic field has decreased from 4 gauss to approximately ½ gauss in a relatively short span of time and the possible effect of this on human health. I was contemplating exposing patients to a 4 gauss field to measure its effects on muscle strength. He made me aware of works by Robin Baker (1), Robert Becker (2), and others , and the possibility of humans having magnetite crystals somewhere in the vicinity of the ethmoid sinuses. These crystals are what give homing pigeons their abilities, etc. Richard had been experimenting using pre 1964 nickels (there may have been a composition change about that time) which he found had a natural field of up to four gauss and placing them on the supraorbital notches (on the eyebrows directly superior to the pupil and in some cases slightly medial) and suggested I do the same.

What I found was that if I placed a pre 1964 nickel on either or both supra orbital notches that any muscle test weakening due to a positive food, fungi, chemical, metal, etc. would be abolished while the nickel was in place. Interestingly these points would not often show positive on regular therapy localization. Searching the cranium I found that GV-20 which I previously recognized to be a pineal NL (3) exhibited the same pattern.

The thought of all my patients wearing nickels on these points was amusing (and it actually did help their symptoms) but its limitations were obvious. Experimenting one day, I tapped all three points and found that they abolished positive findings for foods, fungi, metals, chemicals, electromagnetic fields, if and only if I had prechallenged the patient for these items (brought these problems to the patients memory neurologically). This was getting to be quite interesting so we continued to explore.

We found that any hypersensitive substance for that patient tested within the course of that visit would have the positive test abolished by tapping the points. The patient did not have to be exposed to the substance during the tapping. Emotions that caused muscle weakness, even many cranials if prechallenged would correct. Regular G-1 muscle weaknesses would strengthen only 20% of the time and NL's prechallenged with a diagnostic magnet (4) would not correct.

Nutritionally we saw a pattern emerge. In 90% of the first 100 or so cases testing either iron or manganese picolinate (5) biomagnetically over these points (often nowhere else on the body) would strengthen the weak G-2 as would testing Pyridoxal-5-Phosphate (5) over GV-20 also. This led to our formulating a product called Pineal Plus (5) which tested even better than the individual products (synergy) and we feel it nourishes this electromagnetic system. It also does wonders in correcting sensitivity to electromagnetic fields (6).

Present Status

At this point we have found that the best way to treat the master set points is with a laser (4). We have found that treatment of the points with a laser not only will correct all pretested foods, fungi, chemicals, metals, etc., but it will also correct all prechallenged NL's, most prechallenged G-1 muscle weaknesses, cranials and subluxations and almost any other type dysfunction if prechallenged. The laser gives a "deeper" fix than tapping and is well worth the cost.

We have added GV-21 as a fourth point to be treated. Though it is of a different nature (only responds to the nickel phenomena 20% of the time) than the other three points, it is essential to help stabilize them. Otherwise rubbing GV-21 often bring back the positive findings.

The only problem if you'd like to call it one is that everything is so simple. I have not had to treat a regular set point or go through any of our former desensitization procedures in quite some time. The time saved and simplification is phenomenal. A few hundred other physicians have already been using these and are reporting similar results.

On return visits if some findings return or you get new ones you just need to treat them again. The master set points do not necessarily work better than other treatments but they work as well and are alot quicker. They do tend to be less stable in patients exposed to high EMF's but the Pineal Plus can often take care of that.

THE PATIENT STILL NEEDS TO FOLLOW ALL APPROPRIATE DIETARY INSTRUCTIONS (AVOID POSITIVE FOODS FOR THREE WEEKS, ETC.) AND TAKE ALL APPROPRIATE SUPPLEMENTS THAT NEGATED THE FOODS, FUNGI, ETC. AND/OR STRENGTHENED THE WEAK G-2.

Conclusion: The master set points are higher order CNS circuits that can take the place of many other A.K. treatments thus saving time without sacrificing quality.

SUMMARY OF PROCEDURES

1. Find a G-2 weakness and a strong intact muscle.
2. Do all the diagnostic workup in our protocol, determining sensitivities, dysbiosis, supplement needs, dietary changes needed, etc.
3. Place a bottle of Pineal Plus 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 and help make the patient more stable by electromagnetically balancing them.
4. The master set points are located bilaterally on the eyebrows directly superior to the pupil (occasionally slightly medial). The 3rd and 4th points are GV-20 and GV-21. Treating them with a 670 nm laser for ten seconds each is the best way to treat them. All positive foods, fungi, metals, chemicals, NL's, etc that were pretested during the course of that visit will now test negative (exposure to them during treatment is not necessary). The G-2 muscle will now be strong.
5. Supplement with Pineal Plus (one at breakfast, one at supper) if indicated.

6. On future visits if G-2 findings come up, the master set points can be used again to treat your findings. Rarely do we need to fall back on previous type treatments.
7. Remember master set points do not always therapy localize but should be used regardless. Patients still need supplements and dietary instructions.

Resources and Bibliography

1. R. Robin Baker, Janice Mather, and John Kavanaugh, "Magnetic Bones in Human Sinuses", Nature, Vol. 301, 6 January 1983.
2. Robert Becker and Gary Selden, The Body Electric- Electromagnetism and the Foundation of Life, New York, Quill, 1985
3. Michael Lebowitz, Sporadic Newsletter #3, September 1990
4. available from Mid American Marketing P.O.Box 124, Eaton, Ohio 45320, 800-922-1744, 219-749-6666
5. available from Thorne Research, P.O.Box 3200, Sandpoint, Idaho 83864, 800-228-1966, 208-263-1337
6. Michael Lebowitz, "Proceedings of the ICAK-USA", Summer 1992, paper tentatively titled "Electomagnetic Field Sensitivity Syndrome"

TOTAL LOAD AND MUSCLE TESTING

MICHAEL LEBOWITZ D.C.

ABSTRACT: There are an unlimited number of variables that can effect muscle testing response. Some of the important ones are discussed.

We all have a certain threshold of "stuff" we can handle without developing symptoms of ill health. "Stuff" includes emotional stress both past and present, nutrient deficiencies, environmental toxins, dysbiosis, subluxations and other short circuited neurological reflexes, etc. When the sum total of our "stuff" passes our threshold to handle it, we develop symptoms. Some days eating wheat may cause reactions, other days it does not; again depending on the sum total of all the other "stuff" at the moment of ingestion. Similarly, some days wheat may weaken on a muscle test while other days it does not, again depending on the other "stuff".

As physicians our role is to diagnose as much of the patients "stuff" as we can, correct subluxations, turn on short circuited neurological reflexes, correct nutritional deficiencies and imbalances, etc., and educate the patient on ways to decrease the rest of their "stuff". When testing a patient we do not want to miss anything. Sometimes unknowingly we lower the patients total load in our office and get some false negative readings.

To give an example, a 35 year old man suffering from fatigue and headaches came to my office. We took off his glasses and proceeded to test him and found a sensitivity to microsporum that suggested intestinal dysbiosis. He returned three weeks later to say he was now symptom free Monday through Friday but still symptomatic on weekends. Questioning brought to light that he spent lots of time in the garden on weekends, so I immediately thought of mold exposure being elevated and with dysbiosis, mold sensitivity to inhalant molds is a common accompanying symptom. Testing was negative. More detective work and we learned that his red tinted glasses were worn only on the weekend. We had him put them back on. They did not cause muscle weakness but they brought back the positive test on microsporum. Desensitizing him with his glasses on corrected the remaining symptoms. As an interesting aside, months later I was reading a book by Harry Spitler M.D.[1]. Spitler did an experiment raising goldfish in an aquarium under red lights, comparing it to a group raised under balanced light (all other variables equal). The group raised under the red light developed fungal growth between their scales and eventually died. I'll have more to say about Spitler and his work in future papers.

The point of the case history is if possible have the patient leave their glasses on during testing. Since eyeglasses selectively filter some frequencies of light more than others, it is stressful to the person wearing them and part of their "stuff". Removing them during testing lowers their load and you may miss some findings especially biochemical and environmental that are there.

Another way we may miss positive findings is by having full spectrum lights in our office. If your patient works under fluorescents or incandescents it is not as balanced as full spectrum lights and the therapeutic effect of these full spectrum lights can block positive findings while muscle testing your patient. If the lights are under a plastic covering it is OK since that blocks the full spectrum effect (but they are worthless as a

healthful lighting source at that point). Ideally we should test patients under the type of lighting they live under though realistically I choose incandescent in our office. It is fairly neutral and though I might get a few false negative tests on those patients that spend their days under fluorescents, I do not want the constant exposure to the EMF's and the unbalanced spectrum for myself. It appears that the ultraviolet in the full spectrum is the positive influence that blocks some findings (more on that in a future paper).

A third variable we must consider is the patients air quality at home and at their job. At my office we try to keep our air as clean as possible. We chose materials of low toxicity to build with and we take steps to keep the mold count low. Many homes and offices not only have high mold counts but also substantial outgassing from particleboard, paints, carpets, etc. Many patients away from the toxins of their house or office will test better than in them. We noticed that in our biomagnetic protocol that occasionally a patient would weaken on an air sample from home or work. In that case we had to determine if it was a chemical or mold and treat it accordingly. More interesting is that in quite a number of patients the air sample would test negative but if you left the patient exposed to it (under the south pole of the magnet) when you did your other testing it would make certain NL's, foods, nutrients, etc., test positive that wouldn't if the air sample was removed. In other words, exposing the patient to their air would increase their total load and give you more accurate findings.

These are just three of many possible variables that effect your findings. The expert practitioner must be a detective and make the patient "right at home" so to speak to get the best results possible.

CONCLUSION: Making the patients light and atmospheric environment like that they experience at home and/or work may be critical in bringing forth findings that would otherwise elude us.

References

1.Harry Riley Spitler, The Syntonic Principle, Eaton, ohio, College of Syntonic Optemetry, 1990

A FOLLOW-UP STUDY OF APPLIED KINESIOLOGY IN THE TREATMENT OF LEARNING DISABILITIES

by Harry Lefkowitz, D.C. and Jacob Lefkowitz, M.A.Ed.

ABSTRACT
In the Summer 1989-90 Collected Papers, a paper was presented by the above authors entitled, "A Study of Applied Kinesiology in the Treatment of Learning Disabilities and Dyslexia."[1] A follow-up study is being presented to establish whether or not the treatments used on the original twenty-one patients in our study had a long-term benefit. A number of possibilities are presented.

1. Did the eighteen patients who improved retain their improvement?

2. Of those who did not show improvement originally, did any show improvement later?

3. Did any patients decline?

4. Is there any age group of patients who respond better to applied kinesiology treatment than another in regard to learning disabilities?

INTRODUCTION

This follow-up study of our original paper was done to chart the progress of the twenty-one patients who were in our original study. This is important, as a true test of any procedure or treatment for learning disabilities or dyslexia is the long-term progress of the patient.[2] Short-term results that are found immediately or shortly after treatment procedures can be relegated to a placebo effect. Often an improvement is felt to have occurred because something different is being done or special attention is being paid.[3] Long-term improvement is indicative of improvement due to a change in structure, function of the nervous system and perception.[4,5]

METHODOLOGY

A questionnaire was developed by which an interview could be conducted by phone. The patient or parent was contacted by phone to determine the present academic status or employment status of the patient. Also, the patient or parent was asked if the original problem had improved, stayed the same or declined. Information about current grade level, coordination, sports activity and extracurricular activities was elicited. If the patient was a child, the parent was

asked about home behavior. All patients and parents (if a child) were asked to what they attributed the change in performance.

RESULTS AND DISCUSSION

We were able to contact 20 of the 21 patients in the original study. The information gathered was more of a subjective nature than our previous paper. In the original paper, all our results were based on post-test scores.[6] No information about performance, academic status or how the patient felt was taken into account.

In this study, all information was gathered through an interview with the patient or patient's parent. Although some of the information is subjective, much of it is objective, such as grade status and special education requirements.

An interesting development was that different results were obtained in this type of interview than was gathered by the previous study.

Two patients who scored significantly higher on the original post-test felt that they were not making improvement over the years. These patients fell into a specific age group, and my theory on this will be discussed later.

Three other patients who showed no significant improvement on the original study were significantly improved at this time. My theories on this will be discussed as well.

Out of the twenty patients who were contacted, sixteen reported improvement.

Out of the four who did not show improvement, two were over 50 years of age (51 and 58), one was later found to have a mild retardation problem and one had polycystic kidney disease.

The three patients who showed improvement now but did not at the original testing also have an interesting history.

First there was a boy who was 7 years 11 months of age at the time of his entry into our program. At that time he had a history of problems in reading comprehension, fine motor control, gross motor balance and bedwetting. He had speech and articulation problems as well.

After treatment he showed no significant improvement in his post-test results.

In a recent interview with his mother, she stated that he no longer had speech difficulties, he is able to run, has much improved motor control and does not wet the bed. His mother attributed the results to applied kinesiology treatment and improved conditions at home.

At the time of his entry into our program, he was in a very bad family situation. The parents were in the midst of divorce proceedings and his father was invalidative to the child. He considered the child "stupid" for having these problems. The father was living at home at the time. Currently the divorce is final, and the father is no longer living at home. In this case, the continued invalidation from the father was suppressing the treatment from being effective. When home conditions improved, the improvement in neurologic function resulted in improved performance.

The second case was a child who was mildly autistic. She was 10 years 7 months old at the time of entry into our study. This child talked very little, could not read, and had a very poor attention span. No significant improvement was noted in the original study.

However, in the recent interview with her mother, improvement was reported. She was able to read and talk. The child was still in a special education program, has poor grades, and her attention span is still short. However, considering the severity of her case, the improvement is dramatic.

The third case was also severe. This was a 6 year 8 month old boy. He had finished first grade but could not read, write or recognize the alphabet. He was programmed to repeat first grade. He started treatment in April of 1987 and completed treatment in October of 1987. During that treatment, he also received tutoring over the summer. He was retested in July of 1987 and followed up with monthly treatment until October of 1987. His post-test showed no significant improvement in reading skills. However, in October of 1987 his skills improved to the point that he was placed in second grade.

The recent interview with his father was positive. He stated that his son was doing well in school with average grades and reading skills.

In this case, I feel that it took longer than usual for the nervous system to respond due to the severity of the disorganization and perceptual problems.

This and the previous cases imply the importance of follow-up as improvement and healing may take longer than we expect.

In the four patients who reported no improvement, special circumstances also exist. Two of these patients were over 50. Their post-test showed significant improvement. However, they did not feel that they made any progress. In these cases it is possible that the neurological disorganization was set deeply in their nervous systems and they therefore could not achieve neural organization. Or perhaps they did become more organized but after elapsed time of a certain self-perception of educational ability, change could not occur.

The other two cases were also unique. One had polycystic kidney disease. She had continual toxins flowing into her body from poor kidney function. This was probably causing her disorganization pattern, and it could not be corrected by applied kinesiology.

The other patient was later diagnosed to be moderately retarded and probably should not have been in our study in the first place.

A few other cases are of interest. These patients had major improvement to the point where they now have A averages in school. A few patients who were in Special Education programs are now mainstreamed.

Three patients were college students who were failing or were not doing well. One patient went on to excellent academic performance but fell prey to drugs and alcohol and had to go to a drug rehabilitation program. Another case improved in coordination, comprehension, memory and sports. His academic performance remained poor, but he scored high on academic achievement tests. His parents feel that he had lack of ambition but that the treatment was effective in the aforementioned areas.

CONCLUSION

Sixteen out of the twenty patients contacted showed improvement. This reflects an 80% result. A few conclusions can be made about the results obtained.

Two patients who did not feel they improved but scored higher on the post-test were over 50. It is possible that age may have a bearing on the expectancy of results. One patient who did not respond was found later to be moderately retarded. A diagnosis of retardation should be a reason for a low expectancy of results. Finally, one patient had polycystic kidney disease. It may be that a severe illness such as this should lower expectancy of improvement.

On the other hand, three patients with a severe learning disability who did not show improvement initially improved at a later than expected time. In such cases, it would be wise to tell patients or their parents that results will be forthcoming but to expect a longer time frame for improvement to show.

One of these patients was in a very bad home situation and improvement came when the home situation improved. If such a situation is detected, it would be wise to counsel the parents that results will come when the family situation improves.

This study is a validation of applied kinesiology techniques[7,8,9] in the treatment of learning disabilities and dyslexia and that long-term results are available. However, more studies such as this need to be done.

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Functional Hypoglycogenesis

Philip Maffetone, D.C.

Abstract: Certain individuals, particularly those with blood sugar handling problems, functional hypoadrenia and/or liver function difficulties, may be unable to effectively replace used glycogen stores, thus intensifying their blood sugar problem and disturbing normal muscle performance. This paper provides a simple applied kinesiology protocol to evaluate those susceptible individuals, and proposes effective corrective measures.

Introduction

The process of forming glycogen from glucose is called *glycogenesis*.[1] It occurs in almost every tissue in the body, with liver and skeletal muscle being the most active sites, storing as much as 200 — 300 grams of glycogen.

In the liver, glycogen helps maintain normal blood sugar levels. After 12 — 18 hours of fasting, liver glycogen becomes almost totally depleted. Some depletion also occurs between meals, during daily activity, and during any exercise, with more depletion occurring with more intense activity and exercise. Replacement of liver glycogen stores are rapid following a meal high enough in carbohydrate. For most people, a daily diet of at least 60% complex carbohydrate should provide adequate replacement of glycogen for both liver and muscle.[2]

Muscle glycogen contributes little, if any, to blood sugar. Instead, it is used as a readily available source of glucose within the muscle itself.[3] Under normal situations of activity and exercise, muscle glycogen may take 24 to 48 hours to replenish its stores, depending on intensity. Without available glycogen and glucose within the muscle to meet a demand, muscle dysfunction may occur, and excess blood glucose may be used to compensate.

Potential sources of glycogen stores are numerous, and include all three food groups.[4] Most sources act through their conversion to glucose. As already noted, dietary carbohydrate is the most common source. It should be noted that a diet comprised of simple (refined) carbohydrates will contribute *less* to glycogen stores than complex carbohydrates.[2] Proteins can also contribute to glycogenesis. As much as 58% of the protein intake may be converted to glucose. Also, the deamination of some amino acids, such as alanine, yields pyruvic acid which in turn yields lactic acid. Both acids, as well as lactic acid formed in anaerobic muscle metabolism, are potential sources of glycogen. Fats can also contribute to glycogen formation — the glycerol fraction of fats can be converted to glucose. Also, small amounts of carbon dioxide formed within the body can be used for glycogenesis.

Various hormones are important regulators of glycogen. The most important is insulin which lowers blood glucose and increases glycogen storage. Epinephrine and norepinephrine, through the beta-adrenergic receptors, as well as glucagon, stimulate the release of stored glycogen in the liver to raise blood sugar.

The biochemistry of glycogenesis is a series of complex events. The process of phosphorylation is a key step in converting glucose to glycogen. This begins with the

addition of a phosphate group to glucose, requiring phosphoric acid, magnesium, ATP and insulin dependant glucokinase. After conversion to glucose-6-phosphate, the reaction continues through the uridine pathway with the key enzyme being *glycogen synthetase.*

Another key enzyme, *phosphorylase kinase,* is involved in converting glycogen to glucose, a process called *glycogenolysis.* These reactions also involve phosphorus as well as a calcium binding protein. (This same calcium is shared in the contraction of the muscle fiber.)

There are numerous types of *glycogen storage diseases,* which are inherited traits characterized by abnormal glycogen storage. Type I glycogenesis has glycogen stores which are unavailable. Type II, III and IV have genetic enzyme deficiencies, and usually results in death. Type V, also an enzyme deficiency but only in the muscle, is more mild. Types VI and VII are also described, showing enzyme deficiencies in the liver and erythrocytes. This paper does not address the management of glycogen diseases rather the functional aspect of glycogen metabolism. Whether a functional disturbance in the glycogen mechanisms is related to these disease states is a question for further research.

Discussion

If an individual has glycogen dysfunction resulting in low glycogen stores, termed hypoglycogenesis, or the inability to release glycogen, called glycogenolysis, how can we diagnose it? This paper provides a clinical design only and requires further research.

It is the clinical opinion of the author that glycogenesis is the most common problem as the need for phosphorous or calcium, an integral part of glycogenolysis, is rare, and that the need for carbohydrate is common. Clinically, the symptoms of hypoglycogenesis may include fatigue, chronic injuries and recurrent blood sugar problems, or liver, pancreas or adrenal dysfunction. In the exercising patient, fatigue immediately following exercise and a plateau of benefits, whether in a weight loss program or athletic competition, is common.

Normally, glycogen is used throughout the day (and night) to help maintain blood sugar levels and provide muscle energy. Glucose regulates the whole spectrum of energy production; in order for beta-oxidation (the use of fatty acids for energy) to occur, there must be a continuous supply of glucose. Some of this glucose is derived from glycogen stores. Also, post-exercise stores must be replenished so the next exercise bout can be performed efficiently. Hypoglycogenesis may result in an increased demand on the blood sugar mechanism followed by an increased demand on the adrenal, liver and pancreas. In susceptible individuals — those with existing adrenal, liver or pancreas problems or blood sugar problem — this glycogen replacement may not occur properly. For some, a lack of essential nutrients necessary for glycogenesis is part of the problem. These include phosphorus, magnesium and thiamine. The most common problem is the lack of immediate carbohydrate needed to stimulate insulin. Utilizing the protocol outlined in this paper may be helpful in determining which need might exist for a given patient.

Applied Kinesiology Evaluation

It is presumed that a complete evaluation is made of each patient which includes proper applied kinesiology evaluations, and that basic imbalances are corrected before testing for the problems described in this article. The need for this procedure is more common in patients who show recurrent patterns of functional hypoadrenia, hypoglycemia or liver dysfunction.

The hypoglycogenesis patient will often show an inhibition, or "weakness," of the sartorius, latissimus dorsi or pectoralis major sternal muscle. This makes a good indicator along with any normal ("strong") muscle. Have the patient therapy localize (T.L.) to any muscle belly, (where glycogen is normally stored) such as the biceps. If a previously weak muscle strengthens or a strong indicator weakens, it is considered positive for this procedure. This may also be true of a positive T.L. to a liver reflex. It is recommended that at least three points (in three muscle bellies) be therapy localized. Since a positive T.L. only tells you where the problem is, you now have an indicator to test other factors. The common ones include:

Nutritional: There may be a nutritional need for any of the items mentioned above. These include magnesium, calcium and phosphoric acid. Vitamin B-1 is sometimes a factor because of its association with lactic acid. Oral nutrient testing can be used to help determine existing requirements.

Also, test maltodextrose or fruit juice. For those patients who exercise or have active jobs, 20 to 30 grams of carbohydrate as maltodextrin or fruit juice (6-8 oz.) can be taken immediately following their work or exercise. Since insulin release aids in rapid glycogen replacement, pure fructose, which does not cause insulin release, is not useful. Also, the carbohydrate must be taken within 10 to 15 minutes of the completion of activity or exercise. A solid feeding of carbohydrates may take too long to digest and absorb. (The type of patient with these problems is often cautioned about the consumption of excess fruit juice due to its high potassium content.)

Hormonal: Any indication of adrenal, pancreas or liver dysfunction and factors associated with these indicators should be addressed. For example, a "weakness" created by T.L. to a muscle belly may be negated by T.L. to the liver neurolymphatic reflex. Occasionally, treatment may only require that correction.

Patient lifestyle: Any factor in the patient's daily routine which adversely affects the nutritional or hormonal functions described above should be discussed with the patient. This includes dietary habits, nutritional supplements taken by the patient, and the multitude of potential stress factors which may be encountered. Exercise habits should also be evaluated. Common errors include eating or drinking sweets before exercise (this

depletes the existing glycogen stores at a much faster rate), skipping meals, especially breakfast (not allowing liver glycogen to be replenished) and immediately following exercise or activity (this retards glycogenesis) and exercising too hard too often (not allowing glycogen stores to be effectively replenished).

See the procedure below for a step-by-step outline of the above evaluation and treatment.

Conclusion

While the mechanisms and procedures outlined above are clinically useful, the physiologic confirmation has not been documented. However, the clinical outcome is our main goal, and the following outline is useful for the procedures described in this article:

1. If a "weak" muscle "strengthens" and/or a "strong" muscle "weakens" when the patient therapy localizes to the belly of any muscle or the neurolymphatic reflex of the liver, it may indicate the need for this procedure.

2. Using the above indicator, determine if a T.L. to the adrenal, pancreas or liver neurolymphatic reflex (or other reflex points) negates the change from #1.

3. Test magnesium, calcium, phosphoric acid and vitamin B-1 against the muscle indicator found in # 1 above.

4. Test a source of maltodextrin or fruit juice against the muscle indicator.

5. Correct positive indicators, provide nutritional needs, if any, and recommend 20 — 30 grams of carbohydrate as maltodextrin or fruit juice immediately following exercise, if indicated.

6. Evaluate and make positive recommendations regarding healthy exercise habits.

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A Non-Cranial Light Touch Reflex Point for the Quadriceps Muscle: A Clinical Observation.

Philip Maffetone, D.C.

Abstract: A new light touch reflex point which may facilitate an inhibited quadriceps muscle group is described within the context of applied kinesiology practice.

Introduction

In applied kinesiology (A.K.), various reflex techniques exist, some stimulated by the doctor using a light fingertip contact, with others requiring hard pressure and still others by tapping. The location and proper use of these are well described in the literature.[1,2] Most light touch reflexes used in A.K. are found on the head. Most of these are termed Bennett's reflexes, after Terrence J. Bennett, D.C. who first described them. They are also termed neurovascular reflexes, inferring a connection between nervous and vasomotor function. Some reflexes, such as the neurovascular point associated with the popliteus muscle, is located at the medial aspect of the knee at the meniscus.

Discussion

This author has found, through clinical trial and error, a reflex which may facilitate the quadriceps muscle group. It is located at the medial aspect of the elbow: specifically, between the lateral epicondyle of the humerus and the olecranon of the ulnar, an area where the ulnar nerve passes. The point is located in the area of SI-8 on the small intestine meridian. Successful stimulation is most effective through light touch, identical to neurovascular points. Like all reflexes used in A.K., its therapeutic need is found in some, but not all, cases of quadriceps inhibition. On some occasions, the abdominal muscles will test positive when this point is therapy localized. Whether this point can influence small intestine function is a research question. However, the author has found this reflex clinically useful when the small intestine is a component of the patient's problem.

Properly diagnosing the need involves positive therapy localization to the reflex point. Treatment requires holding it with a light touch for as little as 10 or as much as 30 seconds or more. Re-evaluation with therapy localization is an important follow-up.

Conclusion

Another therapeutic tool available for the applied kinesiologist is a light touch reflex associated with the quadriceps muscle group, and occasionally the abdominal muscle group. This light touch reflex has been successfully used by the author for over seven years.

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NEUROENDOCRINE AXIS TECHNIQUE (NEAT)

William Maykel

Abstract: A screening mechanism and treatment protocol for jamming of the neuroendocrine axis and resultant ligamentous laxity is discussed.

Applied kinesiology challenge to the ligamentous system has, to this point in time, been involved with shock absorber technique; adrenal ligamentous stretch technique and direct challenge to a specific ligament while testing a muscle related to that joint. This author introduces a new method of challenge involving abnormal function of the pineal gland and its subsequent downstream affects resulting in systemic ligamentous laxity.

The pineal gland is a complex neuroendocrine organ which is under photoneuroendocine control. Environmental light acts through the retina and entrains the pineal gland's circadian rhythms by way of the hypothalamus and sympathetic nervous system. This tiny gland (100 - 200 mg.) is considered to be the "regulator of regulators" and important in general homeostasis.

The gland plays an established role in controlling reproduction and is involved in the control of sexual maturation. It has a major influence on the circadian organization of vertebrates, including human beings. The hormone melatonin has a potential therapeutic value in treating disorders that are associated with biological rhythm disturbances like sleep disorders, "jet lag" phenomena and affective disorders. The gland is actively involved in the mechanisms controlling sleep-wakefulness cycle and human mood disorders. It actively participates in the neuroendocrine mechanism controlling stress. (1)

Recent research reveals evidence that suggests that melatonin may protect against the age processes, in part, by attenuating the affects of free radical-induced neuronal damage. Diminished melatonin secretion may be associated with acceleration of the aging process and melatonin may be a natural anti-aging hormone. (2) Pineal extracts inhibit a wide spectrum of experimental tumors. (3)

Other research suggests that the pineal gland may play a central role in the biological response to extremely low frequency (ELF; including 50 - or 60 Hz) electric and magnetic field exposure via alterations in the melatonin signal. Recent epidemiological studies have suggested a possible association between ELF and increased risk of

certain cancers, depression and miscarriage. ELF field-induced pineal gland dysfunction is a possible etiological factor in these effects. (4)

Awareness of the vast implications that pineal gland dysfunction has on the human body certainly places the correction of this gland at the top of any priority list. When examining any new or established patient the presence of pineal gland dysfunction is easily recognized. Therapy localization one inch left lateral to the EOP and/or one-third of the way forward from the back of the saggital suture on the vertex of the skull, are two reflex points for the pineal gland. The finding of a positive challenge, that is the weakening of a strong indicator muscle, upon distortion of a joint found systemically, is an indication that this problem is present. Far too often patients are given hard, osseous adjustments to a soft tissue system not readily capable of accepting high velocity thrusts and may ensue in an extremely dissatisfied patient. When the presence of marked systemic ligamentous laxity is present in a patient, the first thing that needs to be done is correction of the cranial faults in a systematic manner that allows freedom of accumulated stress from this axis. Failure to look for, recognize and correct this condition can lead to tremendous amounts of frustration for both the doctor and the patient. In the last year in which we have been using this technique, we have found it to be present in approximately 70% of all new patients. In those patients with multiple complaints and acute pain, we have found it to be present in greater than 90% of those cases.

Once you have identified the presence of this problem, it is relatively straight forward and easy to correct. First of all jamming of the midline sutures, including the cruciate and sagittals, must be found and fixed. Secondly, re-establishment of normal spheno-basilar flexion and extension must be achieved. The lower mandible must be spread and the pterygoid processes of the sphenoid must be lifted one at a time. (It is best to inform the patient that these areas are sensitive and one should make an effort to get and in and out of the mouth as quickly as possible.) Correction of the supportive vault bones including parietal descent and/or temporo-parietal bulge, along with any external rotation of the zygomatic bones or internal rotation of the frontals is next. A reduction in the hypertonicity of the masseter, temporalis and pterygoid muscles must be done. As long as it is not contraindicated, the patient should be given the hatha yoga exercise, the "LION" to do. This is basically having the patient stick their tongue out of their mouth as far as they can for three seconds several times a day to help reduce recurrence of tension in these muscles. Conditions which

would contraindicate this exercise would be those of a tearing of the capsular ligament and/or acute, severe trauma to the temporo-mandibular joint itself, to name the most common ones. The patient should be instructed also to increase their water intake to help detoxify the body. Prior to the correction of the cranial faults the patient should be tested for the need of tyrosine or pineal substance to help jump-start the function of this gland.

Besides clenching and/or bruxing, severe trauma, physically, chemically or emotionally may evoke this problem. Sensitivity to various electromagnetic fields may also precipitate this problem. If there is a recurrence in the pineal jamming, further steps must be taken to investigate the cause. If the person is clenching during the day, it's a good idea to have that patient consciously tongue thrust forward to try and break up the cycle of tension and stress in the temporo-mandibular joint muscles. If they brux at night repetitively, they may need to have a splint made to help disocclude the rear molars. Obviously stress reduction is helpful by making some simple lifestyle changes. Correction of the faults previously mentioned will turn on the downstream endocrine indicators, including the thyroid, adrenal and male or female gonads. This technique lends itself to a very impressive show and tell and is an excellent way to teach people about the need for positive lifestyle changes.

Summary:

In summary, this author considers the evaluation and treatment of pineal gland dysfunction to be a priority strategy in the care and treatment of all patients. The presence of systemic marked ligamentous laxity is a contraindication to high velocity manipulative techniques. The undiagnosed presence of this condition may contribute to the failure of any physical medicine technique. Once you start looking for and finding presence of this condition your therapeutic expertise will be greatly enhanced.

:neat

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Thoracic Spine Evaluation and Treatment Techniques

William Maykel

Abstract: The identification and correction of thoracic intervertebral disc and facet imbrication problems is discussed.

Just like the middle child, the thoracic spine is caught in the unenviable position of being stuck in the middle. Applied Kinesiology challenge allows the astute practitioner mastery over this key area to sympathetic nervous system function. Many pain syndromes not readily approachable by other methodologies may be assessed and treated with precision. The thoracic intervertebral discs and facet joints are probably two of the greatest overlooked areas in the entire spine.

The thoracic intervertebral discs may be challenged with a direct compressive challenge using a spinous-lamina contact. They should be challenged in the following positions as they seem to relate to the severity of injury and amount of treatment necessary to remedy the situation.

- Non-weight bearing - patient is prone - represents greatest potential damage and thus the greatest amount of therapeutic intervention
- Weight-bearing - patient sits or stands.
- Torque - patient sits and twists or stands in a gait position
- Flexion - patient bends forward
- Flexion torque - patient bends forward and turns or assumes gait pose
- Flexion torque with left brain activity - same as above with left brain activity

This progression is one that you may count on. If you correctly direct your therapy the patient will progress through these stages of healing. You may judge a re-aggravation quite readily once you have worked with this system a short while. This progression itself is based on compressive discal and meningeal forces and is apparent in the other spinal areas as well. I have no idea of why the left brain activity is the last component to clear. This system allows for a great doctor-patient communication tool to evaluate treatment efficacy and progress.

Once a thoracic intervertebral disc problem has been identified, treatment can ensue. Usually the pelvis and feet have been balanced and spinal rotations and anteriorities have been reduced. Hypertonicity in the surrounding erector spinae muscles should be removed with spray and stretch and/or spindle techniques. You are now ready to have the patient bring their head and arms over the edge of the treatment table. Have them cross the table so that they are perpendicular to the long axis of the table. The weight of the upper torso and head aids in the distraction process.

You may need an assistant to hold their legs so they don't fall. You contact the appropriate spinous-lamina contact and respiratorally traction the segments three to four times each, applying force on the inspiration phase of respiration.

Interferential current through the involved segments is done next. The patient is educated in terms of correct posture and corrective exercises. For exercise we ask that the patient stand and bend into flexion while inhaling. Then they laterally bend to one side, being careful not to flex their knees. The arm opposing the side to which they are lateral bending is elevated straight cephaled as high as possible. This action ensures maximum motion in the thoracic intervertebral discs. Once again the patient inhales while laterally flexing. This is done next to the opposite side. The patient has now done one repetition. We recommend two to three sets of ten repetitions a day.

Before you evaluate the thoracic facets make sure you have cleared their spine of rotations and anteriorities. Do not forget to challenge and clear the ribs themselves. Usually the last 5 ribs move infero-laterally with respect to the costo-vertebral joints. The first five ribs will move superolaterally and jam medially at the anterior. Any combination is obviously possible if trauma is superimposed upon a torqued spine. Just don't forget to reduce the sterno-costal articulations anteriorly as well. They are usually opposite the costo-vertebral joints.

Evaluation of the thoracic facet joints for imbrication may now be done. Have the patient seated with legs out straight towards the end of your table. Ask the patient to rotate and extend their head and thoracic spine to the side you are checking. For instance, if you suspect a problem in the left T3-5 facets, have them look over their left shoulder while extending their upper back. You challenge the lamina in this area while they are in this posture. If positive you now have the patient lie back fully supine. Have them bend their knees to 90 degrees and roll them to the right while they also turn their head to the right. Meanwhile you stand at the left side of the table at the head and traction their left arm headwards by tucking it in your right axilla. Your left hand contacts the lamina on the left side starting at the lowest imbricated facet and you give a quick cephalad thrust while you simultaneously tug their arm in the same direction. This is repeated for each of the imbricated levels. Your job is now done and usually does not need to be repeated barring any new injury.

Summary:

(1) Intervertebral disc problems are common in the thoracic spine.
(2) These problems may be readily monitored for progression or regression.

(3) Imbrication of the thoracic facet joints may occur as well.
(4) Both problems are relatively easy to find and fix.

:thoracic

SLIPPED BICIPITAL TENDON SYNDROME REVISITED

Bernard M. Nonte, D. C.

ABSTRACT: The frequency of lateral subluxation of the bicipital tendon is greater than previously thought. The angle of the tendon during injury will dictate the direction of slippage. Proper identification of direction of subluxation is paramount for success of treatment. Patient education can prevent re-occurrance.

The biceps muscle of the upper arm, as its name implies, originates in two tendons. The short tendon attaches at the coracoid process of the scapula. The long head attaches at the superior surface of the glenoid cavity. Both muscles run somewhat independent of one another until they are joined in a common tendonous insertion about three inches above the elbow joint. The action and misaction of the long head of the bicep is the subject of this paper.

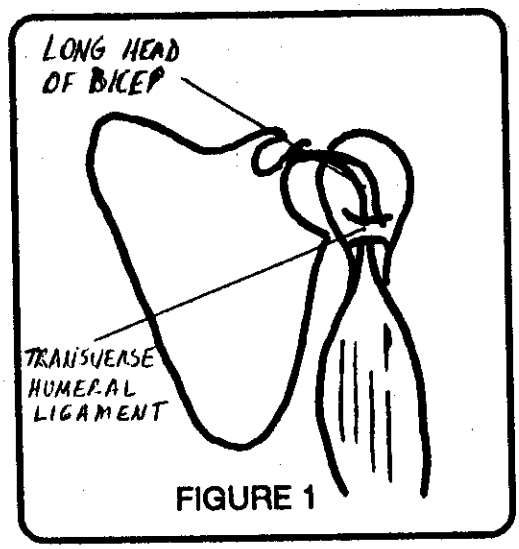

FIGURE 1

When at rest, after originating at the glenoid cavity, the long head of the bicep runs laterally at 90° angle to the bicipital groove where it is held in place by the transverse humeral ligament, then runs inferior and inserts into the belly of the biceps. (See Figure 1) In adduction the angle of the bicep tendon through the groove decreases. (Figure 2) However, with abduction, when the elbow and shoulder are level, the angle of the tendon increases to 180° (Figure 3) Rene Cailliet, MD, in his **Pain Series - The Shoulder**, states that the bicep tendon does not move within the bicipital groove. However, due to the movement of the humeral head, the tendon does change its attitude within the bicipital groove. Cailliet speaks of the movement of a fixed point along the bicipital tendon superior to the transverse humeral ligament that will move inferior with abduction and superior with adduction. In the event of rupture of the transverse humeral ligament, the long tendon always displaces medially.

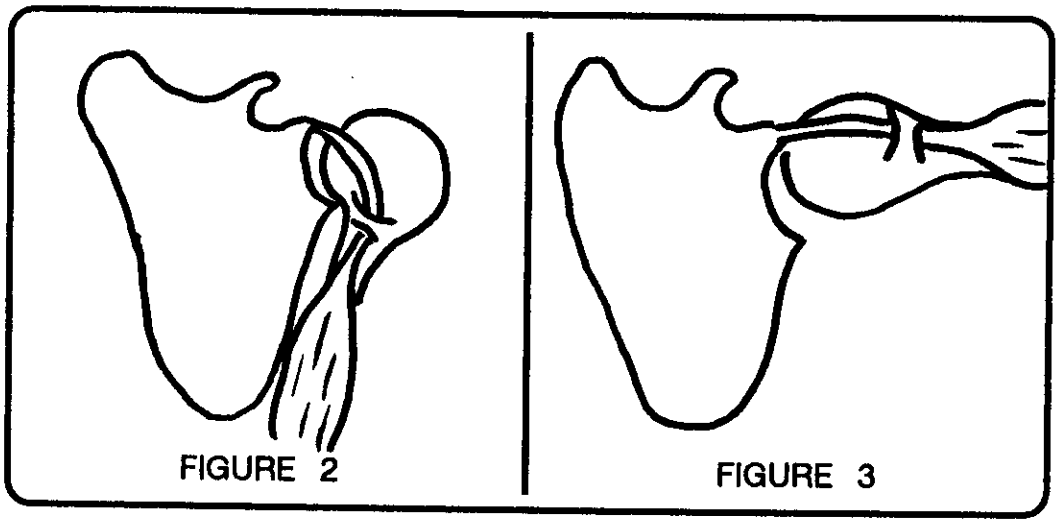

FIGURE 2 FIGURE 3

Since we, as applied kinesiologists, are certainly functional interpretors, I think proper examination can reveal more useful information about the misaction of the bicipital tendon. Walther's text, **Applied Kinesiology - Synopsis** has an excellent discussion on the bicipital tendon. In it, Dr. Walther states that most common displacement of the tendon is medially. It has been my observation that the direction of subluxation of the bicipital tendon is dependent on the angle of the tendon, in reference to the groove, during trauma. If the angle of the bicipital tendon is less than 180° during injury, the tendon generally displaces medially, however, if the angle is greater than 180°, the tendon will slip and/or become entrapped laterally by the transverse humeral ligament. (Figure 4) Over 14 years of treating athletes involved in throwing sports, individuals with "Lazy Boy®" shoulder (more later) and stomach sleepers, has led to the assertion that subluxation of the bicipital tendon within the **lateral** attachment point of the transverse humeral ligament is much more common that previously thought.

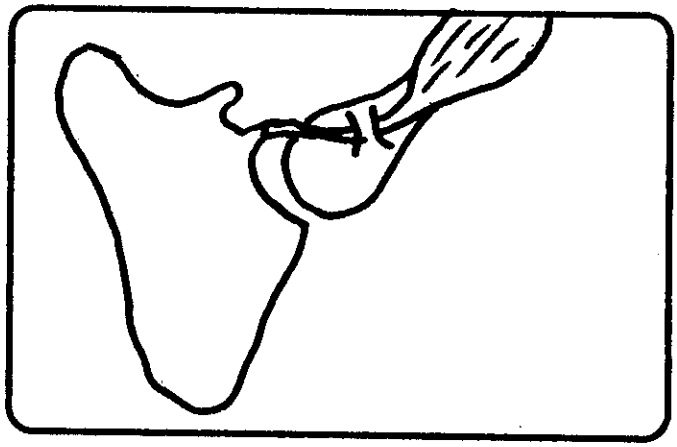

FIGURE 4

In a neutral position, the bicep tendon in forced firmly into the groove by bicep contraction, in an anterior to posterior direction. With the shoulder in abduction and externally rotated, and the bicep tendon at 180°, this A to P vector is continued, however now the bicep tendon is forced against the lateral portion of the bicipital groove. If

abduction is over exaggerated or held for an extended period of time, the tendon will subluxate and can become entrapped by the transverse humeral ligament. Since, as we learned earlier, the bicipital tendon does not move *per se*, this may be one of the few examples of "subluxation by association".

As previous stated, lateral subluxation or entrapment of the bicipital tendon by the transverse humeral ligament occurs when the angle of the tendon is greater than 180° during injury. Some of the more common case histories I see include: falling asleep in a reclining chair with the arms up above the head and hands locked (Lazy Boy® shoulder), stomach sleepers who get their arm abducted and externally rotated under their pillow, carpenters and painters working above their head (Michelangelo shoulder). I have been Team Doctor for the local high school baseball team for 11 seasons, shortstops and pitchers who throw sidearm or "submarine" are particularly prone to this type of injury. Players who throw "over the top" rarely are bothered by bicipital tendon injury due to proper shoulder mechanics that align the tendon within the groove more efficiently. Swimmers, particularly butterfly and reverse crawl participants, are also prone to lateral subluxation of the bicipital tendon.

Depending on the severity of injury, the biceps muscle may or may not test weak in the clear. By directly challenging the bicipital tendon you may determine not only whether the tendon is involved but also the direction on subluxation. **Tendons response to direct applied kinesiologic challenge in the same manner as spinal segments, adjust into weakness.** Using an indicator muscle other that the bicep is more accurate because it rules out the effect of pain on testing results, I prefer the latissimus dorsi. Push the bicipital tendon medially with your thumb and release, if your indicator muscle weakens, the tendon is entrapped laterally. If the challenge does not weaken the indicator muscle, push the tendon laterally, release and re-test. If the indicator weakens, the tendon is entrapped medially. Occasionally, the arm must be abducted greater than 180° during testing to display the subluxation. Doctors may wish to challenge the entire humerus internally and externally to rule out bony subluxation. Yergason's sign, in which the patient attempts to supinate the forearm against resistance with the elbow in flexion, is indicative of bicipital tendon involvement. However, this test is not nearly as accurate as directly challenging the tendon. Out of curiosity, I have manipulated the bicipital tendon opposite of the indicated direction to observe reaction and have found no change on the indicator muscle.

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SLIPPED BICIPITAL TENDON REVISITED
NONTE

Correction of the slipped bicipital tendon is as described by Walther in his **Synopsis** text. With the patient supine, to lessen bicep contraction and increase stabilization, flex the elbow to 90° while supporting the forearm. Apply pressure in the direction of correction steadily while moving the elbow posteriorly. When you have moved the elbow posteriorly as far as possible, then slowly abduct the arm to 180°, all the while maintaining corrective pressure. If the tendon was displaced medially, rotate the humerus internally when at 180°. If the tendon was displaced laterally, rotate the humerus externally at 180°. Following correction re-challenge the tendon to confirm results. Frequently, one correction is all that is required but occasionally the problem will reoccur and more treatment is needed.

All shoulder muscles should be tested to insure proper alignment of the shoulder girdle. Frequently, the rotator cuff muscles, supraspinatus, teres minor and major, subscapularis, and infraspinatus, will be involved. The pectoralis major tendon inserts onto the outer bicipital ridge, offering some stability to the bicep tendon and therefore should be carefully examined. According to **Gray's Anatomy**, there is a third head of the bicep found in 12% of the population, arising from the upper and inner part of the Brachialis muscle. All factors of the IVF for the bicep muscle should be checked and treated properly. Spindle cell adjustment is very useful to strengthen the bicep. Lyso-Lyph-Forte™, available from Nutri-West,[1] is an enzyme mixture that reduces inflammation and speeds recovery. It is recommended that the supplement be taken between meals, two tablets at 10:00 am and two tablets at 3:00 pm. The musculocutaneous nerve, which innervates the biceps muscle, arises from C5 & C6 and vertebral subluxation at this level is common. Occasionally, the patient will complain of pain in the elbow, at the insertion point of the biceps, rather than at the shoulder. (Another reason to "fix what you find")

Weakness of the muscle encourages an excessive amount of motion in the entire biceps complex, that in turn encourages subluxation of the tendon. After correction, the patient should begin strengthening exercises at home. The traditional bicep curls with 5 to 10 lbs weight is helpful. I instruct the patient to do sets of 12 curls at 1/3, 2/3, and full extention of the elbow every day, starting at one set increasing to two sets after one week. Cho-Pat, Inc [2] produces an excellent bicipital tendon strap called the Swimmer's Arm Brace. This strap is supportive by shifting the focal point of

1 NUTRI-WEST P.O. BOX 1298 DOUGLAS, WYOMING 82633 (307) 358-5066

2 CHO-PAT, INC P.O. BOX 293 HAINESPORT, NEW JERSEY 08036
(800) 221-1601 FAX (609) 261-7593

tendon stress away from the bicipital groove. Athletes are able to continue participation with reduced likelihood of re-injury. Modification of everyday activity also reduce exacerbations. Instruct the patient when dressing to place the injured limb into shirt, blouse, or jacket first, reducing internal and external rotation of the affected side. It is always good to tell the patient not to sleep on their stomach, no matter how obvious it is to us, some patients know nothing of the proper bio-mechanics of sleeping.

I encourage all AK members to review the bicipital tendon sections of Dave Walther's texts, Cailliet's **Shoulder Pain**, Hammer's **Functional Soft Tissue Examination and Treatment by Manual Methods**, and the old standby, **Gray's Anatomy**. Most of the technical information in this paper was taken from these books. Lateral entrapment or slipping of the bicipital tendon is much more common than previously thought. Direction of subluxation of the bicep tendon is often dependent on the angle of the tendon during trauma. If the tendon angle is less than 180° during injury, the tendon will subluxate medially. If the angle is greater than 180°, the tendon will subluxate laterally. After corrrecting the subluxation, educating the patient on proper shoulder mechanics can prevent re-injury. When treating the shoulder pain patient it is paramount to be sure the individual is ... "in the groove".

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THE USE OF CRANIAL TENDER POINTS TECHNIQUE TO FIX COMPENSATORY PROBLEMS

BY SHELDON A. SINETT BA, MA, D.C.

Abstact

The purpose of this paper is to show you how the use of Cranial Tender Points Technique will correct adaptive or compensatory problems.

Introduction

The use of Tender Cranial Points Technique was introduced by Dr. George Goodheart. Dr. Goodheart stated that one should visualize a football or a motorcycle helmet being the skull and the helmet wearer's head being the brain within the skull suspended by dura tentorium and falx etc. Finding cranial tender points when the patient is supine may indicate excessive backward movement by normal gravity pulling on the straps of the interior of the helmet suspensory arrangement.[1]

The cranial dura consists of two layers; an inner meningeal and outer periosteal. The carnial is divided into falx cerebri, falx cerebelli, tentorium cerebelli and the diaphragma sellae. The dura has different poles which attach to ethmoid, frontal, temporal, occiput, and sphenoid bones. The dura as you all know is a reciprocal tension membrane whose function is to cause movement to the articulation and at the same time regulate or limit the normal range of articular mobility.[2] We are also aware that if cranial dysfunction takes place cranial nerve entrapment can occur, along with endocrine dysfunction. I have an opportunity to see a lot of T.M.J. patients in my practice. I think the root of their problems are meningeal or dura. I would examine and evaluate these patients and would find a number of cranial problems, an imbalance to cervical muscles and an imbalance to muscles mastication. After doing Cranial Tender Points Technique I was amazed to see a lot of these procedures negative on retesting. The ones that were not corrected by Cranial Tender Points Technique were corrected by standard A.K. procedures. It is my opinion that whatever problems were left were primary and not compensatory factors.

Procedure

1. Test sartorius, gracilis - should be strong. In the clear, if not - correct by standard A.K. procedure.
2. Test sartorius, garcilis against breath cessation - should be strong.
3. Test sartorius, gracilis against breath cessation while two handed contact therapy localization to any cranial suture.

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Weakness will occur at any cranial suture with breath cessation.

4. Treat cranial tender points by Jones's strain and counter strain technique.
5. Recheck sartorius, gracilis muscle against T.L. to suture with breath cessation - muscle should remain strong.
6. T.L. to lamboidal suture and ipsilateral sacroiliac joint. If weakness occurs - tap lamboidal suture and sacroiliac joint, not necessary in unison. Tap both sides for 30 seconds to 60 seconds.
7. Now test and treat B&E points, if necessary.[3]

Conclusion

The proper application of Cranial Tender Points Technique will correct many cranial problems, cervical muscle hypotonicity and muscles of mastication dysfunction. It also helps endocrine dysfunction. (Pineal and Pituitary).

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A NEW APPROACH IN TREATMENT OF EMOTION RELATED PROBLEMS.

Harry P. Stassen, D.D.S.
The Hague, The Netherlands

ABSTRACT:

A new approach in treatment of emotion related problems is reported, using Applied Kinesiology, acupuncture, Bachflowers, and laser technique.

INTRODUCTION:

Most of the Pain Dysfunction Syndrome (PDS) patients, or the also called : Tempero - Mandibular Joint (TMJ) patients, do have in their history an emotional trigger, that may interfere with treatment.

Misalignment of any bony structure in the body can be caused by a trauma or be a trauma itself. This trauma than, is not a privilege for only the physical body, but may also affect the psycological side of our patients.

The following concept in treatment is a very helpfull tool, when you want to treat emotions in a different way.

PROCEDURE:

Though treating mainly TMJ problems, or TMJ-related problems in my office, the following procedure used, can be applicated in any problem that has a emotional component.

* Find a strong indicator muscle (IM).
* TL on the Right TMJ. If the IM stays strong, check what happens to the IM while the patient:
  - clenches
  - chews
  - opens wide
  - protrudes
  - etc.

* Repeat this at the other side aswell.
* Where a strong IM weakens, double TL the involved side and the NV points on the frontal bone, as discribed in the emotional stress release treatment.
* When this negates the weakend test muscle, it shows an emotional problem in conjunction to the TMJ.
* Now test the PMC muscle in the clear to use as Indicator Muscle (THE emotional related muscle).
* Ask the patient to think on hers\his TMJ problem, and test the PMC.Because there is a emotionalrelated problem, the IM should turn weak.
* Find the Bachflower that negates the weakening.
* As listed further on, most Bachflowers have a relation with one or even more acupuncture points.
* When more than one acupuncture point is indicated, find the acupuncture point,most appropriate for the treatment, by having the patient think on the problem again and TL the indicated points.A previous weak muscle should turn strong on one of the indicated points.
* ON this acupuncture point, you apply one drop of the Bachflower found before, and " beam " the Bachflower through the acupuncture point into the body,with the laser (please use a He/Ne laser)
* Using a dognut magnet may even increase the effect.

N.B. **EXEPT FOR GV AND CV ALL ACUPUNCTURE POINTS DO HAVE A LOCATION ON THE RIGHT AND ON THE LEFT SIDE OF THE BODY.**

CONCLUSION:

Usually I find only ONE bachflower.The time that the laser is engaged is 45 seconds per acupucture point.
One should use a He/Ne laser.
After this the patient is checked again.
As a standard one or two trearments will do.
This procedure turns out to be manyfold more powerfull than expected,when I started this procedure.Many patients have benefitted by this treatment.

SUMMARY

Treatment of emotion related problems have been discussed. The use of a combined application of Applied Kinesiology, acupuncture and Bachflowers, along with laser beams has been reported.

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ACKNOWLEDGEMENT

I like to express on this place my gratitude to my Professor in acupuncture,Dr. Alphons van der Burg,who thaught me the principles of acupuncture.

BACHFLOWERS — ACUPUNCTURE POINT RELATION CHART

ASPEN	L i	17
	Bla	15
BEECH	Sto	19
	S i	1
	Lu	10
CENTAURY	Bla	13
	Bla	19
	Bla	65
	C/S	6
CERATO	Bla	18
CHERRY PLUM	sto	44
	S i	2
	S i	6
	Spl	1
	TrH	1
	TrH	6
CHESTNUT BUD	Hea	8
	S i	4
	C/S	5
	Lu	5
	Lu	11
	Spl	4
	Spl	21
	K	7
	Ga	24
	CV	17
CHICORY	Liv	4
	Liv	13
	Spl	8
CLEMATIS	Liv	8
	L i	15
	C/S	4
	TrH	2
CRAB APPLE	Sto	4
	Liv	6
	L i	1
	L i	4
	Bla	58
	CV	12
ELM	Hea	3
	Sto	41
	Lu	5
	C/S	9
	K	2
GORSE	Ga	42
HEATHER	Hea	3
HOLLY	Ga	41
HORNBEAM	Spl	9
IMPATIENS	Sto	41
MUSTARD	Ga	41
OAK	Hea	6
OLIVE	Spl	5
	Bla	41
PINE	Hea	9
	Lu	7
	Bla	27
RED CHESNUT	Sto	42
	Liv	2
	Lu	8
	GV	2
ROCK ROSE	L i	2
	S i	3
	K	10
	TrH	10
	Ga	43
ROCK WATER	Sto	34
	Liv	1
	L i	6
	L i	7
	Spl	2
	TrH	4

EMOTION RELATED PROBLEMS Harry Stassen

SCLERANTHUS	Sto	43
	L i	1
	Lu	1
	Spl	3
	K	1
	K	5
	Bla	60
	CV	14
	CV	17
STAR OF BETHLEHEM	Sto	25
	S i	5
	C/S	7
	Ga	36
	CV	3
	CV	5
	Bla	20
	Bla	25
	Bla	66
	K	3
SWEET CHESNUT	Sto	40
	Liv	14
	L i	11
	Lu	6
	Tr H	3
	"	5
	Ga	44
	GV	11
	Bla	28
	"	64
VERVAIN	l i	3
	S i	7
	C/S	3
	C/s	8
	Ga	34
	GV	8
	Bla	21
	"	22
	"	67
VINE	Liv	3
	Lu	9
	Ga	25
	Ga	38
	Bla	63
WALNUT	K	4
	Bla	40
EAU VIOLET	Hea	5
	"	7
	S i	8
	Spl	6
	Tr H	7
WHITE CHESNUT	Sto	34
	"	45
	Ga	37
	GV	10
WILD OAT	Sto	6
	Bla	23

"Enhancing Your Neurolymphatic Treatment"
Working with the body's Electro Magnetic Polarity Energy Fields

Dr. Ron C. Wagner, D.C.
4222 Milwaukee st.
Madison, Wisconsin 53714
(608) 244-0211

ABSTRACT:
Neurolymphatic (NL) stimulations are very important for receiving a complete Applied Kinesiology treatment. I have seen doctors, including myself (when I first started practice), avoiding or leaving out the stimulation of these NL points. This would happen when many people were too sensitive with congestion and tenderness in their chest, neck and thoracic areas. When these NL points were too heavily rubbed or stimulated with a forceful massage, it caused too much pain for many people and they had asked not to have this done again.

I kept searching for and correlating new ideas and techniques and here are some of my findings. Some doctors, in their treatments, have used magnets.[1] Since magnets are not accepted for the use of any treatment in Wisconsin, our hands can be used as magnets. I would like to share a stimulating and relaxing NL treatment that most everyone would enjoy.

INTRODUCTION:
Everything in life is energy, from the smallest atom with the electron flowing around, to the universe with the planets in our solar system. All life has an electrical-magnetic-polarity-energy field. This energy is like magnetic or electrical currents flowing like rivers of energy in and around our bodies.

"As soon as that circuit is interrupted, changes begin to appear which, in the human body, are interpreted as pain or dis-ease. The interrupted current cannot reach the core to flow through and out again. In the meantime, the opposing currents pile up energy particles at the point of interruption and act as blocks in the area where they occur. This pressure of energy particles in any of the five fields of matter registers as intense pain, or obstruction of normal energy flow, called dis-ease."[2]

Many natural healing arts, from chiropractic to acupuncture, voice their opinion of the importance of "that too much or not enough energy is disease"[3]

The whole body is like rivers of energy flowing. When there is a blockage or dam in the river, the energy cannot flow. In front of

the dam there may be an emptiness, aching, numbness, tingling, stiffness, soreness, lack of energy, lack of feeling, lack of sensation, lack of function, degeneration, etc.

Behind the dam, the blocked up energy may cause congestion, pain, swelling, tightness, pressure, tension, headache, excess growth, colds, sinus, asthma, pimples, irritations and inflammations, etc. We need to find and release the blocks, so the Chi Life Force Energy can flow evenly throughout our whole Being.

Christopher Hills states it as: "When this energy flows in a balanced way, we are healthy. When the flow is unbalanced, slowed or blocked, we are not healthy. To acupuncturists, then, bodily symptoms of disease indicate that the flow needs to be corrected."[4]

"Meridian therapy works to balance and release the "life force" within the body."[5]

Dr. Randolph Stone also mentions that one of the main principles of polarity is the attraction and repulsion (like a magnetic field) of everything in life.

There is also in life a positive and negative, Yin and Yang, male and female, light and dark, etc.[6,7,8,9]

"Stimulation of bioenergy flows, as in acupuncture treatment, cures many ailments in our physical body. Application of pulsed electric currents speeds healing of broken bones...." [10]

"This energy, called Chi...is electromagnetic."[11]

Christopher Hills further states that Chi or Qi "is the continuous, unblocked flow of this life energy which carries the pulse of life,....some researchers explain this flow in terms of an electrical impulse (polarization) or wave of energy..."[12]

When a person has pain, they constrict and tighten up muscles causing blockage in their body, with a lack of circulation, and energy flow, resulting in sluggishness, congestion, spasm, subluxation and dis-ease

When the blocks are released, the chi life force energy can flow more freely and increase all of the body circulations.

By using acupuncture needles, or using acupressure on these acupuncture points, we are increasing all of the body's circulations and energy fields, especially through the acupuncture meridians.

DISCUSSION:
When I was studying chiropractic at Palmer from 1972 to 1975, I was introduced to Polarity. The classes were given by Dr. Jarvis D.C. from California. He was using materials and texts written by Dr. Randolph Stone, D.O., D.C., D.N.

It was taught in the Polarity classes, that when someone had a rib problem, the treatment would be to hold the front and back points on the rib heads. By doing this, the patient felt more relaxed and seemed to get all the tension and tightness out of the chest area and many times seemed to heal faster.

In examining the location of these points on the chest area, I found it very interesting that many of these points were the same points as the anterior and posterior neurolymphatic points that Dr. George Goodheart, D.C. was teaching us in his Applied Kinesiology classes and material. Throughout my 17 years of practice, I have researched and studied this concept in detail.

With each treatment, I examine, loosen up and do a firm gentle massage adjustment *[1] on the whole spine. Opening up the whole spine is very important for the nerve supply to all parts of the body, especially in the chest, thoracic and cervical areas.

After adjusting and releasing all blocks in the spine, I tonify all the acupressure points. Then I recheck and test all the muscles that were weak or switched off, many of these muscles, I find become stronger and now show a firmer resistance with the muscle tests.

After a good treatment, all of the muscles are switched on and test strong. There are always those few times, though, that some people are not receiving results. These people, that are not responding to the treatment, I have found, will have a deeper or serious condition.

Many of these conditions in the thoracic and chest related areas can be of many causes: worry, emotional stress (carrying the burdens of the world on their shoulders), holding tension, tightness, congestion or toxins in the back, neck and chest areas.

Instead of testing all of the muscles for the chest area each time, I have found that to save time, I could test the chest area with two general muscles, the supraspinatus and the anterior deltoid muscles.[13] When one or both of these muscles are switched off, I many times correlate this, a person can have a lung congestion and possible gall bladder problems.

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*1 Massage adjustment to be discussed further in another paper.

I found a way to save treatment time. The next step in my research was to discover that instead of rubbing or stimulating only one anterior and posterior NL points[14] for a specific muscle or organ correlation, it was beneficial to use all of my five fingers on both hands that God gave me. I could then stimulate all the points that I could contact in the area.

I also discovered that rubbing too hard on any point can possibly irritate or bruise the person. I have found greater results by holding the anterior and posterior NL points simultaneously. Thus balancing the electrical-magnetic-polarity-energy fields of the body.

Many of the muscles use the same NL points and even different organs and meridians overlap and use some of the same NL points. Another point of interest is that the "Muscles of Mastication and Mandibular Movement"[15] are also using many of the same anterior and posterior NL points.

To balance the thoracic and chest areas, I correlate and hold the anterior and posterior NL reflex points with the acupuncture points on the front and back of each person.

I have found that skin contact has the best effect in treatments. Yes, we are working with an energy field and we can energize any person with our hands above the body. This is not as effective as with using skin contact.

It is very important to have skin contact when finding the exact locations of any acupuncture points. When we have skin contact, we are using the conduction of an electrical energy field, the magnetic energy field, the polarity energy field and the aura or your hallo energy field. This is why I always state that the body is an electrical-magnetic-polarity-energy field. Holding points through any material can be a reduction of an electrical energy flow, unless the material is wet to conduct the electrical energy current.

EXPLANATION OF PROCEDURE AND TREATMENT

The person is laying supine (on their back) and I retest the supraspinatus and the anterior deltoid muscles. When only one, both or all four of these muscles show weakness or are switched off yet, I balance both sides of the chest and thoracic area.

When I find both right and left sides or only one side weak or switched off, I balance both sides to keep the body balanced. Usually, I do the weak side first and then retest to show that it is now stronger.

Sometimes, I notice that the person is showing signs of doubts or questioning what this treatment is all about. As I balance the NL points on the chest, I would balance one side, front and back. I would retest at this time to show the person that now one side is strong and the other side is still switched off yet.

There may be a situation where I am balancing the patient by myself. I have balanced one side and the other side happens to get switched on, (not often), I still balance both sides of the body, to keep everything in balance.

APPROACHING THE PATIENT

I say to them: "There are many acupuncture points, trigger points and reflex points on your body. I would like to help you more with these points on your chest. Is it ok for me to palpate these points (through the gown) along the sternum on your chest? Are any of these points tender? (While the person is squirming from pain.) Would you like to get the tenderness and soreness out of these points and help your body to feel better? To give you the best results we need to palpate and find the exact point location on the skin."

To avoid the gown from putting pressure on our hands toward the breast area, we slide the person's arms out of the gown sleeves (keeping the breast covered).

Most of the time when I balance these NL points, I have a female assistant. She holds the points on one side, while I hold the points on the other side and we balance the body simultaneously. Depending on which side of the patient I stand, to balance their posterior NL points, I lift the shoulder with my hand and I slide my other hand under the upper back across the shoulder blade to the Bladder meridians along the spine.[16, 17, 18]

The medial Bladder meridian is on the Back Shu points, also called the Associated points.[19, 20, 21] The lateral Bladder meridian is the emotional points for the organ level on the medial Bladder meridian.

With my right hand on the person's back, I contact with my fingers between the two Bladder meridians. My index finger is placed on the corner of the superior medial border of the scapula, by vertebrae level T 3 and the meridian point is Bl 13. My middle finger is by T 4 and Bl 14. My ring finger is by T 5 and BL 15 and my little finger is by T 6 and BL 16. The right hand thumb is resting comfortably between SI 12, TW 15 and LI 16.

When my right hand is in position on the back, I use my left hand on the front of the body to balance the anterior NL points.

We reach under the gown by the diaphragm area, sliding our hands up the center line to the sternum. We always keep our hands cupped to avoid any contact with the breast area.

When our fingers are above the sternum, we contact the intercostal spaces points along the sternum, on the Kidney meridian.[22], [23], [24]

The first contact point location is with the little finger on K 22 at the medial corner of the breast of the 5th rib intercostal space. The next NL point is on K 23 of the 4th intercostal space. The middle finger is on K 24 the 3rd intercostal space and my index finger is on K 25 the 2nd intercostal space. My left hand thumb is either placed on CX 1 or LU 1, in that area wherever my hand feels comfortable. My assistant holds the same NL points on the opposite side. (When I am by myself, I repeat the same points on the opposite side.)

When the person has Subscapular muscle weakness or heart problems, I slide my fingers one or two spaces up higher to stimulate more energy to the heart area. I hold the anterior NL points starting with my index finger on K 26 the 1st intercostal space. On the posterior NL points my index finger is on T 1 (Bl 11) or T 2 (Bl 12) and in sequence with the rest of the fingers on the anterior and posterior NL points.[25]

Simultaneously using magnetic and polarity energy fields and holding these anterior and posterior NL points, we are increasing the energy and circulation to the lungs, heart, gall bladder, stomach and balancing everything in the chest and thoracic areas.

I also found that balancing with this magnetic Chi energy is very good for the whole chest area and many times helps to relax the neck, face and jaw muscles and flows down into the diaphragm. This Chi energy seems to flow wherever it is needed.

Some people do not feel anything while holding these points. Some feel a cooling effect, whereas most people feel a warming and/or relaxing sensation in their chest or even a current flowing through their the body, wherever the energy is needed.

DISCUSSION OF FINDINGS:
I would hold these points for approximately one minute on an average. When a person has a lot of congestion, I hold these points longer.

For example, one patient came in and asked me to loosen up his neck so he could relax better in the hospital. I had asked him why he was going to the hospital. He stated that his medical doctor said

he had pneumonia and needed to go to the hospital immediately. I suggested to him, instead of just loosening up his neck, how would it be if I loosened up your whole body, making it feel better. He said "Oh Good". After I treated him, I had rechecked the anterior deltoid and supraspinatus muscles and found these to be switched off yet. I then asked the patient if he wanted me to work further with his lungs, chest and breathing. He said go ahead. I held the anterior and posterior NL points simultaneously on the right side and on the left side. I rechecked the muscles after doing the right side and the right side muscles were strong but the left were still weak. I thus came to the conclusion, that when treating only one side of the body, the person may feel off balanced and I behoove everyone to work with both sides.

When I was done treating the left side, while still treating, the patient stated, after taking a deep breath, "Wow, I can breathe better, I'm not going to any hospital". I strongly encouraged the patient that, if there is any congestion coming back, he should come back to the office right away, see his medical doctor or go to the hospital. The patient stated that he would continue with me. He was rescheduled for two days later, when he came back, I worked with his whole body and especially the chest and lungs again. He was doing 75% better. I worked with him for 3 treatments, one per week for the next 3 weeks and one month later for a recheck. He had no further congestion, never went to any hospital, and he was a very happy man. He told me it's much cheaper to come in than to lay in the hospital.

We are receiving 60% to 75% results after holding these anterior and posterior NL points and the person feels much better when leaving.

At times, a person has to come in several times for treatments, especially when the congestion and tightness keeps reoccurring or does not fully clear out, then I proceed to work with the retrograde lymphatic.[26],[27]

CONCLUSION:
Ashley Montagu tells us the importance of touching and in his summary tells us "...the evidence points unequivocally to the fact that no organism can survive very long without externally originating cutaneous stimulation.[28]

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Touching, The Human Significance Of The Skin
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"ENHANCING YOUR RETROGRADE LYMPHATIC MASSAGE"

The importance of doing a retrograde lymphatic massage.

© March 7, 1992

Dr. Ron C. Wagner, D.C.
4222 Milwaukee st.
Madison Wisconsin 53714
(608) 244-0211

ABSTRACT:
Our goal is to search further and help each person receive the best results, eliminate the recurrence of symptoms and many other problems. An additional, very important, technique, that should never be forgotten, especially when not receiving results, is the Retrograde Lymphatic Technique.[1,2]

INTRODUCTION:
I have been studying and using Applied Kinesiology since 1973. When starting my practice, due to the lack of funds, I put AK classes aside and took state board class credits. AK credits are not accepted in Wisconsin for license renewal.

I decided to review basic AK classes to catch up on any material that I have missed. I was first introduced to Retrograde Lymphatic Massage in 1986-87 by Dr. G.E. Achilly, D.C.
Dr. Richard J. Caskey, D.C.
Dr. Jerold I. Morantz, D.C.
They state in their notes:
"Indications for use
a. any signs of lymphatic congestion
b. edema
c. anemia
d. respiratory problems
e. infections"

I find that the retrograde lymphatic should be checked after the body has been treated and the NL points balanced. Yet, we are still finding a recurrence of symptoms.

"Certain patients who show evidence of lymphatic congestion by edematous extremities, decreased resistance to infections, and .... there may be no outward appearance of lymphatic congestion." [3]

I have found you can use retrograde lymphatic with a wide range of symptoms that keep reoccurring. The symptoms may be a combination of tension, tightness, soreness, fatigue, tiredness, to toxins and congestion in the body. The person may be experiencing headaches, stiffness in the neck, upper back, upper arms, shoulders, upper trapezius muscles, chest congestion and pain on the sternum and

into the diaphragm. Also, there can be soreness in the low back, pelvic and hips tenderness radiating into the low abdominal and groin areas. (This will be discussed at another time.)

SIDE NOTE:
I have found that many people are using their back muscles very little and are continuously over using their arms, front of the shoulders and chest muscles. With this type of patient, I find that they may have tightness, soreness, and tension in the chest to the front of the shoulders, upper back and neck areas.

This could be the result of repetitive lifting, to typing on the computer. The pectoris and other chest muscles are being overused in the front and not being balanced with the muscles on the back. The front muscles get tighter and tighter, and the back seems to weaken and become more fatigued.

I have an exercise for my patients to do on a regular basis, so they can improve their posture and relax the neck and shoulder tensions. The exercises consists of repeating 5 to 10 times each morning and night.

The shoulder exercise that I recommend is to lean back against a door or wall (a paneled wall will leave less smudge marks), put hands on the wall with palms out, pushing your body away from the wall. This will help strengthen the upper back muscle and may improve some rounded shoulders. (You may want to check the person to see if there is a need for iodine.) While doing this exercise, if you are not feeling your muscles being worked, then put your feet out further from the wall and push again. When there is too much strain, bring your feet back, till you are comfortable with this rebuilding shoulder posture exercise.

The second part of this exercise is for the neck posture and for strengthening the neck muscles. Put the back of your body against the wall, this time only using the back of your head to push your whole body away from the wall. Again, while doing this exercise, if there is too much strain, move feet closer to the wall. If you don't feel much resistance then move your feet out further again. Remember, you should enjoy the exercises and please do not over strain or cause your body more problems.

HELPING LYMPHATIC WITH EXERCISE AND EATING:
It is very important to burn fat and increase the circulation with exercise as Covert Bailey explains on his Fit or Fat tapes presented at the fund drive on public TV. Al Carter tells us the importance of exercise on a trampoline and especially the lymphatic systems, chapter 6 in his book The Miracles of Rebound Exercise.

He states that the lymphatic system has several descriptive names: ""The Auxiliary Circulating System," "The Garbage Collector of the Body," "The Vacuum Cleaning System Within," and, most recently, "The Immune System." Each one of these nicknames is very descriptive for a particular property of the lymphatic system."[4] Also C. Samuel West N.D. tells about doing exercise on the trampoline, increasing lymphatic circulations, clearing out "Trapped Protein" and eating a good diet by avoiding excess fats oils and meats.

In an effort to help the body function better there are many other good books to read and here are a few:
New Life Through Nutrition by Dr. Sheldon C. Deal
How To Get Well by Paavo Airola, Ph.D., N.D.
The McDougall Plan by John A. McDougall, M.D. & Mary A. McDougall
The New Vegetarian by Gary Null
Mucusless Diet Healing System by Prof. Arnold Ehret

There are many anatomy and physiology books explaining the lymphatic system. I like David S. Walther Applied Kinesiology Volume 1 book. pp. 217- 231. This is one book that I feel is easy to understand and is outstanding in explaining the function of the lymph system, to its correction.

"Protein and fat leave the blood capillary, moving into the interstitial spaces. These large molecules cannot return to the blood capillary and move into the lymph capillary through the one-way valves. The lymph, with the protein and fat, is returned to the blood by way of the thoracic duct and right lymphatic duct."[5]

Anatomy books state that the major drainage of the lymphatic is through the thoracic duct on the left side of the body. "The only exceptions are the right side of the head, neck, and thorax, the right upper limb, the right lung, right side of the heart, and the diaphragmatic surface of the liver. These areas are drained by the right lymphatic duct."[6]

"The pectoral (or anterior) group of nodes is located along the lower border of the pectoralis major inside the anterior axillary fold. These nodes drain the anterior chest wall and most of the breasts."[7]

By massaging the chest area, we are increasing the lymphatic circulation and the acupuncture meridians to the chest and thus benefiting the whole body.

Enhancing Retrograde Lymphatic, Wagner, p 4

TECHNIQUE:
The original Retrograde Lymphatic Technique is to have the head lower on the table and raise the table slightly, so the person is at a slant. We now test and find a strong indicator muscle (example: Tensor Fascia Lata or Quadriceps). Then you have the person raise the arms over the head and again test the indicator muscle. When the indicator muscle tests weak, then congestion is present in the body and we need to do the retrograde lymphatic. For more reference, see Applied Kinesiology Volume 1, by David S. Walther, pp. 224-228, or Applied Kinesiology Synopsis by David S. Walther, pp. 183-186.

With the person laying on the table, I find a strong indicator muscle, usually a facia lata or a quadriceps muscle. Instead of rotating the person on the table, I have found that it is quicker to lower the headpiece, having the person's head lower than the rest of their body. I now retest the strong indicator muscle. If the strong indicator muscle switches off, on one side or both sides, then I know there is congestion and the lymph is not moving freely.

I leave the head in the lowered position, I ask the person for permission if it is ok to assist them in getting the congestion out and massaging the chest. When I get a "no", then, I tell them the importance of getting the congestion out of the chest area and tell them how to massage their own points.

Most people come for results and definitely say "yes". Then I proceed to massage and work with the left chest first, because this is the main lymphatic drainage system to 3/4 of the body. I retest and now the left should be strong and whether the right indicator muscle is strong or not, I always massage both sides of the chest.

Massaging both sides of the chest is very important, even when one side shows strong. Otherwise the person will feel lopsided or off-balanced yet and tell you the next time they come in that you did not do a complete job and now this side still feels tight.

TREATMENT AND BENEFITS:
The first procedure is to stimulate all of the trigger points on the origin and insertion of all the chest muscles, especially the pectoralis major, minor and anterior deltoid muscles.[8] This will increase the circulation in the muscle area.

Many of these origins and insertions are cross-referenced with Receptor Tonus Technique by Dr. Raymond L. Nimmo, D.C. ref.Nimmo

Enhancing Retrograde Lymphatic, Wagner, p 5

While massaging the chest, I keep in mind all of the related organs with the Neurolymphatic (NL) points that are being stimulated.[9]

I would like to refer back to my first book on Applied Kinesiology by Dr. George J. Goodheart, D.C., 1972 workshop manual on page 24. "Findings: STOMACH PROBLEMS; DISTURBANCES OF LIVER, GALL BLADDER, DUODENUM AND PANCREAS BROUGHT ABOUT EMOTIONALLY. ALSO ULCERS." and then on the bottom of page 24 "IN EMOTIONAL PROBLEMS THIS MUSCLE IS OFTEN FOUND WEAK. USE N.V. CONTACT UP TO 4 MINUTES."[10]

Several of us were experimenting in 1973 with this new information. We found that by holding the anterior NL and NV at the same time we could clear out these emotional points much faster. When I am on the left side, I place my right hand resting comfortably and lightly on the anterior NV points on the forehead, with my thumb on ST 7 in the hollow of the cheek. My left hand contact is on the 5th rib intercostal space on the anterior NL points for the pectoralis major, clavicular muscle. When I am doing this procedure by myself, I do one side then the other side. I always do both sides for the full balance of the body.

When I have an assistant doing this procedure with me, we have contact on the right and left sides holding the NL and NV simultaneously. We use the anterior NL points as a barometer. As long as there is pain or tenderness in the NL points, we hold the points until the pain decreases 90% or is totally eliminated. We then proceed to the next point under the breast area, along the fifth intercostal space.

Many times I find these NL points under breast area are very sore and sensitive. I hold these rib points with a firm gentle touch for about 30 seconds to a minute, then lightly rub and ask the person are these points relaxing or is the tenderness gone? If no, then I proceed to hold these points with a firmer pressure for another 30 seconds. If these points are too sensitive, please back off, because it is much better for the comfort of the patient to be relaxed and not tightening up from the discomfort and/or pain. When the point is no longer sensitive, I go on to the next point. If a point is not sensitive at all, I hold for a quick 15 seconds and move on to the next point. Remember to point your fingers up into the rib space under the breast.

There are 4 main points under the 5th intercostal space. When these points are extremely tender or sensitive, I hold and balance these points out longer, using the following technique.

Enhancing Retrograde Lymphatic, Wagner, p 6

On the left side, the first point, K 22, is by the sternum, around 7:00 on the left breast. (5:00 on the right breast). When tenderness is gone, go lateral to the second point on the 5th rib space intersecting with the ST meridian. This point is ST 18 located around 6:00 under both of the breast. The Stomach meridian is the only Yang meridian that flows directly through the chest area. The ST meridian starts under the eye orbit at ST 1 and flows down through the chest, from the clavicle ST 13 through the nipple ST 17 to ST 18 and on down to ST 45 on the 2nd toe of the foot.

While holding each point for 30 sec. to 1 min. or even longer, I ask the person if there is anything bothering them, or does any stressful thoughts come to their mind, or what are they thinking of. This might not seem important to us, many times these questions are very important to the person and possibly that this is their main stress or what's bothering them. I find, that talking about their problems, many times this helps the person to clear out some emotional problems.

Continuing on the lateral breast area, the third point on the left breast is SP 18 at 4:00 (8:00 right breast), on the Spleen meridian. The fourth point is SP 19, also on the Spleen meridian, at 3:00 left breast (9:00 right breast).

When these four NL emotional points (this is what I called them) are not overly tender, I would omit balancing the emotional points. I proceed to stimulate the origins and insertions of the muscles, the points along the sternum, under the breast and all the points in between. I would even trigger SP 20 and at times I would massage SP 21. SP 21 is located on the mid-axillary line, 6 cun below the axilla, midway between the axilla and the free end of the 11th rib. SP 21 is a Major Luo-Connecting Point of the Spleen, and is the Master Luo point for extremities functions. It is very important to stimulate, especially when the person had any type of stroke, difficult walking or any problems with their extremities function.

The meridians around the chest area are all very important. By doing the retrograde lymphatic, we are stimulating and increasing the flow of the Chi Energy or Life Force through the whole body. There are many important NL and acupuncture points[11],[12] being stimulated on or near the chest area.

Conception Vessel Meridian (CV) is very important, especially when there is tenderness on the sternum, from congestion in the lungs and chest area to tightness in the diaphragm. CV is a Yin meridian flowing up the center line from the pubic to the lower lip.

CV 12 is 4 cun (body inch) above the umbilicus and is the Front Mu Point for the Stomach or the Stomach Alarm Point.
CV 14 is 6 cun above the umbilicus and is the Front Mu Point for the Heart or the Heart Alarm Point.
CV 15 is 7 cun above the umbilicus and is located below the xiphoid process. This point is also the Luo-Connecting Point.
CV 16 is the on the level with the 5th intercostal space.
CV 17 is between the nipples, on the level with the 4th intercostal space. This is the Front Mu Point for the CX meridian or the Circulation Sex meridian Alarm Point. This is also the Master Luo point for Chi Energy and Circulation.
CV 18 is the on the level with the 3rd intercostal space.
CV 19 is the on the level with the 2nd intercostal space.
CV 20 is the on the level with the 1st intercostal space.
CV 21 is between CV 20 and CV 22.
CV 22 is in the center of the suprasternal fossa.

While massaging the origin and insertion, there are many NL points being stimulated, especially along the Kidney (yin) meridian.

K 22 location in the 5th intercostal space, 2 cun lateral to CV 16.
K 23 location in the 4th intercostal space, 2 cun lateral to CV 17.
K 24 location in the 3rd intercostal space, 2 cun lateral to CV 18.
K 25 location in the 2nd intercostal space, 2 cun lateral to CV 19.
K 26 location in the 1st intercostal space, 2 cun lateral to CV 20.
K 27 location in the depression on the lower clavicle border, 2 cun lateral to CV 21.

There are three more yin meridians running from the chest to the fingers. These are Circulation seX (CX) (Pericardium), Lung and Heart.

Lung 1 is the Front Mu Point for the Lung or the Lung Alarm Point. L 1 is 1 cun below L 2 and is 6 cun lateral to CV 21, below the acromial extremity of the clavicle.

L 2 is in the depression below the acromial extremity of the clavicle, 6 cun lateral to CV 22.

Heart 1 is located in the center of the axilla, on the medial side of the axillary artery. I trigger the points under the pectoris muscles, starting from the armpit to under the breast area.[13]

CX 1 is located on the breast, 1 cun (body inch) up and 1 cun lateral from ST 17 the nipple. CX starts at CX 1 and flows to the shoulder and down the inside of the are to the middle finger

Enhancing Retrograde Lymphatic, Wagner, p 8

In the acupuncture techniques, when a person has a skin ulceration, inflammation or irritation, then we use the technique "Surround the Dragon"[14] This is putting the acupuncture needles around the area being involved to promote faster healing.

When there is congestion or a lump in the breast, I hold the area with the fingers like surrounding the dragon. I hold this area to increase the energy; by doing this, the lumps many times get smaller in size and usually are less tender, if calcified, there will be no changes. Always ask if they feel any tenderness, if so, be gentle with the area. There are a lot of NL and tender spots in the breast area.

When CX 1 is very sensitive or tender, many times I also find the pubic bone area to be tender, then I balance CX 1 with the pubic bone. I am on the left side of the person, my right hand on the CX 1 point and my left hand on the lateral side of the pubic bone, above the inguinal ligament. These two points CX 1 and the pubic bone help to balance and relax some painful menstrual cramps. These two points are along the mammary line, from the armpit, through the breast, abdominal, past the groin and into the inside of the leg.

It is possible for a person to have an extra nipple or breast on the mammary line.[15],[16] I have seen a man with a left extra nipple located on the lower rib area, he said that it is just a birth mark.

When CX 1 is not overly sensitive, I would omit balancing these two points and proceed to massage and stimulate the rest of the muscles and points on the chest.

When one side feels all relaxed and cleared out, I proceed on the other side doing the same thing. Before I do, I recheck a muscle for strength and see if there is weakness on the opposite side yet. Even if there is no weakness, I will continue to massage and balance both sides.

When the right leg shows strong, we know that we have cleared that out. If there is a lot of congestion in the L 1, H 1 area, I will ask the person to raise their arms (raise the head piece). Sitting or standing at their head, I contact under the pectoris muscles and then I hold H 1 with my fingers and hold my thumb pads on L 1. Holding these points helps relax a lot of tension in the neck and shoulders, upper back and the whole chest area. It is very beneficial and feels good to hold these points, increasing the energy and circulation throughout the whole area, clearing out any congestion that we may have worked loose.

Enhancing Retrograde Lymphatic, Wagner, p 9

CAUTION:
If you have a room that has two inches of dust all over, you can't open any windows or doors to clean, you go in and start to stir up the dust, what happens? The dust will be so stirred up that it will suffocate the cleaner. This is the same with the body. I do not do the retrograde lymphatic if it is the first treatment. I usually concentrate on working with getting the elimination organs functioning better first. (kidney, bladder, intestinal tract)
On the second or third treatment, I work with the NL on the chest area. When these NL points are not clearing out, then I proceed to the retrograde lymphatic on the third or fourth treatment.

There may be quicker results when we put the person at a 20 degree retrograde position. I find that tilting the head back starts the congestion to begin to clear out and is not as dramatic on the patient. I check the patient several times in a row. Sometimes, the person needs to have the retrograde lymphatic done each time, then at the third or fourth retrograde lymphatic treatment, many times, I find the chest not to be very sensitive. The retrograde lymphatic still tests to be needed at this time, or the person is asking for me to massage the congestion out of the chest area again. I will then rotate the person to 20 degrees retrograde position and balance the person with the original technique.

SUMMARY:
When the indicator muscle still shows not having the best strength after the retrograde massage, I will do one or at times even both of the following: I will hold the NL points to increase the energy and clear out any congestion that we have worked loose or the person might be dehydrated and needs to lubricate and flush out the excess congestion and toxins. (For this I would have the person drink water and retest.)

By stimulating all the points on the chest area, we are also increasing the whole body circulation. By increasing circulation, we are clearing out congestion. This in turn will bring in oxygen and nutrition to help the body to heal and repair itself. It will also help get the congestion out of the lungs and they will have less tenderness.

We are receiving 65% to 85% effective results in our office, with the retrograde lymphatic massage.

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DIVISION III - COMMENTS ON PUBLISHED PAPERS

CORRECTIONS IN MERIDIAN THERAPY
A letter to Dr. David Walther, D.C. Diplomate
By Dr. Ron C. Wagner, D.C.

Dr. David Walther, D.C. Diplomate
At-Large Member
257 West Abriendo
Pueblo, CO 81004-1870

March 8, 1992

Dear Dr. David,

ABSTRACT:
Ever since I started to study and review your acupuncture meridian therapy section, I been concerned about some differences and concepts of the material presented.

INTRODUCTION:
I have heard Dr. George Goodheart, D.C., Dr. Walter H. Schmitt Jr., D.C. and many other Applied Kinesiology doctors, tell the story of a family, who for generations, always cut two inches off of the roast, because great grandma always did it. Then, they find out that this was started because the pan was too small for the roast and two inches had to be cut off to fit in the pan.

Many times we do this with daily habits, techniques or even updating material.

I have studied Acupuncture and Meridian Therapy and some of my findings are different than yours. Please correct me if I am wrong and send me your source of information, so I can correct my notes.

FINDINGS:
From your SYNOPSIS 1988 blue book, page 207.
I am positive that Yin is Solid and Yang is Hollow and this I believe is just a misprint.

From your 1976 three ring binder, page 187.
The Body section states, that:
Yang is Spine/Back and Yin is Chest/Abdomen.

From your SYNOPSIS 1988 blue book, page 207.
The Body section states, that:
Yin is Chest and Yang is Abdomen,
Yin is Spine and Yang is Back,

Please set me straight if there's been a change in this material.

ADDITIONAL FINDINGS:
Also, another difference in our thinking and my findings with my acupuncture books are the LUO or CONNECTING Points.

CORRECTIONS MERIDIAN THERAPY, Wagner, p 2

On page 184 from your 1976, (3 ring binder),
You have K 5 as your LUO point
and page 186, you have your LUO point listed as K 4,
and again in your 1988 SYNOPSIS blue book,
Page 250 you have K 5 as your LUO point
and page 268, you have your LUO point listed as K 4,

Ralph Alan Dale Ed.D., Ph.D. C.A., F.W.A.S.,
states that the Kidney LUO Point is K 4,

Acupuncture A Comprehensive Text
Shanghai College of Traditional Medicine
Translated and Edited by John O'Connor and Dan Bensky
On pages 129, Table 2-5, The Fifteen Connecting (LUO) Points,
states that the Kidney LUO point is K 4 (Dazhong)

Essentials of Chinese Acupuncture 1980
Foreign Languages Press, Beijing, Page 201,
states that the Kidney LUO-Connecting point is K 4 (Dazhong)

Dr. Fred Stoner, D.C. has his 1975, 1976 notes listed as K 6.
This is probably an incorrect numbering of the flow on the Kidney points around the ankle.

Another misprint is the acupressure tonifying points,
in your 1988 SYNOPSIS blue book, Page 257,
you have K 5 instead of K 3,
on both the 2nd. part of Tonification and Sedation points.

Dr. John F. Thie, D.C. Touch For Health books copyright in 1973:
Revision copyright in 1974 on page 58 had only the point locations and no numbering,
Revision copyright in 1979 on page 64 have SP 3 & K 5 listed for on both the 2nd. part of Tonification and Sedation points.
Revision copyright in 1987 on page 64 have corrected and updated this third revision to SP 3 & K 3 listed for on both the 2nd. part of Tonification and Sedation points.

I know K 3 is correct. Because K 3 is the Earth point on the Kidney meridian and SP 3 & K 3 controls the Water element, through the Ko cycle.

Thank You for your time.
Best In Health,

Dr. Ron C. Wagner D.C.

Dr. Ron C. Wagner, D.C.
4222 Milwaukee st.
Madison, Wisconsin 53714
(608) 244-0211

DR. GEORGE J. GOODHEART
RESEARCH REPORT

DR. GOODHEART'S RESEARCH TAPES
TAPE 121
-1-

-Dejarnette's Category 2 is an osseous sacroiliac lesion. The indicators in posture are a lateral pelvic sway and a high or low hip. The posterior ilium side has pain upon pressure over the origin and insertion of the sartorius/gracilis on the short leg side, and the upper groin. The posterior ischium side will have pain over the origin and insertion of the hamstrings, lateral portion of quadriceps (vastus lateralis), lower groin. UOMS: U = upper, O = obturator, M = medial, S = short for the posterior ilium. LLL: L = lateral thigh, long leg, lower groin for the posterior ischium. Category 2 is therapy localized with one hand over the sacroiliac joint.

-Modification of Category 2: patient is prone and placed in a Patrick-Fabere position (knee is flexed and laterally rotated with the foot on the opposite knee). This tests for the internal and external PSIS rotation.

-Sacrotuberous Syndrome: described by Danish researchers, translated by Mark Newton for GJG. They state that the Patrick-Fabere test will often be positive on the affected side. Fabere: F = flexion, AB = abduction, ER = external rotation, E = extension. While patient is in this position, place a pressure on the flexed knee while stabilizing the opposite hip; may be pain along the sacroiliac joint on the same side. Bilateral pressure at the coccygeal-sacral area in a cephalad and lateral direction may give sacrotuberous pain and may also give symphyseal pain as a result of the pelvic torsion. Often will have a problem between C1 and C2 which is due to the compensatory pattern. Axis of rotation on the affected side, instead of rotating at the sacroiliac joint, will rotate at the symphysis pubis; the culprit is the sacrotuberous ligament. GJG has found:

1. Symphysis pubis on the affected side will therapy localize, whereas the sacroiliac joint will not; this is done in the supine position.
2. Leg length is equal with patient supine, no evidence of a UOMS or LLL palpatory tenderness.
3. When patient therapy localizes the symphysis pubis and has a positive response, there is immediately evidence of the presence of a posterior ilium or ischium in that the leg shortens or lengthens, and the UOMS or LLL palpatory pain appears. Rather than adjusting in the classic osseous way, use a non-high-velocity technique.

-For a posterior ilium, the patient is supine, place a block under the posterior ilium side and the opposite ischium. Flex the knee of the long leg, bring across midline from lateral to medial; flex the short knee and bring it away from the midline from medial to lateral. Both knees are moving in the same direction.

-For a posterior ischium you do a stretch of the sacrotuberous ligament. Prone or side-lying patient, challenge the coccyx-sacral junction in a cephalad and lateral direction. Note palpatory pain in the C1-C2 area on the same side as the positive symphysis TL. Hold the sacrotuberous contact (like a basic contact) in the direction of positive challenge for 10-15 seconds and note removal of pain from C1-C2. Use 5-8 pounds of pressure. In difficult cases, you have to go

rectally to stretch the sacrotuberous ligament.

-Dvorak and Dvorak, Manual Medicine Diagnostics, second revised edition, has a section on the sacrotuberous ligament. "It is a fan shaped ligament whose fibers pass in a propeller-like fashion from origin to insertion, thus the fiber tracts undergo a change over the fibers arising at the most superior portion cutting across anteromedially going practically vertical, and inserting in the ramus of the ischium most anteriorly. Fibers arising at the most inferior portion in contrast ascend posterolaterally to the ischial tuberosity reaching a line of insertion of the most posterior portion. The sacrotuberous ligament lies posterior to the sacrospinous ligament which is weaker and shorter. Portions of the sacrotuberous ligament are close to the origin and serve as part of the origin of the gluteus maximus muscle. Palpating the origin at the coccyx and the sacrum, the examiner must be aware of the anatomical relationship of the sacrotuberous ligament to the sacrospinous ligament and the gluteus maximus. The sacral zones of irritation lie more medially and should not be the cause of confuction. The sacrotuberous ligament is spondylogenically related to the upper thoracic spine and is similar to the sacrospinous ligament which has also a cervical spondylogenic reflex."

-This is a common occurance, i.e. trauma where there is a fall on one leg, or an impact on one side of the pelvis, stretching of the sacrotuberous ligament in pregnancy and childbirth. Injury to the sacrotuberous ligament will cause local pain and tenderness, and also makes the nervous system refractory in an effort to alter the muscle balance, the pain remains and there is no mechanical stabilization of the area, there is a locking of the sacroiliac joint. Normal proprioceptive impulses from the ligament and articularis sacroilii, there will be pain at the insertion of the ligament, and secondary pains in the abdominal viscera. In the standing position, the iliac crest will move up on the side of the tension while the trochanteric line is unaltered, there will be an apparent lengthening of the affected side's extremity. The sacrum will be rotated so the apex will point downward towards the disordered side and there will be a compensatory lateral scoliosis in the spinal column with the convexity to the opposite side. This causes neck pain when the head tries to maintain itself in a level position despite the scoliosis. The basic method of treatment was to distend the ligament so as to alter its tension as was described in the Danish article. GJG has found the additional requirement of movement of the pelvis as described earlier.

-This explains why Logan's basic contact has gotten goof results.

-GJG describes an 18 year old female patient with hematuria, abdominal pain, pain with voiding, but no sign of infection. Upon treatment of the sacrotuberous ligament, the abdominal pain stopped.

-Torticollis associated with upper respiratory problem may respond to the sacrotuberous ligament treatment.

-Patient's with lumbar pain, iliac crest pain with some radiation into the inguinal region, difficulty with sitting, headaches (forehead), neck pain, menstrual difficulty (like something is dropping), continual urge to defecate without defecation with the urge, dyspareunia may reveal this problem. Check the x-rays, may

reveal level trochanters, but a scoliosis in the lumbar spine with the convexity away from the affected side.

-Key to applying the described sacrotuberous ligament contact is not to contact the sacrum as in the pure Logan basic contact, but to take a sacrotuberous contact which is sacrococcygeal with a pressure cephalad and lateral. Change the direction of pressure to negate pain in the cervical spine. Make the adjustment using the blocks, this helps to remove the pain from the symphysis.

-There will be positive therapy localization to the sacrotuberous ligament area. Be sure to place the patient's hand correctly between the coccyx and ischial tuberosity. Correction of the sacrotuberous ligament will negate the therapy localization.

-Check muscles for aerobic weakening; weakening of the muscle with repeated testing. This indicates the need for anti-inflammatory supplements, i.e. Linum B6, vitamin F perles, evening primrose oil, etc. If there is a great deal of pain associated with the sacrotuberous ligament, use up to 6 per day.

-Another key diagnosis to the sacrotuberous ligament is to look at the gluteal crease. Normally it will point the upper end toward the inferior sacrum. In the sacrotuberous ligament dysfunction, the lower end of the gluteal crease will point itself towards the involved side.

-In difficult cases, if the contact on the middle of the ligament does not do the job, sometimes it requires directional pressure over the ligament in order to increase the tension (as you would a muscle) by spreading the ligament apart. Less often it may require origin-insertion directional pressure by pressing them together.

-Manual Medicine Diagostics, Dvorak and Dvorak, an earlier edition. They state that the sacrotuberous ligament is spondylogenically related to the upper thoracics C7-T5. The propeller-like arrangement of the fibers find the expression in the SRS correlation and its manifestations. The most lateral portion of the lower section relates to C7 and the most medial portion relates to T5, whereas the most medial portion of the upper portion next to the coccyx relates to C7 and the most lateral portion on the sacrum relates to as high as T8. This may explain why there may be strange symptoms associated with the sacrotuberous ligament. The sacrotuberous ligament overlies the sacrospinous ligament. The spondylogenic reflex area for the sacrospinous ligament starting at its portion just on the ischium above the obturator is the occiput, and as it goes upwards towards its inserion on the coccyx and sacrum is as high as C6.

-Walter Schmitt, D.C. speaks of the clorox sniff test as a way to discover if there are superoxide radicals in the body. Clorox is an oxidizing agent so if there is an excess of oxidizing agents in the body, sniffing clorox will weaken muscles.

-The body makes its own superoxide radicals and there are superoxide radical quenchers, for instance DMSO on a low order, and vitamin E on a high order. Bilirubin is a normally acting free radical quencher.

-SOT describes the right thumb web as being associated with gall bladder reflexes and the left thumb web being associated with stomach reflexes.

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-There is an area just ahead of the malleolus on the sole of the foot on the right hand side that is associated with gall bladder reflex. These reflex points are described in CMRT technique.

-GJG feels that this point is related to the large intestine.

-Mary Austin, Acupuncture Therapy, 1972, Doctor's Supply Center, 24028 Union, Dearborn, MI 48124, 313-278-2840. "One of the greatest points in acupuncture is the fourth point of the large intestine meridian, LI4, it has been called the Great Eliminator. LI4 is included in a wide variety of couplings or special combinations of points for specific purposes. If I were asked, "If one wished to memorize one point only, is there any one point of such importance that you would choose it?" - there is little doubt in my mind I would go for LI4. As a sinlge point it can be used to regularize and tone up the entire large intestine function: also, as an eliminator, it is used to control the elimination of mental as well as physical toxins from the organism. It is useful in fevers when there is intense thirst, or fever with shivering. All kinds of skin conditions respond to this point, such as acne and boils; all kinds of headache arising out of faulty elimination. The effect on headaches is often quite remarkable. You will also realize there is a wide range of pains in the upper part of the body, in the neck, chest and limbs, eyes, sinuses, teeth, etc. amenable to treatment at LI4; also inflammation of eyelids and conjunctiva, mouth, lips, tonsils, nose, etc. LI4 is also diagnostically quite useful. If you place your thumb well into the point LI4 and your middle finger opposite to it in the palm, near the thenar eminence, so that your thumb and finger serve as pinchers, you will, by working the finger and thumb about, be able to feel the condition of the tissues. If, for example, it feels as if there were a rubbery lump inside, or a small sausage-shaped lump inside, deep in the tissues, you would know that the congested, bound-up unfree condition here mirrors the state of the actual colon. If the flesh between your finger and thumb feels limp or toneless, it will be but a reflection of the large intestine lack of tonicity. If we can alter the condition of the deep tissues at LI4 by massage, and bring them back to a living, pliable, supple condition, free of congestions and adhesions, then whatever is achieved here will have comparable beneficial repercussions on the colon and the entire large intestine function. Regular use of this point is generally extremely beneficial. Now for some special combinations of points or couplings. When children have such symptoms as cough, fever, breathing difficulty, or throat troubles of any kind, the combination at LI4, LI1, and Lung 11 is especially useful. In adults too, this can be useful to relieve bronchitis. LI4 and LI11 is a combination that may be used for all afflictions of the head, face, eyes, and nose. LI2 is a special point for constipation. I have known a few moments' massage treatment at this point to bring about a good bowel movement within ten minutes; the patient was constipated, congested, and without urge immediately before the treatment. Actually I did this particular treatment in order to lower a temperature that, in my opinion, had been too high for too long and must be reduced. But think of LI2 primarily as a special point for constipation rather than fever. For all disorders involving trachea and throat the combination of LI11 and LI15 may

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safely be used with good effects expected. The combination of LI4, LU11, and LU9 has particular influence over the mouth. In the common cold think of LI19 and LI20."

-GJG had a patient with gall bladder symptoms; he tested her with the clorox sniff which was positive. He massaged LI4 for 30-40 seconds and found that this negates the clorox sniff test. He has repeated this 50-100 times since then.

-In SOT, check for occipital fiber 3, line 2, right or left, with nodulation on T4. There is consistently an irritability at LI4 in a large number of patients. Right/gall bladder, left/stomach. Because of the high prevalance, there may be a lot of unresolved stomach and/or liver/gall bladder problems.

-GJG feels this may be a reaction of the large intestine perhaps due to the failure of the stomach or gall bladder. Journal of Science states that bilirubin is one of the best free radical quenchers that the body produces.

-From Harry Eidenier's Newsletter, #48, Dec. 87 - Jan. 88, cited Bilirubin As An Antioxidant, Stocker, Science 1987, "Bilirubin is an antioxidant of possible physiological importance. To prevent the formation of oxidants and repair oxidative damage to tissues, all aerobic living organisms process antioxidant defenses that include the enzyme superoxide dismutase, catalase, glutathione, vitamin C, E, and A. Bilirubin, the end-product of heme catabolism is generally regarded as a potential cytotoxic, liposoluble waste product which needs to be excreted. In this part of the country, the indians used to kill the hedgehog at a certain phase of the moon and take the gall bladder and dip their arrows in it to make poison arrows. However, in a micromolar concentration in vitro, bilirubin efficiently scavanges peroxide radicals. Its antioxidant sensitivity increases as the oxygen is decreased from 20% (normal air) to 2%. In addition, under 2% oxygen in liposomes, bilirubin suppresses oxidation more than alpha tocopherol which is thought to be the best antioxidant for lipid peroxidation. This study supports the beneficial role of bilirubin at low levels."

-The lab values for bilirubin are wide, i.e. 0.1 - 1.2.

-GJG describes performing K27 and SP21 technique on a student at a Chicago seminar. There happened to be a laboratory seminar going on in the same hotel. They obtained a bilirubin prior to the treatment, the value was high; following the treatment the bilirubin decreased to a normal level. This ocurred off and on, not consistent.

-Walter Schmitt, D.C. found that when K27 and SP21 no longer therapy localized, if you place a mild alkali in the body, such as organic minerals on the tongue, or a mild acid, such as betaine hydrochloride on the tongue, if K27/SP21 had not reached its zenith of stimulation, the therapy localiztion would then become positive again and treatment to K27/SP21 would be done longer.

-David Cheetham, D.C. treated a severe allergy patient with 8 minutes of tapping K27/SP21 with no return of the allergy problem.

-John Schmitt's class at Logan did a study on K27/SP21 trying to reproduce the same effects on pH, etc. It varied with the length of time that K27/SP21 was stimulated.

-GJG can change pH of mouth or LAAT about 6 out of 10 times after tapping K27/SP21 for 3-4 minutes. Reinstitute positive TL to K27/SP21

after putting an alkali or acid in the mouth.

-Stimulating LI4 for about one minute has good effects on upper thoracic pain, reocurring upper thoracic subluxations, fixations, "gall bladder" headache, persistent fat indigestion, lack of hydrochloric acid/gas, undigested food in stool.

-LI4 will negate the clorox sniff test. In difficult patients, or patients in a lot of pain, recommend a source of SOD three per day, or whole dessicated spleen. The patients who took the nutritional support did better than those that did not.

-Anyone with a resting potential away from the midpoint of Isaacs' concept, either parasympathetic or sympathetic dominance, will have positive therapy localization to SP21/K27.

-Increased sympathetic drive patient will have positive K27/SP21 therapy localization that is negated by Organic Minerals (alkaline ash minerals) on the tongue. Needs support for the parasympathetic system. Tap SP21/K27. This helps to bring the resting potential back to the mid-potential.

-Increased parasympathetic drive patient will have positive K27/SP21 therapy localization that is negated by Betaine Hydrochloride (acid ash minerals) on the tongue. Tapping SP21/K27 brings the resting potential back to the mid-potential.

DR. GOODHEART'S RESEARCH TAPES
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-Pain at the right thumb web associated with gall bladder and pain at the left thumb web is associated with stomach.

-There are no specific references to these points in SOT except for some simplified hand reflexes.

-Manipulation of these areas seems to enhance the recovery of difficult gall bladder patients or gastric patients along with other important reflexes, i.e. foot reflexes, NL, etc.

-Particular patient with a lot of digestive disturbances, pain at the T2,3,4 area right and left, and reflex pain at the sternocostal junction of the second, third, and fourth ribs. Some foods would aggravate the condition, but would not reproduce the aggravation each time the food was taken. Patient was on supplements to thin the bile (vitamins A, F, and betaine) and bile salts. GJG manipulated both the right and left thumb webs of this patient while she was in an acute episode of pain in the right sternocostal joint and costovertebral areas, and the pain stopped immediately. This was after there had been some other treatment by GJG and analgesics taken by the patient. An outside lab measured the patient's bilirubin prior to treatment and after treatment and there was a change in the level. (Recall GJG treatment of Dale Sandvall who had an increased bilirubin level that was reduced with tapping of SP21 and KI27 from 2.2 to 1.7 in 10 minutes.)

-Common name for the thumb web point is Large Intestine 4 or heaku.

-One of the main functions of the liver is to detoxify the bowel.

-After World War II, there was a large outbreak of hepatitis in Texas due to the hepatotoxic effects of DDT. The Texas treatment was a non-absorbable intestinal antibiotic, seven-up, and hard candy for about a week. This sterilized the lower bowel, the candy provided glucose for liver function to maintain itself, and the seven-up provided sodium citrate.

-The patient won't be aware of a pain over LI4, but if you palpate LI4 with 7 pounds of pressure, the patient will note a very painful point, and the point may feel like a little sausage. Manipulate the point for 2-3 minutes. The sausage sensation disappears and the pain diminishes. This helps with digestive distubances.

-GJG describes holding a contact over the gall bladder and manipulating right LI4, and he would note a gurgle or sound of intestinal function. Manipulating left LI4 and holding a contact over the stomach, he would note a gurgle over the stomach. The contact on the abdomen is not necessary, just the manipulation of LI4.

-Mary Austin, Acupuncture Therapy, 1972. See quote in previous tape number 121, pages 3-4. In addition, there is a lung point (LU4) that is very close to LI4, located slightly palmward from the web of the thumb. This point is treated along with LI4.

-Schmitt's clorox sniff test detects problems with producing too many superoxide radicals. If the patient has increased oxide radicals, sniffing clorox causes general muscle weakness. This shows need for

superoxide radical quenchers, i.e. superoxide dismutase, glutathione, vitamins C and E.

-Refer to Bilirubin As An Antioxidant quote in previous tape number 121, page 5. Article in Science, 1987, 235:1043, Stocker et al.

-Bilirubin seems to act like a free radical quencher and it seems to act better than SOD, vitamins A, E, C, glutathione.

-LI4 and LU4 have almost a confluens at the web of the thumb. You will manipulate both at the same time. There is a combined effect.

-Superoxide radicals (produced by white blood cells) attacks friend or foe. The body makes SOD in the liver from manganese, copper, and zinc. SOD, A, C, E, and bilirubin act as antioxidants.

-GJG describes the IFF system used during WWII in England. While flying, you needed to have the IFF system in use (identification friend or foe) while crossing a channel. If the IFF was not in use, the British antiaircraft would shoot you down. Germans would fly suicide missions in order to bomb the aircraft factories in southern England, so it was necessary to use the IFF.

-Sometimes people don't have their IFF working for them (SOD, A, E, C, etc.) and their own superoxide radicals produced from the white blood cells can produce devestation in the body.

-Unused bile is relatively cytotoxic, but it is reabsorbed from the blood by the liver. There may be failure of the liver to reabsorb bilirubin because the liver is too busy dealing with the large intestine and cannot tend to normal duties. Stimulation of LI4 produces good effects in patient symptoms and it changes bilirubin levels.

-LI4 is tender to palpation and will therapy localize.

-Check patient for clorox sniff test. If positive, check the various antioxidants for neutralization of the clorox sniff test. Then manipulate LI4 until pain has reduced. Rechallenge with the clorox sniff and many times it will be negative. Also check for therapy localization of L5 which is the associated point for the large intestine. If TL is negative, have the patient stop breathing and recheck to see if TL is know positive. Challenge and correct as usual.

-From Bilirubin As An Antioxidant, "Under 2% oxygen in liposomes, bilirubin suppresses oxidation more than alpha tocopherol which is thought to be the best antioxidant for lipid peroxidation."

-Patients with pain problems or spinal cord injury patients are related to lipid peroxidation.

-A normal amount of bilirubin is influenced by large intestine. Bilirubin levels taken pre and post-treatment show a change in their levels. The levels can go up or down in the change, most often by going up but remaining in the normal range.

-These changes may occur in urobilinogen, but in GJG's opinion, the bilirubin levels must have to be chronically high before this occurs.

-Guyton's Physiology: "Two basic conditions are necessary for the gall bladder to empty. Number one, the sphincter of Odi must relax in order to allow bile to flow from the common bile duct into the duodenum, and second, the gall bladder must contract to provide the force needed to move the bile along the common duct. After a meal, especially a high fat concentration, both of these effects take place.

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First the fat and the partially digested protein in the food entering the small intestine causes a release of cholecystokinin (CCK) from the intestinal mucosa, especially from the upper regions of the small intestine. Then the CCK is absorbed into the blood which passes to the gall bladder which causes specific contractions of the gall bladder muscles which provides the pressure to force the bile toward the duodenum. Second, vagal stimulation associated with the cephalic phase of gastric secretion (anticipation of food) or other intestinal reflexes causes an additional weak contraction of the gall when the gall bladder contracts, the sphincter of Odi relaxes and this may be neurogenic, myogenic, or the effect of CCK in the presence of food produces a peristalsis in the duodenum. Each time a peristaltic wave travels towards the sphincter of Odi this sphincter along with adjacent intestinal wall momentarily relaxes due to the phenomenon of receptive relaxation which travels ahead of the peristaltic contraction wave. If the bile in the common bile duct is under sufficient pressure, a small quantity of bile squirts into the duodenum. Therefore, the gall bladder empties its store of concentrated bile into the duodenum mainly in response to CCK and when there is no fat, the gall bladder empties poorly, but with adequate quantities of fat, the gall bladder empties completely in about an hour. There is an enterohepatic circulation of bile salts. (Precursor of bile is cholesterol. The liver cells must have cholesterol in order to make bile (cholic acid and chemodesoxycholic acid in about equal quantities). These acids combine with lysine and to a lesser extent taurine to form glyco- and tauro- conjugated bile acids. The salts of these acids are then secreted into the bile.) Approximately 94% of bile salts are reabsorbed by an active transport process in the distal ileum intestinal mucosa. They enter the portal blood and upon reaching the liver, the bile salts are absorbed almost totally on the first passage through the liver into the venous sinusoids and into the hepatic cells, and then resecreted into the bile. Bile salts are reused approximately 16 times before being excreted in the feces. The small amount of bile salts lost in the feces are replaced by new bile that is continuously formed by the liver cells. Recirculation of these bile salts is called the enterohepatic circulation. In addition to the strong stimulating effects of bile acids themselves on bile acid secretion, the intestinal hormone secretin also increases bile secretion, sometimes more than doubling the secretion rate for several hours after a meal, however, this increase in secretion represents mainly a secretion of a bicarbonate rich watery solution by the epithelial cells and increased secretion of bile acid by the liver parenchymal cells. The bicarbonate in turn passes into the small intestine and joins the bicarbonate from the pancreas in neutralizing the acid from the stomach, thus the secretin feedback mechanism for neutralizing duodenal acid operates not only through the effects in promoting pancreatic secretion, but also through the effects of the secretion of the liver ducts as well."

-TL LI4; if positive, check the clorox sniff test; if clorix test is positive, see what antioxidant negates (A,C,E, SOD, etc.), manipulate LI4; then recheck for negation of the clorox sniff test.

-GJG had a patient who therapy localized LI4 with only the index

finger. He thought he had stumbled on a new way to identify acupuncture points since the index finger has the large intestine meridian in it, but he found that the patient was simply switched.

-GJG would find a switching pattern in patients who TLed to LI4 with only the index finger. The pattern was both activation of left brain with counting and right brain with humming. He noted that ribonucleic acid neutralized this switching pattern. He would also find indication of switching via TL of the umbilicus and right or left KI27, or both KI27 together. Manipulation of these points neutralized the indication of switching via right or left brain activity as long as the RNA was on the tongue. Once the RNA was removed, the right or left brain switching pattern returned. Manipulation of LI4 right and left would not neutralize the switching pattern. Give the patient 180 milligrams of RNA three times a day for a week in order to "prime the pump". This produced good results.

-Research of Kim Bo Han utilized radioactive phosphorous to identify the meridians. He stated that the meridians were one cell wall thick and contained RNA. The French are now using techtinium 90 to identify meridians.

-Sometimes when you treat the meridian system, you move the energy around and move the "empty place" into another area. In 24 hours, the same empty place would return. Using RNA helps to provide the raw materials (similar to the Schmitt theory of "what if there is no water in the hose").

-Standard need for RNA is the inability to stand on one foot with the eyes closed. Another indicator for the need of RNA is now switching.

-Melzack Wall Pain Control technique: tapping the first tonification point of the meridian involved shows good results with control of pain. If you don't get results, it is possible that there is not enough RNA/memory chemicals.

-Jugular foramen contains the jugular vein, vagus, spinal accessory, and glossopharyngeal nerves. Compression of the occiput and the temporal bone or a stretch of the dural sleeve can initiate vagal disturbances. Need to perform Jugular Decompression. Good technique to use in digestive problems.

-Usual routine for digestive distubances is to be followed. Add to this the LI4 treatment.

-You may find that the tissue between the little finger and ring finger is tender to palpation in these patients. TL and treat if positive.

-Check KI1 on the bottom of the foot for palpatory tenderness. If painful, this patient may have repressed fears or anxieties that are being held in check by a force of will. The patient does not complain of pain at KI1, and will find it hard to believe that it hurts that much on palpation. Manipulation of KI1 may cause an emotional catharsis, not from the pain of the treatment. Mary Austin speaks of this in her book, Acupuncture Therapy, 1972, Doctor's Supply Center, 24028 Union, Dearborn, MI 48124.

-SP21 is a back up for the spinal gate system. If SP21 and KI27 have positive therapy localization when TLed together, treat them by tapping for 3 minutes; this will lower blood sugar and blood pressure.

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Prior to treatment, see if organic minerals or hydrochloric acid negates the positive TL (based on pH of patient). If you prescribe organic minerals, dosage is one-third of a tablet three times a day.

-Fecal pH should be 7.0. Use bromthymol to check stool pH. If turns yellow, the stool is acid. If bromthymol turns blue, the stool is alkaline. Normal is a greenish-yellow.

-Can obtain bromthymol from Seltzer's Pharmacy in Detroit.

-Have the patient bring a sample of toilet paper with stool on it in an appropriate container. Put bromthymol on the tissue to determine if the toilet paper is acid or alkaline itself. Then drop bromthymol on the stool and check for color change.

-Refer to chart of Management of the Colon pH by Food Intake.

-Treatment of LI4 will show a change in the pH of the stool.

-Be sure to supplement the antioxidant nutrient that negated the clorox sniff test after manipulating LI4. Check L5 for subluxation.

MANAGEMENT OF THE COLON pH BY THE FOOD INTAKE

OVER-ACID COLON ←	Most Favorable Area For Normal Bowel Action	Bromthymol Olive-Green	NEUTRAL	→ OVER-ALKALINE COLON
ATONIC COLON ←				→ SPASTIC COLON
Specimen Turns Bromthymol YELLOW ←	Bromthymol Turns Yellow-Green			→ Specimen Turns Bromthymol BLUE

pH Scale 6.0 6.1 6.2 6.3 6.4 6.5 6.6 6.7 6.8 6.9 7.0 7.1 7.2 7.3 7.4 7.5 7.6 7.7 7.8 7.9 8.0 pH Scale

RESULT: FERMENTATION

SYMPTOM: Excessive Intestinal Gas (Methane or Marsh Gas) Almost Odorless

PATHOLOGY: DIARRHEA
Hemorrhoids
Intestinal Ptosis
Colitis (if excessively acid)

ETIOLOGY**

1. Excessive Intake of Foods Which Cause Acidity:
(Aids Lactobacillus Acidophilus Activity Creating Lactic Acid)
Fruits, Vegetables, Starches, Sugars, Buttermilk.
2. Factors Which Tend to SPEED Intestinal Rate:

RESULT: PUTREFACTION

SYMPTOM: Moderate Amount of Intestinal Gas with Putrefactive Odor

PATHOLOGY: CONSTIPATION (alternating diarrhea may be present)
PRURITUS ANI
INTESTINAL TOXEMIA (with symptoms of Hay Fever, Ulcers, Mucous)
Colitis (if excessively alkaline)

ETIOLOGY**

1. Excessive Intake of Foods Which Cause Alkalinity:
(Due to alkaline guanidine created by bacterial activity on proteins)
Animal Proteins: Meats, Poultry, Cheese, Sweet Milk, Eggs, Sea Foods.
2. Factors Which Tend to RETARD Intestinal Rate:

Overactive	LIVER	Sluggish
Sluggish	PANCREAS	Overactive
Insufficient	FAT INTAKE	Excessive
Excessive	HYDROCHLORIC ACID	Insufficient
Insufficient	VITAMIN B COMPLEX	Excessive

THERAPY

1. DIETARY REGULATION: Select More Foods From List Which Causes ALKALINTY.
2. Correct Item 2 by proper supplementation.

THERAPY

1. DIETARY REGULATION: Select More Foods From List Which Causes ACIDITY.
2. Correct Item 2 by proper supplementation.

**Note: It should be recalled that the Colon is a food tube and is affected differently than the blood serum pH by foods.

DR. GOODHEART'S RESEARCH TAPES
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-Changes in weather/barometric pressure affect certain people. There are obvious effects of perspiring with heat and shivering with cold. Changes in weather/barometric pressure are unaccompanied by any science as obvious as the effect of heat or cold on the body. With a change in weather, there is an increase in the circulating level of serotonin. To balance the body with the increase in serotonin, the body then makes 5-hydroxyindolacetic acid and then this person is not affected by the weather. Some patients are able to tell that the weather is changing due to return of pain in an old injury.

-This is especially evident in patients with sinus problems. Common neurolymphatic reflex for the sinuses is located half way between the proximal and distal clavicle, just underneath. This neurolymphatic is associated with differences in head level. Attention to the anterior and posterior neurolymphatic reflexes is accompanied by marked changes in the progression and activity of the sinus, but there is a certain hardcore of patients that continue to have difficulty. They have blocked nasal turbinates, head pain, etc., and alteration of the sternocleidomastoid and upper trapezius.

-GJG has had a series of patients who are affected very strongly by the weather.

-Previous tape discusses the use of LI4 therapeutically when there is a positive clorox sniff test.

-Triple warmer meridian associated with the teres minor/thyroid.

-DeJarnette has stated that disturbances in thyroid function may manifest themselves as sinus trouble.

-We often recommend the use of iodine, especially patients with thick mucous that is difficult to get rid of. Use iodine, tablet or liquid (Ioaquosol).

-Also balance the head on the neck, i.e. Pitch, Roll, Yaw, Tilt, Auricular Technique.

-There is still a hardcore series of patients that do not respond to the usual treatment.

-Patient's say: their hands and feet are cold, that they put they shoulders up in October and don't put them down until April.

-Patients who have difficulty with cold or changes in weather, you would check their axillary temperature. Some patients would reveal low thyroid function with a low axillary temperature. However, there are some patients who have symptoms of low thyroid but have normal temperatures.

-Mary Austin, Acupuncture Therapy, 1972: "The temperature organ regulation function amounts to an automatic self regulating or cooridination of three master or chi switches, one triggered by heat, another triggered by chemical reaction, and the third triggered by pressure/rythmn. The temperature organ effect or function will be concerned with the transfer of heat or heat energy from one part of the body to another. Either to retard or accelerate this conservation or dissipation of heat alread there and the production of heat within the body either to retard or accelerate this process of manufacture.

Therefore, there are three duties to be performed. Where is this point located? It will need to be where the blood temperature is representative of the total body temperature of the inner organs. If there is such an organ, if such an organ exists, it would almost be perfectly protected from all risk of trauma or disturbances from external circumstances. The diagram to illustrate this is the traditional placing of the triple heater based on the illustrations of Ni Ching."

-The diagram and the ancient Ni Ching closely approximate that part of the hypothalamus that rests upon the circle of Willis. It is of the same tissue matrix as the optic nerves and retina. Located just posterior to the pituitary. The circle of Willis is named after the english anatomist in 1675, it was called then the arterial circle. It is formed by the internal carotids and the basilar arteries.

-There is a thermostat in the hypothalamus where the circle of Willis is located. This discovery resulted from experiments by Dr. Bensinger and his team at the head of the Calometric Branch and Biomedical Energetic Division of the Naval Medical Research Institute at Bethesda, Maryland.

-Try this the next time you see a difficult patient with head/neck range of motion problems, chronic sinus irritability, symptoms associated with weather or temperature changes, symptoms associated with under or over active thyroid. This is most acutely noted in patients who react with a change in weather. The chinese used to say "you have January rheumatism", and there is a certain amount of truth in this.

-DeJarnette discusses palpating the upper trapezius. The upper trap has a dual innervation.

-GJG determines on patients before they leave that with the left leg forward the left upper trap and right sternocleidomastoid turns off. This is normal. Beginning point for the PLUS technique.

-Patient who presents with a torticollis often says, "I must have slept wrong". The average case responds well to conservative manipulative treatments. However, some of the more difficult cases proceed to include an arm or a shoulder and a great deal of distress.

-Next time you have a patient with a difficult sinus or neck condition, check triple warmer 15 (TW15) which is located on the trapezius muscle, on the posterosuperior shoulder about half way between the point of the shoulder and the base of the neck.

-GJG notes that TW15 requires dual treatment.

-(Auricular Pull Technique of Dr. Watkins: obtain palpatory pain over the neck or back, look for the low occiput side, pull down on the ear on the low occiput side and see if this relieves the palpatory pain.)

-If you have a patient with the head/shoulder unlevel, check the upper trapezius and sternocleidomastoid as usual. Under most circumstances, you would find these involved and correct as usual. In a certain number of patients, they will have postural evidence of upper trap/SCM imbalance, but it is not weak. In this case, have the patient therapy localize TW15 and check for weakening of the upper trap/SCM. This does not respond to the 5 IVF factors, but rather to strain/counterstrain with the trigger point at TW15.

TAPE 123
-3-

-GJG has had patients who were always cold, hands and feet cold, or patients who had difficulty in maintaining a normal temperature, or temperature normality between one part of the body and another, i.e. between the hands and the feet, or hands and the head, etc. Patients may sweat only at the head and not anywhere else (this may be a vitamin D deficiency or an indication to check TW15), or a patient that is cold in your treatment room and stnading next to the space heater when everyone else is comfortable.

-Take the pain out of TW15 by strain/counterstrain of the upper trapezius. After the pain is out of TW15, put a thermistor on the hand or foot, or whatever the patient complains is too hot or too cold. Then tap TW15 and note change in temperature.

-GJG uses the liquid crystal thermography unit to show temperature change.

-Occasionally the strain/counterstrain will change the temperature, but you usually need to tap TW15 to make the change.

-Sometimes this works for patients whose hands and feet perspire. This is usually a problem with a lack of perspiration all over the rest of the body. This is treated by rubbing lemon over the skin and then taking a hot bath with a couple of handfuls of epsom salts. In some patients the sweaty hands or feet is due to alteration in TW.

-Draw a horizontal line through the top of the spinous process of T1 and a vertical line touching the inner border of the scapula, the distance between the T1 spinous process line and the point where the horizontal and vertical lines cross is an area referred to by Austin as AB. Extend the horizontal line a half inch lateral of line AB which then locates the hydrometric point (TW15).

-Acupuncture points are able to be found on cadavers with a point finder.

-Have patient therapy localize the pulse points to find if triple warmer is indicated. Regular therapy localization may not be positive until you have the patient stop breathing (breath cessation).

-Loss of hearing and tinnitus: check TW1,2,3,15,16.

-To improve circulation to the brain in patients with cerebral palsy, epilepsy, poor development we do cranial technique. While performing the cranial technique, the patient respires their own air, carbon dioxide is a potent vasodilator. Place a plastic glove over the nose and mouth of the patient and have them respire while you perform cranial technique.

-Check for diaphragm dysfunction, if positive, one of the factors to check and correct is a subluxation at C3. Have the patient therapy localize C3 and check for weakening of a strong indicator muscle. Often times straight TL to C3 is negative, but check TL of C3 against inspiration or expiration and the TL now becomes positive.

-Prior to correction of the diaphragm, check the patient's ability to tolerate rebreathing their own air, i.e. how many times they can breathe in the plastic glove. After correction of the diaphragm and related factors (NL, C3, thoracolumbar fixation, psoas reactivity), the patient will be able to increase the number of breaths in the glove, and the vital capacity will increase.

-A weak muscle that strengthens or a strong muscle that weakens on rebreathing carbon dioxide indicates a vitamin B deficiency.

Placing a tablet of Standard Process Cataplex B on the tongue will neutralize the breathing reaction in the muscle.

-Recurrent switching: needs K27/umbilicus, lack of magnesium (chronic tension in subclavius), stimulation of memory circuit (give them RNA), difficulty distinguishing right and left (needs folic acid), reading rapidly, backward, say multiplication tables, hum, etc. reveals switching.

-Relationship between levels of oxygen to the brain and switching.

-If a patient weakens when they rebreathe their own carbon dioxide five times, vitamin B or manganese may negate this weakness; but GJG has found that there is a subclinical subluxation at C3 that would only therapy localize with patient inspiration or expiration, most often expiration, and this correction negated the rebreathing weakness.

-The diaphragm drives the acupuncture system.

-New neurotransmitter concepts from the University of Michigan, Dr. Michael Marietta. The most common neurotransmitter is not what we think (acetylcholine, etc.), but is actually nitric oxide (key signalling for cells). Nitric oxide is one of the gases they check as an emitter from automobiles.
This neurotransmitter does not last more than 2-3 seconds, acts to facilitate transmission. Many vital functions of the body are mediated through nitric oxide. Blood vessels contain a smooth muscle relaxing factor (known for the last 4-5 years). The endothelial smooth muscle lining of blood vessels constantly are emitting little puffs of nitric oxide. In the immune system, the phagocytes (scavengers that kill bacteria), liver cells, and fibroblasts all use nitric oxide. Phagocytes produce nitric oxide.

-Nitric oxide has escaped physiologist's attention because it survives in the body for only seconds and because it bears no resemblance to any of the other biologic regulators (neurotransmitters).

-Nitric oxide is a messenger molecule that is involved in a wide range of activities: mediates blood pressure, helps immune system to kill especially invading parasites, stops cancer cells from dividing, can cause large scale death of brain cells that can debilitate people with stroke or Huntington's disease even though the cells of the brain that make it are immune to it.

-Dr. Salvatore Moncatta, research director at the Burroughs Welcome Research Laboratories in London wrote an article in 1989 suggesting that nitric oxide is the universal signal transducer (an intermediary that converts messages from one form to another). He feels that they have stumbled on something very big.

-Investigators have known for a long time that there was a substance made by the endothelial cells that line the blood vessels and that substance would diffuse into the muscle cells that grip the blood vessels and make the muscles relax. Yet this substance, which was then known as the endothelial-derived relaxing factor, appeared only fleetingly and no one had been able to characterize it before it was gone.

-The existence of this agent was then further documented by Dr. Robert Furchgut at the Down State Medical Center in Brooklyn at the

State University of New York. He and a Dr. Louis Ignaro at the University of California speculated that it may be nitric oxide since the biochemical properties of nitric oxide were very similar. There was some evidence that people would use nitroglycerin for heart disturbances, nitroglycerin breaks down to nitric oxide. Moncatta used the same machine that measures nitric oxide from car exhaust and aligned it to look for the chemical from the exhalation of cells. Putting nitric oxide on smooth muscle had exactly the same effect as the endothelial-derived relaxing factor.

-In addition to the nitric acid controlling blood pressure, they found that blood pressure soared when the endothelial cells were prevented from making this substance. The usual thought that the major signals that controlled blood pressure were signals that caused blood vessel constriction was something that was accepted, but Dr. John Hibbs of the University of Utah School of Medicine said that now it is the major signal that dilates blood vessels, nitric oxide. This is exactly the opposite view of the prevailing theories.

-A second threat of the nitric oxide study started with a question about substances that are thought to cause cancer. Researchers at MIT wondered whether mammals could synthesize nitrates or nitrites, and if so, were they being converted by cells into cancer causing nitrosamines. Cured meats and some vegetables are sources of this. Dr. Steven Tanenbaum of MIT gave student volunteers a diet low in nitrates and nitrites and analyzed the urine to see how much they were excreting. During the experiment, one of the students developed a viral disease with diarrhea and suddenly she began to excrete nine times the normal amount of nitrites and nitrates. This suggested to Dr. Marletta of MIT that the white blood cells of the immune system that are recruited to fight the viral infection, might be making nitrates and nitrites. They found this in the macrophages.

-Dr. Hibbs had been studying macrophages since 1970 trying to figure out how they killed infected cells. He knew that the macrophages needed one crucial amino acid to do the job. It turns out to be arginine.

-In the urea cycle, arginine is converted by arginase into citruline. How is the conversion of citruline associated with the killing of cells? When arginine is converted to citruline, it loses a nitrgen atom and this nitrogen atom is turned into nitric oxide which is what kills the infected cells and allows the macrophage to do this. Not only did they find that second link, but the compound that relaxed the blood vessels could be made on demand by the macrophages to kill invading organisms and parasitic invaders like the mycobacterium that causes tuberculosis and leprosy, or the cryptococcus in toxoplasmic organisms that plague people with AIDS. These bacteria can slip inside cells, then those cells can burn up the person with puffs of nitric oxide.

-Dr. Hibbs said that cancer cells use nitric oxide in making it to control their own growth. When cancer cells start producing the compound they stop growing. The cancer cells are prodded to produce the nitric oxide by immune system hormones, especially gamma interferon and the tumor necrosis factor. This allowed Dr. Hibbs to explain how the immune system helps defend the body defend against

cancer.

-Snider (who worked with Candace Pert and did the radioimmune histology stuff where they tagged the morphine and other opiates with radioactivity and found where they went in the brain), a neurobiologist at Johns Hopkins, said that nitric oxide is too nice not to be in the brain. He soon found that nitric oxide had a major role in neuron function. The enzyme that makes the nitric oxide arginase is greater in quantity in the brain than any where else. He states that the brain enzyme arginase is in the cytoplasm, the substance that makes up the body of the cell, but it can move to the membrane that encases itself when the cell needs to make nitric oxide and thus the chemical can be released inside of the cell without hurting the cell that makes it. The nitric oxide acts as a neurotransmitter passing messages between brain cells, but it is unlike any neurotransmitter ever found before. All the other neurotransmitters are stored in little bags inside the cells, like acetylcholine, and then released when they are needed. Nitric oxide in contrast is created only when needed. Sometimes the nerve cells can produce too much. Snider found that when the nerve cells would die, certain nerve cells that were actually making nitric oxide, not only were those neurons uniquely resistent to the neurotoxicity that was around them, but they caused it. The real damage from strokes occurs after the initial event of blockage. Initial blockage starves a group of brain cells that quickly die, then nerve cells in a larger area 10 times the original size of cell death begin to react and they release glutamic acid (part of the glutathione molecule) that stimulates the other cells to release a cloud of nitric oxide. The nitric oxide then kills all the cells in the area except those cells that are releasing it. If the cells have enough arginine, this stops the nitric oxide from killing them.

-Types of patients to check this in: stroke patients, failure to respond patients, patients that weaken on rebreathing carbon dioxide.

-GJG had a stroke patient who was unable to flex his knee while lying prone even after one and a half years of treatment. Placing two Arginex (Standard Process Labs) (contains arginase) on the patient's tongue caused him to immediately bend the knee.

-Another patient with a spinal cord injury. GJG placed a plethysmograph on the dorsalis pedis. The patient weakened with arginine on the tongue (zinc arginate, potassium arginate, charged amino acids) and also weakened with glutamic acid on the tongue. With Arginex on the tongue with either the arginine or glutamic acid, the weakness was negated. Patient kept a few Arginex in his mouth and checked for a reaction on the plethysmograph (made sure first that a chewing motion of the TMJ did not affect the plethysmograph), upopn chewing the Arginex, there was a doubling of the width of the tracing (indicates an increased circulation to the area). Also did B&E treatment (common to do this in spinal cord injuries).

-Arginex as a source of arginase is useful in kidney, bladder, and liver problems; low specific gravity, elevated BUN (above 25), or elevated creatinine.

-Arginase is a factor in urea metabolism for excretion (manganese dependent). The Arginex works best, GJG does not find a need for manganese.

-Restrict the patient to one glass of fluid at night. Urinate and

discard the last evening urine. Upon arising, save the first, second, and third urine specimens (note times of voiding), after the third specimen, the patient can resume eating and drinking. This is the concentration phase. On the second night, no food or drink after the evening meal and kept inactive. Upon awakening, the patient discards the first urine specimen, then after this urination the patient is given 5 glasses of fluid to drink within 15 minutes. Urine is collected at 1, 2, 3, and 4 hours after the patient has started drinking. These samples are saved and recorded. After the 4 hour specimen, the patient may food/drink. This is the dilution phase.

-The specific gravity is measured for each specimen. The range should be 1.026 or over for the concentration phase, and 1.003 and gradually increasing for the dilution phase.

-In general, GJG just measures the specific gravity of one sample, and if it is very low or very high, test the patient against liver and/or kidney for need of Arginex.

-Test for available nitric oxide by the arginase splitting off the nitrogen from the arginine, if arginine weakens the patient, they need Arginex (source of arginase).

-Some patients are taking protein supplements and are overdosing themselves with arginine. This is a popular concept with people who are trying to stimulate human growth factor to increase muscle size.

-Most patients who weaken on rebreathing carbon dioxide will also weaken on arginine. GJG still working on where the structure, chemistry, and psychology is in relation to this.

-Test protocol: if arginine in the mouth weakens the patient, put Arginex (source of arginase) in the mouth to check for negation of weakening. Recommended dosage is to chew 3-6 per day.

-There are biochemical abnormalities that cause hyperammonemia. Can measure plasma arginine levels. The literature also describes if there is a deficiency of arginosuccinic acid. GJG has not done this. This is not a discussion of arginine and its relationship to the urea cycle, but rather that the arginase splits off a nitrogen molecule from the arginine to provide the base for the nitric oxide.

-Arginine is a normal part of the urea cycle for elimination of ammonia. GJG has checked patients for sniffing ammonia and this had no effect on the patient. This is not an ammonia problem, but that it relates to the level of nitric oxide.

-If there is no reaction, there may be a need for manganese or a failure of small intestine absorption.

-When the arginine can't be broken down, the body will find an alternate pathway. This involves the arginine combining with available glycine and eliminating ammonia via the creatinine which spills into the urine (Schmitt). This is different from the discussion of nitric oxide.

-GJG has been testing patients (under the supervision of a dentist) for sniffing nitrous oxide.

-There is no effect of glycine on these patients, either strengthening or weakening.

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* Diplomate
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