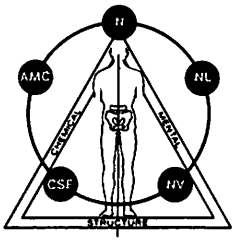


COLLECTED PAPERS OF THE MEMBERS OF THE INTERNATIONAL COLLEGE OF APPLIED KINESIOLOGY-U.S.A.

Volume II
Winter, 1988-89

PRESENTED JANUARY 2 THROUGH JANUARY 7, 1989

KALAPAKI BEACH
LIHUE, HAWAII

Introduction

David S. Walther, D.C.
Chairman, Education Committee

This twenty-sixth collection of papers of members of the International College of Applied Kinesiology-USA contains thirty-nine papers by twenty-four authors. The papers will be presented by the authors to the general membership at the Winter Meeting of the ICAK in Kauai, HI, January 2-7, 1989. 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 on the Table of Contents page.

The manuscripts are published by the ICAK as presented by the authors. There has been no effort to edit them in any way; however, they have been reviewed by members of the Education Committee for originality and to determine that they follow the "Instructions to Authors of Collected Papers" published by the ICAK. The primary purpose of the ICAK in publishing the *Collected Papers* 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 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.

With this edition of the *Collected Papers of the Members of the International College of Applied Kinesiology-USA,* there are two format changes adopted by the Executive Board. First, there are three divisions of papers. Division 1 consists of papers for members' information. Division 2 contains papers where the author invites constructive comments to be published in future editions of the *Collected Papers,* and division 3 is for constructive comments on papers published in division 2. Papers will be put in division 1 or 2 at the author's request. It is expected that authors will choose division 1 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 2 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 of 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 will only be published in this area on papers presented in section 2 inviting constructive criticism.

This issue of the *Collected Papers* has two commentary articles, authored by the Chairman of the Education Committee, on papers published in this issue. These are samples to initiate this section of the *Collected Papers.*

The second change to appear in this edition of the *Collected Papers* is an opportunity to vary the format of paper presentation from that required in the instructions to authors. For example, some papers read better when certain portions are single-spaced rather than the required double-spacing. Deviation from the instructions to authors is only obtained by special application to the Education Committee, which currently is responsible for screening the papers for publication. As the ICAK-USA reorganizes with new by-laws, there will be a Publications Committee that will take over this duty. Whether a paper can vary from the usual guidelines will be the decision of the committee responsible for screening the papers.

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

MESSAGE FROM THE CHAIRMAN

The International College of Applied Kinesiology-USA continues to flourish as a forum in which doctors in the healing professions can present their ideas and their research. By contributing to the collected papers, members have the opportunity to be heard and to be guided in their further efforts through the feedback of their colleagues.

The collected papers include a compilation of research reports, validation studies, case reports and intellectual discourses on various aspects of Applied Kinesiology. Some of the papers represent "seeds" which will grow into powerful diagnostic and therapeutic procedures.

The members of the International College of Applied Kinesiology-USA are to be congratulated, not only for contributing to this collection of papers, but for receiving them, studying them and assisting their authors in the further development of their ideas, concepts and procedures. Through the synergistic effects of helping ourselves and each other to grow, we become a more powerful team, and our contribution to the healing arts and to the health of the world's people multiplies in an exponential manner.

Robert M. Blaich, D.C.
Diplomate
Chairman, ICAK-USA

*Diplomate

****TABLE OF CONTENTS****

DIVISION I - PAPERS FOR MEMBERS INFORMATION

Table of Contents
Cont'd

Table of Contents
Cont'd

Table of Contents
Cont'd

Instructions to Authors of Collected Papers — ICAK-USA

The *Collected Papers of the Members of the ICAK-USA* are published twice annually, prior to the summer and winter meetings. Manuscripts are reviewed for format, originality, and quality for reproduction. There is no review for authenticity of material.

The ICAK-USA recognizes that the usual procedure for selection of scientific papers is a blind review. The purpose of the *Collected Papers of the Members of the ICAK-USA* is to stimulate creative thinking among its members. These papers are distributed only to the members of the ICAK-USA 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 *Collected Papers of the Members of the ICAK-USA* are those of the author(s); the editor(s) and the ICAK-USA disclaim any responsibility or liability for such material.

The current ICAK-USA Status Statement is published with the *Collected Papers*. It is recommended that procedures presented in papers conform with 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-USA Status Statement be supported by statistical studies, literature references, and/or any other data supporting the procedure.

Papers are published in three divisions: 1) papers intended by the author as informative to the membership and not inviting critical review; 2) 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. 3) The third section is for review comments on papers published in division 2.

Manuscripts are accepted by the ICAK-USA for consideration to publish with the understanding that they represent original unpublished work. Acceptance of the manuscript by the ICAK-USA does not necessarily imply acceptance for publishing. The author may appeal any paper rejected to a committee composed of members of the Education 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. The paper must begin with the title, author's name, and an abstract. The abstract should be a brief description of the content of the article.

3. The body of the article should follow the abstract and include an introduction, discussion, research procedure, and discussion of findings. Any or all of these topics may need to be addressed, depending on each paper.

4. The paper is to end with a short summary of the author's conclusions.

5. Quotations should be short, usually no longer than three lines, and should be referenced, giving credit to the original author. All referenced articles, books, and persons other than the author must be properly referenced at the end of the paper, e.g., David S. Walther, *Applied Kinesiology, Volume I — Basic Procedures and Muscle Testing* (Pueblo, CO: Systems DC, 1981). If an article in a journal is referenced, the notation should read as follows: Walter H. Schmitt, Jr., "Fundamentals of Fatty Acid Metabolism — Part II," *The Digest of Chiropractic Economics*, Vol. 28, No. 2 (Sept/Oct 1985).

6. 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 *Collected Papers of the Members of the International College of Applied Kinesiology-USA*, copyrighted by the International College of Applied Kinesiology-USA.

7. All artwork 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 *Collected Papers*. Photographs must be original black-and-white glossy prints.

8. Terminology or procedures that might be unfamiliar to some readers should be referenced at the end of the paper.

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

10. The body of the article should be double-spaced on plain paper. No papers typed on office letterhead will be accepted. The manuscript must be clear copy with dark print to ensure adequate reproduction in the *Collected Papers*. The margins on both sides of the paper must be a minimum of 3/4", and the top and bottom margins 3/4" when relating to 8-1/2" x 11" letter-size paper. European authors should make note of the copy height of the American standard 11" paper size, which relates to approximately 28 cm. Each page of the paper should be identified by an abbreviated title and the author's last name centered at the top of the paper with a 3/4" margin.

11. By special application to the Education Committee, paper format can deviate from that described in #10. Deviation will be allowed only when in the Committee's judgement it improves the readability of the paper.

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

13. Currently the articles to be published should be sent to the Education Committee Chairman in triplicate (the original and two copies). The Education Committee Chairman is David S. Walther, D.C., 275 West Abriendo Avenue, Pueblo, CO 81004.

It is planned to establish a Publications Committee in the near future to review all ICAK-USA publications.

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.

DIVISION I - INFORMATIVE SECTION

CRANIAL FAULTS AND THE CONCEPTION VESSEL

Hans W. Boehnke, D.C.

Abstract: A study of the effectiveness of running the conception vessel in reverse to see if it indeed locks in a cranial correction on the following patient visit.

In Dr. Walther's writing on acupuncture in his first book, he states, "The conception vessel, when stimulated (run in reverse) locks in a cranial correction or upper cervical subluxation correction." I thought it would be informative to study a series of patients making a cranial correction and running the conception vessel in reverse and make a comparison to a control group who had a cranial correction without running the conception vessel. They would be checked on their following visit to see if the cranial correction was maintained.

I had ten patients in each group and the findings indicated that all but one patient in each group maintained their correction on the second visit.

My impression is that it matters little to run the conception vessel reverse after cranial corrections to maintain the corrections on future visits. It is possible that a larger sample of one hundred patients or more in each group would show a different pattern. It may also give more information if the

correction were checked over a series of visits. I was hoping for a clear advantage in doing this procedure on the conception vessel; however, my findings did not show it.

REFERENCES

1) Walther, David. Applied Kinesiology - The Advanced Approach to Chiropractic. Systems D.C., Pueblo, Colorado, 1976.

2) G. J. Goodheart, D.C. 1973 Applied Kinesiology Research Manual.

SYMPATHETIC DOMINANCE AND LATERAL CURVING OF THE SPINE

Hans W. Boehnke, D.C.

Abstract: A pilot study of Gamma II muscle testing that responds to putting the spine convex to the right with some common symptoms and signs of sympathetic dominance.

In Dr. Schmitt's work on centering the spine he makes reference to sympathetic dominance being associated with a left convex spinal position and that a Gamma II weakness will be temporarily neutralized by putting the spine in a right convex position. This is a short study to compare Gamma II response, ie strengthening to placing the spine in a right convex position with some classic sympathetic dominant signs and symptoms.

I selected some commonly accepted sympathetic dominant symptoms and signs and attempted to either validate or challenge Dr. Schmitt's statements with regard to centering the spine and its relation to sympathetic patients.

I selected some 16 patients who I suspected had sympathetic dominance, then found a Gamma II weakness on them and tested it in the clear and with the spine placed convex to the right, that is, in a position approximating the left shoulder and left hip.

When the Gamma II weakness strengthened in a right convex pattern, I asked them two questions.

1) Do you suffer from constipation?

2) Do you have trouble with digestion, ie fullness, belching, etc.?

These questions were followed by answers yes, no or sometimes on my research sheets. Then I checked them for pupil size and observed if they appeared dilated, average or constricted. I then would feel the palms of their hands for moisture and used a plant moisture meter to see if a reading could be obtained. Their pulse was then recorded and their tongues were examined to see if the saliva was thick and viscous or the tongue dry as opposed to the saliva being thin and copious and the tongue wet. The findings were as follows.

1) Do you suffer from constipation?

yes 6 no 7 sometimes 3

2) Do you have trouble with digestion?

yes 12 no 1 sometimes 2

Gamma II Weakness.

Strengthened by spine convex to the right.

yes 16 no 0

Pupil

dilated 13 constricted 0 average 3

Palm moisture (reading taken from moisture meter)

reading between .8 - 1.7 15 reading of 0 was 1.

Pulse

pulse of 72 or higher - 11 lower pulse - 5

Saliva

thick and viscous or dry tongue - 15

thin, copious or wet tongue - 0

normal - 1

DISCUSSION AND CONCLUSIONS.

I feel that the findings do indicate strong support that Dr. Schmitt's ideas are correct with regard to sympathetic dominant types. I feel that the only findings that were not consistent were the answers on the question of constipation. I think that I should likely have worded my question here differently. I should have asked how often they had bowel movements and make my own decision of constipation by frequency.

REFERENCES

1) Schmitt, Walter H. Centering the Spine. Functional Neurological and Biochemical Considerations, ICAK Collected Papers, Summer 1987.

2) Schmitt, Walter H. Cassettes and notes summarizing his three seminar series on the links between the nervous system and body chemistry.

3) Guyton, Arthur C. Textbook of Medical Physiology, Third Edition 1966. W.B. Saunders Company, Philadelphia and London.

4) Chusid, Joseph G. & McDonald, Joseph J. Correlative Neuroanatomy and Functional Neurology, 1967. Lang Medical Publications, Los Altos, California.

5) Kuntz, Albert. The Autonomic Nervous System, Fourth Edition, 1953. Lea and Febiger, Philadelphia.

REFERENCES (continued)

6) Goodheart, George J. Article reprints from Chiropractic Economics related to Acid Alkaline Balance and Patient Management.

APPLIED KINESIOLOGICAL MANAGEMENT OF A KIDNEY STONE: A CASE HISTORY

Cecilia A. Duffy, D.C.

ABSTRACT: Clinical management of a patient with a kidney stone using Applied Kinesiology methods.

INTRODUCTION: Urine pH should normally shift from acid in the morning to alkaline at night (1). Non-changing urinary pH can predispose to stone formation. If the kidney stone is from persistent alkaline urine, acidification is necessary; if due to persistent acid urine, alkalinization is necessary (2). If a kidney stone is supected, urinalysis should include a dip stick (3) and slide examination with stain (Sedi-Stain (4)) under high power. The slide will identify red blood cells, white blood cells, debris, casts, and crystals. In the laboratory section of our office we have charts to help differentiate the various types of crystals.

DISCUSSION: A 38 year old male veterinarian presented complaining of pressure and cramping in the lower abdomen that radiated to the low back and frequency of urination. A urinalysis obtained at approximately noon revealed a pH 7, 3+ blood, a mixture of calcium phosphate and calcium carbonate crystals, and debris. Diagnosis of kidney stone was made. Treatment consisted of correction of the ileocecal valve, right anterior tibialis/bladder meridian over activity/tap right BL58, and a right psoas reactive to the diaphragm, diaphragm NL, and thoracolumbar fixation. Standard Process (SP) Cataplex A every ½ hour and SP Phosfood 10 drops three times a day was prescribed to help acidify the urine and break up the stone (2) (5).

At this point, daily or even twice daily urine samples should be examined to observe changes in pH, crystals, blood, debris, etc., and alter treatment as necessary. In this case, the patient is a veterinarian and capable of examining his own urine. He called daily with the urinalysis and slide examination of his first morning urine.

Over the next four days the urine revealed pH 5-6, 2+-3+ blood, calcium oxalate crystals, heavy debris (stone breaking up), and one day showed ketones. Overall the lower abdominal pain had improved, but there was considerable low back pain. Treatment rendered on the fourth day was for a left anterior tibialis/bladder meridian overactivity/tap left BL58, tapping kidney and conception vessel connecting points simultaneously (K5 and CV15 to move the energy from yang to yin), and an upper thoracic fixation.

The following day (fifth day) there was a trace of blood and debris only. At this point the SP Cataplex A was cut to three a day, SP Phosfood remained at 10 drops three times a day.

The sixth and seventh day there was a increase in blood to 2+, ph 5, heavy debris, and calcium oxalate crystals, and on the seventh day he experienced an increase in frequency and a new symptom of burning urination. It was determined that there was over-acidification and the patient was instructed

to take a tablespoon of baking soda immediately, two teaspoons throughout the day, and stop the Phosfood. This decreased the frequency, burning, and brought the pH up to 7 by evening. He resumed the Phosfood on the eighth day.

On the tenth day a KUB (anteroposterior abdomen) radiograph was obtained. It revealed small calcifications in the region of the right kidney and an increase in the bladder density. First morning urine revealed pH 5, trace blood, and calcium oxalate crystals. The thyroid was evaluated for its role in kidney stones (6). Correction was made of a left teres minor weakness, right teres minor fascial flush, and left sacrospinalis weakness. He was prescribed SP Thytrophin PMG three times a day and taken off of the Cataplex A and Phosfood.

The following day (eleventh) he reported an improvement in symptoms. Treatment rendered was for a right anterior tibialis cranial stress receptor and a right sacrospinalis fascial flush.

Urinalysis gradually improved over the next two weeks. The patient unfortunatly became frustrated and self-administered ampicillin which stopped the residual frequency for a few days and then became ineffective. He then used Keflex with the same results.

A Magnetic Resonance Imaging was performed to rule out kidney and bladder abnormalities two months after resolution of symptoms. It revealed normal kidneys and bladder with no evidence of masses or urinary tract obstruction.

CONCLUSION: This case history of a kidney stone was presented to give direction and treatment rationale based on Applied Kinesiology and proper nutritional supplementation for conservative management of similar cases. It is interesting to note that two major texts state that the urinary pH is of little importance (7) (8).

REFERENCES:

(1) National College of Chiropractic, Physical Diagnosis Course, Spring 1985.

(2) Therapeutic Food Manual, Kidney Stones, reprinted by AK Printing, Geneva, Ohio.

(3) Multistix-Reagent Strips for Urinalysis Insert, Ames, Miles Inc., Diagnostics Div., Elkhart, IN.

(4) Sedi-Stain-Concentrated Stain For Urinary Sediment Insert, Clay Adams, Parsippany, N.J.

(5) A Specific Nutritional Guide And Some Applied Kinesiology Suggestions For Patient's Signs And Symptoms, Lance West, D.C., pages 62-63.

(6) Personal Communications, George J. Goodheart, Jr., D.C., March 10, 1988.

(7) Fundamentals of Internal Medicine, Kaye & Rose, CV Mosby Co., St. Louis, 1983, page 925.

(8) Clinical Laboratory Medicine 4th Edition, Ravel, Year Book Medical Publishers Inc., Chicago, 1984, page 112.

G. V. 20 and Pineal

Edward E. Evans D. C.

Following is a procedure which treats subjective symptoms of hormonal dysfunction which are mostly post-surgical or menopausal related in women.

Many women complain of hot flashes and emotional lows which interfere with them functioning normally. Most of these women are post-surgical or have menopausal related symptoms. Herein is an explanation of the procedure I found which helped most of these cases.

Testing for Gamma I, Gamma II, Structural, and Set Points, I corrected what I found. Some women continued to have symptoms. I decided to try to find another relationship to the sartorius and gracilis muscles. I had the patient T.L. between the eyes about one inch above the brow line for the pineal. The left sartorius being tested became very weak.

I now had a T.L. which I could test nutrition off of, but also wanted to find a Set Point relation to the pineal T.L. I had the patient T.L. all the B. and E. points, but the sartorius still tested weak. I then had the patient T.L. G. V. 20 and the weak sartorius became very strong.

I began to check all patients with this type of symptom and found that some would weaken upon T.L. to the pineal and others needed to T.L. the pineal and G. V. 20 to weaken a sartorius or gracilis.

GV20/Pineal-Evans

Summary of Proeedures

1. Test sartorius or graeilis in elear. If weak, eorreet using five I.V.F. factors.
2. If strong, T.L. pineal and test again. If muscle weakens T.L. G.V. 20 as a two point.
3. If musele then strengthens, tap G.V. 20 100X while patient touches pineal point.

Nutrition

1. If T.L. to pineal or pineal and G.V. 20 weakens a strong sartorius or graeilis:

   Test:

   A. Pineal-pituitary substanee
   B. Adrenal substanee
   C. Tyrosine and tryptophane
   D. Other neurotransmitters

Conclusion

This is another simple proeedure to test hormonal dysfunetion.

HOW ACCURATE IS THE LEG CHECK?

Hannes L. Hendrickson, B.Ch.E., P.E., D.C.

ABSTRACT: Members of ICAK compare the length of legs whether it be for Category analysis or evaluating pre and post treatment of patients etc. This paper is a study of the various factors which must be taken into account when performing these tests. The chief purpose of this paper is to determine "How Accurate Are These Measurements"!

INTRODUCTION: The author of this paper first began to use leg measuring after studying with Dr. John Grostic (1). Besides using as precise x-rays as possible of the upper cervical spine, very careful procedures were used to measure the length of the patient's legs. The patient had to empty out the back pockets of any bulging wallets and then he had to stand at the foot of the table (which, again had a very small padding to circumvent the patient from sinking into table which modern tables will allow). The wallet was taken out so that the patient's leg measurements would be more accurate.--And, so the patient carefully sat down on the table and pulled himself up to a fully extended supine position on the table.

Pre and post measurements were made of the patient's legs; also, pre and post x-rays were made to see changes in the occipito-atlanto-axial region of the cervical spine. And if the x-rays were shown to be in alignment i.e. the occiput, atlas and axis were in a normal position with no laterality or rotation-then the treatment was a success.

DISCUSSION: This author was interested in the accuracy of the leg measurement's in general. Webster (2) defines accuracy as "State or quality of being accurate; freedom from mistake or error; precision; exactness".

PROCEDURE: The measurement of the leg lengths involve three CHIEF factors: THE PATIENT
THE EQUIPMENT (table etc.)
THE DOCTOR

(CONTINUED ON PAGE 2)

IDEAL LEG CHECK PROCEDURE: In order to study errors in checking the length of legs, let us look at an ideal leg check: (note, this paper is only considering a supine position test)

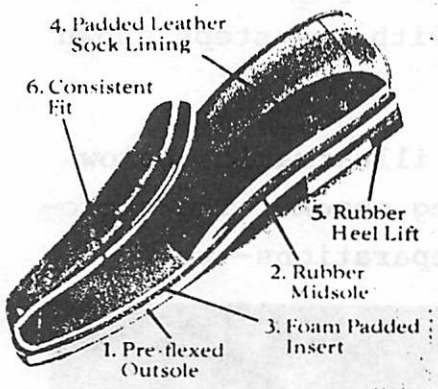
a. The table must have no padding but must have a frictionless surface. This means that the limbs and the rest of the body can move effortlessly on the surface of the table when physiological changes take place.
The table must be level for any tipping toward the sides or tipping from the top or bottom will effect the readings. (the measurements between the differences of the feet can vary from 1/8th of an inch to ½ an inch. In some cases there can be readings up to 3/4 of an inch-which is a rare occassion.)

b. The patient must be positioned on the table in the mid-line preferably--but must always be placed so that the body is as straight as possible--for example, using a drawn mid-line the legs must be equal distance from this line,etc. And it must be repeated in successive leg measurements--that is-the patient must be again be positioned in the same starting set up.
The patient's clothing must be frictionless so that there will be no lessening of the physiological changes. There should be no wallets or any other bulges in the back pockets to produce a torquing of the body. Men should were shorts whenever possible so that the doctor can ascertain any physical defects such as a permanently flexed knee or other deformities such as genu varum, genu valgum etc. Can the feet be dorsiflexed? If you are going to use any part of the foot, such as the internal malleolus as a measuring point,you must be able to move both feet equally.

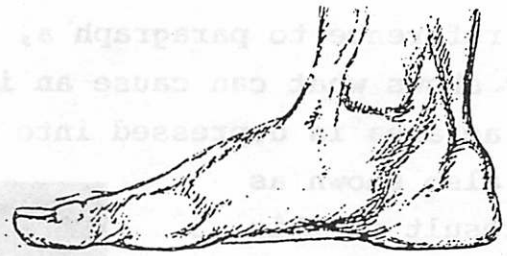
c. Most doctors request that the patients were their shoes when the doctor checks the leg lengths. A picture of a typical shoe is shown in illustration (A).
(continued on page 3)

HOW ACCURATE IS THE.....HENDRICKSON
Page 3

A picture of the medial side of a foot is shown in illustration (B).



(A)



(B)

The shoe pictured in (A) shows many layers at the heel as well as the sole. The ideal should have zero softness with no wearing away of the heel if the heel should be used as a measuring point ie the bottom. The shoe should fit snuggly against the bottom of the foot. There should never be any separation of the inner sole ------ from the bottom of the planter surface of the foot no matter in which way the foot may be moved--dorsiflexed or planter flexed.

How much'give', or play, there exists between the planter surface of the foot and the inner sole of the shoe can cause many discrepencies in measuring leg lengths.
For example, the plantar muscles of the foot (3) include: Plantar Aponeurosis, first layer of Abductor hallucis, Flexor digitorum brevis, Abductor digiti quinti, second layer-Quadrutus plantae, Lumbricales, third layer-Flexor hallucis brevis, Adductor hallucis, Flexor digiti quinti brevis.

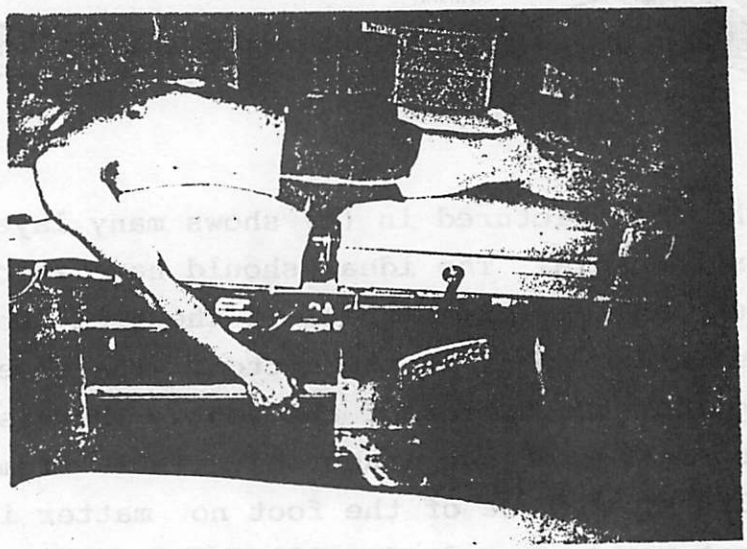
So, to be as accurate as possible the doctor must apply exactly the same pressure with both hands cephalically on the bottom of the foot to insure that the shoes remain in perfect contact with the planter surface of the foot. It is of utmost importance that the dorsiflexion or other position be identical on the

HOW ACCURATE IS THE.....HENDRICKSON

(continued from page 3) Page 4

right as well as on the left sides. It should be noted that there is much softness on the bottom of the foot as explained in the previous paragraph. Then again, if repeated measurements are used, the doctor must conform with the steps shown in the previous paragraphs.

In reference to paragraph a, Page 2, the illustration below (C) shows what can cause an inaccurate leg check. The sacro-iliac area is depressed into the table separations--there is also shown as a result of this--- a flexion of the knee articulations from this one condition.



(C)

A soft table brings in too many inaccuracies to the measurements.

CONCLUSIONS:

As a result of the study of the leg check as reviewed over these 4 pages it is felt that the leg check is not accurate because of the many variables.

REFERENCES

1. Grostic, John, Private Lectures, 1952
2. "Websters New Collegiate Dictonary", 2nd ED. 1960
3. Gray, H. "Anatomy of the Human Body",Lea & Febiger, 1948

LINKS BETWEEN THE NERVOUS SYSTEM AND THE BODY CHEMISTRY
" A Simple Approach"

By
Michael V. Muench
Chiropractic Intern L.A.C.C.

ABSTRACT

This paper has been designed to help doctors and students become more proficient at treating patients with Dr. Schmitt's protocol. This paper attempts to break down each procedure from the Links Seminar Series and simplify it so that anyone who has completed the basic 100 hours A.K. course can utilize Dr. Schmitt's Procedures.

ACKNOWLEDGEMENTS

All material contained within this paper is the work of Dr. Walter Schmitt, with supplementation from Dr. Richard Belli. I would like to thank both of them personally in helping myself make this paper possible.

LINKS BETWEEN THE NERVOUS SYSTEM AND THE BODY CHEMISTRY

****** Postural Analysis ******
***** T-S Line ******
1. Test for gamma 1 and gamma 2 weakness
If it shows on the T-S line, it should test as a Gamma 2, if it test as a Gamma 1, treat as if all muscles are Strong.
a. TL to I.C.V. check for open / closed
Open - Parasympathetic Dominant
Closed - Sympathetic Dominant
If I.C.V. does not show, Check Valve of Houston
+ Patient has switching problem
b. NO WEAKNESS FOUND go to ALL MUSCLES STRONG
c. ALL MUSCLES WEAK go to GLUTATHIONE Test

****** Pre Image Testing ****** PAGE 5
1. Structural
a. cranials b. sutures
2. Rebreathing (If CO2 strengthens always check Citric Acid, and Alpha-Keto Gluterate)
\+ Go to step 3
\- Check patient for Zn++, white dots on nails
3. Biochemical Test Citric Acid and A-KG
a. B-1 e. Niacinamide "G"
b. Manganese f. Niacin
c. Pantothenic Acid g. Lipoic Acid
d. B-2 h. Phosphorus
If C.A. and A-KG Does not Strengthen; Test:
1. B-6 3. Biotin
2. P5P (Zn, Cu, B-2, Mg) 4. Fe++

***** Tonic Labyrinthine Reflex ***** PAGE 6
If reoccurs check endocrine function, (CW/CCW) May show need for Pituitary or Pineal.
1. Supine
a. Extensor muscle weakness (use Lat. or other extensor)
2. Prone
a. Flexor muscle weakness (use Iliacus or other flexor)
Correction
T.L. and Challenge mastoid process
\+ Fix in position found
\- Check TMJ and/or Tilt from P.R.Y.-T. technique

***** ALL MUSCLES STRONG ****
1. SP21-K27
Therapy Localize to
SP21 on the left in a right handed patient
SP21 on the right in a true left handed patient
\+ Check Spinal Laterality (Logan Basic) PAGE 7
2. Retrograde PAGE 8
\+ Strong muscle weakens to TL of Pect Minor NL's.
3. Anterograde PAGE 9
\+ Strong muscle weakens to spinous inferior challenge
(Anterograde-coccygeal lift technique with body torque)

****** SWITCHING FACTORS ****** PAGE 10

1. ICV-Antronex go to Spinal Laterality
2. SI 19 Bilateral ---Check SI19 with eyes open and closed
3. T.L. Thymus
4. Hyoid

******* Neurotransmitter Testing *******

1. CCK PAGE 11
2. Clorox PAGE 12
3. Acetone PAGE 13
4. Aldehyde PAGE 13
5. Glutathione
   a. Cysteine PAGE 14
   b. Glycine PAGE 15
   c. Glutamic Acid PAGE 16
   d. 2 or 3 of these strengthens a weak muscle
      B-6, Magnesium, Potassium, Parathyroid
6. Ammonia (PMS symptoms) PAGE 17
7. Aspirin PAGE 18
8. Sugar PAGE 19
9. Toxic Metals (test exo-tox +, blood test for metals)

******* CENTERING THE SPINE ********

1. Lateral flexion PAGE 20
2. Flexion-Extension PAGE 21
3. Gait patterns (CW and CCW torque patterns) PAGE 22

******** SEATED TESTS *********

Check the following muscles for gamma 1, and gamma 2 weakness
Any gamma 2 weakness still present recheck

a. Tonic Labyrinthine Reflex
b. Neurotransmitter
c. Switching
** d. Centering the Spine --ORIGINAL GAMMA 2 WEAKNESS RETURNS

Gamma 1 weakness, check the following
If no subluxations are found,
recheck with patient's eyes closed

1. T.F.L. Bilateral weakness - Upper Cervical Fixation
2. T.F.L. Unilateral weakness - Sacral or S.I. Involvement
   Colon Neurolymphatic
3. Psoas Bilateral weakness - Occiput Fixation
4. Psoas Unilateral weakness - Lumbar Subluxation
5. Quadricep weakness - T.M.J., Lumbar Subluxation
6. Lower Trapezius weakness bilaterally
   a. Dorsal Lumbar Fixation
      1. Recurrent - Vitamin A / Cataplex A
7. Test all other fixation muscles
   ** IF NO FIXATIONS ARE PRESENT COMPRESS SPINE BY DOWNWARD PRESSURE
8. Primary subluxations
   a. Adjust most painful subluxations
   b. Check lovett brother for subluxation
   c. Check for Spondylogenic Patterns

***** ILIOLUMBAR LIGAMENT *******

1. Test for the presence of a ilio lumbar ligament
   - Their is a foot subluxation on the back foot
   + Challenge the Ilio Lumbar Ligament
     L-5 lateral fibers on the right, then left
     L-4 30-40 degree fibers on the right, then left

   Correct on the phase of respiration that negates the challenge

********** SPECIALIZED SEATED TESTS ********** PAGE 23

Place the patient into the seated position.

1. Have the Patient Flex Forward
   In this position the Right Hand Dominant patient, the following muscle should be WEAK:

   Left Latissimus
   Left Trapezius
   Right SCM
   Right Piriformis

   If Not, Then Have the Patient Therapy Localize the Following:
   a. Pelvic Faults (Category I and II)
   b. Lumbars
   c. Sacrum
   d. Upper and Lower Cervicals
   e. Occasionally Thoraco-Lumbar Fixation

2. Have the patient Extend Backward in a seated position:
   Recheck the same muscles.
   They should be WEAK, if not Recheck the same areas.

********** ACUPUNCTURE **********

1. Pulse Point DX
   a. tonification point
   b. TL alarm point and:
      1. associated spinal level
      2. lovett of associated spinal level
      3. spinal level of dermatome of the tonification point
   c. extremity subluxation near course of meridian
   d. Therapy Localize B and E head point against
      1. NL 2. NV 3. associated vertebra

********* UPPER CERVICAL PROBLEMS ********

1. T.L. to Upper Cervical
2. Challenge Upper Cervicals
   a. Right Lateral - adrenal and/or gonadal NL
   b. Left Lateral - thyroid NL

****NOTE****

TRACTION FOR 8 SECONDS TO CERVICAL SPINE IN RANGES OF MOTION TO UNCOVER HIDDEN PROBLEMS.

NASOSPHENOID FAULT WILL HIDE A LATERAL ATLAS

******* PRE AND POST CORDIAL TAP ********

1. Test any residual weak muscles with right (humming) and left (multiplying) brain activity.
    a. + check weak muscles for strengthening with right nostril or left nostril breathing.
        1. Right nostril breathing strengthens - tap right front and left back.
        2. Left nostril breathing strengthens - tap left front and right back.
    b. Preform Pre and Post Cordial Tap technique with opposite brain activity from that which strengthened.
    c. If Pre and Post Cordial Tap returns, Check for the need of Water Fearing (Hydrophobic), and Water Loving (Hydrophilic) Amino Acids.

***** LOCALIZED PROBLEMS ********

Muscle Stretch Reaction (Fascial Flush)

If localized treat by fascial flushing muscle
If present all over the body, Check B-12, Small Intestine NL's, then tap SP21-K27 to reset the alkaline-acid balance in the body.

PRE TEST IMAGING PAGE 5

CITRIC ACID CYCLE

1. GAMMA 2 WEAKNESS
   Have patient think about the muscle test, then test the muscle.
   + muscle is now strong.
2. REBREATHING
   a. Have patient rebreath his own CO2 from a paper bag
      + Muscle is now strong,
        Check Citric Acid and A-Kg, go to step 3
      - Test patient for the need of Zinc
        + Muscle is now strong, continue in flow chart. (White dots on fingernails)
3. BIOCHEMICAL CORRECTION
   Test the following nutrients to neutralize gamma 2 weakness.

   Test Citric Acid and Alpha Keto Glutamic Acid:

1. C.A.C. No effect then Test A-KG

   A-KG No effect Test:
   a. Biotin
   b. B-6, P5P(Zn, P, Mg, B2)
   c. Iron

   A-KG Strengthens Test:
   a. Manganese
   b. Niacin

2. C.A.C. Strengthens then Test A-KG:

   A-KG Strengthens Check:
   a. Cataplex B
   b. Manganese
   c. B-2
   d. Niacinamide "G"
   e. Lipoic Acid
   f. Magnesium
   h. Phosphorus (acid oral PH use Na2PO4)

   A-KG Weakens Check:
   a. Cataplex B
   b. Pantothenic Acid
   c. B-2
   d. Niacinamide "G"
   e. Lipoic Acid

   If 2 or 3 strengthen use Nutriwest CAC factor
   *Iron may be needed for cytochrome enzymes in Mitochondria.

NOTE When using Cataplex B and G, if a Phonocardiograph is not available, check Cataplex B and G using the Subscapularis Muscle.

***NOTE** If the patient is doing fine for a week or two, then complains of being very tired or worse then before, there is an oxidative stress problem. Look for the need of Co Enzyme Q10 immediately. Co Enzyme Q10 is utilized by the electron transport system, it allow for 36 ATP molecules to be produced in the citric acid cycle. If there is no Co Enzyme Q10 present then there is not enough ATP being produced for the body.

4. STRUCTURAL CORRECTION
   a. T.L. for cranial faults and sutures
      + negate weakness of gamma 2.
   b. T.L. for cranial faults and sutures
      + negates weakness from PTI of strong indicator muscle.

CORRECT CRANIAL FAULTS AND SUTURES WHILE PATIENT REBREATHS CO2.

****Temporal tap and retest the muscle, if weakness returns then nutrient must be given.

TONIC LABYRINTHINE REFLEXES

SUPINE
Extensor muscle weakness
(Use Latisimus muscle or other extensor)

PRONE
Flexor muscle weakness
(Use Iliacus muscle or other flexor)

**weakness will be a gamma 2 type muscle weakness

Step 1
Therapy localize to the mastoid process on the Ipsilateral side of muscle weakness
+ negates weakness, then Challenge mastoid process for direction of correction. Go to Step 2.
- Then Therapy Localize to the mastoid process on the Contralateral side of muscle weakness.
  + Patient is switched at the Endocrine Level. Check patient for a Sphenoid Tilt. Challenge for Nasosphenoid type correction. Recheck T.L.R.
  - Check TMJ or Tilt from P.R.Y.-T. Technique.

Step 2
Therapy localize to the Neurolymphatic points for the endocrine related muscles:
a. Right Thyroid / Left Thyroid
b. Right Adrenal / Left Adrenal
c. Right Gonadal / Left Gonadal
+ Therapy localization will negate the gamma 2 weakness.

Step 3
Therapy localize to the positive Neurolymphatic point and challenge the mastoid at the same time.
+ Negates the challenge
  Treat the Neurolymphatic reflex found.
  Recheck T.L.R. Supine and Prone.

This means that the endocrine system is more important than the T.L.R..
- Treat the T.L.R. in the position it is found.
(Found Supine treat Supine, found Prone treat Prone)

ALWAYS CHECK Therapy Localization to the Neurolymphatic against Pituitary and Pineal Drives.

NOTE
Sometimes you have to fix the same mastoid process first A to P supine, then P to A prone, any combination is possible.

ALL MUSCLES STRONG PAGE 7

SP21-K27

Acid-Alkaline Balance

1. Have patient Therapy Localize to SP21.
   SP21 on the left in a right handed person.
   SP21 on the right in a true left handed person.
   + Strong Muscle Weakens, go to Step 2.

2. While the patient TL's SP21, check the following positions:
   a. Left Convexity (Feet and Head to the right)
      + Negates TL to SP21, go to step 3.
   b. Right Convexity (Feet and Head to the left)
      + Negates TL to SP21, go to step 4.

3. LEFT CONVEXITY STRENGTHENS
   CHEMICAL CORRECTION
   With the patient in a neutral position check for the need of acid ash minerals.
   + Will negate TL to SP21.

   STRUCTURAL CORRECTION
   a. Challenge sacral tuberous ligament on the right Torque patient with blocks CW/CCW, find the torque pattern that enhances the challenge
   b. Check for the phase of respiration that negate the challenge with the blocks in place. Correct on that phase on respiration. This allows the spine to return to its normal position.
   c. Retest original Gamma 2 Muscle.

4. RIGHT CONVEXITY STRENGTHENS
   CHEMICAL CORRECTION
   With the patient in a neutral position check for the need of alkaline ash minerals.
   + Will negate the TL to SP21.

   **May also show the need for EFA's, Ca++, or "B", correlate with oral PH. Cataplex B is important in Pancreatic Enzymes (Alkaline) if pt does respond check patient for taking to much Vitamin C.

   STRUCTURAL CORRECTION
   a. Challenge sacral tuberous ligament on the left. Torque patient with blocks CW/CCW, find the torque pattern that enhances the challenge.
   b. Check for the phase of respiration that negates the challenge with the blocks in place. Correct on the phase which negates the challenge. This allows the spine to return to its normal position.
   c. Retest original Gamma 2 Muscle.

NOTE* If alkaline ash minerals strengthens in step 2 or if acid ash minerals strengthens in step 3, then the patient is switched.

ALL MUSCLES STRONG PAGE 8

RETROGRADE

Sympathetic-Parasympathetic Balance (Hypothalamic Outflow)

Parasympathetic Dominant

This patient has to much Flexor Tone, not enough Extensor Tone.

STEP 1. Therapy Localized to the Neurolymphatic point of the Pectoralis Minor Muscle.

\+ Strong muscle weakens, go to step 2

\- Muscle remains strong go to Anterograde page 9

STEP 2. CHEMICAL CORRECTION [supplement patient if reoccurs]

While the patient Therapy Localizes to the Neurolymphatic for the Pect. Minor, Check for the of the following nutrients.

\+ Nutrient will abolished weakness induced by the TL. to the NL's of the Pect. Minor.

a. Iron / Fe++

b. Molybdenum / Mo++

Anytime there is a need for Fe++, there may also be need for Molybdenum.

c. Neurotransmitter GABA and Co-factors

```
                [ B-6---->
                [Niacin-->          [B-6-->
C.A.C.------A.K.G.------GLUTAMIC ACID-----GABA
                [NH3---->                   [CO2--->
```

d. Whole Adrenal (if Choline weakens a strong muscle)

NOTE Fe++ is utilized in aerobic muscle activity, and extensor muscles are generally aerobic in nature because they are postural muscles.

STEP 3. STRUCTURAL CORRECTION

A. Treat the Pect. Minor Neurolymphatic.

B. Check for the following

a. Upper Cervical Fixation

b. TMJ

ALL MUSCLES STRONG
ANTEROGRADE

PAGE 9

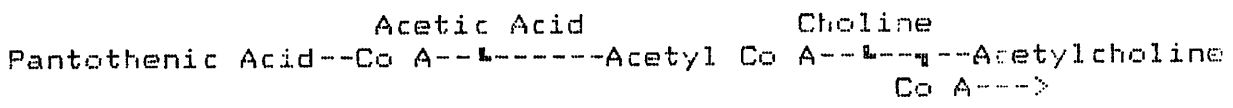
Sympathetic Dominant

This patient has to much Extensor Tone, not enough Flexor Tone.

STEP 1.
Challenge spinous processes inferior
\+ Weakens Strong Muscle, go to step 2.

STEP 2. CHEMICAL CORRECTION [supplement patient if reoccurs]
Check for the need of the precursor and/or Co-factors for the neurotransmitter Acetylcholine.

The proper nutrient will negate the challenge.
a. Vitamin G
b. Pantothenic Acid
c. Choline



Vitamin G [SPL] (Vit. G is involved in the breakdown of Acetylcholine to allow the body to recycle choline)
** Lecithinase enzyme is the key ingredient of Vit. G (calf brain), this frees choline from lecithin and makes it available for Acetylcholine production.

STEP 3. STRUCTURAL CORRECTION

A. Coccyx Lift Technique
Patient TL's coccyx and pushes inferior
Place blocks under patient to create a torque pattern
\+ Proper torque pattern will enhance the weakness
Correct by pulling coccyx superior and upper cervicals inferior

B. Upper Cervical Fixation

C. TMJ (Especially closing and wide opening faults with temporoparietal jamming)

SWITCHING FACTORS

Gamma 2 Weakness

Test each of the following to strengthen the weak gamma 2.

ANTRONEX:

Place Antronex in the patients mouth

\+ Gamma 2 muscle is now strong, go to SP21-K27 (Spinal Laterality)

*If Antronex is needed on a return visit, check for food allergies.

SI19:

Have the patient therapy localize to SI19 or the right side and the left side of the patient's head.

\+ 1. Test Co Enzyme Q10 for strengthening of gamma 2 muscle.

2. Treat the neurolymphatic points for the Small Intestine on the side of SI19 involvement.

\- Recheck SI19 On both sides with the eyes closed. if + treat as above.

THYMUS:

Have patient therapy localize to the thymus.

\+ Strengthens weak muscle:

Treat the neurolymphatic points for the thymus (lateral border of the ribs at the 5th intercostal space), and Recheck.

\- T.L. the thymus with clockwise torque then counter-clockwise torque to produce a weakness of the muscle and insure correction.

HYOID:

Doctor challenges the hyoid.

\+ Strong indicator muscle weakens, Go to step A.

\- Challenge with head in flexion and extension

\- Cross TL to the T.M.J. for ligament interlink

\+ Hold hyoid indirection of challenge while rubbing T.M.J.

Step A

Check the following to negate the challenge.

1. Folic Acid
2. Upper Cervical Subluxation
3. TMJ involvement
4. Thymus

NEUROTRANSMITTER TEST PAGE 11

CCK

KININ MEDIATED ALLERGIES

1. Identify any gamma 2 muscle weakness

2. Test strong indicator muscle against oral insalivation of CCK + weakness implies kinin mediated allergy problem

   A. therapy localize to the pancreatic neurolymphatic point (left 7th intercostal space) to neutralize CCK induced weakness

3. Test the following substances to neutralize CCK induced weakness
   A. zinc (Rarely Cataplex A as a synergist to zinc)
   B. pancreatic PMG
   C. whole pancreatic tissue
   D. pancreatic enzymes / check for protein deficiency
   ** while using pancreatic enzymes or zinc watch for the need of folic acid.

4. Clean CCK out of the mouth

5. Test the positive nutrients found in step 3 for strengthening of the weak gamma 2 muscle found in step 1.

STRUCTURAL CORRECTION

1. Treat the pancreatic neurolymphatic point and check for other pancreatic reflexes.

   ** If the patient continues to react to CCK on subsequent visits, test for food hypersensitivity reaction for offending foods.

NEUROTRANSMITTER TEST
CLOROX

Step 1.
Find a weak gamma 2 muscle

Step 2.
Find a strong indicator muscle.

Step 3.
Have patient sniff Clorox
- \+ Strong indicator muscle weakens, got to step 4.
- \- No reaction proceed on in flow chart

Step 4.
Test the weak gamma 2 muscle for strengthening with the following nutrients

a. Taurine
   - \- Go to b
   - \+ Test Cysteine, Strengthens check
     1. B-6
     2. Molybdenum, (work by negative feedback mechanism)

b. Methionine
   - \- go to step 5
   - \+ Strengthens check
     1. B-6
     2. Folic Acid
     3. B-12
     4. Methyl Donor (Choline)
     5. Magnesium

Step 5
Check Each of the following to strengthen muscle

a. Niacinamide or Niacin (B2, G, Cu++)
b. Selenium
c. Vitamin E (High dose and Low dose)
d. Aspirin
   1. Evening Primrose Oil
   2. Linseed Oil and/or Fish Oils (EPA)
   3. Other essential fatty acid products
e. Vitamin C
f. Others (rare)
   - Bioflavonoid
   - S.O.D. (check Cu++, Mn++, Zn++)
   - Vitamin A
   - Beta Carotene
   - Endocrine Imbalances

Step 6
Test each positive testing substance against sniffing Clorox SUPPLEMENT ONLY THE NUTRIENTS THAT STRENGTHEN BOTH THE GAMMA 2 WEAKNESS AND THE WEAKNESS INDUCED BY SNIFFING CLOROX

PAGE 13

NEUROTRANSMITTER TEST
ACETONE TEST
Pentose Phosphate Pathway

1. Identify a weak gamma 2 muscle.

2. Identify a strong indicator muscle.

3. Test patient by having them sniff Acetone.
   + Strong indicator muscle weakens.

4. Test the following nutrient to neutralize the acetone induced weakness.
   While the patient sniffs Acetone test:
   a. B-1
   b. B-2
   c. Niacin/Niacinamide

5. Now test the nutrient that neutralizes the weakness induced by the Acetone, to strengthen the weak gamma 2 muscle.

NEUROTRANSMITTER
ALDEHYDE TEST

*** Check on patients, who are fragrance intolerant, or with Candida Albicans infection.

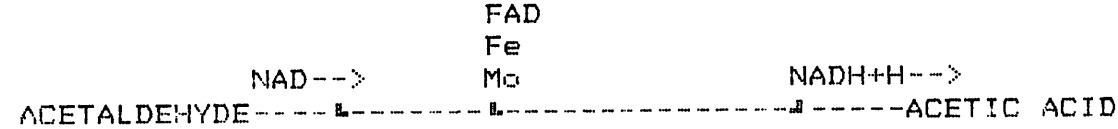
STRONG INDICATOR MUSCLE

1. Have patient sniff aldehyde.
   + Weakens strong muscle
2. Test patient with the following nutrients, find the nutrient/or nutrients that neutralize the weakness.
   a. Molybdenium
   b. Iron
   c. B-2
   d. Niacin/Niacinamide

GAMMA 2 WEAKNESS

1. Test the nutrients that neutralized the aldehyde weakness, against the gamma 2 weakness.
   + Strengthens gamma 2 weakness.

Note
Supplement only the nutrients that negate the Aldehyde test and strengthen the gamma 2 weakness.



NEUROTRANSMITTER TEST PAGE 14

GLUTATHIONE

CYSTEINE

If Cysteine Strengthens:

Test Clorox:

Weakens, go to step 1

No reaction go to step 2.

Step 1 Check the following:

a. Taurine

\+ Strengthens Test B-6

Taurine is needed to neutralize free radicals (OCL-)

B-6

CYSTEINE-----┗--------> TAURINE

b. Di-Cysteine (CYS-CYS)

\+ Weakens strong muscle --then Test

1. Niacin 2. Cu++ 3. Riboflavin

This patient is over-oxidized, and cannot reduce CYS-CYS to Cysteine, for eventual conversion to Taurine.

B-3, Cu++, B-2

CYSTEINE-CYSTEINE--┗-----┗-----┗-----> CYSTEINE----------> TAURINE

c. Methionine

\+ Strengthens -- then Test

1. B-6
2. Folic Acid
3. B-12
4. Magnesium
5. Methyl Donor (eg. Choline, Betaine)

Rebreathing usually strengthens this patient, it increases the need for single carbon groups.

METHIONINE ----------->CYSTEINE ----------> TAURINE

Step 2 CLOROX NO REACTION

a. Methionine

\+ Strengthens -- then Test

1. B-6
2. Folic Acid
3. B-12
4. Magnesium

METHIONINE ----------> CYSTEINE

b. If GLYCINE and GLUTAMIC ACID Strengthen

Test the following:

1. Magnesium 2. Potassium
3. Need for Parathyroid treatment

CYSTEINE + GLUTAMIC ACID + GLYCINE -------> GLUTATHIONE

c. Di-CYSTEINE (CYS-CYS)

Strong muscle Weakens then Test:

1. Niacin 2. Cu++ 3. Riboflavin

Patient may be over-oxidized, and not able to convert (CYS-CYS) to CYSTEINE.

NEUROTRANSMITTER TEST PAGE 15
GLUTATHIONE
GLYCINE

GLYCINE STRENGTHENS WEAK GAMMA 2

Test Arginine
No effect, go to step 1
Weakens a strong muscle, test Ammonia - weakens go to step 2
Strengthens the weak gamma 2, go to step 3

Step 1 No effect
Test the following nutrients for strengthening of the weak gamma 2 muscle

a. Magnesium c. Folic Acid
b. B-6 d. B-2

Step 2 Arginine and Ammonia both weaken a strong muscle
Test the following nutrients to strengthen the weak gamma 2 and to neutralize the weakness induced by sniffing Ammonia.

a. Arginex b. Manganese c. B-12

Step 3 Arginine strengthens the weak gamma 2 muscle.
Test the following nutrients to strengthen the weak gamma 2 muscle.

a. B-6 c. Aspartic Acid (check like glutamic acid)
b. Magnesium d. Biotin

B-6 in its active form (P5P) is necessary for the conversion of

SERINE---------->GLYCINE

THREONINE is the most difficult Amino Acid to be absorbed

B-6
THREONINE-----L-------->GLYCINE

CHOLINE and BETAINE can be converted into GLYCINE

B-2 B-12 B-2
CHOLINE----L------> BETAINE----L------> N1N-DiMethylGlycine--L--

B-2
-------> SARCOSINE-----L--------> GLYCINE

NOTE: 5-Adenosyl/Methionine
GLYCINE + ARGININE ---->GUANIDOACETATE---------->
(5 SAM ---> 5 SAH)

Mg++
---->CREATINE---L------>PHOSPHOCREATINE------>CREATININE (Urine)

Phosphocreatine is created for the production of high energy Phosphorus bonds -- this may be overworked in highly trained or over trained athletes or athletes after a long event (eg, marathon)

NEUROTRANSMITTER TESTS PAGE 16

GLUTATHIONE

GLUTAMIC ACID

GLUTAMIC ACID STRENGTHENS

Test AMMONIA:

Weakens- May indicate a need for more available Alpha Keto Gluterate from the Citric Acid Cycle:

Check Rebreathing to strengthen the gamma 2 muscle:

Test the Citric Acid Factors:

1. Pantothenic Acid
2. Cataplex B (SPL)
3. B-1
4. Manganese
5. Niacin/Niacinamide
6. Riboflavin
7. B-6
8. P5P (Zn, Mg, P, B-2)

Strengthens:

Check for the need of Glycine

\+ Check:

1. B-6
2. P5P (Zn, Mg, P)

GABA Strengthens:

Check:

1. B-6
2. Zn

NOTES

BRAIN:

B-6

GLUTAMIC ACID---┗-------> GABA

KIDNEY:

Mg++

GLUTAMIC ACID----┗----> Glutamine------> GLUTAMIC ACID + NH3

LIVER:

Niacin

GLU----┗----> a-KG + NH3----->Carbamyl Phosphate----> Urea Cycle

┗-----> Citric Acid Cycle

B-6

***GLU <---┗------> a-KG

Is a major transaminase reaction for many other reactions of Amino Acids throughout the body

NEUROTRANSMITTER TEST PAGE 17

AMMONIA

*** Check on Patients with active PMS symptoms

Step 1

Find a weak gamma 2 muscle

Find a strong indicator muscle

Test the patient by having them sniff Ammonia

No effect, Continue in the flow chart

Weakens a strong muscle, go to step 2.

Step 2 Weakens a strong indicator muscle

Test Arginine

No effect, go to step 3

Strengthens a weak Gamma 2 Muscle and neutralize the Ammonia induce weakness, go to step 4

Step 3 No effect

Test the following nutrients to strengthen a gamma 2 muscle and neutralize the weakness induce by sniffing the Ammonia:

a. B-6
b. P5P
   + Check
     1. Zinc
     2. Phosphorus
     3. B-2
     4. Magnesium
c. Molybdenum
d. Iron
e. Arginex [SPL]
f. Manganese
g. Alpha Keto Glutaric Acid, go to step 5

Step 4 Arginine Strengthens gamma 2 weakness

Test the following nutrients to strengthen the gamma 2 weakness and neutralize the Ammonia induced weakness:

a. B-6
b. Magnesium
c. Biotin
d. Phosphorus
e. Aspartic Acid, go to Citric Acid Cycle, Step 5

Step 5 Strengthens a weak gamma 2 muscle or Neutralized the Ammonia induced weakness

The patient has a problem with the Citric Acid Cycle factors.

Test the following nutrients to strengthen the weak gamma 2 muscle and to neutralize the Ammonia induced weakness:

a. B-1
b. B-2
c. Niacin/Niacinamide
d. Vitamin G [SPL]
e. Manganese
f. Pantothenic Acid
g. Phosphorus
h. Lipoic Acid
i. Zinc
j. B-6
k. Iron
l. Molybdenum

NEUROTRANSMITTER TEST PAGE 18

ASPIRIN / EFA's

GAMMA 2 WEAKNESS

Test patient by having them insalivate aspirin

No effect, Continue in the flow chart

Strengthens Gamma 2 muscle, go to step 1

Step 1 Aspirin strengthens gamma 2 muscle

Remove the Aspirin from the patient mouth.

Test the following nutrients for strengthening of the gamma 2 weakness:

a. Black Current Seed Oil
b. Evening Primrose Oil
c. Linseed Oil
d. Fish Oils (EPA)
e. Other EFA's
f. Zinc
g. Magnesium
h. B-6
i. Niacin

Step 2

Test for Aerobic Muscle Problem

Strong Muscle weakens on repeated testing (1 test per second)

Test the following nutrient for neutralizing the aerobic muscle weakness:

a. Black Current Seed Oil
b. Evening Primrose Oil
c. Linseed Oil
d. Fish Oils (EPA)
e. Other EFA's
f. Zinc
g. Magnesium
h. B-6
i. Niacin

Supplement the nutrient that abolishes the Gamma 2 weakness and the Aerobic muscle weakness

NEUROTRANSMITTER TEST
SUGAR

Find a strong muscle
Have patient insalivate sugar
No effect, Continue in flow chart
Weakens strong muscle, go to step 1

Step 1 Sugar Weakens strong muscle
Have patient therapy localize to the Neurolymphatic for the Thymus, will the insalivate the sugar.
Strengthens, treat neurolymphatic, then go to step 2
No effect, go to step 2

Step 2
Have patient therapy localize to the Neurolymphatic for the Thymus and the Acupuncture point Triple Warmer 23, at the same time.
Strengthens sugar induce weakness

Treatment
Have patient two hand therapy localize to the Neurolymphatic point for the Thymus, While the Doctor taps the acupuncture point Triple Warmer 23, (tap 50-60 times).

Recheck Sugar

FOOD DESENSITIZATION

GAMMA 2 MUSCLE STRENGTHENS TO ANTRONEX

1. Check strong muscle against L-Histidine
   + Strong muscle weakens

   Therapy Localize to SI 19
   + Negates weakness

   Therapy Localize to the Neurolymphatics for the Small Intestine
   + Negates weakness (should be same side as SI 19)

2. With L-Histidine in the mouth, Advance the Right Leg (increasing Pituitary Action)
   + Negates weakness induced by L-Histidine, go to 3
   - No Effect, Therapy Localize to the thymus
     + Negates weakness, treat by Thymus treatment System (Switching Factors - Thymus)

3. Now with L-Histidine in the mouth
   Check the Cofactors for the neurotransmitter norepinephrine:

1.	B-6	2.	Folic Acid	3.	Fe++
4.	Niacin	5.	Cataplex C	6.	Cu++

4. With L-Histidine in the mouth, treat the Neurolymphatics for the Small Intestine.

5. Check for the need of Yin-Yang Synchronization technique (TL SI 19 and the NL's for the Small Intestine at the same time, + Muscle goes Weak, Treat by Tapping SI 19 while the patient TL's the NL's for the small intestine.

6. Administer Pituitary Drive Technique with the L-Histidine in the patient's mouth.

7. If reoccurs check for the following
   a. Cofactors of B-6
      P5P (Zn, Mg, P, B-2)
      **If patient show a need for Zinc, and they are taking it, Check for the need of Cataplex A (synergist of Zn)
   b. Hypothalamic Set Point Technique.
   c. The need for desensitization to individual foods.

NOTE

Check the patient temperature before and after. Usually you will see an increase of 2 degrees or more. These patient are the type, that there temperature will not increase using normal techniques.

SPINAL LATERALITY PAGE 20

ACID-ALKALINE BALANCE

Sympathetic-Parasympathetic Tissue Balance

GAMMA 2 WEAKNESS

1. Laterally deviate the patient in a C-shaped curve with a
   a. Left Convexity
      + strengthens gamma 2 weakness go to step 2.
   b. Right Convexity
      + strengthens gamma 2 weakness, go to step 3.

2. LEFT CONVEXITY STRENGTHENS

   CHEMICAL CORRECTION
   With the patient in a neutral position check for the need of acid ash minerals.
   + will neutralize the weak gamma 2 muscle.

   STRUCTURAL CORRECTION
   a. Challenge sacral tuberous ligament on the right Torque patient with blocks CW/CCW, find the torque pattern that enhances the challenge
   b. Check for the phase of respiration that negate the challenge with the blocks in place. Correct on that phase on respiration. This allows the spine to return to its normal position.
   c. Retest with the patient laterally distorted.

3. RIGHT CONVEXITY STRENGTHENS

   CHEMICAL CORRECTION
   With the patient in a neutral position check for the need of alkaline ash minerals.
   + will neutralize the weak gamma 2 muscle.

   **May also show the need for EFA's, Ca++, or "B", correlate with oral PH. Cataplex B is important in Pancreatic Enzymes (Alkaline) if pt does respond check patient for taking to much Vitamin C.

   STRUCTURAL CORRECTION
   a. Challenge sacral tuberous ligament on the left. Torque patient with blocks CW/CCW, find the torque pattern that enhances the challenge.
   b. Check for the phase of respiration that negates the challenge with the blocks in place. Correct on the phase which negates the challenge. This allows the spine to return to its normal position.
   c. Retest with the patient laterally distorted.

NOTE* If alkaline ash minerals strengthens in step 2 or if acid ash minerals strengthens in step 3, then the patient is switched.

SPINAL FLEXION - EXTENSION PAGE 21

E.O.O.D.

Sympathetic-Parasympathetic Balance (Hypothalamic Outflow)

GAMMA 2 WEAKNESS

Eyes superior strengthens the weak muscle, go to step 1

Eyes inferior strengthens the weak muscle, go to step 2

STEP 1. **Parasympathetic Dominant**

This patient has to much Flexor Tone, not enough Extensor Tone.

CHEMICAL CORRECTION [supplement patient if reoccurs]

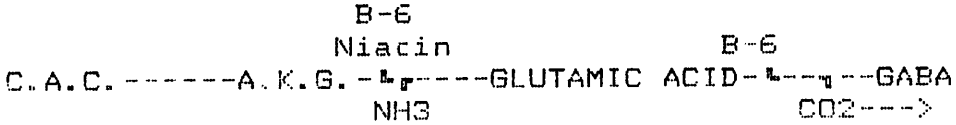
Check for the of the following nutrients

a. Iron / Fe++

b. Molybdenum / Mo++

Anytime there is a need for Fe++, there may also be need for Molybdenum.

c. Neurotransmitter GABA and Co-factors



d. Whole Adrenal (if Choline weakens a strong muscle)

NOTE Fe++ is utilized in aerobic muscle activity, and extensor muscles are generally aerobic in nature because they are postural muscles.

STRUCTURAL CORRECTION

Check for the following

a. Pectoralis Minor Neurolymphatic

b. Upper Cervical Fixation

c. TMJ

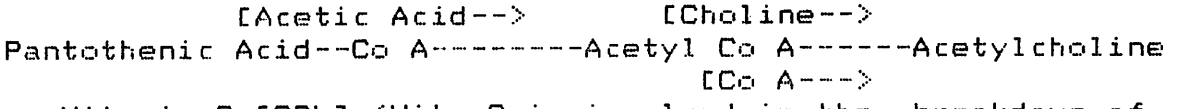
STEP 2 **Sympathetic Dominant**

This patient has to much Extensor Tone, not enough Flexor Tone.

CHEMICAL CORRECTION [supplement patient if reoccurs]

Check for the need of the following nutrients

A. Neurotransmitter Acetylcholine / and Co-factors



B. Vitamin G [SPL] (Vit. G is involved in the breakdown of Acetylcholine to allow the body to recycle choline)

** Lecithinase enzyme is the key ingredient of Vit. G (calf brain), this frees choline from lecithin and makes it available for Acetylcholine production.

STRUCTURAL CORRECTION

1. Coccyx Lift Technique

Patient TL's coccyx and pushes inferior

Place blocks under patient to create a torque pattern

+ Proper torque pattern will enhance the weakness

Correct by pulling coccyx superior and upper cervicals inferior

2. Upper Cervical Fixation

3. TMJ (Especially closing and wide opening faults with temporoparietal jamming)

SPINAL TORQUE PATTERNS PAGE 22

WEAK GAMMA 2

Test for the strengthening of the weak gamma 2 muscle

A. Place patient's right leg forward, + go to step 1

B. Place patient's left leg forward, + go to step 2

STEP 1. The patient has an increase in pineal activity and a decrease in pituitary activity.

CHEMICAL CORRECTION

Test for the need of the cofactors and/or precursors for the production of the Neurotransmitter Norepinephrine, also Pituitary Tissue.

TYROSINE ----------NOREPINEPHRINE
B-6, Folic Acid, Niacin, Fe++
Cataplex C (Tyrosinase), Ascorbic Acid, Cu++, Methyl Donor

The proper nutrient will negate the gamma 2 weakness.

STRUCTURAL CORRECTION

Challenge for the need of pituitary drive technique.

NOTE Patients with a decreased temperature will require pituitary drive technique. Patients with a normal or increased temperature will require chemical correction. Often patients will require both the structural and chemical correction.

STEP 2. The patient has decreased pineal activity and increased pituitary activity. (Increased Thyroid and Steroid activity, adrenal cortex -- ovarian activity).

CHEMICAL CORRECTION

Test for the need of the precursors and/or co-factors involved in the production of the Pineal Hormone.

TRYPTOPHAN----------SEROTONIN----------MELATONIN
B-6,Folic Acid Pantothenic Acid
Niacin, Fe++ Methyl Donor (Choline)

** If Choline weakens a strong muscle, as in the need for Whole Adrenal, use Betaine as Methyl Donor.

The proper nutrient will negate the Gamma 2 weakness.

STRUCTURAL CORRECTION

Challenge for the need of the Sphenoid Spread Technique.

** A patient with a low or normal temperature will need chemical correction. A patient with an increased temperature will require the Sphenoid Spread Technique.

NOTE Pineal activity decreases or dampens endocrine function. Melatonin dampens the endocrine system, while Serotonin dampens the hormonal effects at the tissue level.

P.L.U.S. TECHNIQUE (3) PAGE 26

Have the patient Stand.

1. Have the Patient Flex Forward;
   In this position, the Right Hand Dominant patient, the following muscle should be WEAK:
   - Left Latissimus
   - Left Trapezius
   - Right SCM
   - Right Piriformis

   If not, Then have the patient Therapy Localize the following:
   a. Pelvic Faults (Category I and II)
   b. Lumbars
   c. Sacrum
   d. Upper and Lower Cervicals
   e. Occasionally Thoraco-Lumbar Fixation

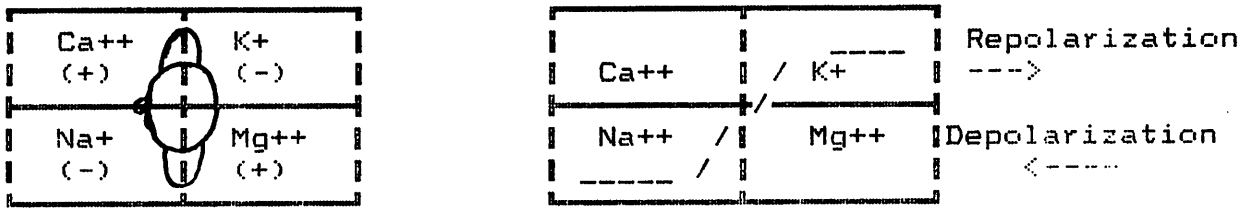
2. Have the patient Extend Backward in a Standing position:
   Recheck the same muscles.
   They should be weak if not Recheck the same areas.

3. Have the patient Lateral Flex to the Right and to the Left.
   Recheck the same muscles.
   They should be weak, if not Recheck the same areas.

NOTE

In real difficult patients, place them in a prone position and have the get up on there elbow in order to arch there back, Hidden Lumbar and Pelvic problems will show.

BODY QUADRANT WEAKNESS



1. Check for weakness of a quadrant muscle (use Abdominal Obloquies and Lats)
   Right Flexor_____|_____Right Extensor
   Left Flexor | Left Extensor
2. Check the following nutrients to strengthen the weak muscle:
   Ca++ (+) Should strengthen a Rt Flexor weakness
   Na+ (-) Should strengthen a Lt Flexor weakness
   K+ (-) Should strengthen a Rt Extensor weakness
   Mg++ (+) Should strengthen a Lt Extensor weakness
   *** If opposite nutrient strengthen, check the patient for a sphenoid tilt (use nasosphenoid type correction), Recheck Quadrant weakness pattern and correct.
3. While the patient has the strengthening nutrient in their mouth, check the muscle quadrants for a hidden weakness of the opposite group of muscles
   + Weakness of opposite muscle group found, recheck nutrients
   Both nutrients may be needed
   Check Sp-21 while patient has nutrient in the mouth

STRUCTURAL CORRECTION Remove Nutrients:

1 Therapy Localize to the Neurolymphatic points for the Endocrine glands (Thyroid, Parathyroid, Adrenal, Kidney, and Gonadal).
   + Abolishes weakness, check for opposite muscle weakness with TL present
   + Treat both points - Treat one point

Note ** The control of these nutrients is by dietary intake, intestinal absorption, hormonal and other regulations.
Thyroid effect the ratio of Ca++ : K+
Steroid effects the ratio of Na++ : Mg++

Ca++ and Na+ are important extracellular Ions necessary for depolarization and muscle contraction. Na+ and Ca++ enhances depolarization as does epinephrine (Sympathetic Dominant)
Mg++ and K+ enhance Repolarization (Parasympathetic Dominant), Intracellular Ions, Alkaline Ash, Counteracts Sympathetic Dominance, supports Parasympathetic

If Na+ and Mg++ strengthen and Ca++ and K+ weakens all over this is a classic case of Hypoadrenia.
Some patients who don't weaken to Ca++ - Mg++, will show ligament stretch with Ca++ - Mg++ in the mouth or in the clear. Whole Adrenal and Anti-Oxidants (Vitamin E, Selenium, Super Oxide Dismutase) will abolish the Ligament Stretch problem.
Weakness induced by Na++ - Mg++ Will be abolished by Thyroid. These patients are Under-oxidized, Iodine, and other oxidants will strengthen.

HYPOTHALAMIC SET POINT TECHNIQUE

This technique is used for any reoccurring problems.

VISCERAL PROBLEMS

Step 1 Therapy Localize to the acupuncture head point for that organ:

\+ Strong Indicator weakens

Therapy Localize to Neurolymphatics or Alarm Point for that Organ

\+ Negates weakness, Treat Neurolymphatics or Meridian

Step 2 Therapy Localize simultaneously to the Head Point and Neurolymphatic or Alarm Point

\+ Strong Indicator Weaken

Treat by tapping Head Point while Patient Therapy Localizes (two Hands if Possible) to Neurolymphatic or Alarm

SPINAL PROBLEMS

Step 1 Therapy Localize to a vertebra

\+ Strong Muscle weakens, then challenge and Adjust

Step 2 Therapy Localize simultaneously to the Vertebra and the Associated Head Point Meridian

\+ Strong Indicator Muscle weakens

Treat by Tapping (50-60 times) Associated Head Point While patient Therapy Localize (two Hands if Possible) to the Vertebra.

MUSCLE PROBLEMS

Step 1 Test a Muscle

If weak treat using appropriate factors

Step 2 Therapy Localize Simultaneously to the Head Point for the Associated Meridian and to the Muscle Location

a. Origin b. Insertion c. Belly

\+ Strong Muscle weakens

Treat by Tapping (50-60 times) while Patient maintains Therapy Localization to the Muscle Location

REOCCURRING HYPOTHALMIC SET POINT PROBLEMS

If a Head Point needs to be corrected after it has been previously treated, and there is no apparent cranial fault, check the nutritional component of the associated neurotransmitter:

Small Intestine -- NOREPINEPHRINE
Bladder ---------- SEROTONIN
Triple Warmer ---- SUGAR METABOLISM, POSSIBLY INSULIN
Gall Bladder ----- ACETYLCHOLINE
Large Intestine -- GAMMA-AMINOBUTARIC ACID (GABA), GLYCINE, GLUTAMINE
Stomach ---------- HISTAMINE, KININS (CCK., POSSIBLY BRADYKININ)

SPECIAL TECHNIQUES

HIDDEN PROBLEMS:

To find hidden problems the following techniques can be used:

1. ANTAGONISTIC TESTING (2)
   Antagonistic testing can be achieved by placing the opposite nutrient in the patients mouth. This can be thought of as a chemical B.I.D..
2. OCULAR LOCK
   Have the patient follow your finger as you move it in a circular motion, watch his eyes for any abnormality in the smooth motion. If there is a sudden movement, have the patient look in that direction, this will bring back a gamma 2 weakness or weaken a strong muscle.
3. DOMINANT EYE
   Have the patient close their dominant eye. This takes out the bodies ocular righting reflex, and allow for hidden problems to present themselves, the same as B.I.D..

NUTRITIONAL

TEMPORAL TAP AUDITING TECHNIQUE (3)

Temporal tap the patient to see if the gamma 2 muscle weakness returns. If the muscle weakness does return then test for the nutrition component. Once the nutrient has been found have the patient chew that nutrient or keep it in their mouth throughout the treatment.

BIBLIOGRAPHY

1. Dr. Walter Schmitt,
The Links Seminar Series, The Links Between the Nervous System and the Body Chemistry,
January, February, and March 1988 Los Angeles.
The Effect of Gait and Torque on Enhancing and Suppressing Immune System Function,
I.C.A.K. 1987 Winter Collected Papers.
Centering the Spine, Functional Neurological and Biochemical Considerations,
I.C.A.K. 1987 Summer Collected Papers.
Pre Test Imaging, A Screening Test for Cranial Faults,
I.C.A.K. 1987 Summer Collected Papers.
Kinin Mediated Allergies and Pancreatic Activity,
I.C.A.K. 1986 Winter Collected Papers.
Tonic Labyrinthine Reflexes in the Weight Bearing Position,
I.C.A.K. 1986 Summer Collected Papers.
2. Dr. Richard Belli,
Personal Communication.
3. Dr. George Goodheart,
P.L.U.S. Technique
I.C.A.K. Summer Convention
Detroit, Michigan Summer 1988.
4. Dr. Michael P. Lebowitz,
Some Helpful Additions to Gamma 2 Muscle Testing,
I.C.A.K. 1987 Winter Collected Papers.

LINKS BETWEEN THE NERVOUS SYSTEM AND THE BODY CHEMISTRY

"Expanding the LINKS"

By
Michael V. Muench
Chiropractic Intern L.A.C.C.

ABSTRACT

This paper has been designed for doctors and students, who are experienced in the "LINKS" work by Dr. Schmitt. This paper allows the doctor to advance his understanding of the "Links". Each section in this paper uses not only Gamma 2 testing but also Gamma 1. This alternative testing by the Gamma 1 procedures allows the doctor to find hidden weaknesses by inducing stress into the nervous system and the body chemistry.

ACKNOWLEDGEMENTS

All material contained within this paper is the work of Dr. Walter Schmitt, with supplementation from Dr. Richard Belli. I would like to thank both of them personally in helping myself make this paper possible.

The author of this paper invites all comments, suggestions, or new ideas, to further this research. As updated material is recieved, followup papers will be published. This will allow doctors to understand where new procedures fit into the "Links".

Please send to Michael Muench
1880 Northwest Dr.
Point Pleasant, NJ 08742

LINKS BETWEEN THE NERVOUS SYSTEM AND THE BODY CHEMISTRY

****** Postural Analysis ******

***** T-S Line ******

1. Test for gamma 1 and gamma 2 weakness
   If it shows on the T-S line, it should test as a Gamma 2, if it test as a Gamma 1, treat as if all muscles are Strong.
   a. TL to I.C.V. check for open / closed
      Open - Parasympathetic Dominant
      Closed - Sympathetic Dominant
      If I.C.V. does not show, Check Valve of Houston
      + Patient has switching problem
   b. NO WEAKNESS FOUND go to SP21-K27
   c. ALL MUSCLES WEAK go to GLUTATHIONE Test

****** Pre Image Testing ****** PAGE 6

1. Structural
   a. cranials
   b. sutures
2. Rebreathing (If CO2 strengthens always check Citric Acid, and Alpha-Keto Gluterate)
   + Go to step 3
   - Check patient for Zn++, white dots on nails
3. Biochemical
   Test C.A. and A-KG

a.	B-1	e.	Niacinamide "G"
b.	Manganese	f.	Niacin
c.	Pantothenic Acid	g.	Lipoic Acid
d.	B-2	h.	Phosphorus

If C.A. and A-KG Does not Strengthen; Test:

1.	B-6	3.	Biotin
2.	P5P (Zn, Cu, B-2, Mg)	4.	Fe++

***** Tonic Labyrinthine Reflex ***** PAGE 7

If reoccurs check endocrine function, (CW/CCW) May show need for Pituitary or Pineal.

1. Supine
   a. Extensor muscle weakness (Test Lat. or other extensor)

2. Prone
   a. Flexor muscle weakness (Test Iliacus or other flexor)

Correction
   T.L. and Challenge mastoid process
   + Fix in position found
   - Check TMJ and/or Tilt from P.R.Y.-T. technique

****** ALL MUSCLES STRONG ****

1. SP21-K27
   Therapy Localize to
   SP21 on the left in a right handed patient
   SP21 on the right in a true left handed patient
   + Check Spinal Laterality (Logan Basic) PAGE 8
2. Retrograde PAGE 9
   + Strong muscle weakens to TL of Pect Minor NL's.
3. Anterograde PAGE 10
   + Strong muscle weakens to spinous inferior challenge
   (Anterograde-coccygeal lift technique with body torque)
4. Gait Patterns CCW/CW Torque Patterns PAGE 11
   + Strong muscle weakens with right or left leg forward.

****** SWITCHING FACTORS ****** PAGE 12

1. Antronex go to ALL MUSCLES STRONG (SP21-K27)
2. SI 19 Bilateral ---If SI19 shows with eyes closed,
   fix Pineal and Cofactors (Torque Patterns)
3. T.L. Thymus
4. Hyoid
5. K27-Cross K27
   K27 hidden Cat 1 pelvic fault

******* Neurotransmitter Testing *******

1. CCK PAGE 13
2. Gamma 2 Ligament Test
3. Clorox PAGE 14
4. Acetone PAGE 15
5. Aldehyde PAGE 15
6. Glutathione
   a. Cysteine PAGE 16
   b. Glycine PAGE 17
   c. Glutamic Acid PAGE 18
   d. 2 or 3 of these strengthens a weak muscle
   B-6, Magnesium, Potassium, Parathyroid

7. Ammonia (PMS symptoms) PAGE 19
8. Aspirin PAGE 20
9. Sugar PAGE 21
10. Food Desensitizing PAGE 22
11. Toxic Metals (test exo-tox +, blood test for metals)
12. Acetic Acid

******* CENTERING THE SPINE ********

1. Lateral flexion PAGE 23
2. Flexion-Extension PAGE 24
3. Gait patterns (CW and CCW torque patterns) PAGE 25

****** SEATED TESTS *********
Check the following muscles for gamma 1, and gamma 2 weakness
a. T.F.L. c. Quadricep
b. Psoas d. Lower Trapizius

Any gamma 2 weakness still present recheck
a. Tonic Labyrinthine Reflex
b. Neurotransmitter
c. Switching
** d. Centering the Spine --ORIGINAL GAMMA 2 WEAKNESS RETURNS

Gamma 1 weakness, check the following
If no subluxations are found, recheck with patient's eyes closed
1. T.F.L. Bilateral weakness - Upper Cervical Fixation
2. T.F.L. Unilateral weakness - Sacral or S.I. Involvement
Colon Neurolymphatic
3. Psoas Bilateral weakness - Occiput Fixation
4. Psoas Unilateral weakness - Lumbar Subluxation
5. Quadricep weakness - T.M.J., Lumbar Subluxation
6. Lower Trapezius weakness bilaterally
a. Dorsal Lumbar Fixation
1. Recurrent - Vitamin A / Cataplex A
Test all other fixation muscles
** IF NO FIXATIONS ARE PRESENT COMPRESS SPINE BY DOWNWARD PRESSURE

****** PRIMARY SUBLUXATIONS *******
Can be palpated prone or seated.
a. Adjust most painful subluxations
b. Check lovett brother for subluxation
c. Check for Spondologenic Patterns

***** ILIOLUMBAR LIGAMENT *******
1. Test for the presence of ilio lumbar ligament dysfunction
+ Therapy Localize to the Iliolumbar Ligament
+ Challenge the Iliolumbar Ligament
L-5 lateral fibers on the right, then left
L-4 30-40 degree fibers on the right, then left
Correct on the phase of respiration that negates the challenge
If Gait Test is Positive, and Iliolumbar Ligament does not T.L. the patient has a Foot Subluxation.

********* P.L.U.S. Technique ********** PAGE 26
The patient should be Standing for this procedure, but can be done seated.

1. Have the Patient Flex Forward
In this position the Right Hand Dominant patient, the following muscle should be WEAK:

Left Latissimus Right SCM
Left Trapezius Right Piriformis

If Not, Then Have the Patient Therapy Localize the Following:

a. Pelvic Faults (Category I and II)
b. Lumbars
c. Sacrum
d. Upper and Lower Cervicals
e. Occasionally Thoraco-Lumbar Fixation

2. Have the patient Extend Backward:
Recheck the same muscles.
They should be WEAK, if not Recheck the same areas.

3. Have the patient Laterally Flex to the Right and to the Left
Recheck the same muscles.
They should be WEAK, if not Recheck the same areas.

********* EXTREMITY SUBLUXATION TESTING (Patient Supine)*******
(Testing Gait Muscles)

1. Test the patient's Right leg and Left arm at the same time
Weakness of either the arm or leg implies a subluxation of either extremity.
2. Test the patient's Left leg and Right arm at the same time
Weakness of either the arm or leg implies a subluxation of either extremity.

********** ACUPUNCTURE **********

1. Pulse Point DX
a. T.L. to Alarm Point
b. tonification point
c. TL alarm point and:
1. associated spinal level
2. lovett of associated spinal level
3. spinal level of dermatome of the tonification point
d. extremity near course of meridian
e. B and E head point against
1. NL
2. NV
3. associated vertebra

2. Muscle Meridian
Pulse Point DX with patient in GAIT Position
T.L. the associated Head Point against each other of the Muscle Meridians found.

3. Then and Now DX
Have Patient TL Pulse Points on the Left hand, and at the same time TL the Pulse Points on the Right Hand.
\+ Strong Muscle goes WEAK
TL Alarm Points to find the meridian involved
Treat Same as in step 1.

******** UPPER CERVICAL PROBLEMS *******
1. T.L. to Upper Cervical
2. Challenge Upper Cervicals
   a. Right Lateral - adrenal and/or gonadal NL
   b. Left Lateral - thyroid NL

*****NOTE***** NASOSPENOID FAULT WILL HIDE A LATERAL ATLAS

********* PRE AND POST CORDIAL TAP ********
1. Test any residual weak muscles with right (humming) and left (multiplying) brain activity.
   a. + check weak muscles for strengthening with right nostril or left nostril breathing.
      1. Right nostril breathing strengthens - tap right front and left back.
      2. Left nostril breathing strengthens - tap left front and right back.
   b. Preform Pre and Post Cordial Tap technique with opposite brain activity from that which strengthened.
   c. If Pre and Post Cordial Tap returns, Check for the need of Water Fearing (Hydrophobic), and Water Loving (Hydrophilic) Amino Acids.

********* MUSCLE QUADRANT WEAKNESS ********* Page 27

If Pre and Post Cordial Tap has been cleared and a flexor or extensor muscle remains weak, or PPCT doesn't strengthen the flexor or extensor there may be an imbalance of positive cations.

Test the Abdominal Obliques and the Lats
+ Weakness indicates a imbalance in cations.

Check the following nutrients to Strengthen

Rt Flexor --- Ca++	Rt Extensor ---- K+
Lt Flexor --- Na+	Lt Extensor ---- Mg++

** Strengthening to opposite nutrient indicate that there is a Sphenoid Tilt present

Supplementation is rarely needed, Correct the regulating mechanism
Correction----Therapy Localize to the Endocrine Glands (Thyroid, Parathyroid, Adrenal, Gonadal, and Kidney) to abolish weakness

********** HYPOTHALAMIC SET POINT TECHNIQUE ********* PAGE 28

Check when any type of problem reoccurs
TL. Head point
+ Treat Neurolymphatic
- Simultaneously TL. Head Point and one of the following:

Visceral Problems -------TL. Nuerolymphatics or Alarm Point
Vertebral Problems ------TL. Vertebra (adjust vertebra First)
Muscle Problems ---------TL. Muscle Locations
a. Origin
**Fix muscle first if weak b. Insertion
c. Belly

Correct by tapping (50-60 times) the head point while patient TL's the problem area.

***** LOCALIZED PROBLEMS ********
Muscle Stretch Reaction (Fascial Flush)
If localized treat by fascial flushing muscle
If present all over the body, Check B-12, Small Intestine NL's, then tap SP21-K27 to reset the alkaline-acid balance in the body.
******SPECIAL TECHNIQUES ********* PAGE 22

PRE TEST IMAGING PAGE 6

CITRIC ACID CYCLE

1. GAMMA 2 WEAKNESS

a. have patient think about the muscle test, then test the muscle. + muscle is now strong, go to step 2.

STRONG INDICATOR

a. have patient think about the muscle test, then test the muscle. + muscle is now weak, go to step 2.

2. REBREATHING

a. Have patient rebreath his own CO2 from a paper bag

\+ Muscle is now strong, Check Citric Acid and A-Kg go to 3a.

\- Test patient for the need of Zinc

\+ Muscle is now strong, continue in flow chart. (White dots on fingernails)

3. BIOCHEMICAL CORRECTION

a. test the following nutrients to neutralize gamma 2 weakness.

b. test the following nutrients to neutralize muscle weakness while patient images test.

Test Citric Acid and Alpha Keto Glutamic Acid:

1. Citric Acid---No effect then Test A-KG

A-KG No effect Test:

a. Biotin
b. B-6, P5P(Zn, P, Mg, B2)
c. Iron

A-KG Strengthens Test:

a. Manganese
b. Niacin

2. Citric Acid--Strengthens then Test A-KG:

A-KG Strengthens Check:

a. Cataplex B, B-1
b. Manganese
c. B-2
d. Niacinamide or "G"
e. Lipoic Acid
f. Magnesium
h. Phosphorus (acid oral PH use Na2PO4)

A-KG Weakens Check:

a. Cataplex B, B-1
b. Pantothenic Acid
c. B-2
d. Niacinamide or "G"
e. Lipoic Acid

If 2 or 3 strengthen use Nutriwest CAC factor

*Iron may be needed for cytochrome enzymes in Mitochondria.

NOTE When using Cataplex B and G, if a Phonocardiograph is not available, check Cataplex B and G using the Subscapularis Muscle. If weakens use B-1, or B-3 accordingly.

***NOTE** If the patient is doing fine for a week or two, then complains of being very tired or worse then before, there is an oxidative stress problem. Look for the need of Co Enzyme Q10 immediately. Co Enzyme Q10 is utilized by the electron transport system, it allow for 36 ATP molecules to be produced in the citric acid cycle. If there is no Co Enzyme Q10 present then there is not enough ATP being produced for the body.

4. STRUCTURAL CORRECTION

a. T.L. for cranial faults and sutures

\+ negate weakness of gamma 2.

b. T.L. for cranial faults and sutures

\+ negates weakness from PTI of strong indicator muscle.

CORRECT CRANIAL FAULTS AND SUTURES WHILE PATIENT REBREATHS CO2.

****Temporal tap and retest the muscle, if weakness returns then nutrient must be given.

TONIC LABYRINTHINE REFLEXES

PAGE 7

SUPINE

Extensor muscle weakness

(Use Latisimus muscle or other extensor)

PRONE

Flexor muscle weakness

(Use Iliacus muscle or other flexor)

**weakness will be a gamma 2 type muscle weakness

Step 1

Therapy localize to the mastoid process on the Ipsilateral side of muscle weakness

\+ negates weakness, then Challenge mastoid process for direction of correction. Go to Step 2.

\- Then Therapy Localize to the mastoid process on the Contralateral side of muscle weakness.

\+ Patient is switched at the Endocrine Level. Check patient for a Sphenoid Tilt. Challenge for Nasosphenoid type correction.

\- Check TMJ or Tilt from P.R.Y.-T. Technique.

Step 2

Therapy localize to the Neurolymphatic points for the endocrine related muscles:

a. Right Thyroid / Left Thyroid

b. Right Adrenal / Left Adrenal

c. Right Gonadal / Left Gonadal

\+ Therapy localization will negate the gamma 2 weakness.

Step 3

Therapy localize to the positive Neurolymphatic point and challenge the mastoid at the same time.

\+ Negates the challenge

Treat the Neurolymphatic reflex found.

Recheck T.L.R. Supine and Prone.

This means that the endocrine system is more important than the T.L.R..

\- Treat the T.L.R. in the position it is found.

(Found Supine treat Supine, found Prone treat Prone)

** If reoccurs check Endocrine Neurolymphatics. If positive check with Pituitary and Pineal Drives.

NOTE

Sometimes you have to fix the same mastoid process first A to P supine, then P to A prone, any combination is possible.

ALL MUSCLES STRONG PAGE 8

SP21-K27

Acid-Alkaline Balance

1. Have patient Therapy Localize to SP21.
   SP21 on the left in a right handed person.
   SP21 on the right in a true left handed person.
   + Strong Muscle Weakens, go to Step 2.

2. While the patient TL's SP21, check the following positions:
   a. Left Convexity (Feet and Head to the right)
      + Negates TL to SP21, go to step 3.
   b. Right Convexity (Feet and Head to the left)
      + Negates TL to SP21, go to step 4.

3. LEFT CONVEXITY STRENGTHENS
   CHEMICAL CORRECTION
   With the patient in a neutral position check for the need of acid ash minerals.
   + Will negate TL to SP21.

   STRUCTURAL CORRECTION
   a. Challenge sacral tuberous ligament on the Left Torque patient with blocks CW/CCW, find the torque pattern that enhances the challenge
   b. Check for the phase of respiration that negate the challenge with the blocks in place. Correct on that phase on respiration. This allows the spine to return to its normal position.
   c. Retest original Gamma 2 Muscle.

3. RIGHT CONVEXITY STRENGTHENS
   CHEMICAL CORRECTION
   With the patient in a neutral position check for the need of alkaline ash minerals.
   + Will negate the TL to SP21.

   **May also show the need for EFA's, Ca++, or "B", correlate with oral PH. Cataplex B is important in Pancreatic Enzymes (Alkaline) if pt does respond check patient for taking to much Vitamin C.

   STRUCTURAL CORRECTION
   a. Challenge sacral tuberous ligament on the Right. Torque patient with blocks CW/CCW, find the torque pattern that enhances the challenge.
   b. Check for the phase of respiration that negates the challenge with the blocks in place. Correct on the phase which negates the challenge. This allows the spine to return to its normal position.
   c. Retest original Gamma 2 Muscle.

NOTE* If alkaline ash minerals strengthens in step 3 or if acid ash minerals strengthens in step 4, then the patient is switched.

ALL MUSCLES STRONG PAGE 9

RETROGRADE

Sympathetic-Parasympathetic Balance (Hypothalamic Outflow)

Parasympathetic Dominant

This patient has to much Flexor Tone, not enough Extensor Tone.

STEP 1. Therapy Localized to the Neurolymphatic point of the Pectoralis Minor Muscle.

+ Strong muscle weakens, go to step 2

- Muscle remains strong go to Anterograde page 9

STEP 2. CHEMICAL CORRECTION [supplement patient if reoccurs]

While the patient Therapy Localizes to the Neurolymphatic for the Pect. Minor, Check for the of the following nutrients.

+ Nutrient will abolished weakness induced by the TL. to the NL's of the Pect. Minor.

a. Iron / Fe++

b. Molybdenum / Mo++

Anytime there is a need for Fe++, there may also be need for Molybdenum.

c. Neurotransmitter GABA and Co-factors

```
                [ B-6---->
                [Niacin-->          [B-6-->
C.A.C.------A.K.G.------GLUTAMIC ACID-----GABA
                [NH3---->                   [CO2--->
```

d. Whole Adrenal (if Choline weakens a strong muscle)

NOTE Fe++ is utilized in aerobic muscle activity, and extensor muscles are generally aerobic in nature because they are postural muscles.

STEP 3. STRUCTURAL CORRECTION

Check for the following

a. TMJ

b. Pect. Minor Neurolymphatic

c. Upper Cervical Fixation

Correct Accordingly

ALL MUSCLES STRONG PAGE 10

ANTEROGRADE

Sympathetic Dominant

This patient has to much Extensor Tone, not enough Flexor Tone.

STEP 1.

Challenge spinous processes inferior

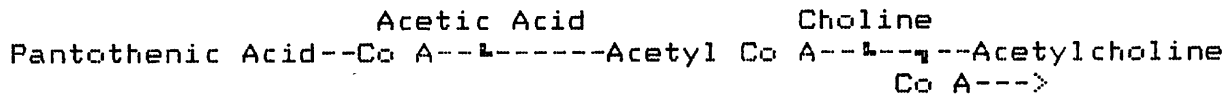
\+ Weakens Strong Muscle, go to step 2.

STEP 2. CHEMICAL CORRECTION [supplement patient if reoccurs]

Check for the need of the precursor and/or Co-factors for the neurotransmitter Acetylcholine.

The proper nutrient will negate the challenge.

a. Vitamin G
b. Pantothenic Acid
c. Choline



Vitamin G [SPL] (Vit. G is involved in the breakdown of Acetylcholine to allow the body to recycle choline)

** Lecithinase enzyme is the key ingredient of Vit. G (calf brain), this frees choline from lecithin and makes it available for Acetylcholine production.

STEP 3. STRUCTURAL CORRECTION

A. Coccyx Lift Technique

Challenge coccyx superior and spinous prosesses inferior.

Place blocks under patient to create a torque pattern

\+ Proper torque pattern will enhance the weakness

Correct by pulling coccyx superior and upper cervicals inferior

B. Upper Cervical Fixation

C. TMJ (Especially closing and wide opening faults with temporoparietal jamming)

ALL MUSCLES STRONG PAGE 11
SPINAL TORQUE PATTERNS

STRONG INDICATOR MUSCLE

With a strong indicator muscle test the patient for the torque pattern that weakens the strong muscle.

1. Place the patient's left leg forward
   + weakens muscle go to step 1.
2. Place the patient's right leg forward
   + weakens muscle go to step 2.

1. The patient has an increase in pineal activity and a decrease in pituitary activity.

CHEMICAL CORRECTION

Test for the need of the cofactors and/or precursors for the production of the Neurotransmitter Norepinephrine, also Pituitary Tissue.

TYROSINE ----------NOREPINEPHRINE
B-6, Folic Acid, Niacin, Fe++
Cataplex C (Tyrosinase), Ascorbic Acid, Cu++, Methyl Donor

1. The proper nutrient will negate the weakness induced by the left leg forward torque.

STRUCTURAL CORRECTION

Challenge for the need of pituitary drive technique.

NOTE Patients with a decreased temperature will require pituitary drive technique. Patients with a normal or increased temperature will require chemical correction. Often patients will require both the structural and chemical correction.

2. The patient has decreased pineal activity and increased pituitary activity. (Increased Thyroid and Steroid activity, adrenal cortex -- ovarian activity).

CHEMICAL CORRECTION

Test for the need of the precursors and/or co-factors involved in the production of the Pineal Hormone.

TRYPTOPHAN----------SEROTONIN----------MELATONIN
B-6,Folic Acid Niacin, Fe++ Pantothenic Acid Methyl Donor (Choline)

2. The proper nutrient will negate the weakness induced by the right leg forward torque.

** If Choline weakens a strong muscle, as in the need for Whole Adrenal, use Betaine as Methyl Donor.

STRUCTURAL CORRECTION

Challenge for the need of the Sphenoid Spread Technique.

** A patient with a low or normal temperature will need chemical correction. A patient with an increased temperature will require the Sphenoid Spread Technique.

NOTE Pineal activity decreases or dampens endocrine function. Melatonin dampens the endocrine system, while Serotonin dampens the hormonal effects at the tissue level.

SWITCHING FACTORS PAGE 12

Gamma 2 Weakness

Test each of the following to strengthen the weak gamma 2.

ANTRONEX: Place Antronex in the patients mouth

\+ Gamma 2 muscle is now strong, go to SP21-K27 (Spinal Laterality) *If Antronex is needed on a return visit, check for food allergies.

SI19: Have the patient therapy localize to SI19 on the right side and the left side of the patient's head.

\+ 1. Test Co Enzyme Q10 for strengthening of gamma 2 muscle.
2. Treat the neurolymphatic points for the Small Intestine on the side of SI19 involvement.

\- Recheck SI19 On both sides with the eyes closed. if + treat as above.

THYMUS: Have patient therapy localize to the thymus.

\+ Strengthens weak muscle: Treat the neurolymphatic points for the thymus (lateral border of the ribs at the 5th intercostal space), and Recheck.

\- T.L. the thymus with clockwise torque then counter-clockwise torque to produce a weakness of the muscle and insure correction.

HYOID: Doctor challenges the hyoid.

\+ Strong indicator muscle weakens, Check the following to negate the challenge.

1. Folic Acid 2. Upper Cervical Subluxation
3. TMJ involvement 4. Thymus

K-27, CROSS K-27: Have patient TL K27 in both a homolateral position, and cross K27 position

\+ Hidden category I pelvic fault

Strong Indicator Muscle

ANTRONEX: Have patient T.L. to the alarm point for the stomach meridian, or place L-Histidine on the tongue. (If Antronex is needed on a return visit, check for food allergies)

\+ strong muscle weakens; test antronex + negates muscle weakness

SI19: Have the patient therapy localize to SI 19 on the right side and the left side of the patient's head.

\+ Weakens strong muscle

1. Test Co Enzyme Q10 to negate T.L. to SI 19.
2. Treat the neurolymphatic points for the Small Intestine on the side of SI 19 involvement.

\- Recheck SI 19 on both sides with eyes closed.

THYMUS: Have patient therapy localize to the thymus

\+ Weakens a strong muscle, treat the neurolymphatic points for the thymus (lateral border of the ribs at the 6th intercostal space)

\- T.L. the thymus with clockwise torque, then counter-clockwise torque to check for hidden problems.

HYOID: Doctor challenges the hyoid

\+ Check the following to negate the T.L. to the hyoid.

1. Folic Acid 2. Upper Cervical Fixation
3. TMJ involvement 4. Thymus

K-27, CROSS K-27 Have patient T.L. in both a homolateral position, and a cross K-27 position

\+ Weakens a strong muscle: Hidden Category I pelvic fault.

NEUROTRANSMITTER TEST

PAGE 13

CCK

KININ MEDIATED ALLERGIES

1. Identify any gamma 2 muscle weakness

2. Test strong indicator muscle against oral insalivation of CCK or T.L. to Pancreas Neurolymphatic.
   + weakness implies kinin mediated allergy problem
   A. therapy localize to the pancreatic neurolymphatic point (left 7th intercostal space) to neutralize CCK induced weakness

3. Test the following substances to neutralize CCK induced weakness, or T.L. to Pancreas Neurolymphatic.
   A. zinc (Rarely Cataplex A as a synergist to zinc)
   B. pancreatic PMG
   C. whole pancreatic tissue
   D. pancreatic enzymes / check for protein deficiency
      ** while using pancreatic enzymes or zinc watch for the need of folic acid.

4. Clean CCK out of the mouth

5. Test the positive nutrients found in step 3 for strengthening of the weak gamma 2 muscle found in step 1.

STRUCTURAL CORRECTION

1. Treat the pancreatic neurolymphatic point and check for other pancreatic reflexes.

   ** If the patient continues to react to CCK on subsequent visits, test for food hypersensitivity reaction for offending foods.

NEUROTRANSMITTER TEST PAGE 14

CLOROX

Step 1.
Find a weak gamma 2 muscle

Step 2.
Find a strong indicator muscle.

Step 3.
Have patient sniff Clorox
- \+ Strong indicator muscle weakens, got to step 4.
- \- No reaction proceed on in flow chart

Step 4.
Test the weak gamma 2 muscle for strengthening with the following nutrients

a. Taurine
   - \- Go to b
   - \+ Test Cysteine, Strengthens check
     1. B-6
     2. Molybdenum, (work by negative feedback mechanism)

b. Methionine
   - \- go to step 5
   - \+ Strengthens check
     1. B-6
     2. Folic Acid
     3. B-12
     4. Methyl Donor (Choline)
     5. Magnesium

Step 5
Check Each of the following to strengthen muscle

a. Niacinamide or Niacin (B2, G, Cu++)
b. Selenium
c. Vitamin E (High dose and Low dose)
d. Aspirin
   1. Evening Primrose Oil
   2. Linseed Oil and/or Fish Oils (EPA)
   3. Other essential fatty acid products
e. Vitamin C
f. Others (rare)
   - Bioflavonoid
   - S.O.D. (check Cu++, Mn++, Zn++)
   - Vitamin A
   - Beta Carotene
   - Endocrine Imbalances

Step 6
Test each positive testing substance against sniffing Clorox SUPPLEMENT ONLY THE NUTRIENTS THAT STRENGTHEN BOTH THE GAMMA 2 WEAKNESS AND THE WEAKNESS INDUCED BY SNIFFING CLOROX

NOTE IF YOU CORRECT FREE RADICAL RESPONSE, ALWAYS CHECK EFA's, AND IF YOU CORRECT EFA's ALWAY CHECK FOR FREE RADICAL PATHOLOGY

NEUROTRANSMITTER TEST PAGE 15

ACETONE TEST

Pentose Phosphate Pathway

1. Identify a weak gamma 2 muscle.

2. Identify a strong indicator muscle.

3. Test patient by having them sniff Acetone.
   + Strong indicator muscle weakens.

4. Test the following nutrient to neutralize the acetone induced weakness.
   While the patient sniffs Acetone test:
   a. B-1
   b. B-2
   c. Niacin/Niacinamide

5. Now test the nutrient that neutralizes the weakness induced by the Acetone, to strengthen the weak gamma 2 muscle.

NEUROTRANSMITTER
ALDEHYDE TEST

*** Check on patients, who are fragrance intolerant, or with Candida Albicans infection.

STRONG INDICATOR MUSCLE

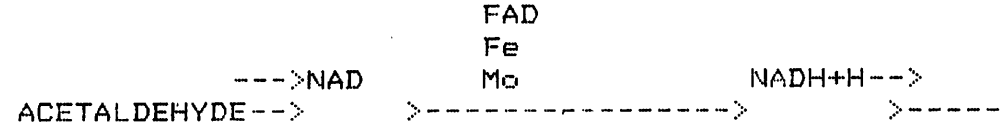
1. Have patient sniff aldehyde.
   + Weakens strong muscle
2. Test patient with the following nutrients, find the nutrient/or nutrients that neutralize the weakness.
   a. Molybdenium
   b. Iron
   c. B-2
   d. Niacin/Niacinamide

GAMMA 2 WEAKNESS

1. Test the nutrients that neutralized the aldehyde weakness, against the gamma 2 weakness.
   + Strengthens gamma 2 weakness.

Note

Supplement only the nutrients that negate the Aldehyde test and strengthen the gamma 2 weakness.



NEUROTRANSMITTER TEST PAGE 16

GLUTATHIONE

CYSTEINE

If Cysteine Strengthens:

Test Clorox:

Weakens, go to step 1

No reaction go to step 2.

Step 1 Check the following:

a. Taurine

\+ Strengthens Test B-6

Taurine is needed to neutralize free radicals (OCL-)

B-6

CYSTEINE-----┗-------> TAURINE

b. Di-Cysteine (CYS-CYS)

\+ Weakens strong muscle --then Test

1. Niacin 2. Cu++ 3. Riboflavin

This patient is over-oxidized, and cannot reduce CYS-CYS to Cysteine, for eventual conversion to Taurine.

B-3, Cu++, B-2

CYSTEINE-CYSTEINE--┗----┗-----┗-----> CYSTEINE----------> TAURINE

c. Methionine

\+ Strengthens -- then Test

1. B-6
2. Folic Acid
3. B-12
4. Magnesium
5. Methyl Donor (eg. Choline, Betaine)

Rebreathing usually strengthens this patient, it increases the need for single carbon groups.

METHIONINE ----------->CYSTEINE ----------> TAURINE

Step 2 CLOROX NO REACTION

a. Methionine

\+ Strengthens -- then Test

1. B-6
2. Folic Acid
3. B-12
4. Magnesium

METHIONINE ----------> CYSTEINE

b. If GLYCINE and GLUTAMIC ACID Strengthen

Test the following:

1. Magnesium 2. Potassium
3. Need for Parathyroid treatment

CYSTEINE + GLUTAMIC ACID + GLYCINE -------> GLUTATHIONE

c. Di-CYSTEINE (CYS-CYS)

Strong muscle Weakens then Test:

1. Niacin 2. Cu++ 3. Riboflavin

Patient may be over-oxidized, and not able to convert (CYS-CYS) to CYSTEINE.

NEUROTRANSMITTER TEST PAGE 17

GLUTATHIONE

GLYCINE

GLYCINE STRENGTHENS WEAK GAMMA 2

Test Arginine
No effect, go to step 1
Weakens a strong muscle, test Ammonia - weakens go to step 2
Strengthens the weak gamma 2, go to step 3

Step 1 No effect
Test the following nutrients for strengthening of the weak gamma 2 muscle

a. Magnesium c. Folic Acid
b. B-6 d. B-2

Step 2 Arginine and Ammonia both weaken a strong muscle
Test the following nutrients to strengthen the weak gamma 2 and to neutralize the weakness induced by sniffing Ammonia.

a. Arginex b. Manganese c. B-12

Step 3 Arginine strengthens the weak gamma 2 muscle.
Test the following nutrients to strengthen the weak gamma 2 muscle.

a. B-6 c. Aspartic Acid (check like glutamic acid)
b. Magnesium d. Biotin

B-6 in its active form (P5P) is necessary for the conversion of

SERINE---------->GLYCINE

THREONINE is the most difficult Amino Acid to be absorbed

B-6
THREONINE-----&-------->GLYCINE

CHOLINE and BETAINE can be converted into GLYCINE

B-2 B-12 B-2
CHOLINE----&------> BETAINE----&------> N1N-DiMethylGlycine--&--

B-2
-------> SARCOSINE-----&--------> GLYCINE

NOTE: 5-Adenosyl/Methionine
GLYCINE + ARGININE ---->GUANIDOACETATE---------->
(5 SAM ---> 5 SAH)

Mg++
---->CREATINE---&------>PHOSPHOCREATINE------>CREATININE (Urine)

Phosphocreatine is created for the production of high energy Phosphorus bonds -- this may be overworked in highly trained or over trained athletes or athletes after a long event (eg, marathon)

NEUROTRANSMITTER TESTS PAGE 18

GLUTATHIONE

GLUTAMIC ACID

GLUTAMIC ACID STRENGTHENS

Test AMMONIA:

Weakens- May indicate a need for more available Alpha Keto Gluterate from the Citric Acid Cycle:

Check Rebreathing to strengthen the gamma 2 muscle:

Test the Citric Acid Factors:

1. Pantothenic Acid
2. Cataplex B (SPL)
3. B-1
4. Manganese
5. Niacin/Niacinamide
6. Riboflavin
7. B-6
8. P5P (Zn, Mg, P, B-2)

Strengthens:

Check for the need of Glycine

\+ Check:

1. B-6
2. P5P (Zn, Mg, P)

GABA Strengthens:

Check:

1. B-6
2. Zn

NOTES

BRAIN:

B-6

GLUTAMIC ACID---┗-------> GABA

KIDNEY:

Mg++

GLUTAMIC ACID----┗----> Glutamine------> GLUTAMIC ACID + NH3

LIVER:

Niacin

GLU----┗----->a-KG + NH3----->Carbamyl Phosphate---->Urea Cycle

┗-----> Citric Acid Cycle

B-6

***GLU <---┗---------> a-KG

Is a major transaminase reaction for many other reactions of Amino Acids throughout the body

NEUROTRANSMITTER TEST PAGE 19

AMMONIA

*** Check on Patients with active PMS symptoms

Step 1

Find a weak gamma 2 muscle

Find a strong indicator muscle

Test the patient by having them sniff Ammonia

No effect, Continue in the flow chart

Weakens a strong muscle, go to step 2.

Step 2 Weakens a strong indicator muscle

Test Arginine

No effect, go to step 3

Strengthens a weak Gamma 2 Muscle and neutralize the Ammonia induce weakness, go to step 4

Step 3 Arginine has No effect

Test the following nutrients to strengthen a gamma 2 muscle and neutralize the weakness induce by sniffing the Ammonia:

a. B-6
b. P5P
+ Check
1. Zinc
2. Phosphorus
3. B-2
4. Magnesium
c. Molybdenum
d. Iron
e. Arginex [SPL]
f. Manganese
g. Alpha Keto Glutaric Acid, go to step 5

Step 4 Arginine Strengthens gamma 2 weakness

Test the following nutrients to strengthen the gamma 2 weakness and neutralize the Ammonia induced weakness:

a. B-6
b. Magnesium
c. Biotin
d. Phosphorus
e. Aspartic Acid, go to Citric Acid Cycle, Step 5

Step 5 Aspartic Acid (Vinegar) Strengthens a weak gamma 2 muscle or Neutralized the Ammonia induced weakness

The patient has a problem with the Citric Acid Cycle factors.

Test the following nutrients to strengthen the weak gamma 2 muscle and to neutralize the Ammonia induced weakness:

a. B-1
b. B-2
c. Niacin/Niacinamide
d. Vitamin G [SPL]
e. Manganese
f. Pantothenic Acid
g. Phosphorus
h. Lipoic Acid
i. Zinc
j. B-6
k. Iron
l. Molybdenum

NEUROTRANSMITTER TEST
ASPIRIN / EFA's

GAMMA 2 WEAKNESS

Test patient by having them insalivate aspirin

No effect, Continue in the flow chart

Strengthens Gamma 2 muscle, go to step 1

Step 1 Aspirin strengthens gamma 2 muscle

Remove the Aspirin from the patient mouth.

Test the following nutrients for strengthening of the gamma 2 weakness:

a. Black Current Seed Oil
b. Evening Primrose Oil
c. Linseed Oil
d. Fish Oils (EPA)
e. Other EFA's
f. Zinc
g. Magnesium
h. B-6
i. Niacin

STRONG INDICATOR

Test for Aerobic Muscle Problem

Strong Muscle weakens

Test the following nutrient for neutralizing the aerobic muscle weakness:

a. Black Current Seed Oil
b. Evening Primrose Oil
c. Linseed Oil
d. Fish Oils (EPA)
e. Other EFA's
f. Zinc
g. Magnesium
h. B-6
i. Niacin

NOTE* IF LINSEED OIL DOES NOT STRENGTHEN AND E.P.A. DOES, CHECK THE COFACTORS.

If patient is POSITIVE for the need of EFA's also check CLOROX TEST.

NEUROTRANSMITTER TEST PAGE 21

SUGAR

Find a strong muscle
Have patient insalivate sugar
No effect, Continue in flow chart
Weakens strong muscle, go to step 1

Step 1 Sugar Weakens strong muscle
Have patient therapy localize to the Neurolymphatic for the Thymus, while insalivating sugar.
Strengthens, treat neurolymphatic, then go to step 2
No effect, go to step 2

Step 2
Have patient therapy localize to the Neurolymphatic for the Thymus and the Acupuncture point Triple Warmer 23, at the same time.
Strengthens sugar induce weakness

Treatment
Have patient two hand therapy localize to the Neurolymphatic point for the Thymus, While the Doctor taps the acupuncture point Triple Warmer 23, (tap 50-60 times).

Recheck Sugar

FOOD DESENSITIZATION PAGE 22

GAMMA 2 MUSCLE STRENGTHENS TO ANTRONEX

1. Check strong muscle against L-Histidine
   + Strong muscle weakens
   Therapy Localize to SI 19
   + Negates weakness
   Therapy Localize to the Neurolymphatics for the Small Intestine
   + Negates weakness (should be same side as SI 19)

2. With L-Histidine in the mouth, Advance the Right Leg (increasing Pituitary Action)
   + Negates weakness induced by L-Histidine, go to 3
   - No Effect, Therapy Localize to the thymus
   + Negates weakness, treat by Thymus treatment System (Switching Factors - Thymus)

3. Now with L-Histidine in the mouth
   Check the Cofactors for the neurotransmitter norepinephrine:

1.	B-6	2.	Folic Acid	3.	Fe++
4.	Niacin	5.	Cataplex C	6.	Cu++

4. With L-Histidine in the mouth, treat the Neurolymphatics for the Small Intestine.

5. Check for the need of Yin-Yang Synchronization technique (TL SI 19 and the NL's for the Small Intestine at the same time, + Muscle goes Weak, Treat by Tapping SI 19 while the patient TL's the NL's for the small intestine.

6. Administer Pituitary Drive Technique with the L-Histidine in the patient's mouth. (ONLY ADMINISTER IF PATIENT'S TEMPERATURE IS LOW)

7. If reoccurs check for the following
   a. Cofactors of B-6
      P5P (Zn, Mg, P, B-2)
      **If patient show a need for Zinc, and they are taking it, Check for the need of Cataplex A (synergist of Zn)
   b. Hypothalamic Set Point Technique.
   c. The need for desensitization to individual foods.

NOTE

Check the patient temperature before and after. Usually you will see an increase of 2 degrees or more. These patient are the type, that there temperature will not increase using normal techniques.

SPINAL LATERALITY PAGE 23

Acid-Alkaline Balance

GAMMA 2 WEAKNESS

Laterally deviate the patient in a C-shaped curve with a

a. Left Convexity
+ strengthens gamma 2 weakness go to step 2a.

b. Right Convexity
+ strengthens gamma 2 weakness, go to step 3a.

STRONG INDICATOR

Laterally deviate the patient in a C-shaped curve with a

a. Left Convexity
+ weakens strong indicator muscle go to step 3b.

b. Right Convexity
+ weakens strong indicator muscle go to step 2b.

NOTE IF NEGATIVE DO ANTAGONIST TEST BY PLACING OPPOSITE NUTRIENT IN THE MOUTH AND RETEST

2. CHEMICAL CORRECTION

a. With the patient in a neutral position check for the need of acid ash minerals.

+ will neutralize the weak gamma 2 muscle.

b. deviated with a right convexity, check for the need of acid ash minerals.

+ will neutralize the weakness produced by the laterally flexed.

STRUCTURAL CORRECTION

a. Challenge sacral tuberous ligament on the Left. Torque patient with blocks CW/CCW, find the torque pattern that enhances the challenge

b. Check for the phase of respiration that negate the challenge with the blocks in place. Correct on that phase on respiration. This allows the spine to return to its normal position.

c. Retest with the patient laterally flexed.

3. CHEMICAL CORRECTION

a. With the patient in a neutral position check for the need of alkaline ash minerals.
+ will neutralize the weak gamma 2 muscle.

b. With the patient deviated to the right check for the need of alkaline ash minerals.
+ will neutralize the weakness produced by the lateral flexion.

**May also show the need for EFA's, Ca++, or "B", correlate with oral PH. Cataplex B is important in Pancreatic Enzymes (Alkaline) if pt does respond check pt for taking to much C.

STRUCTURAL CORRECTION

a. Challenge sacral tuberous ligament on the Right. Torque patient with blocks CW/CCW, find the torque pattern that enhances the challenge.

b. Check for the phase of respiration that negates the challenge with the blocks in place. Correct on the phase which negates the challenge. This allows the spine to return to its normal position.

c. Retest with the patient laterally flexed.

NOTE* If alkaline ash minerals strengthens in step 2 or if acid ash minerals strengthens in step 3, then the patient is switched.

SPINAL FLEXION - EXTENSION PAGE 24

E.O.O.D.

Sympathetic-Parasympathetic Balance

GAMMA 2 WEAKNESS

Eyes superior strengthens the weak muscle, go to step 1

Eyes inferior strengthens the weak muscle, go to step 2

STRONG INDICATOR

Eyes inferior weakens the strong muscle, go to step 1

Eyes superior weakens the strong muscle, go to step 2

STEP 1. This patient has to much flexor tone

Parasympathetic Dominant

CHEMICAL CORRECTION [supplement patient if reoccurs]

Check for the of the following nutrients

a. Iron / Fe++

b. Molybdenum / Mo++

Anytime there is a need for Fe++, there may also be need for Molybdenum.

c. Neurotransmitter GABA and Co-factors

```
                [ B-6---->
                [Niacin-->          [B-6-->
  C.A.C.------A.K.G.------GLUTAMIC ACID-----GABA
                [NH3---->                   [CO2--->
```

d. Whole Adrenal (if Choline weakens a strong muscle)

NOTE Fe++ is utilized in aerobic muscle activity, and extensor muscles are generally aerobic in nature because they are postural muscles.

STRUCTURAL CORRECTION

Check for the following

a. Pectoralis Minor Neurolymphatic

b. Upper Cervical Fixation

c. TMJ

STEP 2 This patient has to much extensor tone

Sympathetic Dominant

CHEMICAL CORRECTION [supplement patient if reoccurs]

Check for the need of the following nutrients

A. Neurotransmitter Acetylcholine / and Co-factors

```
            [Acetyl Acid-->      [Choline-->
 Pantothenic Acid--Co A--------Acetyl Co A------Acetylcholine
                                    [Co A--->
```

B. Vitamin G [SPL] (Vit. G is involved in the breakdown of Acetylcholine to allow the body to recycle choline)

** Lecithinase enzyme is the key ingredient of Vit. G (calf brain), this frees choline from lecithin and makes it available for Acetylcholine production.

STRUCTURAL CORRECTION

1. Coccyx Lift Technique

Patient TL's coccyx and pushes inferior

Place blocks under patient to create a torque pattern

+ Proper torque pattern will enhance the weakness

Correct by pulling coccyx superior and upper cervicals inferior on the respiration which negates the weakness.

2. Upper Cervical Fixation

3. TMJ (Especially closing and wide opening faults with temporoparietal jamming)

SPINAL TORQUE PATTERNS PAGE 25

WEAK GAMMA 2

Test for the strengthening of the weak gamma 2 muscle

1. Place patient's right leg forward, + go to step 1a
2. Place patient's left leg forward, + go to step 2a

STRONG INDICATOR MUSCLE

With a strong indicator muscle test the patient for the torque pattern that weakens the strong muscle.

1. Place the patient's left leg forward
   + weakens muscle go to step 1b.
2. Place the patient's right leg forward
   + weakens muscle go to step 2b.

1A&B. The patient has an increase in pineal activity and a decrease in pituitary activity.

CHEMICAL CORRECTION

Test for the need of the cofactors and/or precursors for the production of the Neurotransmitter Norepinephrine, also Pituitary Tissue.

TYROSINE ----------NOREPINEPHRINE

B-6, Folic Acid, Niacin, Fe++

Cataplex C (Tyrosinase), Ascorbic Acid, Cu++, Methyl Donor

1A. The proper nutrient will negate the gamma 2 weakness.

1B. The proper nutrient will negate the weakness induced by the left leg forward torque.

STRUCTURAL CORRECTION

Challenge for the need of pituitary drive technique.

NOTE Patients with a decreased temperature will require pituitary drive technique. Patients with a normal or increased temperature will require chemical correction. Often patients will require both the structural and chemical correction.

2A&B. The patient has decreased pineal activity and increased pituitary activity. (Increased Thyroid and Steroid activity, adrenal cortex -- ovarian activity).

CHEMICAL CORRECTION

Test for the need of the precursors and/or co-factors involved in the production of the Pineal Hormone.

TRYPTOPHAN----------SEROTONIN----------MELATONIN

B-6,Folic Acid Pantothenic Acid
Niacin, Fe++ Methyl Donor (Choline)

2A. The proper nutrient will negate the Gamma 2 weakness.

2B. The proper nutrient will negate the weakness induced by the right leg forward torque.

** If Choline weakens a strong muscle, as in the need for Whole Adrenal, use Betaine as Methyl Donor.

STRUCTURAL CORRECTION

Challenge for the need of the Sphenoid Spread Technique.

** A patient with a low or normal temperature will need chemical correction. A patient with an increased temperature will require the Sphenoid Spread Technique.

NOTE Pineal activity decreases or dampens endocrine function. Melatonin dampens the endocrine system, while Serotonin dampens the hormonal effects at the tissue level.

P.L.U.S. TECHNIQUE (3) PAGE 26

Have the patient Stand.

1. Have the Patient Flex Forward;
   In this position, the Right Hand Dominant patient, the following muscle should be WEAK:
   - Left Latissimus
   - Left Trapezius
   - Right SCM
   - Right Piriformis

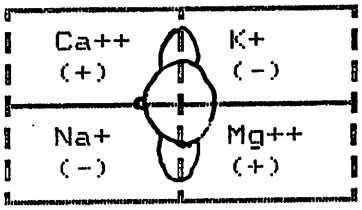
   If not, Then have the patient Therapy Localize the following:
   a. Pelvic Faults (Category I and II)
   b. Lumbars
   c. Sacrum
   d. Upper and Lower Cervicals
   e. Occasionally Thoraco-Lumbar Fixation

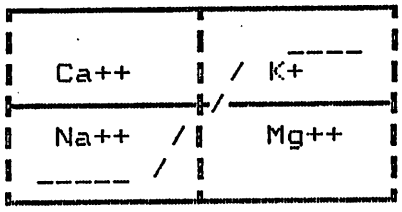
2. Have the patient Extend Backward in a Standing position:
   Recheck the same muscles.
   They should be weak if not Recheck the same areas.

3. Have the patient Lateral Flex to the Right and to the Left.
   Recheck the same muscles.
   They should be weak, if not Recheck the same areas.

NOTE
In real difficult patients, place them in a prone position and have the get up on there elbow in order to arch there back, Hidden Lumbar and Pelvic problems will show.

BODY QUADRANT WEAKNESS PAGE 27





1. Check for weakness of a quadrant muscle (use Abdominal Obliques and Lats)
   Right Flexor_____|_____Right Extensor
   Left Flexor | Left Extensor
2. Check the following nutrients to strengthen the weak muscle:
   Ca++ (+) Should strengthen a Rt Flexor weakness
   Na+ (-) Should strengthen a Lt Flexor weakness
   K+ (-) Should strengthen a Rt Extensor weakness
   Mg++ (+) Should strengthen a Lt Extensor weakness
   *** If opposite nutrient strengthen, check the patient for a sphenoid tilt (use nasosphenoid type correction), Recheck Quadrant weakness pattern and correct.
3. While the patient has the strengthening nutrient in their mouth, check the muscle quadrants for a hidden weakness of the opposite group of muscles
   + Weakness of opposite muscle group found, recheck nutrients
   Both nutrients may be needed
   Check Sp-21 while patient has nutrient in the mouth

STRUCTURAL CORRECTION Remove Nutrients:

1 Therapy Localize to the Neurolymphatic points for the Endocrine glands (Thyroid, Parathyroid, Adrenal, Kidney, and Gonadal).
   + Abolishes weakness, check for opposite muscle weakness with TL present
   + Treat both points - Treat one point

Note ** The control of these nutrients is by dietary intake, intestinal absorption, hormonal and other regulations.
Thyroid effect the ratio of Ca++ : K+
Steroid effects the ratio of Na++ : Mg++

Ca++ and Na+ are important extracellular Ions necessary for depolarization and muscle contraction. Na+ and Ca++ enhances depolarization as does epinephrine (Sympathetic Dominant)
Mg++ and K+ enhance Repolarization (Parasympathetic Dominant), Intracellular Ions, Alkaline Ash, Counteracts Sympathetic Dominance, supports Parasympathetic

If Na+ and Mg++ strengthen and Ca++ and K+ weakens all over this is a classic case of Hypoadrenia.
Some patients who don't weaken to Ca++ - Mg++, will show ligament stretch with Ca++ - Mg++ in the mouth or in the clear. Whole Adrenal and Anti-Oxidants (Vitamin E, Selenium, Super Oxide Dismutase) will abolish the Ligament Stretch problem.
Weakness induced by Na++ - Mg++ Will be abolished by Thyroid. These patients are Under-oxidized, Iodine, and other oxidants will strengthen.

HYPOTHALAMIC SET POINT TECHNIQUE PAGE 28

This technique is used for any reoccurring problems.

VISCERAL PROBLEMS

Step 1 Therapy Localize to the acupuncture head point for that organ:

\+ Strong Indicator weakens
Therapy Localize to Neurolymphatics' or Alarm Point for that Organ

\+ Negates weakness, Treat Neurolymphatics or Meridian

Step 2 Therapy Localize simultaneously to the Head Point and Neurolymphatic or Alarm Point

\+ Strong Indicator Weaken

Treat by tapping Head Point while Patient Therapy Localizes (two Hands if Possible) to Neurolymphatic or Alarm

SPINAL PROBLEMS

Step 1 Therapy Localize to a vertebra

\+ Strong Muscle weakens, then challenge and Adjust

Step 2 Therapy Localize simultaneously to the Vertebra and the Associated Head Point Meridian

\+ Strong Indicator Muscle weakens

Treat by Tapping (50-60 times) Associated Head Point While patient Therapy Localize (two Hands if Possible) to the Vertebra.

MUSCLE PROBLEMS

Step 1 Test a Muscle

If weak treat using appropriate factors

Step 2 Therapy Localize Simultaneously to the Head Point for the Associated Meridian and to the Muscle Location

a. Origin b. Insertion c. Belly

\+ Strong Muscle weakens

Treat by Tapping (50-60 times) while Patient maintains Therapy Localization to the Muscle Location

REOCCURRING HYPOTHALAMIC SET POINT PROBLEMS

If a Head Point needs to be corrected after it has been previously treated, and there is no apparent cranial fault, check the nutritional component of the associated neurotransmitter:

Small Intestine -- NOREPINEPHRINE
Bladder ---------- SEROTONIN
Triple Warmer ---- SUGAR METABOLISM, POSSIBLY INSULIN
Gall Bladder ----- ACETYLCHOLINE
Large Intestine -- GAMMA-AMINOBUTARIC ACID (GABA), GLYCINE, GLUTAMINE
Stomach ---------- HISTAMINE, KININS (CCK., POSSIBLY BRADYKININ)

SPECIAL TECHNIQUES PAGE 29

HIDDEN PROBLEMS:

To find hidden problems the following techniques can be used:

1. ANTAGONISTIC TESTING (2)
   Antagonistic testing can be achieved by placing the opposite nutrient in the patients mouth. This can be thought of as a chemical B.I.D.'
2. OCULAR LOCK
   Have the patient follow your finger as you move it in a circular motion, watch his eyes for any abnormality in the smooth motion. If there is a sudden movement, have the patient look in that direction, this will bring back a gamma 2 weakness or weaken a strong muscle.
3. DOMINANT EYE
   Have the patient close their dominant eye. This takes out the bodies ocular righting reflex, and allow for hidden problems to present themselves, the same as B.I.D..

NUTRITIONAL

TEMPORAL TAP AUDITING TECHNIQUE (3)

Temporal tap the patient to see if the gamma 2 muscle weakness returns. If the muscle weakness does return then test for the nutrition component. Once the nutrient has been found have the patient chew that nutrient or keep it in their mouth throughout the treatment.

BIBLIOGRAPHY

1. Dr. Walter Schmitt,
The Links Seminar Series, The Links Between the Nervous System and the Body Chemistry,
January, February, and March 1988 Los Angeles.
The Effect of Gait and Torque on Enhancing and Suppressing Immune System Function,
I.C.A.K. 1987 Winter Collected Papers.
Centering the Spine, Functional Neurological and Biochemical Considerations,
I.C.A.K. 1987 Summer Collected Papers.
Pre Test Imaging, A Screening Test for Cranial Faults,
I.C.A.K. 1987 Summer Collected Papers.
Kinin Mediated Allergies and Pancreatic Activity,
I.C.A.K. 1986 Winter Collected Papers.
Tonic Labyrinthine Reflexes in the Weight Bearing Position,
I.C.A.K. 1986 Summer Collected Papers.
2. Dr. Richard Belli,
Personal Communication.
3. Dr. George Goodheart,
P.L.U.S. Technique
I.C.A.K. Summer Convention
Detroit, Michigan Summer 1988.
4. Dr. Michael P. Lebowitz,
Some Helpful Additions to Gamma 2 Muscle Testing,
I.C.A.K. 1987 Winter Collected Papers.

A NEW VIEW OF THE PATHWAYS OF THERAPY LOCALIZATION

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

Abstract: An up dating of information of the pathways of therapy locaization based upon our current information.

I first wrote "Pathways of Therapy Localization" about ten years ago. I felt that new information about the nervous system coupled with new techniques in Applied Kinesiology made it imperative that this paper be up dated.

In my original paper the conclusions I drew were the following:

1. That the phenomenon should be one of brain synaptic connection rather then spinal cord reflex becuase of the wide areas of neurological connection involved and that any intact muscle could be used to test.

2. For this to take place there should be a relatively direct pathways available for both sensory and motor connections to take place. These direct pathways are present in the sensory area in the lateral spinothalmic tracts, and ventral spinothalamic tracts. In the motor areas the direct pathways are in the pyramidal tracts, crossed pyramidal of lateral corticospinal tracts.

3. A central area in the brain where the intermixing of both motor and sensory fibers could have effects on either function. This area was found in the pre and post central gyrus and thalamic regions.[1]

I began examining the literature in neurology to see if pathways existed that could explain some of the newer Applied Kinesiology therapy localization enhancement techniques of cerebellar therapy localization, eyes inot distortion (E.I.D.), body into distortion (B.I.D.). These enhancement techniques were not known at the time of my first paper.[2]

Dr. Goodheart explained the cerebellar TL as a rapid touching and braking of the contact to the are of therapy localization. This could be explained in the fact that the cerebellum computes, compares, and compensates, by way of three differnt feedback systems that had transimission rates of 2.5 to 4 m/sec., 2.5 to 6 m/sec., and 18 to 25 m/sec.[3]

The reason that we needed to find a enhancement technique was because of the ability of the cerebellum to compare, compute and compensate, and thereby hiding the problem from the normal method of muscle testing and therapy localization.

Now to look at the cerebellum and its connection to therapy localization.

Vestibulocerebellar pathways-have direct connection from afferent fibers from the vesibular nuclei from the vestibular labyrinth of the inner ear which signal position of the head in space. This pathway could be related to both E.I.D. and B.I.D.

Spinocerebellar pathways have two types of direct tracts from the spinal cord to the cortex of the cerebellum. Each coponent can be devided into forelimb or hindlimb components.

Dorsal Spinocerebellar tract (DSCT) and cuneocerebellar tract (CCT). Convay information from hindlimb and forelimb, and bring afferent feedback of propiroceptors in muscle and joint and exteroceptors of touch and pressure. This could supply the cerebellar therapy localization.

The dsct and CCT receive neurons from the muscle spindles afferent (group 1a and less II fibers) and from the golgi tendon organs afferents (group 1b). They receive signals about muscle lenght and tension.

The Spinocerebellar tracts both dorsal and ventral carry unconscious Proprioception information to the cerebellum. These tracts do not synapse

with higher order neurons, but pass to the cerebal cortex and never reach conscious level.

Neurons from the spinocerebellar tract synapse with neurons in the cerebellum of the Red nucleus and in the midbrain which cause contraction of skeletal muscle. These may be the pathways for cerebellar therapy localization.[4]

The DSCT relyas information from the spindle cells at a rate of 2.5 to 6 m/sec. The ventral spinal cerebellar tracts VSCT relays information exclusively from golgi tendon organs (GTO's) at 2.5 to 4 m/sec., which is slightly faster then the DSCT.

The other pathways which receives information from the muscle receptors are carried to the cerebellum by the olivo cerebellar tracts. These pathways carry muscle afferents from group 1a and 2 from the contralateral limbs, mostly lower limbs. The Olivary tract have a much longer latency of 18 to 25 m/sec.[5]

In Richard Restak book The Brain, he speak of the vestibuloocular reflex (VOR) can be demonstrated by having a person hold his hand about 6 inches from his eyes and moving it back and forth as fast as possible. This motion will cause the image to be blurred. Now if you hold the hand still and move your head back and forth as quick as possible there will be much less blurring of the hand. This is due to the (VOR) and the vestibular center of the inner ear allowing the eye to fix on an object while the head is being turned.[6]

The VOR freflex is due to the cerebellar pathways. The only efferent out of the Purkinje cells discharge in the cerebellar nuclei of Deiter's nucleus-Bulbar reticular formation and to the eye muscles. This would account

for E.I.D which is a compensator or error detector.

The Vestibulospinal system have connections that are carried to the muscles of the neck, trucn, and limbs. During activities these centers (VOS) will cause corrective movement that will bring the head to a level position. These are connected to the VOS by the VOR reflex to the cerebellum by the vestibulocerebellar tracts. These may also have a relationship to E.I.D and B.I.D. by their connection to the Thalamus and Basal ganglion.[7]

Besides the cerebellar connections that have been discussed E.I.D. must have a tie in to the cranial nerves that move the eyes.

To put the eyes into distortion one eye must use the superior oblique muscle, which is enervated by the Trochlear nerve IV that move the eye down and medially. The other eye is moved down and laterally by the inferior rectus muscle, which is controlled by the Oculomotor nerve III. These are tied to cerebellum by Deiter's tracts.

Conclusion:

These pathways that have been diecribed can possibly explain the phenomenon of E.I.D. and B.I.D. and cerebllar therapy localization.

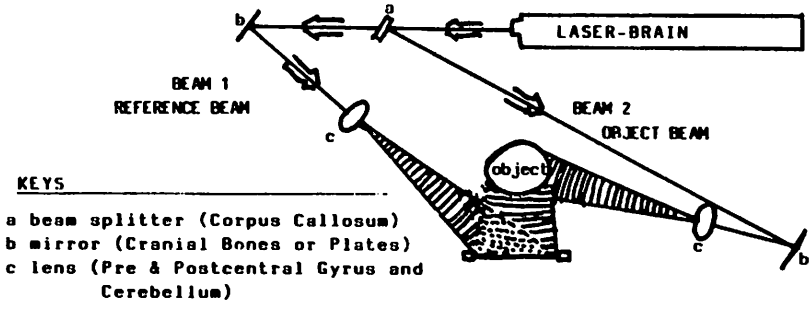
This does not mean that we have found all the possible pathways that may exist.

REFERENCES

1. Sprieser Paul, T. D.C., Pathways of Therapy Localization, The Digest Of Chiropractic Economics, Nov./Dec. 1983.
2. Goodheart George, J. D.C., Applied Kinesiology, Workshop Procedures Manual, Privately Published, Detroit,1983, 1985, 1986.
3. Goodheart George, J., D.C., Applied Kinesiology, Workshop Procedures Manual, Privately Published, Detroit, 1983
4. Netter Frank, M.D., The CIBA Collection of Medical Illustrations, Vol. #1, Nervous System, Cadwell New Jersey, 1983
5. Spence Alexander & Mason Elliott, Human Anatomy and Physiology, Benjamin/Cummings Publishing Company, 1983
6. Restak Richard, M.D., The Brain, Bantam Book, New York, 1984
7. Chusid Joseph, M.D. & Mc Donald Joseph, J., M.D., Correlative Neuroanatomy and Functional Neurology, Lange Medical Publications, Los Altos, Cal. 1964

FIGURE 1

HOW A HOLOGRAM WORKS



The one necessity to have a hologram is to have two beams occurring at the same time that produce an interference pattern. (See Fig. 1).

Any of the Therapy Localization (TL) techniques or enhancement methods have a holoramic quality. The techniques that require dual contacts, or using TL and other sensory stimulus all fit the pattern of a reference beam and object beam: two simultaneous signals at the same time.

Looking at the most basic component of pelvic involvement, we see Category # 1 as being a holoramic representation of the whole pelvis requiring both hands being localized to the PSIS simultaneously. The representation of the whole pelvis as compaired to the pattern of a Category # 2 which requires only one hand contact to the PSIS at one time.[3,4]

Category # 3 disc involvement requires a two point TL to the vertebra above and below the involved disc is holoramic.[5]

In a sacral wobble the patient's both hands are placed in the center of the sacrum palms together, then to find the side of involvement one hand is placed over the PSIS and the other hand on the lateral ilium using two point contact.[6]

We use holoramic information with right and left brain, front and hind brain activity, Basal ganglion and Pons and Medulla techniques, while therapy Localizing the area in question.[7,8,9]

In Meridian therapy we use pulse diagnosis with breath holding (B & H) technique and the Now and Then Technique, which use the currently active meridian based on the Mid-Day/Mid-Night law therapy localized to the alarm point of the meridian and time frame in question.[10,11]

The universal holoramic cranial fault, and the new holoramic cranial faults that I presented at the summer I.C.A.K., Washington, D.C. 1987 meeting.[12,13] Other cranial faults thought to be structural rather then holoramic (impaction) distortion requiring two point TL are the Frontal and Glabellar.[14]

As for as organ involvement we have holographic heart and lung techniques.[15]

Modular distortion of the PRY-T techniques also fit the holoramic variety because they require two body components to be placed into challenge position before muscle indicator will weaken.[16,17] If we compare a singular distortion of rocker action which only require flexion or extension, which doesn't fit the criteria of the reference and object beam. Pitch however, does have the dual component of head and pelvis in simultaneous flexion, extension, or occasionally a combination, which can be considered a reference and object beam.[18]

Other AK techniques that can be considered to fit the holoramic components without belaboring the point are dural torque (Isogai) blocking challenge and Gait Reflex testing,[19] Eye into Distortion (EID), and also body into Distortion (BID),[20] Ligament Interlink,[21] and Pre & Post Ganglionic Technique.[22]

Dr. Ferrari's Neurological Organization Techniques have used the combination of cloacal to righting or vestibular reflexes which would be holoramic.[23]

Conclusion:

It would seem, upon close examination of AK techniques, to have contained the holoramic representation long before we were aware of this theory.

Applied Kinesiology procedures that require two simultaneous points of therapy localization or sensory combination with therapy localization such as E.I.D. or right and left brain and TL are all components of a holographic pattern of memory.

REFERENCES

1. Sprieser, Paul T., D.C., Holographic Memory and Therapy Localization, Digest of Chiropractic Economics, Nov/Dec 1984.

2. Goodheart George J., D.C. Applied Kinesiology, Workshop Procedures Manual, Privately Published , Detroit 1982.

3. Walther David S., D.C., Applied Kinesiology, The Advanced Approach in Chiropractic, System DC, Colorado, 1976.

4. Walther David S., D.C., Basic Procedures and Muscle Testing Vol. #1, System DC, Colorado, 1981.

5. Walther David S., D.C., Applied Kinesiology, The Advanced Approach in Chriopractic, System DC, Colorado, 1976.

6. Walther David S., D.C., Applied Kinesiology, The Advanced Approach in Chiropractic, System DC, Colorado, 1976.

7. Goodheart George J., D.C., Applied Kinesiology, Workshop Procedures Manual, Privately Published, Detroit, 1980.

8. Gooheart George J., D.C., Applied Kinesiology, Workshop Procedures Manual, Privaltely Published, Detroit, 1982.

9. Sprieser Paul, T., D.C., A New Phase of Holographic Memory, I.C.A.K Collected Papers, Privately Published, Winter 1985.

10. Goodheart George J., D.C., Applied Kinesiology, Workshop Procedures Manual, Privately Published, Detroit 1981.

11. Goodheart George J., D.C., Applied Kinesiology, Workshop Procedures Manual, Privately Published, Detroit 1980

12. Walther David S., D.C., Applied Kinesiology, Vol. # 2, Head, Neck, and Jaw Pain and Dysfunction, The Stomatognathic System, System DC, Colorado 1983.

13. Sprieser Paul, T., D.C., A New Class of Holographic Cranial Faults, I.C.A.K. Collected Papers, Privately Published, Summer 1987.

14. Walther David, S., D.C., Applied Kinesiology, The Advanced Approach in Chriopractic, System DC, Colorado, 1976.

15. Goodheart George J., D.C., Applied Kinesiology, Workshop Procedures Manual, Privately Published, Detroit 1986 & 1987.

16. Goodheart George J., D.C., Applied Kinesiology, Workshop Proceudres Manual, Privately Published, Detroit 1980.

17. Walther David, S., D.C., Applied Kinesiology Vol. # 1, Basic Procedures and Muscle Testing, System DC, Colorado 1981.

18. Goodheart George J., D.C., Applied Kinesiology, Workshop Procedures Manual, Privately Published, Detroit 1980.

REFERENCES

19. Walther David, S., D.C., Applied Kinesiology, The Advanced Approach in Chiropractic, SYSTEM DC, Colorado 1976.

20. Goodheart George, J., D.C., Applied Kinesiology, Workshop Procedures Manual, Privately Published, Detroit 1981 & 1986.

21. Goodheart George, J., D.C., Applied Kinesiology, Workshop Procedures Manual, Privately Published, Detroit 1977.

22. Goodheart George, J., D.C., Applied Kinesiology, Workshop Procedures Manual, Privately Published, Detorit 1979.

23. Ferrari Carl, C., D.C., Basic Neural Organization, I.C.A.K. Collected Papers, Winter 1984.

DIVISION II - CRITICAL REVIEW

ORTHOSTATIC HYPERTENSION AS AN INDICATOR FOR HISTAMINE MEDIATED PROBLEMS

RICHARD BELLI, D.C.

Abstract: The physiological and pathological effects of histamine are described. Histamine has far reaching effects on Applied Kinesiology treatment and its outcome. Histamine becomes a problem in some people because of its over production and an inability to degrade it. There are sympathetic spinal patterns as well as other sympathetic override problems caused by histamine. Histamine may also be a factor in orthostatic hypertension. Applied Kinesiology is the treatment method of choice in histamine mediated health problems.

INTRODUCTION

Histamine can be a significant factor and often a real nemesis to the practice of health care. Statistically 76% of my patients present themselves with histamine levels elevated enough to be detected as abnormal through Applied Kinesiology methods.1 Histamine is such a significant factor in many patients' conditions that I make it quite clear to them that they will not experience significant improvement unless we abolish the histamine response. Once this has taken place the patient will notice such a significant difference that they really become aware of when it returns. However, with some patients, getting rid of the histamine response can be a real problem, because they tend to resort back to old nutritional and dietary habits that brought the problem on

in the first place. When the patient resorts back to the old habits, the condition that they presented with will usually exacerbate the next day. This can be a very frustrating scenerio for the doctor who takes pride in getting fast and permanent results.

The complaints that the patient will present will range from swollen ankles to panic attacks and just about everything in between. The most common complaints that are histamine mediated seem to center around four physiological functions of histamine. The vasodilitation of histamine can be a factor in headaches. The inflammatory factor can drastically lengthen the healing time of simple musculoskeletal injuries. The sympathetic nervous system enhancement of histamine can cause a whole meriad of problems. Frequently the histamine patient will complain of foggy head and the inability to concentrate. This is related to the neurotransmitter effects and the right/left brain imbalances associated with histamine and switching.2

It's not uncommon for the patient to have had the histamine related symptoms for many years. At this point, you can expect to be dealing with a very frustrated patient. This patient has probably experienced little relief or insight into their problem. You will also be amazed to find out how little other health care practitioners know or care about the effects of histamine.

This over abundance of histamine seems to be the result of two metabolic faults, over production and/or the inability to degrade histamine into other amino acids. These problems are primarily

nutritionally mediated.3 The most common food culprits in my area, which I'm sure that it varies from region to region, are MSG, artificial sweetners, and caffeine. The vitamin cofactors will be discussed later in this presentation. The encouraging aspect is that over 90% of these patients will respond to the suggested treatment methods.4

Applied Kinesiology is the obvious treatment of choice for the histamine patient. The AK doctor can detect the histamine problem and find out exactly what is needed for its correction. Once the histamine problem is cleared, all of the histamine related symptoms disappear with remarkable haste. If the overt conditions aren't completely healed, you will be amazed at how fast the patient will revert back to pre-treatment status when the histamine response returns.

DISCUSSION

Initially I would like to point out that it is beyond the scope of this paper to go into deep biochemical and physiological explanation of the effects of histamine. However, there is a basic knowledge that is necessary to comprehend the effects that histamine can have on a patient. I would like to spend some time reviewing the basics, then discuss AK findings related to histamine.

Histamine is a neurotransmitter that is derived from the de-carboxialtion of L-histadine amino acid. Histamine is an inflammatory agent as well as a vasodilator. Along with vasodilation, histamine also causes capillary permeability which leads to the edema of inflamation. The combination of

vasodilation and capillary permeability are thought to be the etiology behind some vascular headaches.5 The action of histamine is opposed by norepheneprine.6 And histamine relase from mast cells is inhibited by glucocortocoids.7 Histamine causes the pituitary gland to release thyroid stimulation hormone, lutanizing hormone, follicle stimulation hormone, adrenocorticotrophic hormone, and prolactin releasing hormone.8 Degranulation of mast cells is blocked by bioflavanoids. And finally, histamine is a major part of the inflamation of hives.

The prior paragraph listed many of the basic physiological responses of histamine. These are important processes to normal physiology and healing ect. However, it is very common to see these processes go awry. This is when these physiological responses become pathological responses causing functional illness or illness.The remainder of this discussion will be dedicated to functional illness and applied kinesiological responses.

Histamine for some time has been reputed as being a neurotransmitter. Anyone who has been practicing applied kinesiology for much time at all will attest to this. This observation is often made on the basis of right brain/ left brain switching or neurological disorganization. Upon further observation it became quite apparent to me that histamine may have a sympathetic enhancing effect. This seemed paradoxical in that sympathomimetic agents are vasoconstrictive. So I theorized that histamine is a vasoconstrictive agent locally, and enhances the sympathetic nervous system through the hypothalamus. This may be

done to increase output of adrenal factors to ward off the effects and release of histamine. The sympathetic stimulation may be an artifact of the need for more adrenal output. The fact that histamine is a neurotransmitter really opens up pandora's box. The effects of an overabundance of histamine are vast and possibly far reaching in a patients' overall health picture. This overabundance seems to be the result of overproduction and/or the inability to degrade histamine.

Furthermore, there are several common applied kinesiological findings that I find with the patient that has an overabundance of histamine. Some of them have been written about before and some are new observations. Dr. Walter Schmitt pointed out in his article, "Inducing Right Brain/Left Brain Activity In The Office Setting," histamine is one of the causes of right/left brain imbalances and that Antronex will abolish this activity. Antronex is a natural antihistamine that contains the substance "yakerton", that helps the liver degrade histamine. It is wideley known, in applied kinesiology, that removing switching factors alone will almost always result in improvement of patient complaints.

As my observations about histamine continued, I soon realized that 85% of my patients with "Anterograde" responded to antronex. This lead me to postulate that histamine can be a major factor in the sympathetic pattern that causes the anterograde response. Dr. Goodheart found that anterograde patients responded to the standard process product "Cyruta", which is a source of bioflavanoids.9

As previously mentioned, bioflavanoids will help block degranulation of mast cells. I also observed that the histamine mediated anterograde did not consistantley respond to other anterograde factors as described by Schmitt.10 However, I did find that non-histamine mediated anterograde did consistently respond to the anterograde factors.

Additionally, histamine is one of the chemical agents that depolarize nociceptors. This makes histamine a major factor in pain.11 Histamine is also a strong vasodilator that can be implicated in headache pain. When histamine and kinins work together the vasodilatory effect is greatly magnefied making this combination a sure fire cause of headaches.

Histamine may be a factor in adrenal burnout or functional hypoadrenia. This may be due to the constant stimulation of the adrenal glands to produce histamine blocking substances. This constant stimulation will eventually lead to adrenal fatigue. It is common to see a patient that has a long history of histamine type complaints who is very depressed. This patient has progressed to the point of adrenal exhaustion. On the other hand, the patient that has a shorter history of histamine complaints may show all the signs of hyperadrenia. The adrenal glands aren't fatigued thus the patient becomes the victim of overproduction of adrenal hormones, catacholamines. This is usually short lived; then the adrenal downhill slide starts. The unique aspect of this is that the spine can still be in a histamine/sympathetic mediated anterograde spinal pattern and the patient is suffering all the complaints of hypoadrenia.12 This patient will present a complex

pattern that will respond well if handled correctly.

It has also been reported that histamine may be the culprit in food allergy schizophrenia and agoraphobia.13,14 It has been pointed out by both Abrhams and Levinson that the drugs most effective in these syndromes have antihistamine properties. Abrhams also points out that many patients may respond so well to fasting because allergens are removed from the diet.15

Histamine is converted back into L-histadine in the liver. The main vitamin cofactor for this is B-6, however there are others involved. At this point, I would like to refer you to work on desentization by Dr. Lebowitz. This is an excellent overview of food desentization and histamine production.16

And finally, I would like to point out that I have made an interesting observation in my office in regards to orthostatic hypertension and histamine. Patients who present themselves with a histamine response will 98% of the time have orthostatic hyertension. This is defined as the systolic pressure dropping less than 10 mm/hg when the patient goes from the seated to the supine position. In other words, when the patient was observed to have this finding, 98% of the time the patient would strengthen to antronex and 85% of the time the patient would have a histamine mediated anterograde response. When the histamine response was removed, the orthostatic hypertension cleared in all but one test case. Also, all of the patients that showed histamine mediated anterograde response no longer did so when the histamine response was cleared.

RESEARCH PROCEDURE

Fifty patients were selected that exhibited orthostatic hypertension on the initial visit and subsequently did not on the next visit. Every patient on every visit was tested for the following.

1) Orthostatic hypertension: Blood pressure taken in the seated, then supine and finally standing positions.

2) The patient is then tested for the anterograde response, using the caudal spinous challenge as described by Dr. Schmitt.17

3) If the anterograde test was positive the patient was tested again after the ensalivation of antronex.

4) A Gamma II msucle weakness was then tested after the ensalivation of antronex.

5) The posetive anterograde was then tested after the ensalivation of colinergic factors, "G", B-5, Choline.

6) On the subsequent visit that orthostatic hypertension did not show the patient was tested for anterograde.

RESULTS

I) Patients with orthostatic hypertension.

1) Anterograde test: 86% pos.14% neg.

2) Ensalivation of Antronex: 85% Abolishing anterograde, 15% not abolishing anterograde.

3) Test weak GII muscle with antronex: 83% abolishing,17% not abolishing.

4) Test histamine mediated anterograde With :

a) Cataplex G: 20% abolished anterograde

b) Choline: 11% abolished anterograde

c) B-5: 9% abolished anterograde

5) Tested anterograde on subsequent visit without orthostatic hypertension: 19% posetive, 81% not.

6) 2% of patients tested showed posetive orthostatic hypertension after the histamine response was abolished.

DISCUSSION OF FNDINGS

It is clear that histamine is one of the causes of orthostatic hypertension. This is expressed mainly in increased systolic pressures. However, there are some diastolic changes but the mechanism may be more complex than that of just systolic elevation. The mechanism of the systolic changes are probably along the lines of Royal Lee's theory That histamine mediated swelling of the kidney capsule has an inhibitory effect on profusion of the kidney. This causes the production of angiotensin ect.. The mechanism for the combination of both elevated diastolic and systolic may be more closley related to histamine mediated sympathetic overactivity, which increases pereprial artery tension and cardiac output.

The histamine medaited anterograde response is probably mediated by the action of histamine on the hypothalamus causing sympathetic outflow.

Finally, it must be pointed out that there are many patients that have the anterograde response that is not histamine mediated that respond to parasympathetic enhancing as well as anaerobic nutrients. Also there are patients that do not have orthostatic hypertension or the anterograde response that still respond with a GII muscle weakness strengthening to antronex. It is my experience

here that the histamine is not a major factor in their complaint, however I do not want to downplay the role of histamine under any circumstances.

CLINICAL PROCEDURE

Orthostatic hypertension is used in our office to alert us to the need to check for anterograde, histamine, liver and kidney associated muscles. For example, when orthostatic hypertension exists the iliacus will weaken when going from the seated to the supine position. This will predispose the patient to sacroiliac subluxation when lying down. Desentization techniques as described by Lebowitz are used as well as treatemt to the liver and kidney neurolymphatic reflexes.

CONCLUSION

Orthostatic hypertension screening has turned out to be a convenient and accurate screening tool for histamine mediated health problems. These histamine mediated health problems make up a large percentage of the complaints that patients present in my office. In light of the physiological effect of histamine this is easy to understand. Furthermore, it is apparent that the western diet provides vast opportunity for the overproduction of histamine. Using orthostatic hypertension testing and the antronex screening methods as described by Dr. Schmitt, it is easy to determine the necessary applied kinesiology treatment methods. The proper application of these methods assures speedy results with patients that were at one time considered nightmares. It is very rewarding to get rid of the patients histamine response and watch

the blood pressure normalize and health improve.

BIBLIOGRAPHY

1,2) Schmitt, Walter, D.C., "The Use Of Antronex and Histadine as Screening Tools for Food and other Allergies", I.C.A.K. Collected Papers, Winter 1983.

3,4,16) Lebowitz, Michael, D.C., "A Technique to Abolish all Food Sensitivities", I.C.A.K. Collected Papers, Winter 1989.

5) Peshek, Robert, D.D.S., "Nutrition for a Healthy Heart", Color Coded Filing and Charting Systems, 1979.

6) Guyton, Arthur, M.D., "Textbook of Medical Physiology", W.B. Saunders Company, Philadelphia, 1981.

7,8) Tepperman, Jay, M.D.," Metabolic and Endocrine Physiology", 5th edition, Yearbook Medical Publications, Chicago,1987.

9) Goodheart, George, D.C., "Monthly Research Tape #72", Privately Published,1982.

10,12,17) Schmitt, Walter, D.C., "Centering The Spine, Functional Neurological and Biochemical Considerations", I.C.A.K. Collected Papers, Summer 1987.

11) Schmitt, Walter, D.C., "Functional Neurology of Pain Control", I.C.A.K. Collected Papers, Summer 1988.

13,15) Hoffer, Abrham, M.D., "Nutrition and Behavior", Medical Applications of Clinical Nutrition, Jeffry Bland, Phd., Editor, Keats Publishing,1983.

14) Levinson, Harold, M.D., "Phobia Free", M. Evans and Company, Inc. 1986.

HIDDEN SMALL INTESTINE FAULTS AND THE PINEAL GLAND

Richard A. Belli, D.C.

ABSTRACT: This paper discusses the Applied Kinesiology phenomenon that the small intestine at times will only therapy localize with the eyes closed. Also discussed is a treatment procedure that will eliminate the further need to have the patient close his/her eyes for therapy localization to the small intestine points, or any other points during the treatment session.

INTRODUCTION

Over the last year or so there has been at least a moderate amount of attention paid to the fact that certain dysfunctions with the body will only therapy localize with the eyes closed. As a result of this, there has been several papers written on this subject. Commonly, it is approached by simply checking SI-19 and other points with the eyes closed and correcting what is found. This seems to be a tedious and time consuming way to approach the problem. Furthermore, this method could conceivably double the number of muscle tests during a patient visit.

DISCUSSION

After experiencing frustration with this method, I started looking for a lasting correction to this problem. With trial and error and a little logic, I happened across a permanent solution. In addition, I also found more than one way to challenge for the need for the corrective procedure. It seemed logical to me that if the eyes closed made a difference then this must be a pineal gland mediated problem. I soon discovered that if I did pineal drive on the patient that I no longer needed to have the patient close his/her eyes to elicit the therapy localization to the small intestine or other points. I then reasoned that if

this is a pineal problem, by advancing the left leg and invoking a clockwise torque into the body, I should be able to negate the need to have the patient close his/her eyes to bring out the therapy localization to the small intestine.1 This proved to be true, so it again seemed reasonable to me that pineal cofactors for serotonin/ melatonin would be the nutritional support for this problem.2 Again this proved to be the case, but I also discovered an interesting hitch. An overwhelming majority of the patients,(86%), with this indicator showed the need for iron. Unfortunately time has not permitted a blood panel backup of this finding. This is based on AK findings only.

POSSIBLE EXPLANATION

Greys 30th edition indicates that there is significant evidence that the pineal is an active secratory organ with dampening effects on other endocrine organs. Such as, adenohypophysis, neurohypophysis, endocrine pancreas, parathyroids, adrenal cortex, adrenal medulla, and gonads.3 Also there is both sympathetic and parasympathetic innervation to the pineal gland, with little known about the sympathetic factors.

With this information in mind, I was able to come up with the follwoing hypothesis. The pineal gland is not putting out enough dampening effect to balance the torque induced by pituitary drive.4 In other words there is too much counter clockwise torque and not enough clockwise torque. Closing the patient's eyes increases the production of pineal hormones, increasing the dampening effect, balancing the torque

patterns. This leads me to believe that a 3rd dimension to the all muscles strong pattern exists.5 This is when the patient is stuck in a torque pattern, causing weak muscles to be strong or "hidden". When this torque imbalance is corrected, the weak muscles that are hidden and other hidden problems will come out into the clear. Furthermore, it seems that as the body tries to compensate for the excess pituitary drive, the small intestine, because of its relationship to epinephrine and norephniperine, is subsequently dampened. This inhabition of the small intestine leads to a whole meriad of functional problems. This is probably why the the small intestine is the most common to show up.

RESEARCH PROCEDURE

Patients were tested for therapy localization to SI-19 with eyes open and closed. The patients that showed positive therapy localization with the eyes closed were also tested with the left leg advanced with the eyes open. Next, these patients were screened for the need of serotonin/melatonin cofactors, (B-6,Iron,Folic Acid, B-3, B-5, and Betafood). Data on fifty subjects was collected and tallied. The following percentages abolished the need to close the eyes to get positive therapy localization to SI-19.

B-6 42% Iron 86% Folic Acid 31% B-3 21% B-5 7%
Betafood 0%. Left leg advanced 100%

DISCUSSION OF FINDINGS

At this point, it is apparent that there should have been an oral temperature study done along with the nutrient study. This would have been valuable information. Furthermore, it would have

been equally as valuable to have blood panels on the subjects. I suspect that there would be a majority of subjects that would have shown elevated oral temperatures. This would make for an interesting follow up study.

CLINICAL PROCEDURE

When it is suspected that the small intestine is involved and there is no therapy localization in the clear to SI-19, have the patient close his/her eyes. If this brings out the therapy localization, have the patient advance the left leg, this should bring out therapy localization in the clear (with eyes open or closed). If this is the case, have the patient insalivate cofactors for serotonin/melatonin. One or more of these cofactors should bring out the therapy localization in the clear. The treatment for this will be based on the oral temperature of the patient. If the temperature is low, pineal drive and the cofactors will be administered. If the temperature is normal, only the nutrients will be administered. For the rest of the treatment, the patient may need to keep one of the nutrients in his/her mouth. This is the case when the patient has a normal temperature and pituitary drive is not administered. Additionally, this torque imbalance can be brought out and corrected by having the patient therapy localize the SI-19 and B-1 meredian points. SI-19 represents epinephrine and B-1 represents serotonin.6 Therapy localization to these two points shows the imbalance of torque. Without insalivation of the nutrients, the torque can be brought back into balance by having the patient maintain the therapy localization to the two points and treating the neurolymphatics to

the small intestine. From this point you should not have to have the patient close his/her eyes to bring out therapy localization. Finally, with the proper administration of nutrients and dietary changes, the patient should not present with this problem on subsequent visits.

CONCLUSION

With the application of the presented techniques, the doctor can avoid time wasting procedures and treat the AK indicators that are more directly related to the patient's complaints and pain. This will insure faster and more permanent corrections, while lessening the frustrations that the doctor experiences chasing indicators around the patients body. The addition of each new tool to our AK black bag helps us achieve the fast and lasting results that sets AK ahead of the pack.

REFRENCES

1,2,5) Schmitt, Walter, "Centering the Spine", Functional Neurological and Biochemical Considerations, Collected Papers of I.C.A.K. Summer 1987.

3) Greys Anatomy 30th British Edition, Williams & Warwick. Saunders Publishing Co. 1980, 1447.

6) Lebowitz, Michael and Schmitt, Walter, "Hypothalamic Set Point Technique", Collected Papers of I.C.A.K. Summer 1988.

page 1.

AN INSIGHT INTO THE SUBTLETIES OF BIO-ENERGETICS.

Or, if it works for me - is it valid?

by

Richard L. Cook D.C.

ABSTRACT: There are so many techniques available designed to offset the ills that plague humanity. Different practitioners use their own unique methods in daily practice with clinically good results. This paper is an attempt to try and uncover what is going on, what makes the difference the therapy or the therapist?

There comes a time, I believe, when everyone stumbles across a great, new and revolutionary idea. Most soon recover their composure and blindly carry on as if nothing had happened. A few however, stop, ponder a while, pick up the concept and maybe develop it into something worthwhile.[1] The genius of our own Dr. George Goodheart has given us the beautiful simplicity of Applied Kinesiology (five minutes to learn, a lifetime to master) - as a science it should, in his own words, be reproducible and repeatable anywhere, anytime and by anyone.[2] Within the sphere of kinesiology, there have been over the last twenty years many offshoots, hybrids and further techniques*. Some have added to our armamentarium, others superceded and updated knowledge we already possess and still more has been, by field researchers, invalidated.

There is perhaps a fine line between what is acceptable procedure and what may be viewed with suspicion. Certain facts make logical, sequential and ordered sense, many phenomena are as yet incompletely understood. However, just because we do not comprehend them does not make them unworkable. I have no inkling how a banana tree grows a banana but, that does not prevent the tree

* BK, CK, EK, YOU NAMEIT K!

from doing what it does best nor me from enjoying the end result!

There are many good techniques and many excellent technicians who peddle their wares to other professionals, hungry for new information[3]. Everyone with something new to offer likes to make out that their method is better than anything else, supercedes all previous material and has to be done strictly according to the users (usually expensive) manual. Am I being too cynical ? The late Dr. Alan Beardall discovered, researched and formulated the concept of arm modes and specific hand modes[4]. In a recent paper Dr. Daniel Duffy[5], an equally respected ICAK member, discounts the arm moding concept having tested out 200 individuals. However, not wishing to take sides, Dr. Duffy does not explain in his paper how he arrives at the conclusions he did. There is no mention of methodology - whether 1 or 2 arms were used, whether the patients had any symptomatology, if the eyes were open or closed, which indicator muscle was employed and whether it was checked for reliability, whether the presence of an atlas fixation, TMJ disfunction and factors which may often interfere with the smooth running of the body were present. Therefore, my conclusion would be to take the findings reached with some degree of scepticism.

If two researchers come to the same conclusion independently, the fact is that it was always there but nobody realised.If someone uses the wrong hand mode unwittingly or TL's the incorrect acupuncture alarm point but, believes they are right does it matter? Is the intention or visualisation of the concept sufficient? "As a man believes, so it is".

This could of course explain many things - why in practice different therapists will go their own way fixing what they find in need of correction. Also why colleagues will discover different problems on the same patient (probably in relationship to their belief system and overall knowledge).

GETTING INTO THE SYSTEM -

Most individuals are no problem - they test easily, accurately and the response is quite definite - they are a joy to treat and generally recover well. There are some who, for reasons best known to themselves, are a challenge and defy the usual test procedures. Classically they are those who are under a bout of extreme and acute stress, even a terminal illness.[6] The body seems to throw a blanket round itself as if to say 'there is nothing wrong with me, thank you'.[7] Naturally we know better and all of us have devised tricks to beat the system!

Members of the the ICAK have performed a number of inter-examiner reliability tests[8,9,10] and shown that although results are statistically significant, not everyone arrives at quite the same conclusions. Perhaps differences in the training, ability and knowledge of the practitioner could be the reason.

Many of us have developed or utilised methods to override the barriers and enter the system of the body. Examples would include having the patient chew RNA tablets,[11] pre-stressing,[12] placing the body into distortion,[13] clearing the major fixations[14] or resolving an emotional conflict.[15] To my mind the body can adapt, some of us adapt better and more efficiently than others, thus we practitioners have to work a little harder sometimes to find a way in. What appears to occur is that the body becomes more and more overloaded with stressors and will put up defensive barriers to survive.[16]

Each cell in our body is programmed to survival,[17] "adapt and survive, fail and die" it is the law of the jungle. Healing takes place at a cellular level,[18] and it is the brain and central nervous system that organises, strategises and mediates the appropriate responses.[19] Frequently the terminally ill patient displays no apparent muscle weakness 'in the clear' - this seems odd as they are obviously in such poor health. In trying to help we have to "re-tune" the body back to where we can obtain meaningful data. One possible reason for this dichotomy could be psychological reversal[20,21] (which may be a significant factor in all disease processes).

We still do not comprehend fully the physiology of why an indicator muscle will change with a positive TL or challenge. But, there is no doubt in my mind that it happens - although sometimes we have to be careful with patient positioning, watch for subtle change in their breathing, jaw clenching and recruitment which might negate the response.[22]

Musscle testing, as we know, is a dynamic two-way interactive phenomenon; though something which has always puzzled me is - before we knew about a particular finding it never showed up! In the early days of AK we were quite unaware of many of the factors we now take for granted but, we still achieved good clinical results. Did it matter if the patients eyes were closed, the hands were touching the body, or whether the fingers happened to be in what we now know to be a specific hand modem? Before the quantum leap of recognition, maybe not. Although the gaps in our knowledge force us to continue an almost obsessional search of how the body functions.

It has been our continual srtiving for better, more consistent and lasting results that have kept pioneers working steadfastly, often in the face of ridicule. Others take hypotheses and attempt to disprove them by dubious and inaccurate methodology, or merely dismiss facts because they do not fit in with their frame of reference! However, it is not my intention to point the finger of suspicion, or pontificate upon who is right nor to try and explain the inexplicable, just to get you thinking.

DISCUSSION -

An often quoted notion is that of the 100th monkey phenomenon,[23] whereby when a new idea or skill is discovered once a sufficient threashold number of beings is made aware of it then, it becomes part of the accepted culture. Health and disease may be an offshoot of this phenomenon - it is often stated health can only come from within and occurs at a cellular level. We know the the brain can influence the course of a disease and the cellular responses. What can influence the brain?

The brain has been termed'the ultimate physician',[24] and when one returns to basics, allow the nervous system to function free from impingement and the brain will correct the body at the cellular level. Cells are programmed for life,[25] it is what we do to the body with negative thoughts and actions that causes all the problems.[26] Some medical authorities believe that we are 100% responsible for the ills we suffer.[7,27] There are many documented cases of mind over matter, those who recover from apparently incurable illness. In my opinion there are no incurable diseases,only incurable patients! So far our knowledge does not encompass every possible eventuality. The brain is influen-

-ced by the environment and bearing in mind "Anything can cause Anything"

according to Harper,[28] there is still no complete answer. Truth may be simple but, it is also an elusive quality unless we take the time and trouble to really want to know.

At a recent AK seminar in Bath, England Dr. John Bandy[29] made a comment which at first made little impression but, on reflection was a most interesting and perhaps profound statement. What he said was that he now rarely finds patients to be switched (something confirmed by many of his compatriots) and suggested that some therapists can by their mere presence clear and correct certain electro-magnetic imbalances.

Dr. George Goodheart believes that the more knowledgable the practitioner, the better are the results.[30] "You can only know what you know" and presumably by this token, only treat what you understand. So, what is at work here - the force of personality, the channelling of positive thought, the faith of the patient in you and the therapy?

A host of questions spring to mind; for instance, are people subconsciously guided to the therapist who can help them the most effectively? Different approaches work equally well on the same conditions. There are at least as many varieties of chiropractic technique as chiropractors, and we all get results! Does it matter whether we use patient therapy localisation[31,32] (TL) or doctor TL[33] or surrogate testing?[34] Should nutrition only be tested in the mouth?[35] Many attain results placing it on the body, although there may be a number of other factors at work here, I would suggest if the individual is testing accurately then the body knows best.[36] To quote from the popular song 'There are more questions than answers'.[37]

CONCLUSIONS -

Patient rapport is a most important but an immeasurable factor.[38] Certainly the patient's compliance assists, being "IN TUNE" with that body on the couch is essential but, how much can expectation override the results? Attitude does make a difference - yours and the patient's. A change in the belief system can and will change results. There are many anecdotal tales of practitioners who have mistakenly read X-rays back-to-front for years and ad usted the vertebrae the wrong way - and they still obtain as good a result as some that are doing it right!

The human body is a superbely constructed mechanism that one day we might be privileged to comprehend - but, until that time we must put our trust in a higher guiding power who monitors results with greater control than we can ever aspire to. Perhaps as David Bowie prophetically stated in a song once "knowledge comes with death's release"[39] however, some of us would rather not wait till then! May I leave you with the opening query to ponder on, if it works for me - is it valid?

REFERENCES -

1. Attributed to Winston Churchill.

2. George Goodheart D.C. - Research Manuals.

3. Dynamic Chiropractic - any issue.

4. Alan Beardall D.C. - Clinical Kinesiology (Biocomputer Hand Modes)1986.

5. Daniel Duffy D.C. - Invalidation of a screening technique. ICAK collected
John Heidrich D.C. papers. Summer 1988.
Cecilia Duffy D.C.

6. John Diamond M.D. - Life Energy. 1985.

7. Larry Dossey M.D. - Beyond Illness. 1984.

8. Katharine Conable D.C. - Inter-examiner agreement in AK manual muscle
Bert Hanicke D.C. testing. ICAK collected papers. Winter 1986.

9. Mario Sabella D.C. - An inter-examiner reliability study of manual muscle
Barry Decker D.C.
Terry Krawchuk D.C. testing. ICAK collected papers. Summer 1987.

10. Keith Maitland D.C. et al - Inter-practitioner reliability study. ICAK collected papers. Summer 1987.

11. David Walther D.C - Applied Kinesiology Volume I. 1981.

12. Bert Hanicke D.C. - Pre-stressing and hidden problems. ICAK papers Winter 1984.

13. George Goodheart D.C. - AK Research Manual. 1986.

14. George Goodheart D.C. - You'll be better. The story of AK.

15. Michael Lebowitz D.C - Clearing out internalised emotions that pertuate ill health. ICAK papers. Summer 1988.

16. Carl Ferreri D.C. - BASIC Neural Organisation. ICAK papers. Winter 1984.

17. Reader's Digest - Article. April 1986.

18. Robert Ornstein - The Healing Brain. 1988.
David Sobel

19.Harry Oldfield - The Dark Side of the Brain. 1988.
Roger Coghill

20.Roger Callahan Ph.D. - Psychological reversal and the testing of substances. ICAK papers. Summer 1988.

21.James Hogg D.C. - A system of prioritisation for psychological reversals. ICAK papers. Summer 1987.

22. David Walther D.C. - Applied Kinesiology Volume I. 1981.

23.Lyle Watson - Life Tide. 1980.

24. Nelson deCamp D.C. - SOT Update Seminar. AECC, Bournemouth, England 1987.

25. Satprem - The Mind of Cells.

26. Shakti Gawain - Creative visualisation. 1978.

27.H.E.Stanton M.D. - The Healing Factor. 1981.

28.William Harper D.C. - Anything can cause Anything.

29.John Bandy D.C. - Personal communication. Bath, England. 1988.

30. George Goodheart D.C. - Personal communication. Milan, Italy. 1988.

31.Elmer Cousineau D.C. - Therapy localisation by the doctor instead of by the patient. ICAK papers Winter 1986.

32.Carl Mestman DDS. - Therapy localisation acceptance. ICAK papers. Summer 1987.

33.Gary Klepper D.C. - Patient therapy localisation versus doctor therapy localisation. ICAK papers. Summer 1986.

34.John Corneal D.C. - The use of a surrogate in manual muscle testing. ICAK papers. Summer 1988.

35.David Leaf D.C. - Testing nutrients using chewing of the substance. ICAK papers. Summer 1987.

36.Michael Allen D.C. - The body knows - ask it. ICAK papers. Summer 1984.

37.Jimmy Cliff- "There are more questions than answers."

38.Karl Hynes D.C. - Healing - the doctor/patient relationship. ICAK papers. Winter 1985.

39.David Bowie - "Quicksand" from the album "Hunky Dory".

.....
.....

The Master Neurological Switching Mechanisms Of The Body
K27 - Tip Of Coccyx - Umbilicus

Abstract

K27, Tip of Coccyx and Umbilicus, in combination, have been recognized as major neurological "switches", by those using Applied Kinesiology and Acupuncture. What are they and how they work has never been fully understood. We know they are Acupuncture points and have electrical energy. This doesn't explain why they have such power. The first clue as to what they really are, comes from the fact that rubbing them in certain combinations, can activate or organize neurological balance to influence proper right - left, front - back and top - bottom neurological organization. They are, in fact, neuro-lymphatic reflex mechanisms of major importance.

Discussion

It was first recognized that if K27 in combination with the Umbilicus or bilaterally by themselves, therapy localized negatively, that there was right to left neurological disorganization. It was further recognized that rubbing both K27's with or without the Umbilicus seemed to reestablish this organization. Proper examination and treatment could then follow once this right-left organization was in place. This K27 switching mechanism is an anterior mechanism. The patient must be in the supine position. It does not therapy localize in the prone position, although bilateral therapy localization in the prone position will allow for a more accurate therapy localization of the Limbic System spinal fixation indicators.

Later, the posterior neurological "switching" mechanisms involving the Tip of Coccyx - Umbilicus was recognized as a front - back organization reflex mechanism and the Tip of Coccyx - K27 [both] was recognized as a top - bottom neurological organization reflex mechanism. The patient must be in the prone position.

In utilizing these reflex mechanisms in Neural Organization Technique protocols [an organized treatment protocol, utilizing Applied Kinesiology

methodology with Sacro-Occipital and Cranial procedures, to attain specific treatment goals], certain phenomena was recognized. A patient with an acute Category II condition, who cannot get out of a chair or bed without great pain, and usually in an antalgic position, can get some relief by attempting to walk. The reason for this dilemma is that the lack of motion imposed by sitting or lying on a sacrum already in difficulty, inhibits the circulation of the Cerebral Spinal Fluid in the cord. It accumulates in and swells the lower 1/3 of the cord. The sacrum is the distal pump of the C.S.F. to recirculate it back up the cord. Activation of Tip of Coccyx and Right K27 will act as a neuro-lymphatic reflex to drain this C.S.F. congestion. The pain will be greatly diminished and the patient will be able to mobilize. This observation led to further investigation into the nature of these reflex mechanisms.

If the patient is put into the retrograde position, with the upper body lower than the feet, and a strong indicator muscle weakens, lifting the left arm overhead will negate the weakness. If the head is lower than the feet, lifting the right arm overhead will negate the weakness. If the body including the head is in the retrograde position then raising both arms overhead will negate the weakness. The lifting of the arms overhead relieves pressure on the thoracic ducts thus increasing the lymphatic drainage temporarily. Activating the Pectoralis Minor muscles with spindle cell or golgi tendon activity will also accomplish the same thing. The Pectoralis Minor muscles are rib lifters and are booster mechanisms for the diaphragm which is the master hydraulic pump maintaining the lymphatic circulation.

Under the same circumstances of retrograde lymphatic drainage position, contacting Left K27 will negate the lower body retrograde position congestion. The Right K27 contact will negate the upper body retrograde lymphatic congestion. Rubbing the Left K27 will negate the lower body retrograde indicators and rubbing the Right K27 will negate the upper body indicators. Rubbing both K27's will negate the retrograde indicators for the total body. Therefore, K27 is the master neuro-lymphatic reflex mechanism for the lymphatic system itself. It should be noted that to make the K27 activation meaningful the Universal Cranial Fault, lateral occiput or anterior atlas must be cleared first [neurological priority].

We now have two proven neuro-lymphatic indicators, ie; one for the lower 1/3 of the cord and one for the thoracic ducts themselves.

In analyzing the effect of stimulation to the Tip of Coccyx [T.O.C.] it seems that it is the N.L. for the spine and cord. Right K27 as demonstrated above is for the upper body [above the nipple line] and head. Therefore T.O.C. and Right K27 is the neuro-lymphatic reflex for the spine and skull. To bring it one step further the cord and brain [where the C.S.F. is manufactured]. T.O.C. and Left K27 is therefore the N.L. reflex for the cord and body.

Further investigation indicates that the umbilicus is the general N.L. reflex for the viscera. The posterior switch T.O.C. and Umbilicus is the N.L. for the cord and viscera.

Testing for and correcting these switching faults should be one of the first priorities in any treatment protocol to establish basic neurological organization.

All Cranial corrections are enhanced by a contact to Right K27 while making the corrections. All lower body corrections are enhanced by contacting Left K27 while making the corrections.

If a visceral or organic problem exists as part of the patients overall problems the Umbilicus and K27 should be stimulated. If the organ crosses the nipple line, ie; lungs, heart and liver, both K27's need to be stimulated with the umbilicus. These three organs share both K27's and, I think, both thoracic ducts.

It should be noted that K27 is bilateral, for the most part, under all treatment conditions. If the K27 combination with T.O.C. or Umbilicus T.L.'s unilaterally at first, the other combination will then T.L. after the original fault is corrected.

Understanding what K27, Tip of Coccyx and Umbilicus are in their various combinations is most important to overall success of the treatment protocol.

Conclusion

The posterior "switching" reflex mechanisms of Tip of Coccyx - Umbilicus and Tip of Coccyx and each K27 and the anterior 'switching" mechanism of bilateral K27 or each K27 and Umbilicus are Neuro-Lymphatic Reflex mechanisms for the Lymphatic System itself and are of primary importance.

References

George Goodheart, - Applied Kinesiology - Private Publications and Seminars.

H. Wallace Gunn, -Switching- a Compensatory Response, I.C.A.K. Detroit 1979

David Walther, -Applied Kinesiology Vol. I, Neurological Disorganization

Carl A. Ferreri, -Personal Observations, Neural Organization Technique Research and I.C.A.K. Collected Papers on N.O.T.

TONIC LABYRINTHINE REFLEX CORRECTION AND CENTER OF GRAVITY: PRELIMINARY OBSERVATIONS

MARK FORCE D.C.

ABSTRACT

The tonic labyrinthine reflex (TLR) gives sense of equilibrium and coordinates muscle function of the trunk and limbs in accord with head position. Methods developed within the field of Applied Kinesiology (AK) have been proposed to affect function of TLR. Subjects were evaluated on Metrecom Quadrilateral Weight Scale (MQWS) before and after correction of TLR dysfunction through AK methods. Application of AK methods for the purpose of correcting TLR function demonstrates measurable changes in center of gravity as observed through four quadrant weight scale evaluation.
Key words: Applied Kinesiology, Tonic Labyrinthine Reflex, Center of Gravity, Four Quadrant Weight Scale.

INTRODUCTION

Labyrinthine receptors are mechanoreceptors located within the semicircular canals and utricles. These receptors transmit information to the nervous system with regard to equilibrium, forming the tonic labyrinthine reflexes (TLR). Nerve impulses from labyrinthine receptors are carried by the vestibular nerve to vestibulospinal tracts. At this level, information from TLR affects muscle tone and coordination throughout the body. The specific patterns of muscle facilitation inhibition have been summarized by Schmitt[1] (Table 1).

Table I

Tonic Labyrinthine Reflexes

Face Up	Face Down	Side Lying
Facilitation of limb extensors inhibition of limb flexors	Facilitation of limb flexors inhibition of limb extensors	Facilitates extensors on superior side and facilitates flexors on inferior side

TLR and Center of Gravity: Preliminary Observations - Mark Force D.C.

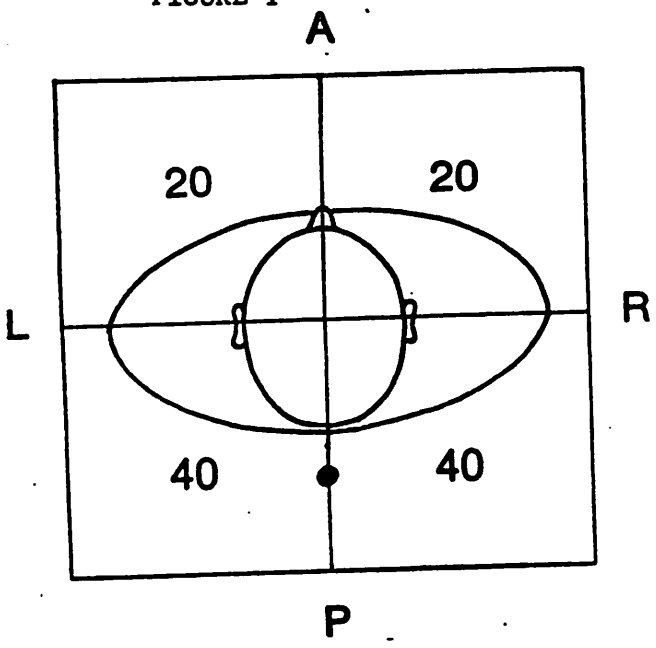
Tonic labyrinthine reflexes are part of a complex network of proprioceptive information that must be coherent for there to be organized function within the nervous system. With the neck righting, visual righting and cloacal reflexes[2], the TLR are involved with coordination of posture and movement. Aberrant function of TLR and other righting reflexes has been proven to alter organization of the nervous system as evidenced by abnormal posture and locomotion.[3,4,5] Evaluation for the presence of and causative factors for neurological disorganization within the body is central to the method of Applied Kinesiology.[6,7] Clinical observation gives support to the assessment that function of TLR can be affected through the application of Applied Kinesiology methods.[2,8,9,10]

It would seem reasonable to observe changes in muscle tone, posture and center of gravity (COG) with the application of techniques that alter TLR function. Methods proposed as specific to evaluation and correction of TLR function have been presented by Schmitt.[1,11,12]

Four quadrant weight scales (FQWS) have been in use by the chiropractic profession for a number of years as an objective measurement of weight distribution and COG[13,14,15,16] and predisposition to biomechanical faults of the spine and pelvis.[17] Standards of accuracy have been proposed.[16,18] Current recommendations are for determining locus plot as a summation of FQWS measurements on an X-Y axis (figures 1 and 2).[16] An average of multiple measurements is recommended for accuracy.[18] Continual measurement of locus plot can be accomplished through measurement with the Metrecom Quadrilateral Weight Scale (MQWS).[19] Figure 3 is an example of this type of measurement.

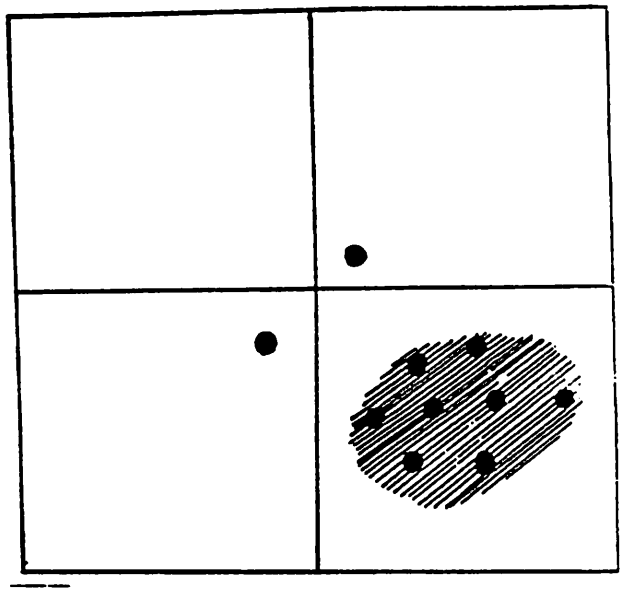
TLR and Center of Gravity: Preliminary Observations - Mark Force D.C.

FIGURE 1



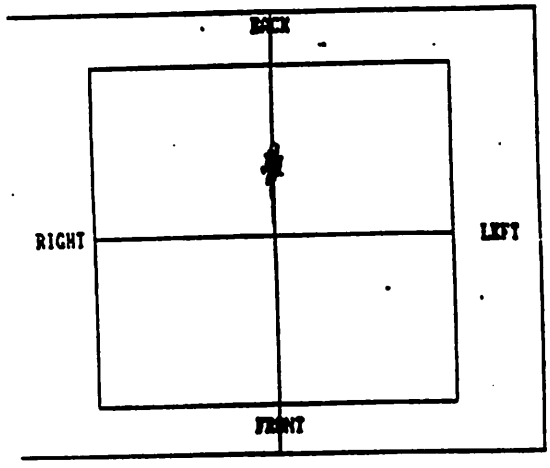
Normal locus plot for average 120 lb. subject.

FIGURE 2



online computer plotting of locus points.

FIGURE 3



It is proposed that theorized effect of TLR technique on TLR function can be evaluated by measurement of subject COG with MQWS before and after application of TLR corrective technique according to protocol as presented by Schmitt.[12]

TLR and Center of Gravity: Preliminary Observations - Mark Force D.C.

Materials and Methods

Subjects were selected at random during the summer 1988 meeting of the International College of Applied Kinesiology. Subjects were informed that they would be participating in a study to *evaluate* the affect of TLR correction or COG. There were 21 subjects in all.

After the initial measurement of subject COG on MQWS, all subjects were evaluated for Gamma II weakness[20] of pectoralis major-clavicular division bilaterally with the subject prone and latissimus dorsi bilaterally with subject supine. Those subjects displaying Gamma II weakness of any muscle tested were then further evaluated for TLR fault and corrected in accordance with protocols as outlined by Schmitt.[12] Correction consisted of a respiratory-assisted manual cranial manipulation to one or both mastoid processes according to methods standard to AK and altered slightly to more specifically affect TLR function as theorized by Schmitt.[1,11,12] There was no evaluation or correction of any other biomechanical or chemical faults. Treated subjects were then re-evaluated with the MQWS. Indications for TLR correction were not present in some subjects and in this group only the initial MQWS measurement was taken.

Subjects stood on the MQWS barefoot and were positioned so that both feet were equidistant from center point of MQWS saggitally and coronally. MQWS measurements were taken for at least 50 seconds. Subjects were not aware of the results of MQWS measurements until after both pre and post-treatment measurements were taken. The clinician who positioned each subject had no awareness of the initial MQWS measurement so that positioning for post-treatment MQWS measurement would not be biased.

TLR and Center of Gravity: Preliminary Observations - Mark Force D.C.

RESULTS

Metrecom Quadrilateral Weight Scale measurements of subjects appear to fall within four groups:

Group I. No tonic labyrinthine fault present. This group numbered 6 of the 21 subjects and represented 28.6 percent of total subject sample. No corrections of TLR fault were made as indication for correction were not present. No second MQWS measurements were made.

Group II. Elicitation of normal locus plot. This group numbered four of the 21 subjects and represented 19 percent of total subject sample. This group represents 26.7 percent of the subjects treated for TLR fault. In this group, there is an apparent change in COG to a COG similar to the normal locus plot as recommended by Grice and Vernon.[16]

Group III. Elicitation of Sagittal Sway. This group numbered four of the 21 subjects and represented 19 percent of total subject sample. This group represents 26.7 percent of the subjects treated for TLR fault. In this group, there is an apparent change in COG such that a decrease of lateral sway and/or accentuation of sway in the sagittal plane is evident.

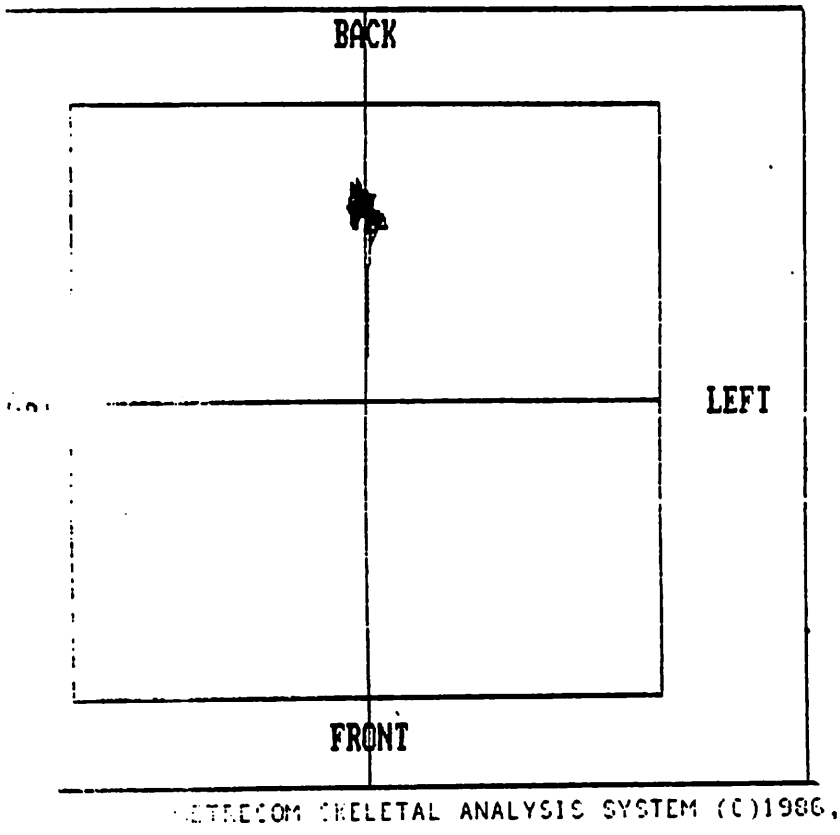
Group IV. Random Response. This group numbered seven of the 21 subjects and represented 33.4 percent of the total subject sample. This group represents 46.6 percent of the subjects treated for TLR fault. There is no apparent change in COG after TLR correction in this group.

Graphs of MQWS measurements for all subjects and ordered according to the four groups, as outlined above, follow this page.

TLR and Center of Gravity: Preliminary Observations - Mark Force D.C.

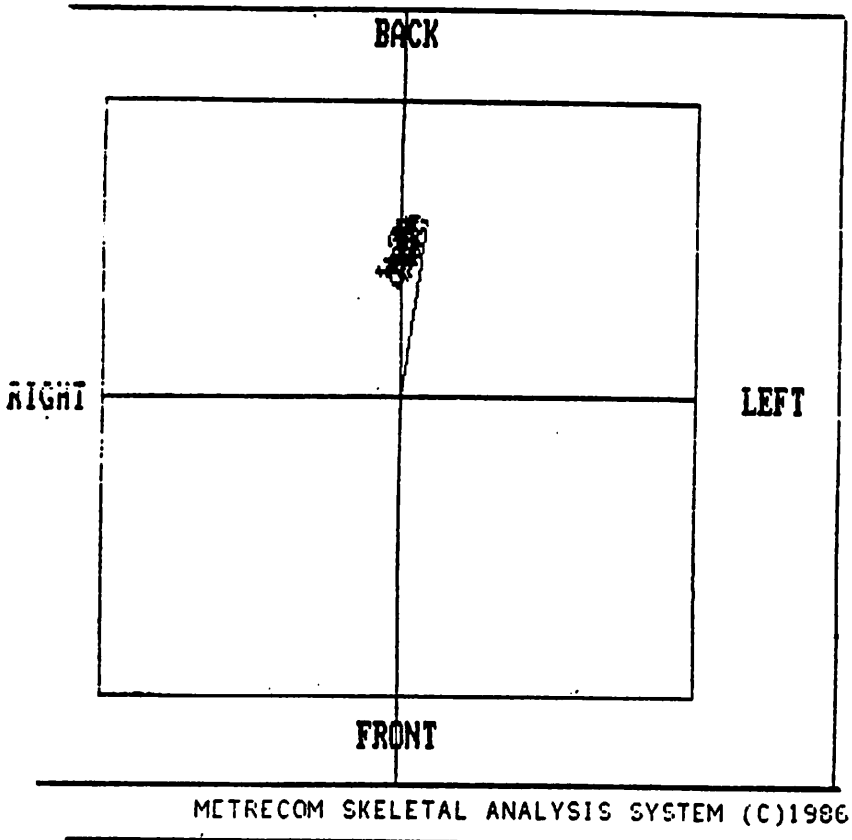
Group I

No Tonic Labyrinthine Reflex Fault Found

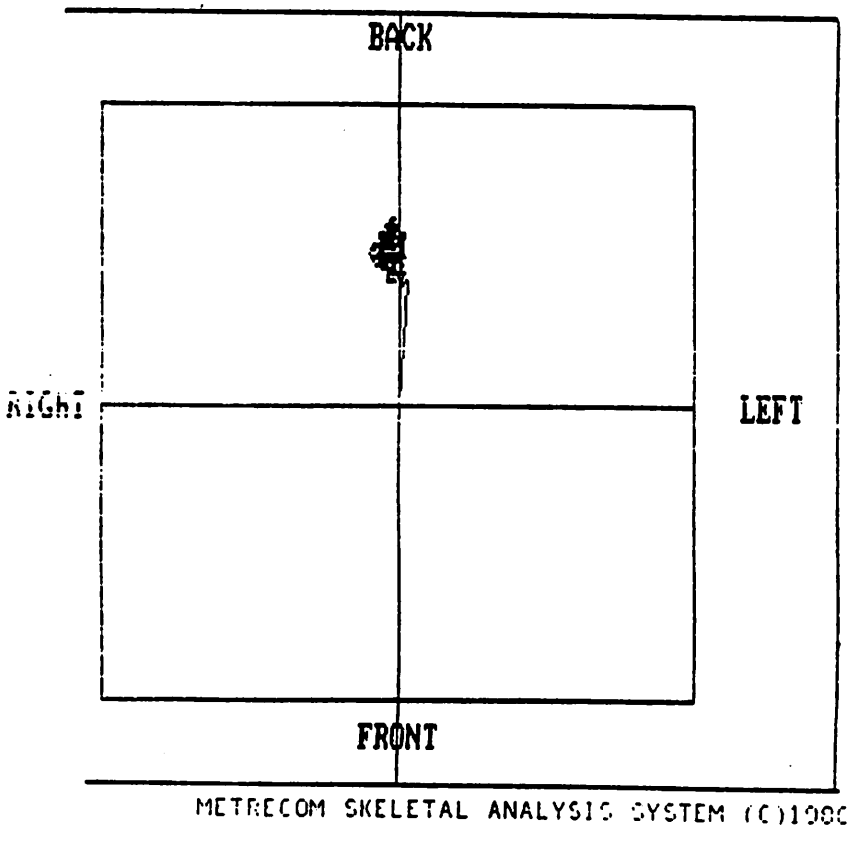


Subject 1

TLR and Center of Gravity: Preliminary Observations

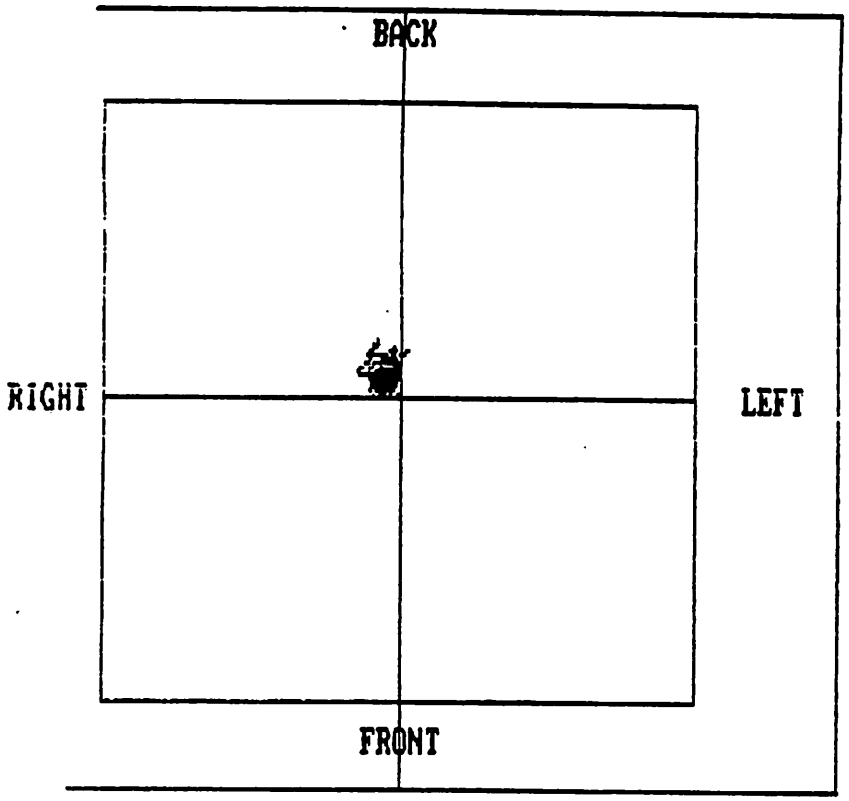


Subject 2

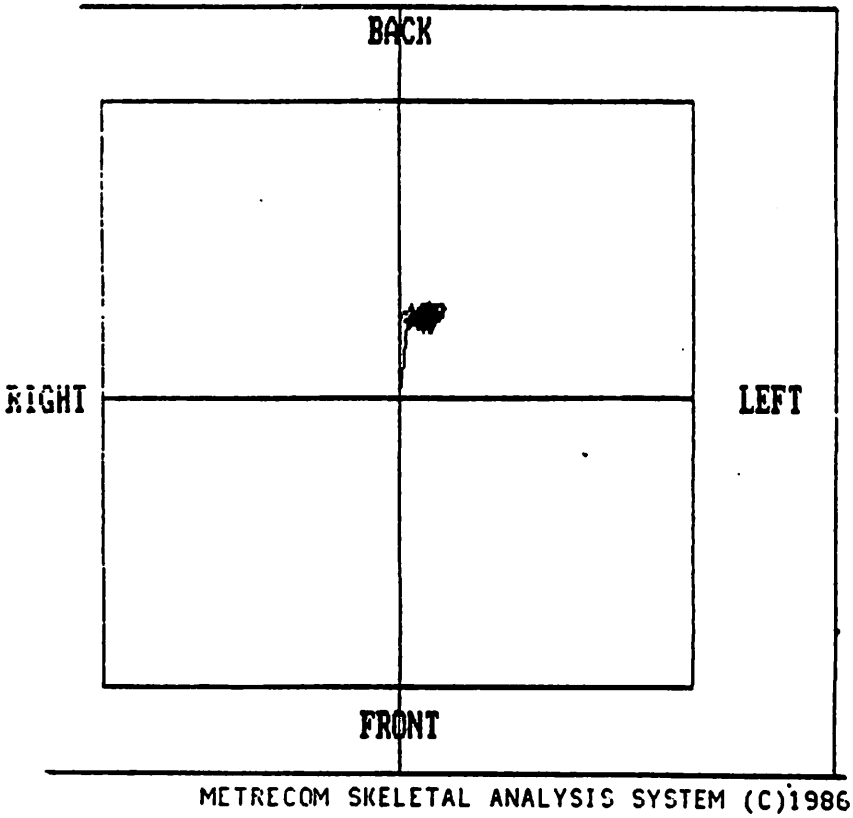


Subject 3

TLR and Center of Gravity: Preliminary Observations

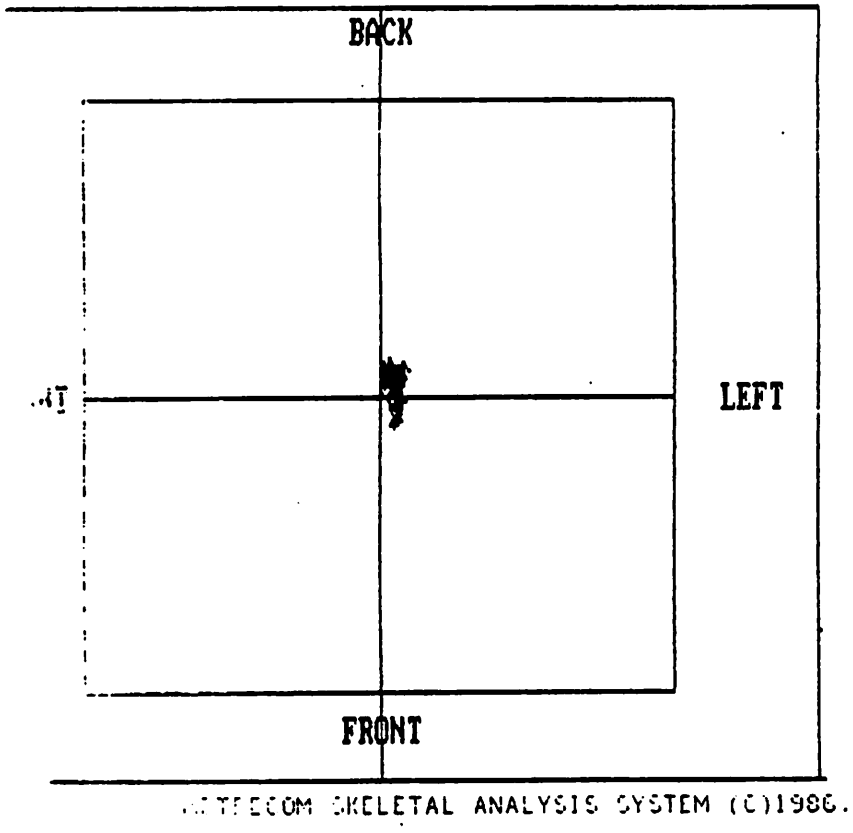


Subject 4



Subject 5

TLR and Center of Gravity: Preliminary Observations



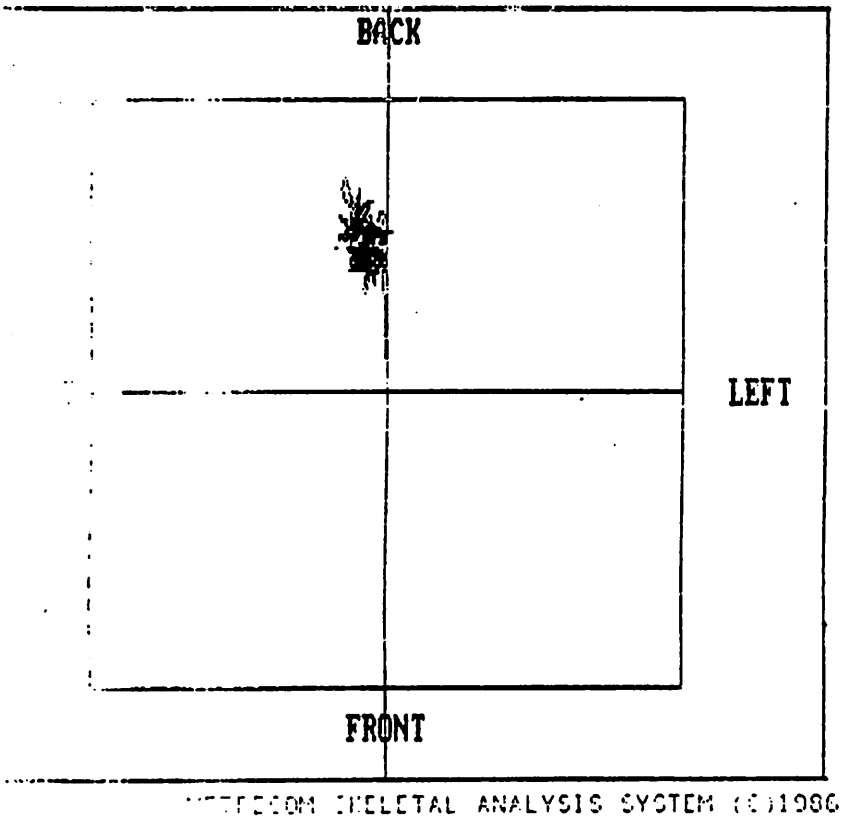
Subject 6

TLR and Center of Gravity: Preliminary Observations - Mark Force D.C.

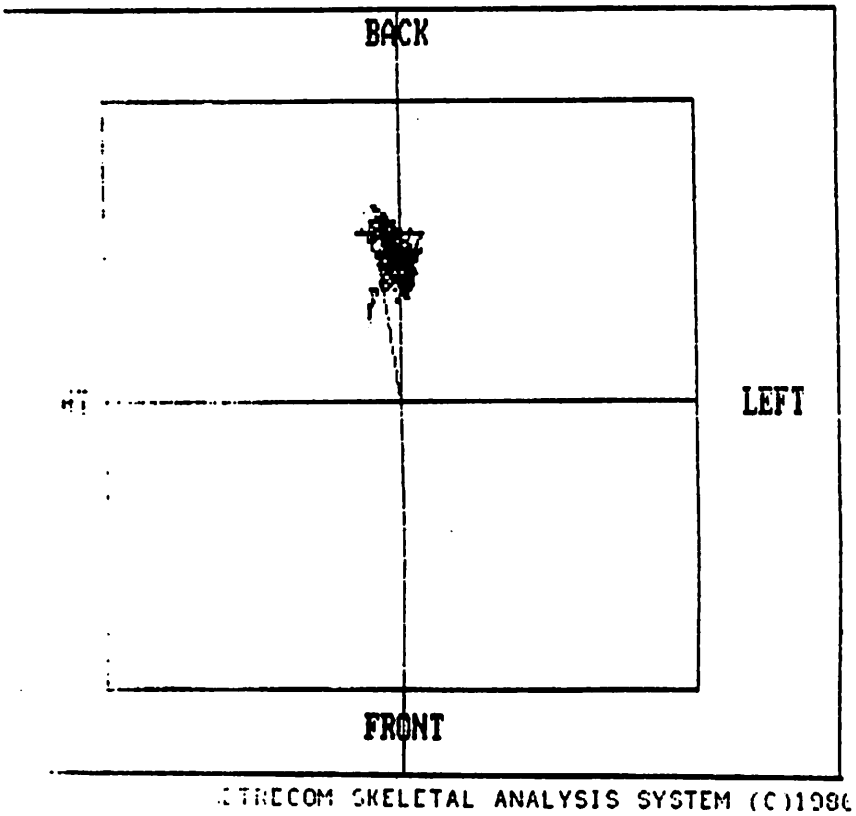
Group II

Elicitation of Normal Locus Plot

TLR and Center of Gravity: Preliminary Observations

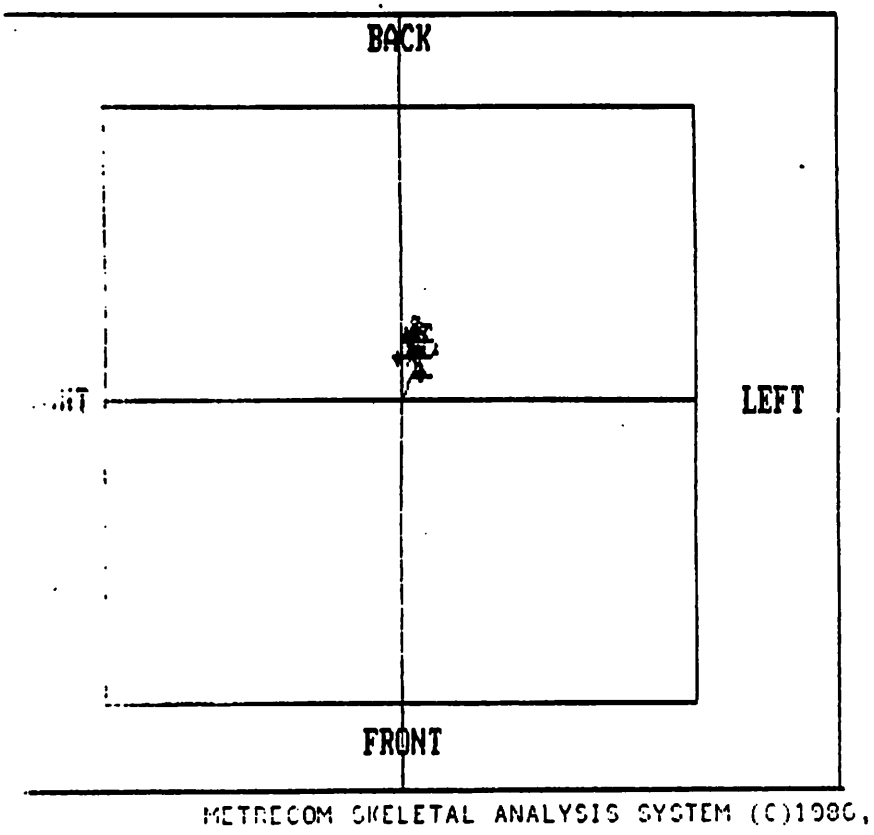


Subject 1
Pre-treatment

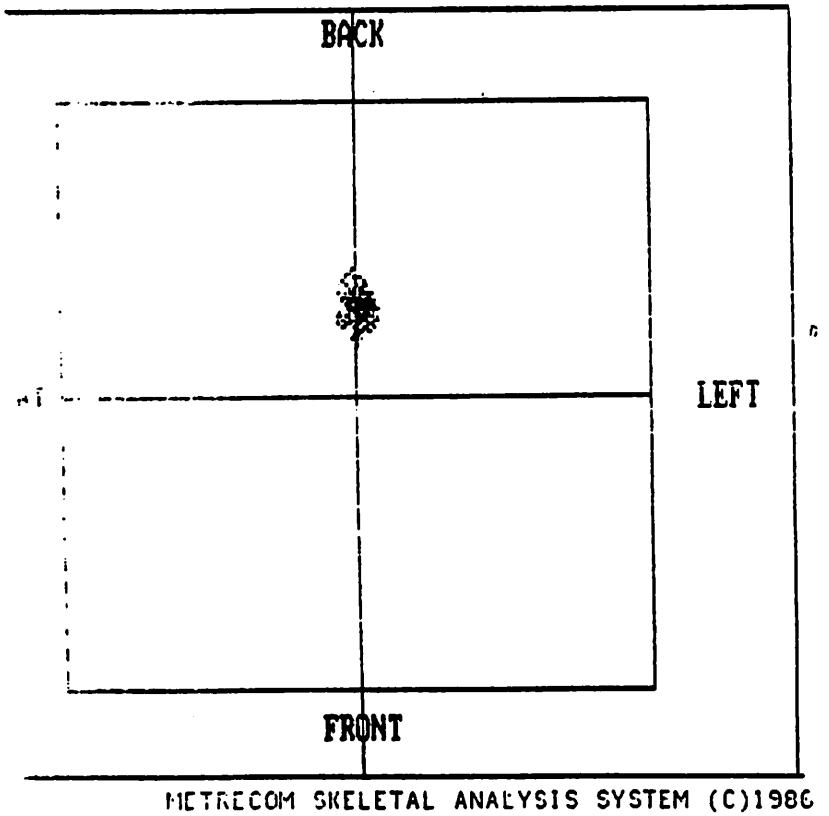


Subject 1
Post-treatment

TLR and Center of Gravity: Preliminary Observations

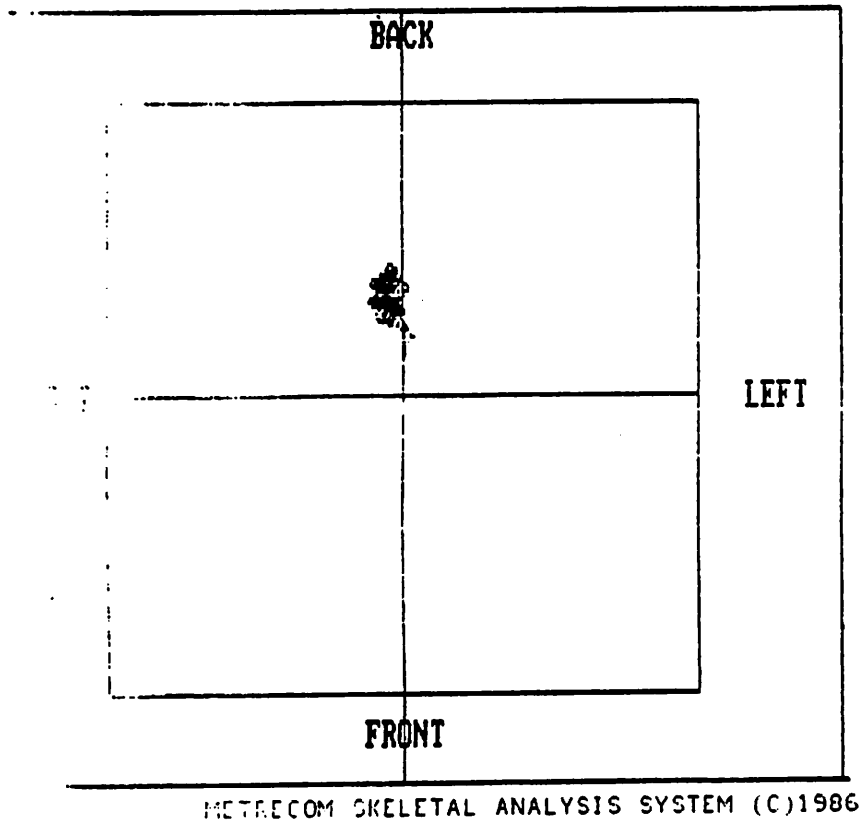


Subject 2
Pre-treatment

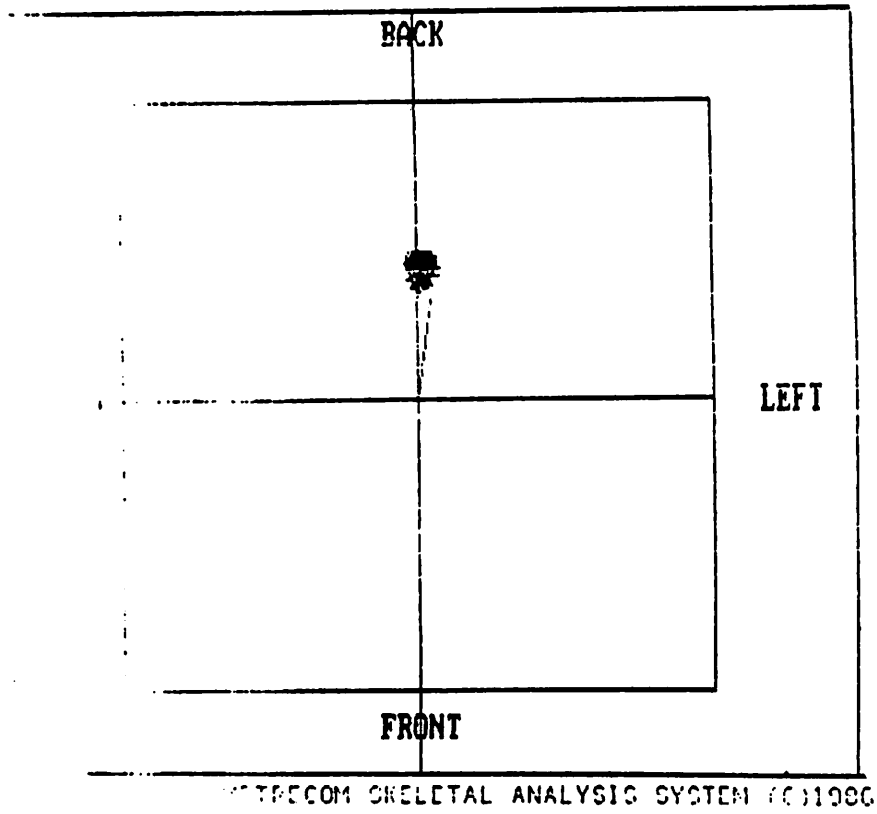


Subject 2
Post-treatment

TLR and Center of Gravity: Preliminary Observations

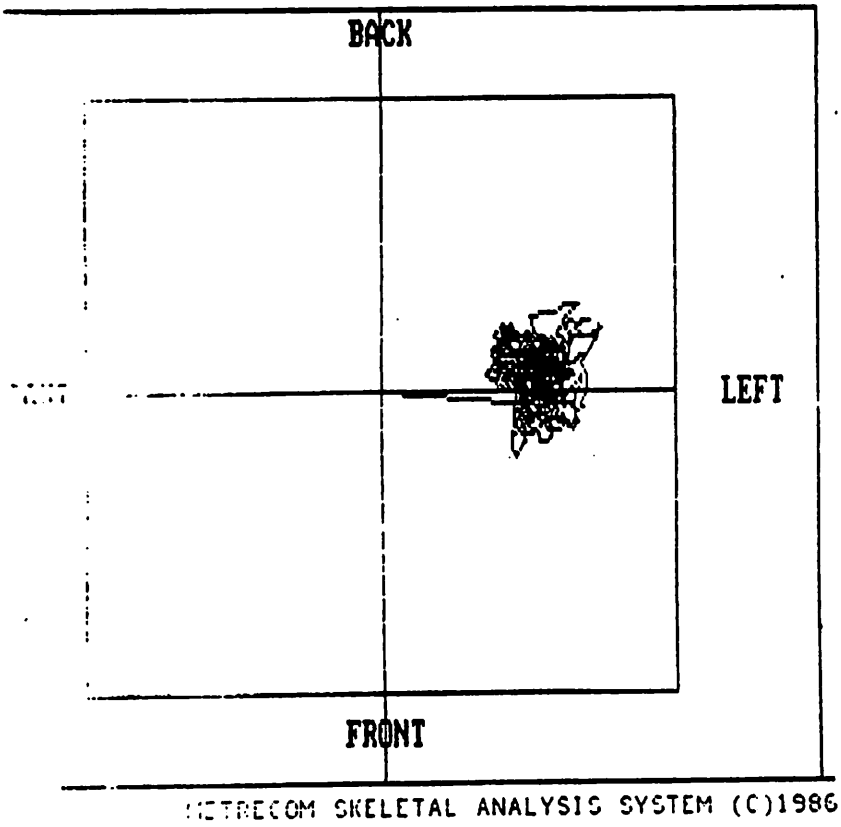


Subject 3
Pre-treatment

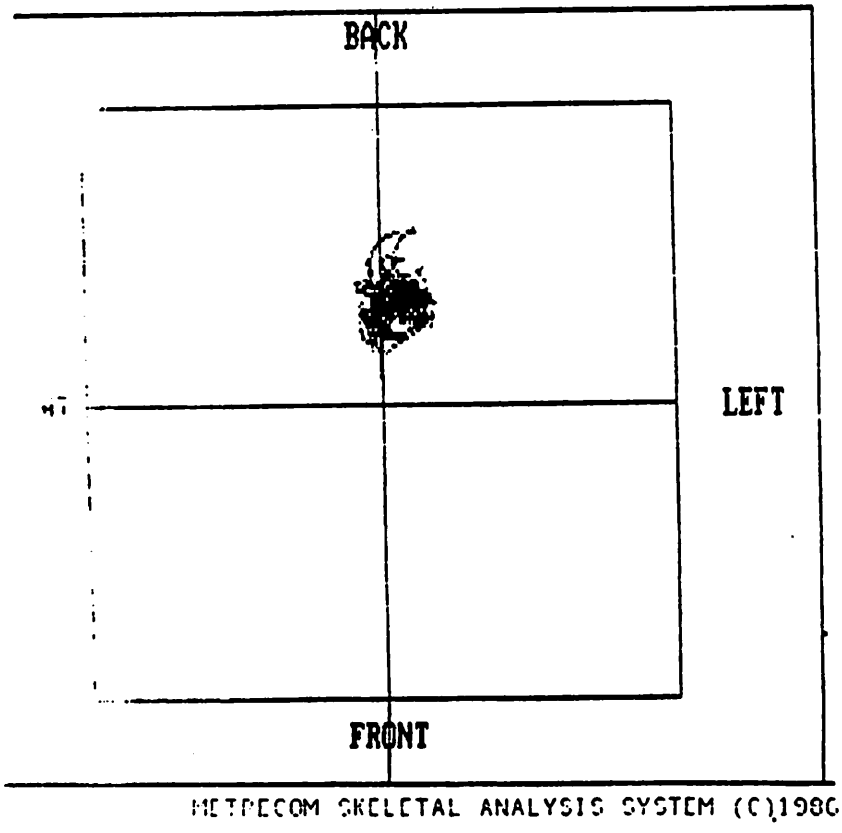


Subject 3
Post-treatment

TLR and Center of Gravity: Preliminary Observations



Subject 4
Pre-treatment



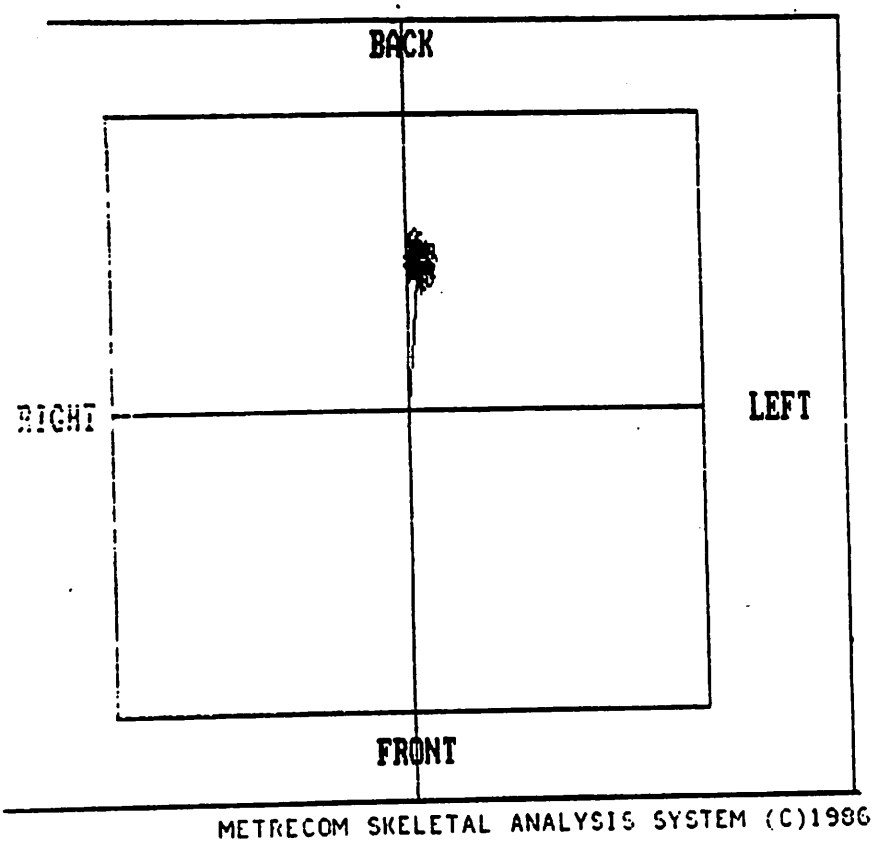
Subject 4
Post-treatment

TLR and Center of Gravity: Preliminary Observations - Mark Force D.C.

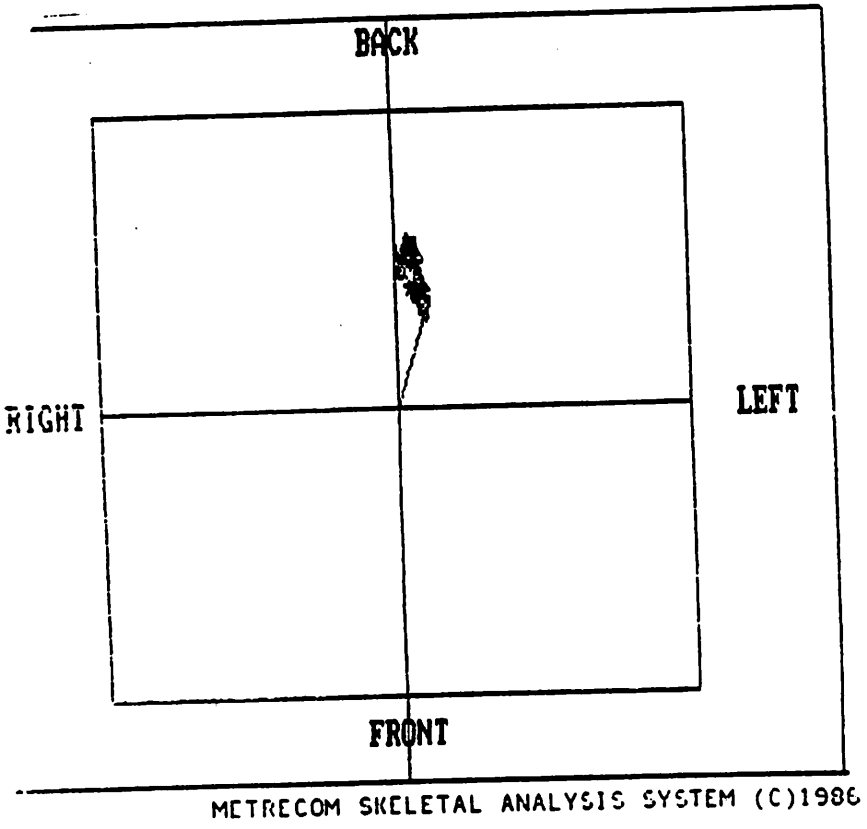
Group III

Elicitation of Saggital Sway

TLR and Center of Gravity: Preliminary Observations

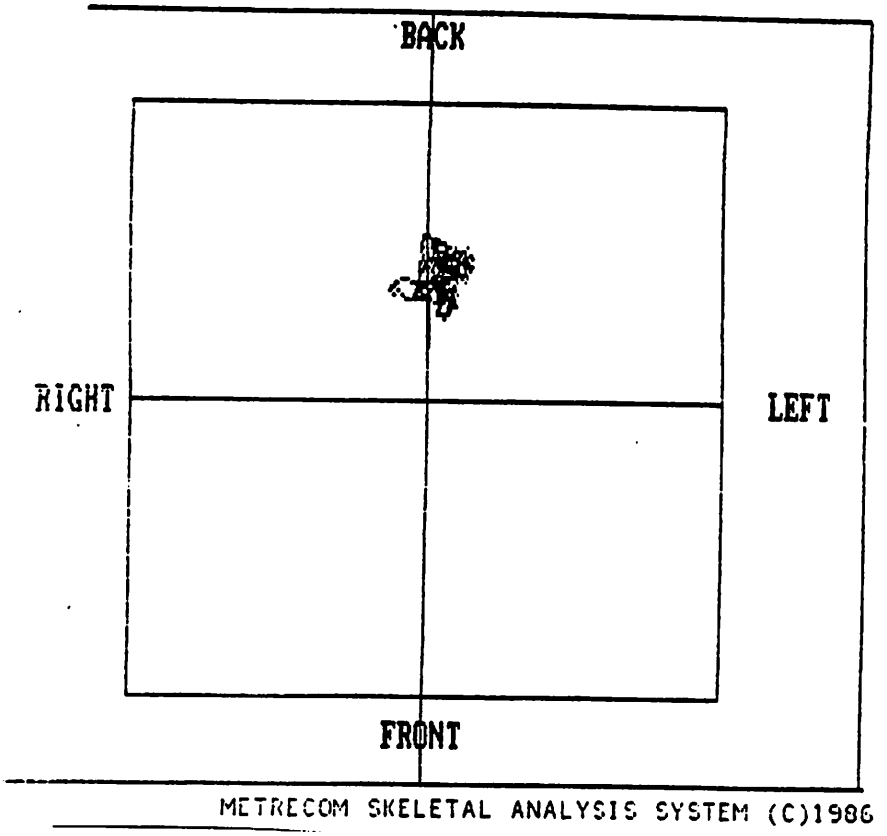


Subject 1
Pre-treatment

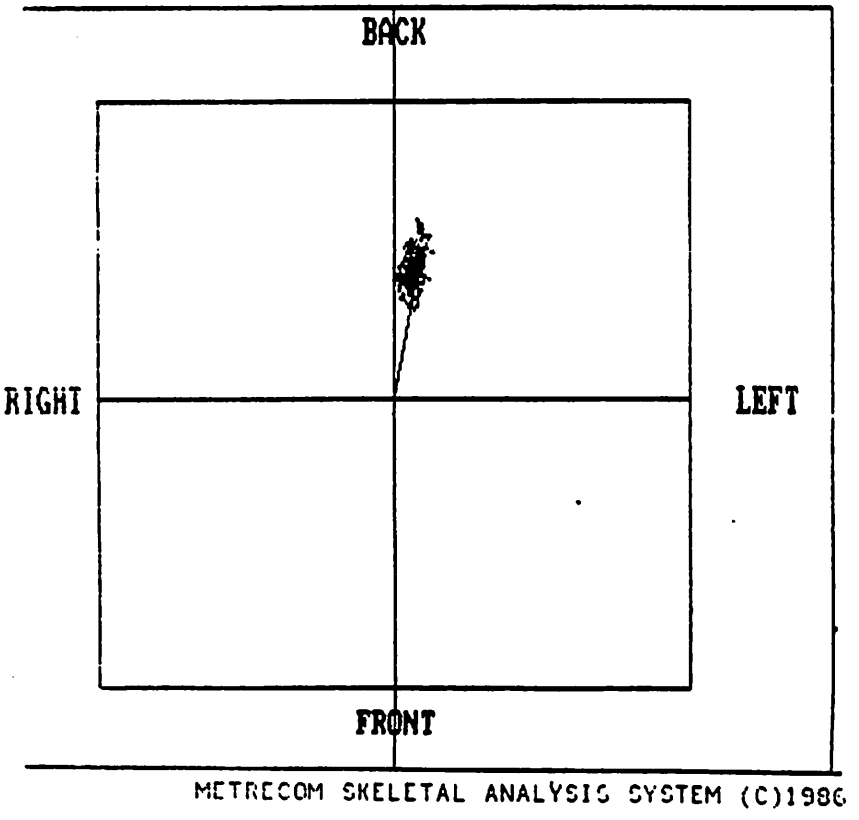


Subject 1
Post-treatment

TLR and Center of Gravity: Preliminary Observations

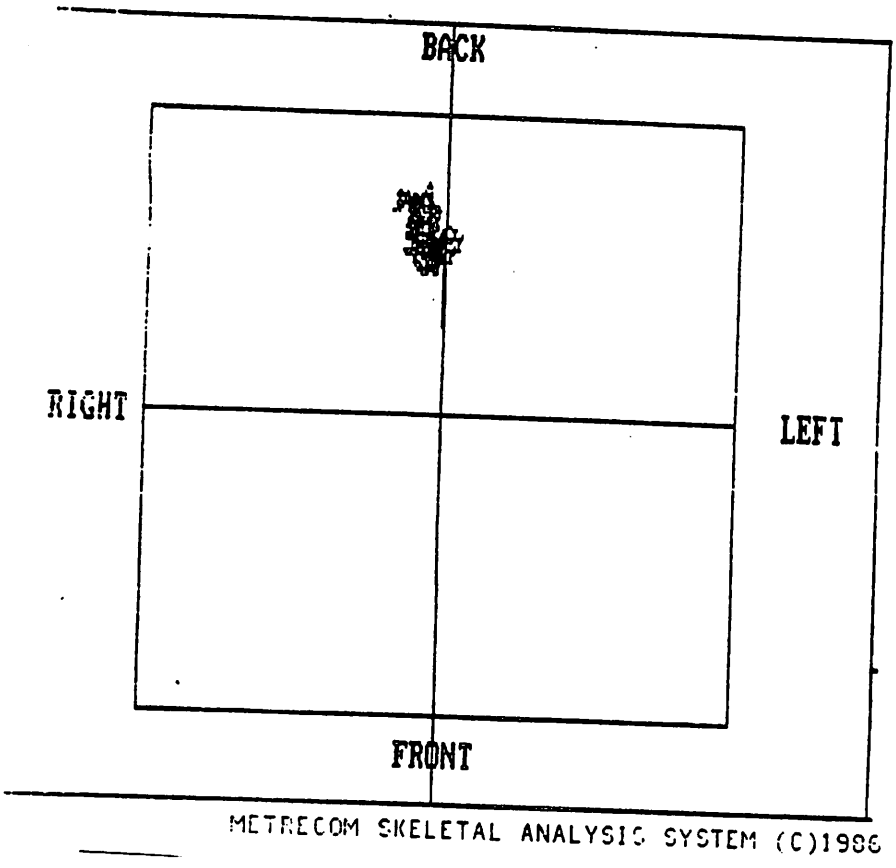


Subject 2
Pre-treatment

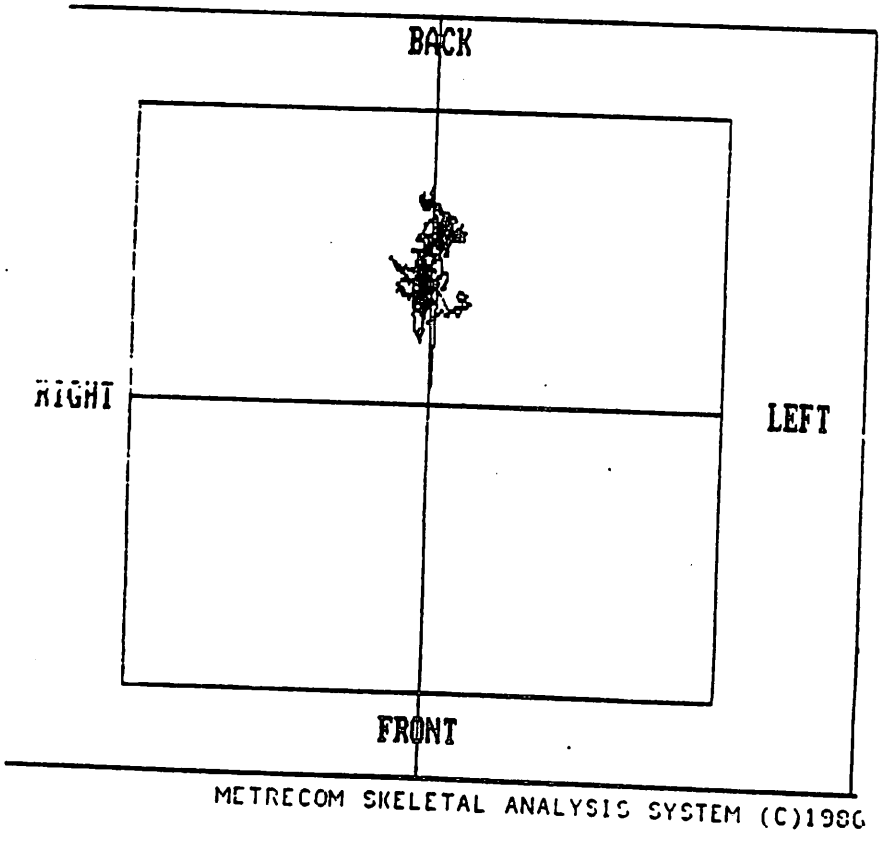


Subject 2
Post-treatment

TLR and Center of Gravity: Preliminary Observations

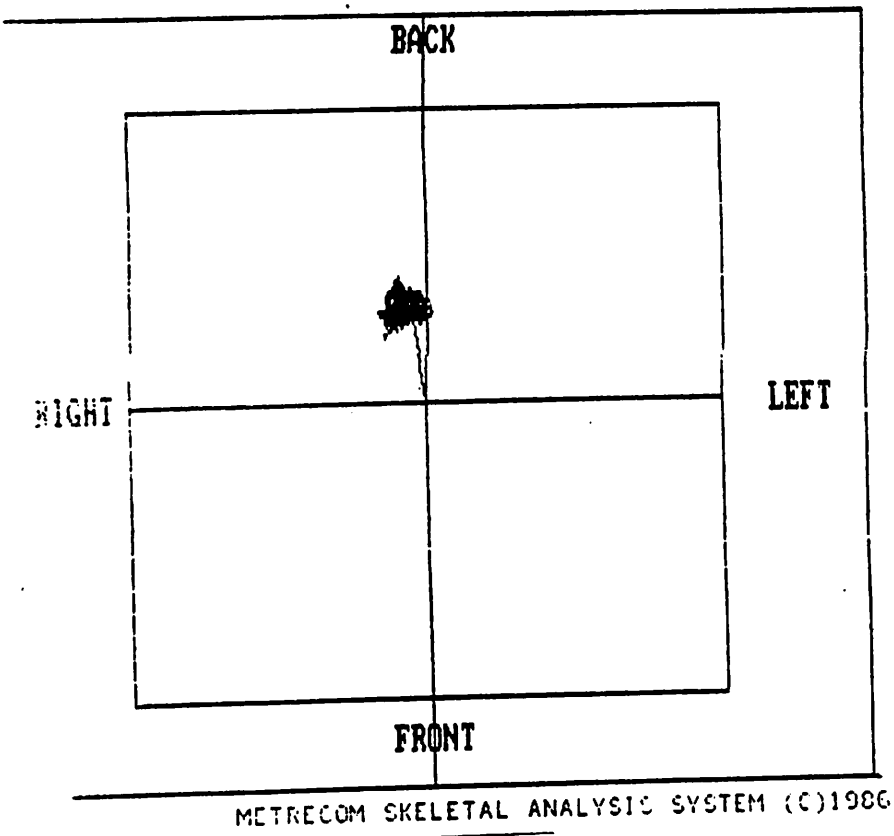


Subject 3
Pre-treatment

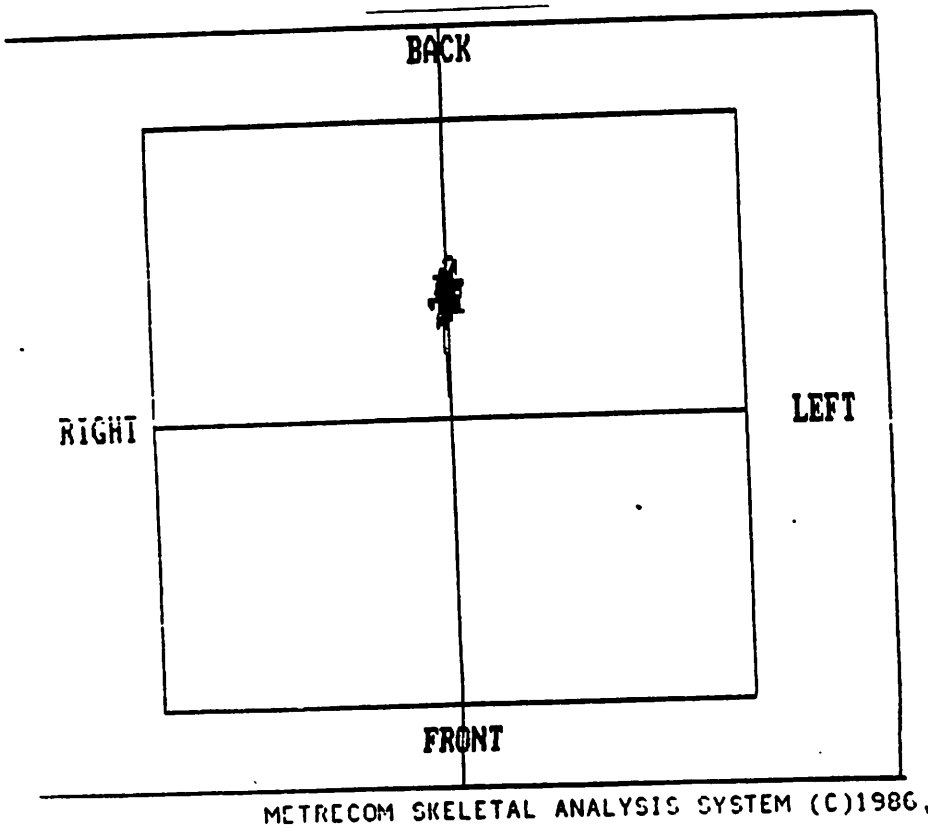


Subject 3
Post-treatment

TLR and Center of Gravity: Preliminary Observations



Subject 4
Pre-treatment



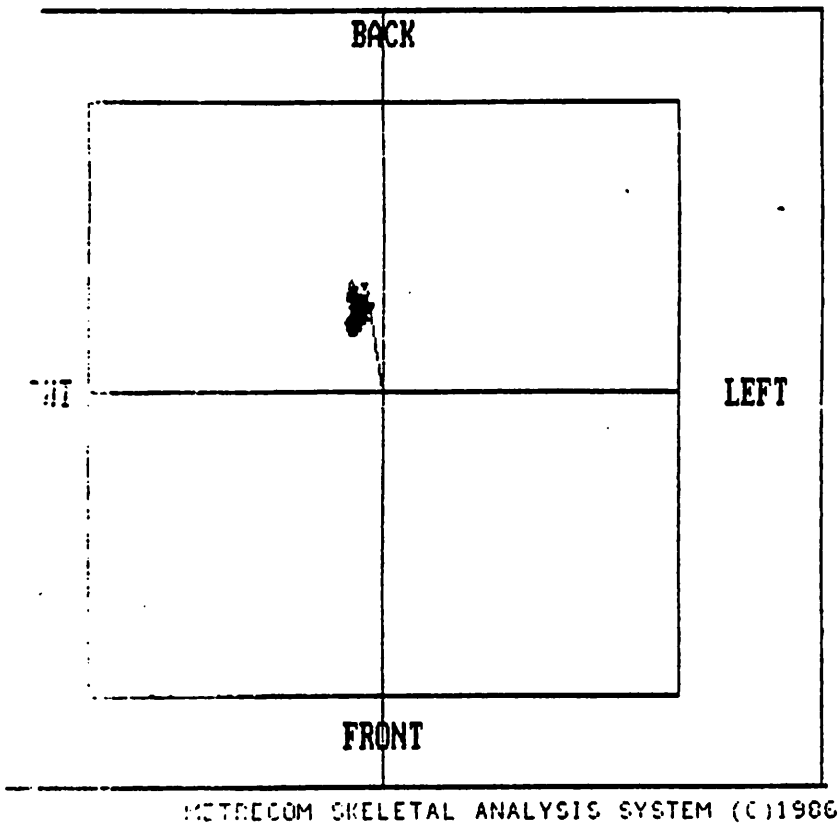
Subject 4
Post-treatment

TLR and Center of Gravity: Preliminary Observations - Mark Force D.C.

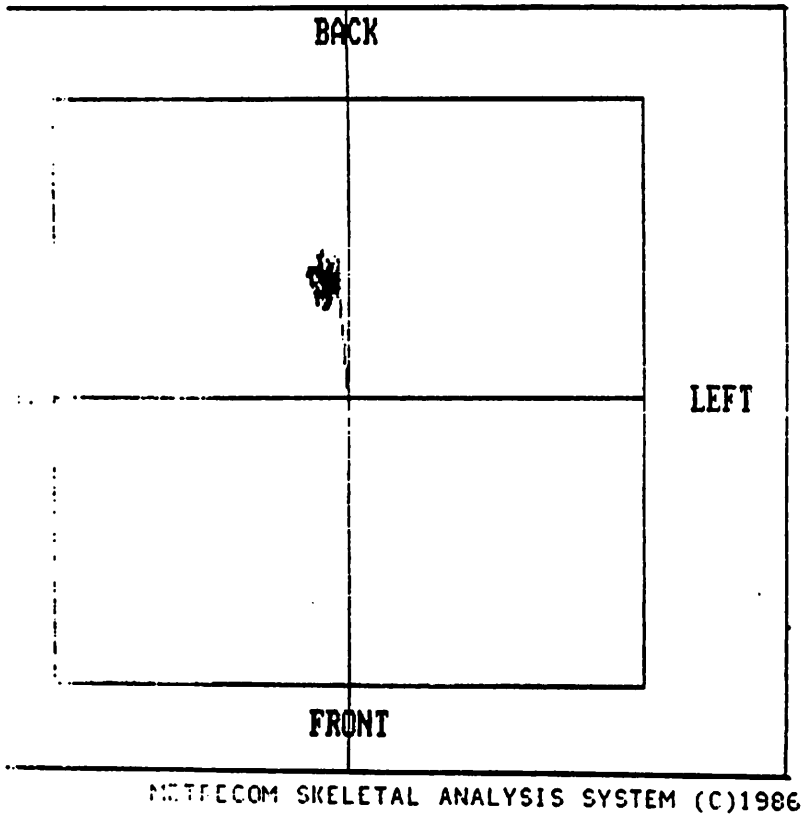
Group IV

Random Response

TLR and Center of Gravity: Preliminary Observations

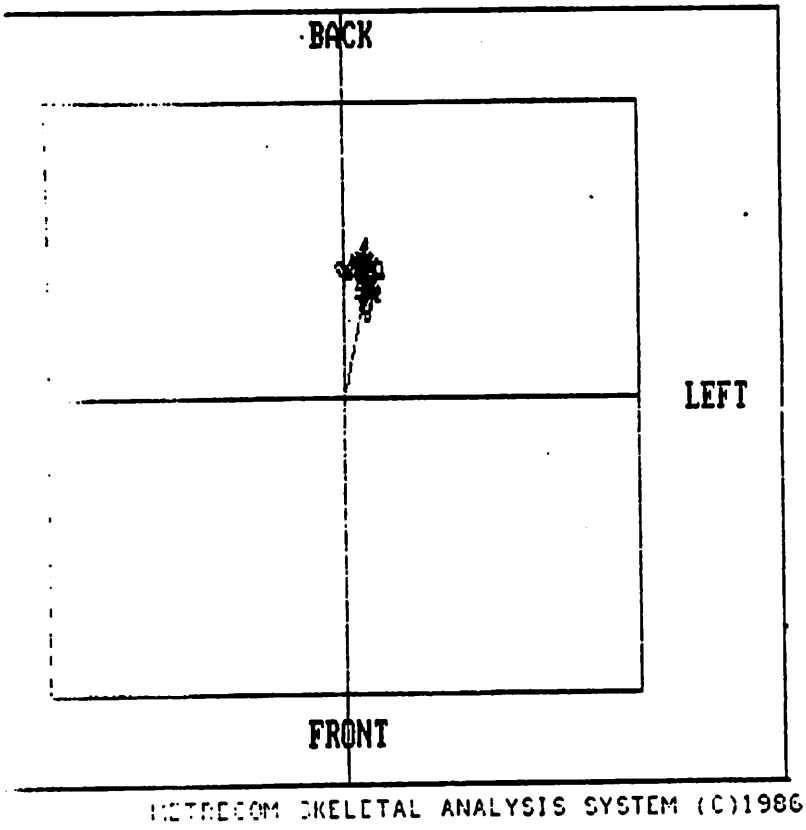


Subject 1
Pre-treatment

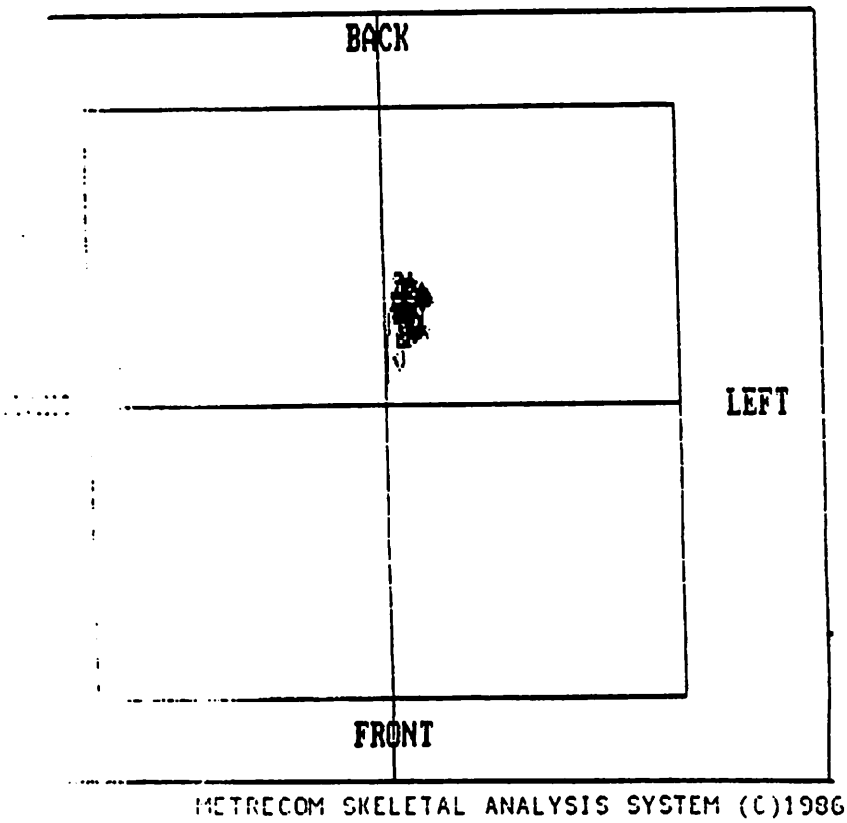


Subject 1
Post-treatment

TLR and Center of Gravity: Preliminary Observations

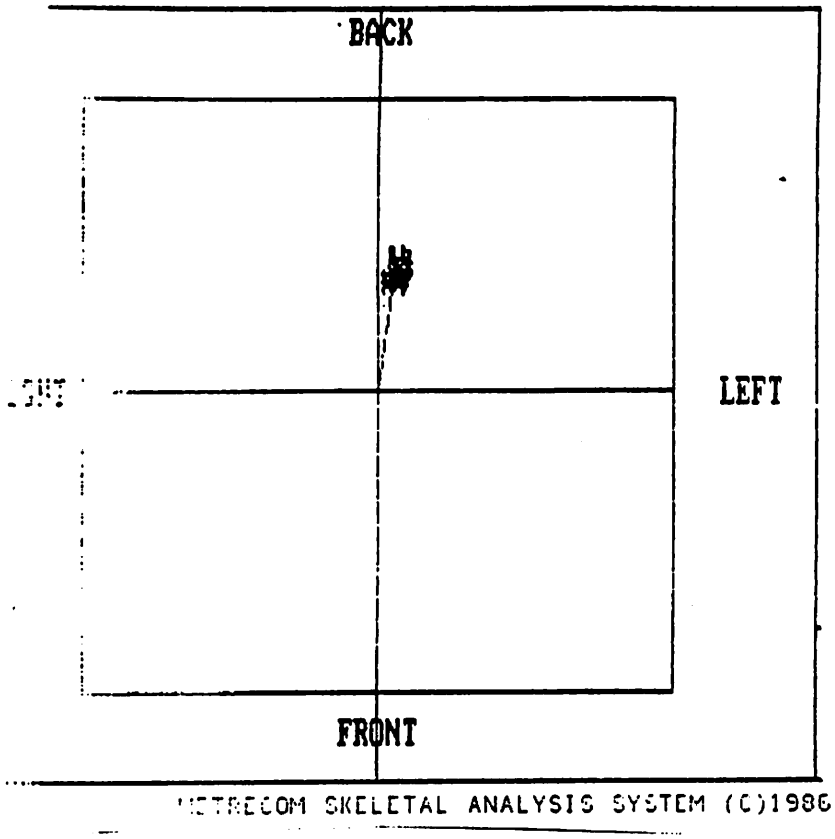


Subject 2
Pre-treatment

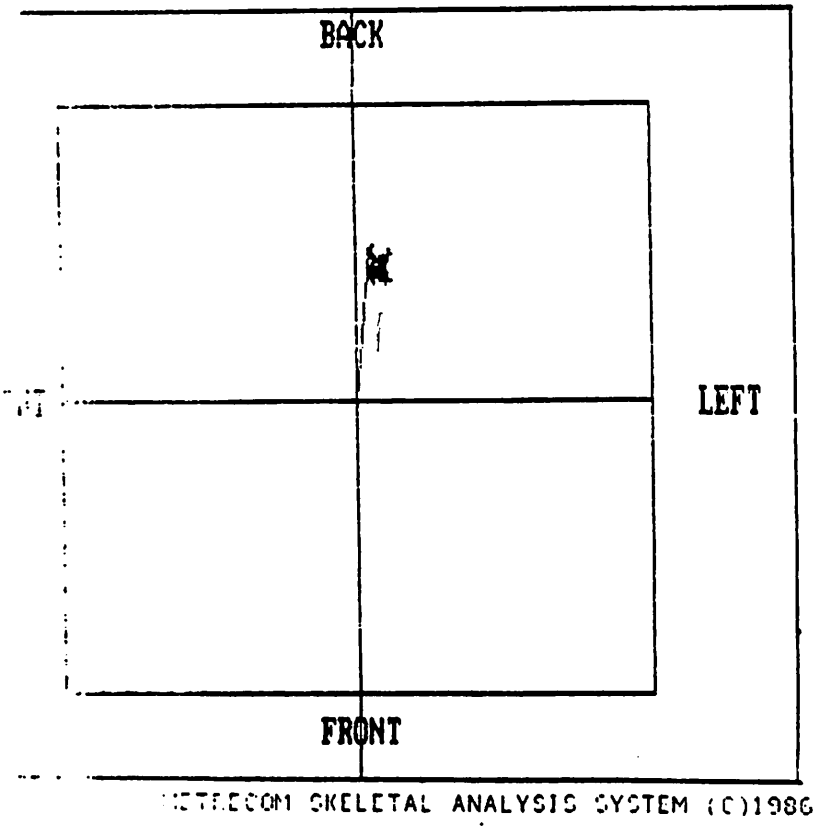


Subject 2
Post-treatment

TLR and Center of Gravity: Preliminary Observations

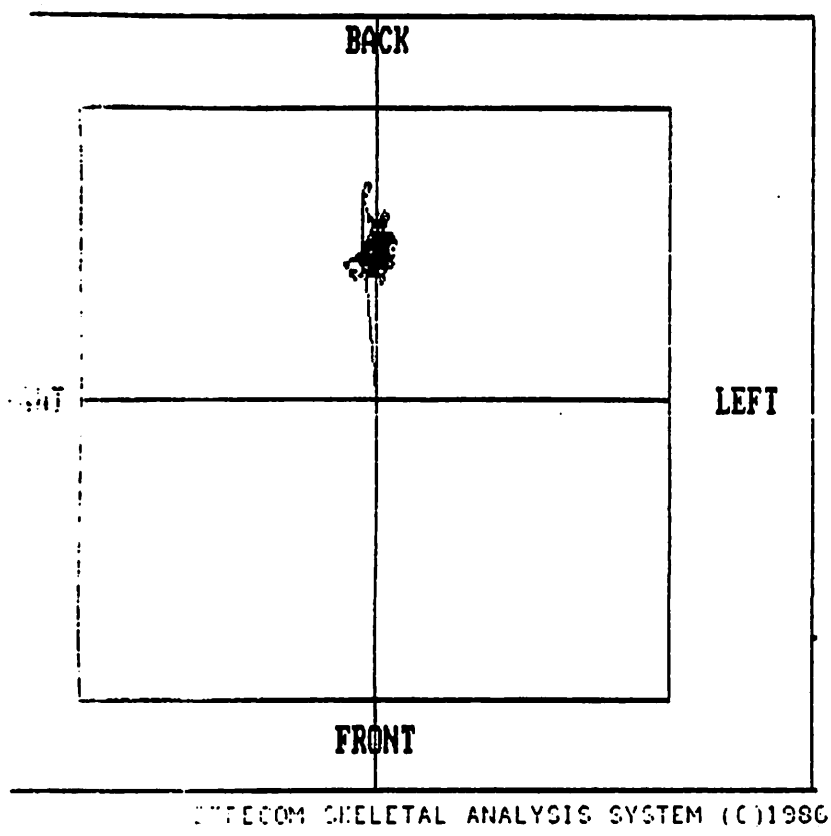


Subject 3
Pre-treatment

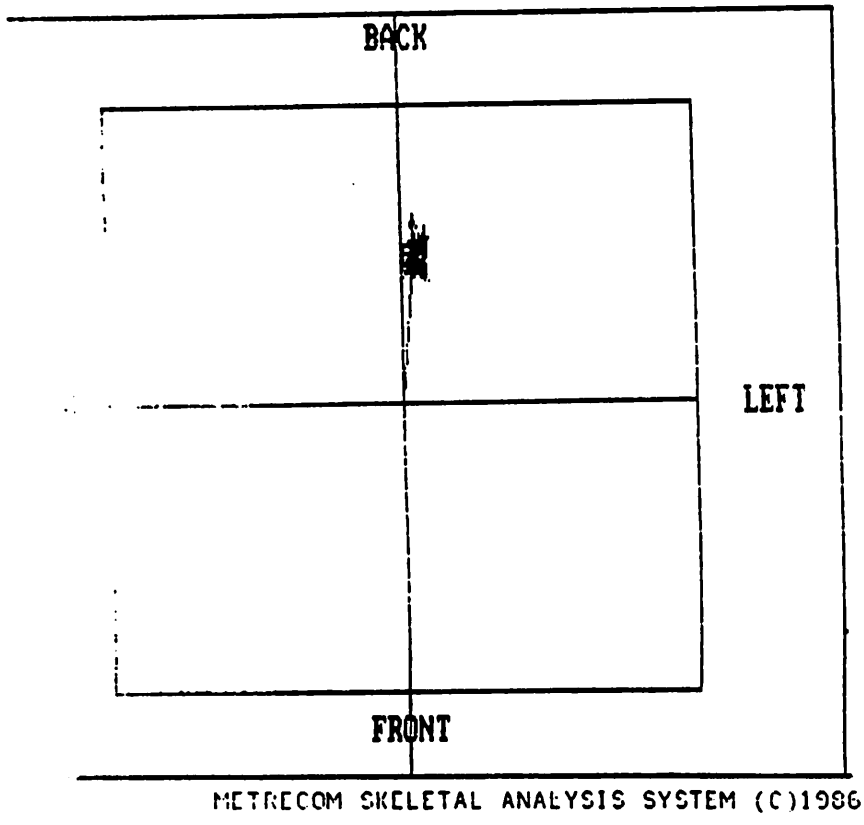


Subject 3
Post-treatment

TLR and Center of Gravity: Preliminary Observations

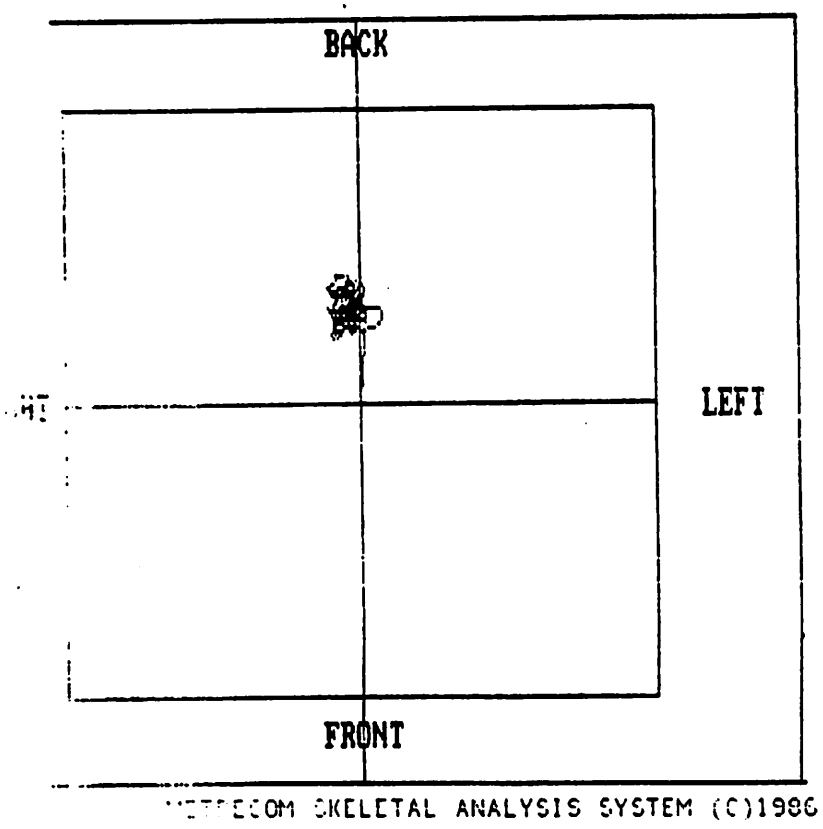


Subject 4
Pre-treatment

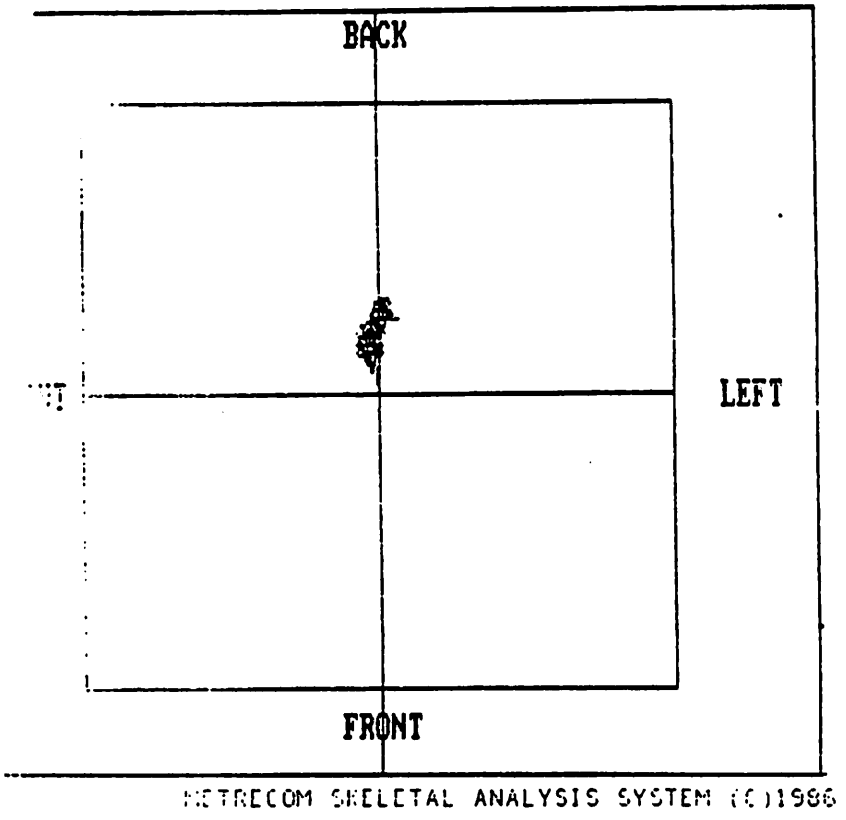


Subject 4
Post-treatment

TLR and Center of Gravity: Preliminary Observations

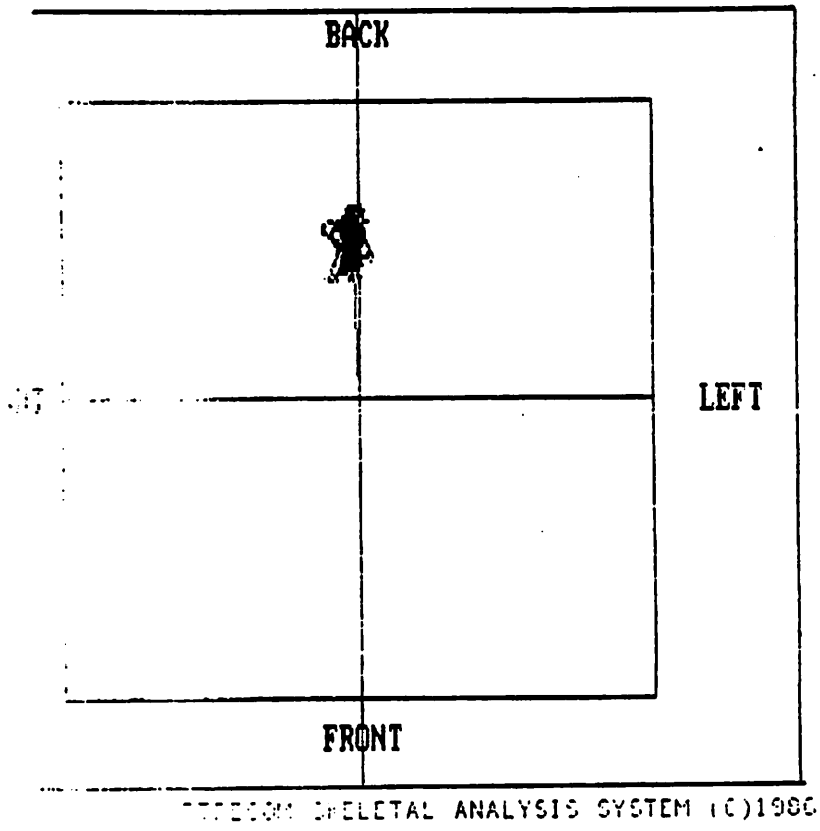


Subject 5
Pre-treatment

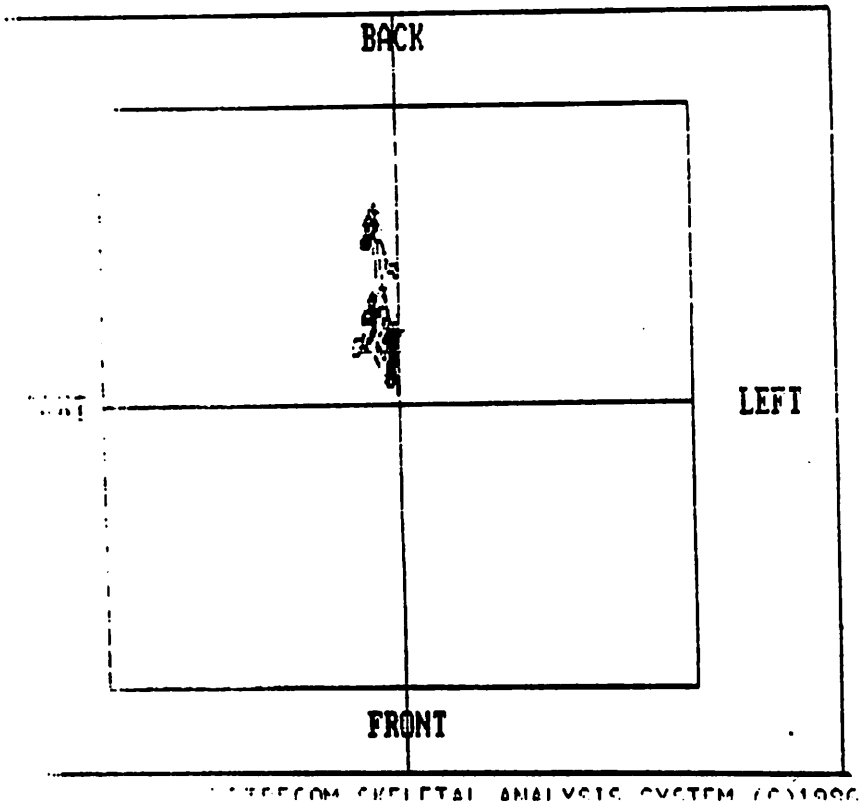


Subject 5
Post-treatment

TLR and Center of Gravity: Preliminary Observations

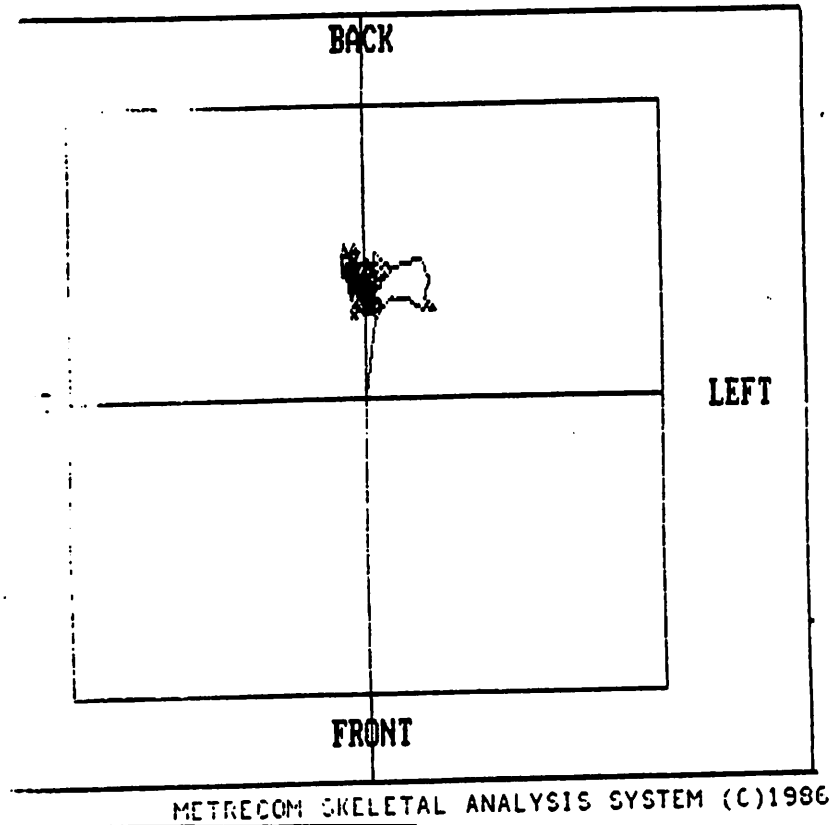


Subject 6
Pre-treatment

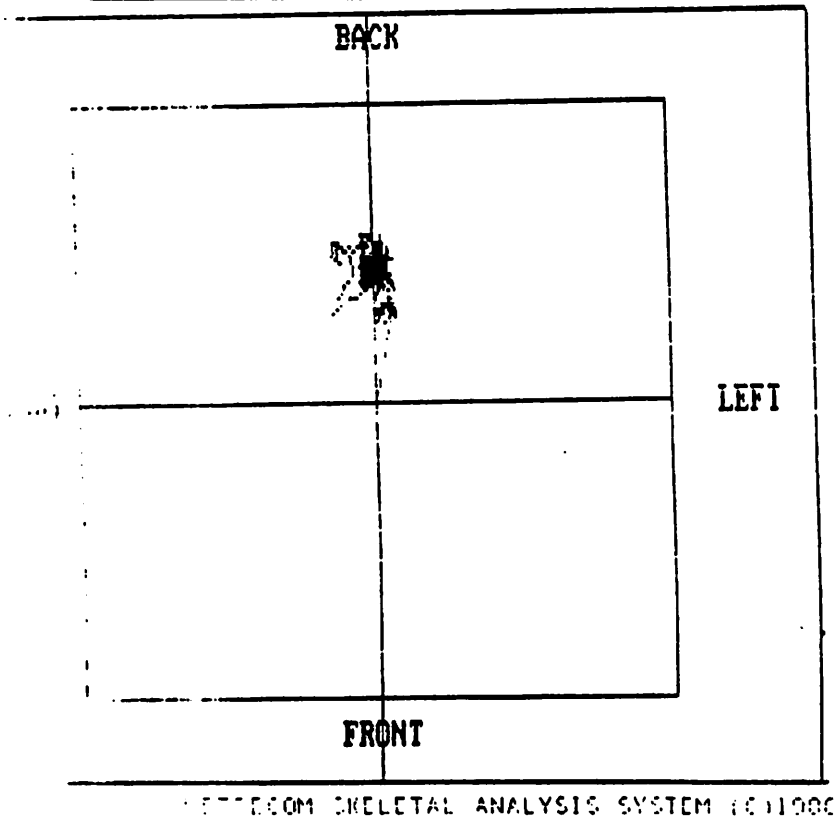


Subject 6
Post-treatment

TLR and Center of Gravity: Preliminary Observations



Subject 7
Pre-treatment



Subject 7
Post-treatment

DISCUSSION

Group I

It is interesting to note that the subjects in this group evidence little deviation from center in the coronal plane. MQWS measurements in this group also show less sway, in general, than the initial MQWS measurements in the other three groups. This may be an indication of the effect of the normally functioning TLR on both COG and sway.

Group II

Change of subjects COG to normal locus plot in this group appears to support the hypothesis that Applied Kinesiology methods as presented by Schmitt optimize TLR function.

Group III

Change of subjects' COG in this group also supports the hypothesis that Applied Kinesiology methods alter TLR function. The initial impression by this author is that this saggital sway may be evidence of a Category I pelvic fault.[21,22,23] Further study is suggested to evaluate the accuracy of this impression.

Group IV

The MQWS measurements in this group indicate an absence of change in COG after TLR correction. A lack of change in COG after TLR correction may be due to no change in TLR function, a fault of transmission of afferent impulses from the TLR through vestibular, vestibulor spinal or other pathways, or a dampening of TLR effects due to aberrant function of other righting reflexes. Further research is recommended to understand the mechanisms affecting response to TLR correction in this group.

CONCLUSIONS

These initial observations demonstrate changes in center of gravity and body sway after application of TLR correction as per Schmitt.[12] These changes support the hypothesis that Applied Kinesiology techniques alter tonic labyrinthine reflex function. More study is needed to establish the consistency of these initial observations and the mechanisms involved.

ACKNOWLEDGMENTS

The author recognizes the tremendous contributions that Drs. Goodheart, Schmitt and Walther have made to the modern healing arts. The author is sincerely and deeply grateful to Dr. Lance West who has been an inspiring mentor and model as physician. To Edward Brennan, the author is thankful for his expertise and efforts in helping to conduct this study.

REFERENCES

[1]Walter H. Schmitt, Using Functional Neurology to Identify Patterns of Facilitation and Inhibitation Arising from Tonic Neck Reflexes and Tonic Labyrinthine Reflexes (Chapel Hill, NC: privately published, 1986).

[2]David Walther, Applied Kinesiology Volume I: Basic Procedures and Muscle Testing (Pueblo, CO: Systems DC, 1981), pp. 190-196.

[3]M. B. Carpenter, H. Fabrega and W. Glinsmann, "Physiological Defcebs Occurring with Lesious of Labyrinth and Vestigal Nuclei," Journal of Neurophysiology, 22:222-234 (1959).

[4]G. P. McCough, I. D. Deering and T. H. Ling, "Location of Receptors for Tonic Neck Reflexes," Journal of Neurophysiology, Vol. 14 (May, 1951).

[5]Leonard A. Cohen, "Role of Eye and Neck Proprioceptive Mechanism in Body Orientation and Motor Coordination," Journal of Neurophysiology, Vol. 24 (January, 1961).

[6]David Walther, Applied Kinesiology Volume I: Basic Procedures and Muscle Testing (Pueblo, CO: Systems DC, 1981), pp. 131-140.

[7]David Walther, Applied Kinesiology Volume II: Head, Neck and Jaw Pain and Dysfunction - The Stomatognathic System (Pueblo, CO: Systems DC, 1983), pp. 515-523.

[8]Ibid., p. 126.

[9]Ibid., p. 188.

[10]Ibid., pp. 512-513.

[11]Walter Schmitt, Tonic Labyrinthine Reflexes in the Weight Bearing Position (Chapel Hill, NC: privately published, 1986).

[12]Walter Schmitt, "Aberrant Firing of the Tonic Labyrinthine Reflexes in the Prone and Supine Position," Proceedings of Summer Meeting, International College of Applied Kinesiology, Detroit (1988).

[13]A. Rozieu, "Determination if the Ideal Weight Distribution Between Heel and Metatorsal Arch in the Human Body," CMCC Research Conference (March 82), Toronto, Canada.

[14]F. Illi, "The Vertebral Column: The Lifeline of the Body," (Chicago National College of Chiropractic, 1957).

TLR and Center of Gravity: Preliminary Observations - Mark Force D.C.

[15]A. Grice, "Posture and Postural Mechanics," Journal of the Canadian Chiropractic Association, (July, 1970).

[16]A. Grice, H. Vernon, "The Four Quadrant Scale: A Technical and Procedural Review," Journal of Manipulative and Physiological Therapeutics, Vol. 7, No. 3, (September, 1984), pp. 165-169.

[17]Alla Zatkin, "Weight Balance and Structural Involvement," (Detroit: Selected Papers of the International College of Applied Kinesiology, 1987), pp. 35-36.

[18]J. P. Moozek, M. R. Wiles, " A Reliability Assessment of Four-Quadrant Weight-Scale Measurements," Journal of the Canadian Chiropractic Association, (1982), 26:97-100.

[19]Information regarding the Metrecom Skeletal Analysis System is available by contacting Faro Medical Technologies, Inc., Montreal, Canada, Telephone 1-800-361-6325.

[20]Walter Schmitt, Muscle Testing as Functional Neurology: Differentiating Functional Upper Motor Neuron and Functional Lower Motor Neuron Problems (Chapel Hill, NC: privately published, 1986).

[21]Major Bertrand DeJarnette, Sacro-Occipital Technique (Nebraska City, NE: privately published, 1979).

[22]David Walter, Applied Kinesiology Volume I: Basic Procedures and Muscle Testing (Pueblo, CO: Systems DC, 1981), pp. 85-90.

[23]David Walther, Applied Kinesiology Volume II: Head, Neck and Jaw Pain and Dysfunction - The Stomatognathic System (Pueblo, CO: Systems DC, 1983), pp. 211-213.

USE OF MYOPULSE IN THE TREATMENT OF MYOFASCIAL ADHESIONS

MARK FORCE, D.C.

ABSTRACT

The use of myopulse therapy appears to be an effective alternative to the standard method of fascial release as presented by Goodheart[1] and Walther[2]. Use of this modality for the treatment of myofascial adhesions as evidenced by an abnormal muscle stretch reaction is more comfortable to the patient than the standard method of therapy, saves time when used by a therapist, and appears to give longer lasting correction.

DISCUSSION

I have been using the Acuscope and Mypopulse instruments for the last few months with very satisfying results. I had stopped using physical therapy in my practice because it's use didn't appear to improve the results beyond Applied Kinesiology techniques alone. However, my frustration in resolving a shoulder injury that I had suffered led me to try Acuscope/Myopulse therapy. The results were very impressive to me with some immediate relief from pain and increase in range of motion. Improvement continued after a single ten minute treatment for two weeks until all pain was gone. This experience prompted me to add the Acuscope/Myopulse[3] system to the methods used in my practice.

The Myopulse has been a useful adjunct to my AK methods for the treatment of musculoskeletal pain and dysfunction. The Myopulse is a microamperage (ua) alternating current stimulator. Myopulse therapy increases muscle strength as evaluated with manual muscle testing and isokinetic-type dynamometry. A double blind study has been conducted to evaluate the effect of Myopulse therapy on the strength of conditioned athletes[4]. Results of this study indicate an average increase in functional strength, after six 30 minute full body treatments, of 13 percent as measured by ability to lift free weights.

Use of Myopulse in the Treatment of Myofascial Adhesions - Mark Force, D.C.

Myopulse therapy is useful in the treatment of myofascial adhesions as diagnosed through abnormal muscle stretch reaction (weakness of a strong muscle after stretching). The standard therapy for myofascial dysfunction is deep kneading massage. Some patients interestingly thrive on this type of attention. Many of my patients begin to wonder why they are paying for what appears to be my pursuit of rather strange pleasures. Myopulse therapy of myofascial adhesions is a painless alternative for this type of patient. The most effective setting in my experience are 100ua at 10 hertz. Abnormal muscle stretch reaction will usually be cleared after one to two minutes of treatment. I find less recurrence of myofascial adhesions after this method of therapy than with the standard manual method. This approach is especially useful when treating the rather sensitive areas of the head, neck, shoulder and groin.

Treatment of muscle spindle cell, golgi tendon organ and origin-insertion dysfunction is also effective with the Myopulse and Myomatic[5]. These uses have been discussed previously by others. I have not found reference to the treatment of myofascial adhesions with Myopulse despite its effectiveness and, so, recommend it for your use.

References

[1]George J. Goodheart, Jr., Applied Kinesiology, 15th ed. (Detroit: privately published, 1979), pp. 77-84.

[2]David S. Walther, Applied Kinesiology: Basic Procedures and Muscle Testing, Pueblo, CO: Systems DC, 1981).

[3]Acuscope/Myopulse are available through Electro-Health, Inc., 3008 No. Dobson, Suite No. 3, Chandler, AZ 85224, (602) 730-1177.

[4]Jack Scott and Robert Picker, A Double Blind Study to Evaluate Muscle Strength in Athletes Treated with the Electro-Myopulse, (Los Angeles, CA: Presented at the Annual Meeting of the International Society of Electro-Acutherapy, 1983).

[5]Myomatic is a microamperage stimulator similar to the Acuscope/Myopulse. Information on this instrument is available through Monad Corp., 460 No. Reservoir St., Pomona, CA 91767, (714) 623-2693.

Pineal Gland Activity in Relationship to Three Common Cranial Faults

James D.W. Hogg, D.C.

Abstract: The cuspal relationship of the upper and lower dental arches create a high probability for the cruciate and symphysis menti cranial faults to occur together. This paper explores the relationship between these faults, the sphenoid compression fault and a stress reaction to darkness which is a common indicator of pineal function. Statistics generated and reported in this paper are taken from a base of 283 patient trials.

In a previous paper by this author[2] a relationship between the cruciate suture and the symphysis menti (also referred to as the mandibular spread) cranial faults was mentioned. Based on conversations with my friend, J. Thomas Howard, D.D.S., a gnathologic orthopedist, it seemed likely that, due to the intermeshing of the cusps of the upper and lower molars, the structural integrity of the upper dental arch would have a profound effect on the structural integrity of the lower dental arch (another instance of "As above, so below"?). Indeed, Dr. Howard frequently makes use of this relationship to widen the lower dental arch by spreading the upper dental arch with various bionator and crozat dental appliances.

The relationship between the sphenoid compression fault and stress reactions to the dark as an indicator of pineal gland function has been well established since it's introduction by Dr. Goodheart in 1980[3]. Schmitt has popularized the mandibular spread as an indirect but very effective method of correcting sphenoid compression/pineal problems as part of his spinal balancing protocol[4].

(Pineal Gland Activity....Hogg)

When I first started using Schmitt's spinal balancing protocol I was very pleased with the improvement in my clinical results but somewhat frustrated with a few patients that experienced recurrence of the symphysis menti fault with it's associated pineal dysfunction. This led, in turn, to insomnia, sleep disturbance, generally poor resting (especially in the dark) and counter-clockwise torquing of the spine. Recalling my conversations with Dr. Howard regarding upper and lower dental arch relationships I began to wonder if an uncorrected cruciate suture fault could be re-creating the symphysis menti fault every time the patient bit down. I started checking for both faults whenever I found either one. I found that most of the time these faults occur together. When I found these faults occurring together, I performed several trials in which I corrected one fault only and then had the patient bite down. Frequently, this would result in cuspal re-creation of the previously corrected fault. Somewhat less frequently this would result in cuspal correction of the fault that had been left uncorrected. I find a generally higher rate of spontaneous correction if the cruciate suture fault is corrected first. This clearly indicated to me an intimate relationship between the two faults on a practical as well as a theoretical level. It seemed to me that if I was correctly interpreting these findings, a statistical study would show these faults occurring together with regularity.

The factors compared in this study were the frequency of the cruciate suture, symphysis menti and sphenoid compression faults as well as stress reactions to the dark. Whenever any one of these were found, all were checked for. The cruciate suture fault was tested for using both a direct[2]

and rebound challenge[3]. The symphysis menti fault was tested for by spreading the mandible apart at the level of the first or second molars, releasing the contact and testing a previously strong muscle for weakening. The sphenoid compression fault was tested for by contacting the greater wings of the sphenoid bilaterally and pressing medially, releasing the contact and testing a previously strong muscle. The stress reaction to darkness was tested in one of two ways. The first 75% of the trials were conducted in an office setting in which I could simply turn off the lights and place the patient in total darkness. In this case I would turn off the lights and test a previously strong muscle for weakening. The last 25% of the trials were completed in my new office which has glass comprising the top four feet of each wall. In this case I placed a folded gown over the patient's eyes, asked them "Is it dark in there?" and if I got an affirmative answer proceeded to test a previously strong muscle for the stress reaction.

The statistical records were used in several different ways. The number of occurrences of each finding were recorded and reported as a total and as a percentage of the total number of trials. These simple totals can lead to false or incomplete interpretations. This is illustrated by the somewhat different or more complete interpretation that can be attained using the comparison statistics that follow. The comparison statistics were arrived at by programming a computer spreadsheet to find each occasion in which, for instance, darkness stress occurred in the absence of a symphysis menti fault. Further formulae were inserted to total these occurrences and to express the totals as a percentage of the 283 trials. Although this may

sound complex, it is actually fairly simple using an "operator friendly" program such as the Appleworks program I use. Although setting up the formulae is time consuming, it is much less so than performing the same functions manually.

One of the interesting results of using the comparison stats is in the relationship of sphenoid compression and symphysis menti faults to darkness stress. In collecting these stats I formed the impression that there was nearly a 1:1 relationship between the sphenoid compression fault and darkness stress and that there was a much greater correlation than between the symphysis menti fault and darkness stress. A quick look at the totals for each finding would seem to support this impression but a more complete picture is yielded by the comparison stats. In fact the rate of occurrence for darkness stress in the absence of sphenoid compression faults (11%) is approximately the same as for darkness stress in the absence of symphysis menti faults (10%) and certainly high enough to rule out the hypothesis of a 1:1 correlation. Using other comparison stats it is easy to see how this initial impression was formed since the rate of sphenoid compression occurring without darkness stress was only 2% while the symphysis menti fault occurred in the absence of darkness stress 22% of the time; a ten-fold difference!

Results of this statistical study are as follows:

Totals out of 283 trials	occurrences	percent of total
cruciate suture:	262	93
symphysis menti:	242	86
sphenoid compression:	186	66
darkness stress:	190	67
Darkness stress w/o cruciate suture or symphysis menti:	5	2
Darkness stress w/o cruciate suture:	8	3
darkness stress w/o symphysis menti:	10	4
symphysis menti w/o darkness stress:	62	22
darkness stress w/o sphenoid compression:	11	4
sphenoid compression fault w/o darkness str:	7	2
darkness stress w/o sphenoid compression or symphysis menti:	2	1
sphenoid compression w/o cruciate suture or symphysis menti:	5	2
cruciate suture w/o symphysis menti:	34	12
symphysis menti w/o cruciate suture fault:	14	5
symphysis menti fault with cruciate suture:	228	81

Conclusions

These statistics indicate a frequency of occurrence between the symphysis menti and cruciate suture faults which must be considered significant at 81%. The cruciate suture fault was the most common finding, occurring with 93% frequency with the symphysis menti fault second at 86%. The symphysis menti fault was less than half as likely to occur without cruciate suture fault than vice-versa. While the incidence of darkness stress without either cruciate suture or symphysis menti faults was very small (2%), it is apparent that the symphysis menti fault may frequently be present without darkness stress being exhibited (22% of the time). By comparison the sphenoid compression fault rarely occurred without darkness stress (2% of the time). The occurrence of the sphenoid compression fault without either cruciate suture or symphysis menti was also very rare (2%). Darkness stress occurred without either symphysis menti or sphenoid compression faults only twice out of 283 trials. I feel that this number has small significance statistically and may reflect either operator or recording error.

Summary and clinical applications

Since the theory that cuspal relationships tend to cause the cruciate suture and symphysis menti faults to occur together is supported statistically, it is reasonable to make a habit of checking for both whenever either is present. The frequency of occurrence for the cruciate suture makes it especially important to use either a direct [2] or indirect

challenge [3] rather than relying on therapy localization [1]. Since darkness stress rarely occurs without either sphenoid compression or symphysis menti faults and since the sphenoid compression fault rarely occurs without the symphysis menti fault it is important to check for the presence of sphenoid compression, symphysis menti and cruciate suture faults whenever there is evidence of darkness stress as demonstrated by manual muscle testing. In actual practice I have found it useful to check for the cruciate suture and symphysis menti faults first and only to look for the sphenoid compression fault if these two are absent (or, of course, if I'm collecting statistics). Usually correction of the symphysis menti and/or cruciate suture faults will correct any sphenoid compression fault although I have no statistics on rate of correction at this time. In spinal centering considerations indicated by a weak gamma 2 [5] muscle strengthening on a clockwise pelvic torque (left leg flexed at the hip) the cruciate suture fault should be checked for in addition to the symphysis menti fault.

The pioneering work of Frank Barr, M.D. as presented to us by Dr. Goodheart at the Summer 1988 meeting and in research tape #109 promises to change the way we deal with body chemistry. The pineal gland is emerging as the primary regulator of many metabolic processes. In light (or perhaps I should say "in dark") of this, the relationship of the symphysis menti, cruciate suture and sphenoid compression crainial faults to pineal function assumes even greater clinical importance.

References:

1. "The Frequency of Three Common Cranial Faults" by James D.W. Hogg, D.C. Collected Papers of the Members of the International College Of Applied Kinesiology Winter, 1987 published by I.C.A.K. Shawnee Mission, KS.

2. "Another Diagnostic Approach to the Cruciate Suture Fault: Applications for Spinal Centering" by James D.W. Hogg, D.C. Collected Papers of the Members of the International College Of Applied Kinesiology Winter, 1987 published by I.C.A.K. Shawnee Mission, KS.

3. 1980 Workshop Procedure Manual by George Goodheart, D.C. privately published.

4. "Centering the Spine; Functional Neurological and Biochemical Considerations" by Walter Schmitt, D.C., Collected Papers of the Members of the International College Of Applied Kinesiology Summer 1987 published by I.C.A.K. Shawnee Mission, KS.

5. "Muscle Testing as Functional Neurology. Differentiating Functional Upper Motorneuron Lesions and Functional Lower Motorneuron Lesions" by Walter Schmitt, D.C. Collected Papers of the Members of the ICAK, Winter 1985.

Determination and Convincing Procedure for a General Foot Problem Screen

by

Thomas J. Kalis, D.C.

Abstract

This paper will deal with a general procedure to determine whether or not a patient has a foot problem. It also is a physical demonstration to the patient that a foot problem exists.

Introduction

This procedure is part of my initial exam. It is usually the second demonstration to the patient of what muscle testing can discover about their body. It is used by me for two reasons: 1. to discover a possible foot problem and 2. to impress the patient with what muscle testing can inform us, the patient and the doctor, about the patient's feet.

Procedure

The patient is gowned and is standing before and facing a large mirror (so that the patient can watch and receive instructions easier from the doctor). The lower legs and feet are without any coverings so that a visual and palpatory exam can be made. Notes are recorded and the patient's attention is brought to focus upon their feet.

Now individual deltoid muscle tests are performed. If these

muscle tests demonstrate strength then it is "generally" assumed that the feet are serving the patient at least adequately. However, if there is a weakness in the muscle testing the following steps are performed:

1. The patient is asked to sit and the individual deltoid muscle tests are performed. If weakness is obtained as in standing then the deltoids must be investigated. If however, strength in muscle is observed then you have generally found a foot/feet problem.
2. This step is used only for a very "skeptical" patient who is having a hard time accepting muscle testing. Have the patient kneel and perform the deltoid tests again. Usually you will find the same strength in muscle tests as was found sitting.
3. All during the testing the only verbal communications are the instructions about the muscle testing and patient position. Now, to emphasize the change in muscle strength with the change in body position, I comment that "if you have a disagreement with someone, sit down because you are stronger sitting than standing". The patient will respond with some laughter or at least a puzzled smile. Instruct the patient to stand and place scaphoid pads (commonly called cookies) under the arch of their foot and test the deltoid muscles again. This will almost always cause the muscle test not to show any changes. Now instruct the patient to step forward and off the scaphoid pads and test

the deltoids again. Usually there will be an even more dramatic change in muscle strength.

At this time I discuss with the patient the importance of structure, and how I and Chiropractic will resolve the foot/feet problem. This entire procedure will take no longer than 2 or 3 minutes.

Conclusion

By demonstrating to the patient the change in muscle strength you gain:

1. The attention of the patient.
2. An increase in the patient's belief in muscle testing.
3. Their respect of your ability to quickly and accurately focus in on their problem/problems.
4. The confidence and trust of the patient.

It is my desire that this sharing of this procedure will help you help your patients to be relieved of some of their pain and discomfort.

Thomas J. Kalis, D. C.
9833 Reeck Road
Allen Park, Mich. 48101
October 11, 1988

A Practical Physical Evaluation Of The Foot/Feet

by
Thomas J. Kalis, D. C.

Abstract

The following evaluation will give the doctor a quick and efficient method to resolve a great majority of the foot problems that you are confronted with. It also is a good teaching tool for use with patients. They are impressed with the treatment because no one else has ever treated their feet in this manner and obtained the relief they will experience.

Introduction

There are usually 26 bones and 35 articulation in one human foot. In both feet the number of bones represent approx. 25% of all the bones in the body. The bones and articulations are stressed each time we walk and or run. With all these bones and articulations it is important to have a "quick" general screen to evaluate and correct the foot. This general screen of the feet should be able to be accomplished in about 4-5 minutes.

Procedure

Test, challange and or correct the following:

Muscles:
1. Anterior Tibialis
2. Posterior Tibialis
3. Peroneus Tertius
4. Peroneus Longus and Brevis

Bones:
1. Calcaneus (anterior and posterior @)
2. Cuboid (lateral and inferior @)

3. Navicular (inferior @)

4. Talus (superior, inferior, lateral and medial @)

@ These are only suggested directions. For best correction find the best vector for the correction. A Chiropractor mallot/activator can be a very helpful tool in adjusting small bones of the feet.

Massage (b): 1. The plantar surface of the foot.

2. The medial surface of the foot.

3. Also the muscles noted above.

(b) This massage (use a lubricant) should be a deep massage so that it will stimulate the origins and insertions of as many muscles in these areas as possible.

Comments

This method has been and is today under constant evaluation for improvement. This method is the result of approx. 6 years of constant reworking.

Conclusion

Using this method I have been able to resolve approx. 95% of all the foot problems that are present to me.

It is my hope that this evaluation can help you with the foot problems that are presented to you.

Thomas J. Kalis, D.C.
9833 Reeck Road
Allen Park, Mich. 48101

October 11, 1988

SPECIFIC POSTURES AND THEIR RELATIONSHIP TO DR. JOHN DIAMOND'S MERIDIAN AFFIRMATIONS

BY DAVID A. KUBICEK, D.C.

ABSTRACT

The correlation of specific habitual postures to the recurrence of the meridian imbalance related to the emotional affirmations of Dr. John Diamond[1] is discussed.

INTRODUCTION

The knowledge of Body Language is not something new. It is something man has instinctively or innately known since his creation or evolution. A person can posturally express their emotional state without uttering a word. One does not have to speak for others to know that they are frightened, happy, sad etc... When man experiences a specific emotion he assumes a specific universal posture that expresses that emotion outwardly. The posture which may include facial expressions is universal or common to all men. This is true due to the fact that all men have the ability to identify the same emotion in a given individual. For example, both Tom and Dick know when Harry is angry by the posture he assumes. The reason for this lies in the conscious recollection or usually unconscious association of the same posture in connection with the same emotion in oneself.

This demonstrates the intimacy that the emotions have with posture and therefore muscular activity which makes up that posture.

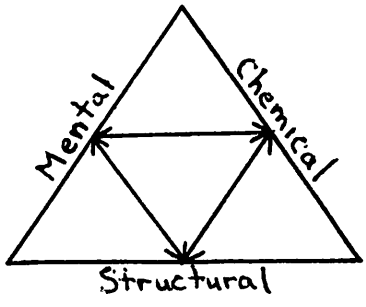
In an ancient psychological or esoteric teaching described by authors Ouspensky, Gurdjieff, and others there is discussed the seven functions or centers of man.[2] These functions or centers are the following:

1. The Intellectual Center - Man's thoughts.
2. The Emotional Center - Man's emotions.
3. The Instinctive Center - Those processes that maintain the life of an individual (example: respiration, digestion, elimination, etc...).
4. The Moving Center - Muscular activity not present at birth, or learned activity (example: walking, writing, riding a bicycle, etc...).
5. The Sex Center - The ability to perpetuate the species.
6. The Higher Emotional and 7. Intellectual Centers - Those experiences only realized in higher states of consciousness.

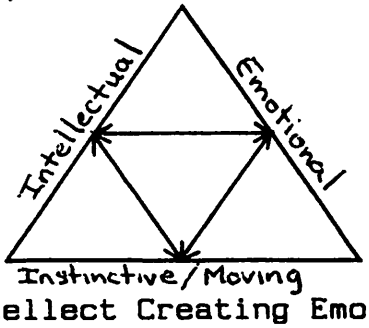
The last 3 centers, Sex, Higher Emotional and Higher Intellectual are not significant in regards to this paper, so will not be discussed. For simplicity and because of the organ /muscle relationship, the Instinctive and Moving centers will be combined to form one center which we will appropriately call the Instinctive/Moving center. So, out of the seven we will concern ourselves with 3, the Intellectual, Emotional and Instinctive/Moving centers. All of the centers are said to have separate concentrations, focus of origin, or "brains" located in different parts of the body. Dr. Goodheart, when discussing the Pre and Post Cordial Tap, speculated that the

heart acted like a second "brain". If the theory regarding the seven functions are correct then there are actually seven "brains" and they are not all located in the head. The Instinctive/Moving center is said to be located in the spine[3]. If this is true, then any weakness caused by a specific posture must be of a gamma 1 nature[4].

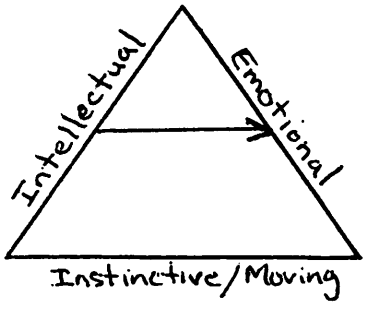
The triad of health is one of A.K.'s main principles. It basically states that man's health is dependent on the interrelationship of the Mental, Chemical and Structural sides of man, and how any one of these sides can effect or influence the others.



The three centers are analogous to the triad of health in that there is an interrelationship between the centers where any one of them may effect, influence or create the others.

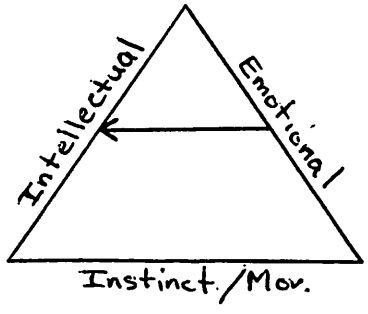


Intellect Creating Emotion



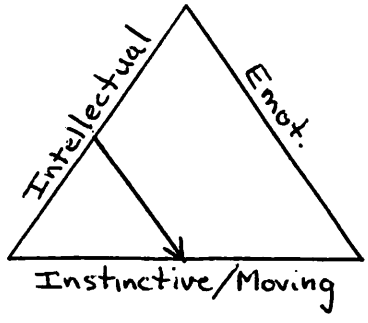
For example: Suppose you are angry with a certain person, until that emotion is replaced by a stronger one, when that persons name is mentioned or brought to your attention (thought), you will most likely, if only temporarily, associatively *become* angry. Or when a couple hears "their song" (thought) they often associatively reminisce or experience a blissful emotional state regarding their young and growing love for one another.

Emotion Creating Thought



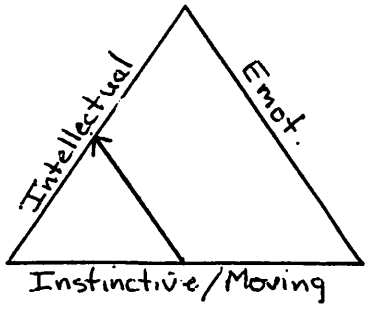
For Example: Again suppose you are extremely angry at a particular individual. That anger may create thoughts about ways of getting even with that individual.

Intellect Creating Movement



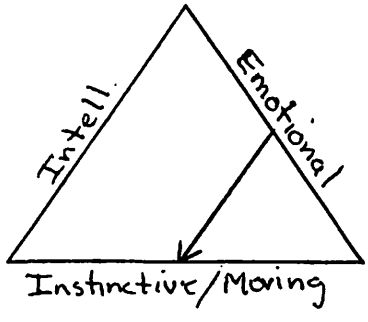
For Example: Suppose a person has a mental image of something he wants to produce. He can intellectually direct his muscles to create that product.

Movement or Posture Creating Thought

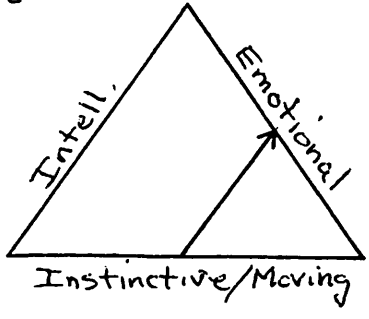


An unusual posture when assumed, may associatively produce the thought of a situation in which that person had assumed that posture before. For example: suppose a person was being extremely confined physically in an overly crowded bus and had to assume a very tight and uncomfortable posture, that posture may associatively produce the thought of an overly crowded elevator in which that person had assumed the same posture earlier.

We have already discussed how Emotions create movement (posture).



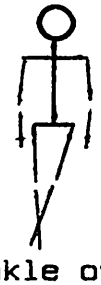
If all of these interrelationships are true, then posture or the Instinctive /Moving Center must be able to create emotions.



Therefore certain postures that are habitually maintained may recreate the meridian imbalance related to the emotional affirmations of Dr. Diamond. After working with Dr. Diamond's meridian affirmations for sometime and knowing of the information regarding the centers, this author decided to try and verify the information clinically by determining if specific postures were associated with the different meridians and their emotions. After a lot of trial and error I finally managed to identify a specific posture for all twelve of the bilateral meridians. If the patient is allowed to continue the habitual posture he will inevitably recreate the emotional imbalance in that meridian. The posture involved is almost always the one the patient admits to assuming often.

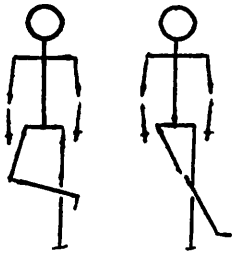
Posture and Their Meridians

Liver Meridian



L ankle on R ankle

Gall Bladder Meridian

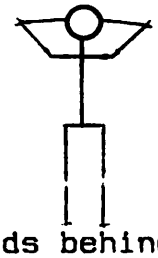


R ankle on L knee
or
R knee on L knee

Spleen Meridian

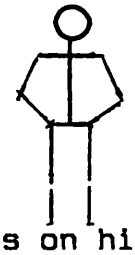
arms crossed

Stomach Meridian



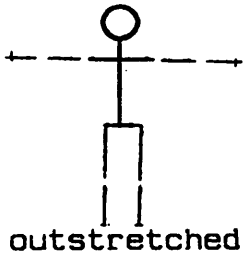
hands behind head

Kidney Meridian



hands on hips

Bladder Meridian

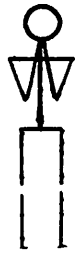


arms outstretched
over the back of
a couch or chair

Lung Meridian

hands together or crossed in front

Large Intestine Meridian



hands under chin

Heart Meridian

hands together or crossed behind

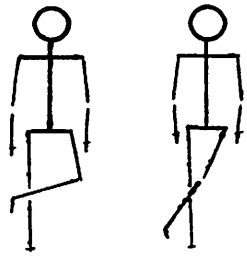
Small Intestine Merid.

R ankle on L ankle

Thyroid Merid.

hands in pockets

Circulation-Sex Merid.



L ankle on R knee
or
L knee on R knee

Materials and Methods

This is basically, with only a few alterations or additions, the procedure described by Dr. Goodheart in one of his research tapes.[5]

1. Identify the meridian involved by checking the pulse points against lips closed and separated. If there is an emotional involvement with a meridian the pulse points will only test positive when the lips are separated. When you find the positive pulse point, go to the muscles associated with the two meridians at that point and test those muscles with lips closed and separated. The positive meridian will show an associated muscle weakness with the lips separated only. The muscle weakness may be bilateral or unilateral. You may also

have the patient T.L. to the alarm points of the involved pulse point with the lips closed and separated and test a strong indicator for weakening. The involved alarm point will weaken the indicator with the lips separated only. Have the patient verbalize the positive emotional affirmations associated with the involved meridian and test it against a strong indicator. If the affirmations are correct they will weaken the indicator.

2. Test a strong indicator against the posture associated with the involved meridian. Interestingly, the indicator will weaken with gamma 1 testing only, which represents a spinal level problem supporting the theory that the "brain" of the Instinctive/Moving center is not in the head. 3. Correction: Tap along the patient's sternum, for approximately 1 minute (to stimulate the thymus), while he verbalizes the positive affirmations. Evaluate the vertebra at the meridians associated point for a subluxation and/or holographic subluxation and correct accordingly. Correction is complete not when the pulse points are clear with lips separated and not when the affirmations no longer create a weakness, but when the posture no longer creates a weakness. At this point the patient will no longer weaken to the posture or the affirmation if performed separately, however if the patient assumes the posture and states the affirmations simultaneously he will create a weakness. This step is simply to educate the patient to the importance of avoiding the posture and helps to verify the contents of this paper.

Results and Conclusions

Clinical trials have supported the contents of this paper and have significantly decreased the number of recurrent meridian imbalances associated with emotional etiologies.

REFERENCES

1. Goodheart, George J., Research Tape # 93-2-400

2. Ouspensky, P.D., The Psychology of Man's Possible Evolution. New York: Vintage 1974 pgs. 23-30.

3. Ibid. page 76.

4. Schmitt, Walter H. Jr., Muscle Testing as Functional Neurology Differentiating Functional Upper Motorneuron and Functional Lower Motorneuron Problems. I.C.A.K. Collected Papers, Winter 1985.

5. Goodheart, George J., Research Tape # 93-2-400

THE ELIMINATION OF NEUROMUSCULAR HYPERSENSITIVITY TO WHOLE FOODS

by DAVID A. KUBICEK, D.C.

ABSTRACT

A procedure for the elimination of Neuromuscular Hypersensitivity to whole foods is discussed.

INTRODUCTION

There are many things that must be considered when trying to eliminate a persons sensitivity to foods. One of those is the genealogical background and therefore cultural diet of the individual. For example, is it possible to desensitize a pure, genealogically speaking, oriental patient to dairy products? Culturally, Orientals generally do not consume dairy, (when was the last time you saw a dish containing cheese at a Chinese restaurant), so the sensitivity may be one of genetics and not a health problem per se. On the other hand, if a patient with a Northern European background were sensitive to dairy then that may be a legitimate health problem, in that genetically that patient should be able to tolerate and actually thrive on dairy, because it is one of Northern Europes main staples. Other examples of inconsistencies may include; American Indians sensitive to corn, the Irish sensitive to potatoes, Orientals

sensitive to rice, etc... All of which are staples for that particular nationality. This paper will deal with neuromuscular hypersensitivity as it relates to a patients sensitivity to whole foods, and not the depletion and over concentration reaction involved with processed or refined foods. The reaction to refined foods (white sugar, white flour, etc...) is not so much a sensitivity, as it is a depletion of the bodies stored nutrients that are removed in the refining process. Not only does the refining process remove most of the nutrients, but it also removes most of the bulk which increases the foods concentration way beyond what the body would normally be able to consume. Natural whole foods contain all of the vitamins and minerals that our bodies need to metabolize them.

This author has not attempted to use this desensitization procedure on the depletion reaction of refined foods because idealistically I feel that people should not consume refined foods any more then they should Strychnine. Refined foods do not exist in nature and should ideally not exist in ones home. Since complete abstinence of refined foods from the typical patient diet is unrealistic, this procedure may have to be used in clinical trials. However, probably a more effective approach to the inevitable depletion reaction would be to research what nutrients are removed in the refining process of that food and supply those nutrients in proportion to the amount of the refined food ingested.

In this paper, sensitivities to foods refers to the health problems that occur when an individual consumes a natural whole food that they should genetically be able to tolerate, if not thrive upon.

The difference between the sensitivity reaction and the depletion reaction is that the former is caused by a nutritional deficiency where as the latter causes a nutrient deficiency.

This author feels that an individual is sensitive to a food because he does not possess all of the nutrients required in the different biochemical pathways involved with the metabolism of that food. These deficiencies may be caused by poor dietary habits (consumption of refined foods) or a genetic predisposition that requires more of a specific nutrient.

The depletion reaction may be dealt with by eliminating refined foods from the diet. And even though the sensitive food may also be eliminated the nutritional deficiencies will still remain causing a variety of health problems. These functional problems may in reality be only exaggerated and not really caused by the offensive food. In essence, we may actually use the sensitivity as a tool to determine the deficiencies in an underlying functional disorder. This desensitization procedure is identical to the procedure this author uses to eliminate the cross K-27 form of neurological disorganization; henceforth known as N.D.. The original procedure has one addition that will be noted in the Materials and Methods section of this paper.

This authors original paper on N.D. will be summarized to help you understand the rational for this approach.[1]

In Dr. Blaich's A.K. and Human Performance paper[2], he discussed how when an individual was pushed beyond his comfort zone or ability to handle some form of stress, physical, chemical or emotional, that individual would show signs of N.D. in the forms of cross K-27 and/or K-27. The examples described were elite bicyclists trying to improve their performance past a certain plateau, and individuals who were asked to read twice as fast as their comfortable reading rate. In other words, if the stress, regardless of its form, (bicyclists attempting to improve their performance, increased reading rate, physical illness, emotional trauma, or sensitivity to foods) is more than the body can tolerate, it will almost always create N.D. in the form of cross K-27. If we can then determine what abolishes the cross K-27 we can identify the bodies deficiencies in its ability to handle that stress. Once these deficiencies are identified and corrected, the body should then be able to ride faster and longer, read faster with better comprehension, fight the infection, deal with the emotional problem, or metabolize foods properly.

One of the main principles in all healing professions is that the body heals itself. This process works automatically and continuously unless some form of stress overwhelms this process creating dysfunction or disease. The N.D. is what interferes with the bodies ability to heal itself. Its as if the confused nervous system is constantly trying to put a round peg in a

square hole. It has trouble sorting out the information coming in and delegating the information going out. When the N.D. is eliminated, the body can then begin to heal itself with the help that you provide.

Before desensitization can be performed, make sure the patient shows no signs of N.D. in the forms of cross K-27 and/or K-27, before insalivating the offensive food.

If the cross K-27 is positive or they weaken to the cross crawl, then the procedure described in this authors original paper on N.D. and slightly modified in this paper must be performed to eliminate the cross K-27.

When the patient is negative to the cross K-27, you may begin the desensitization procedure.

First determine the offensive food by having the patient insalivate the suspected food and test against a strong indicator associated with some phase of digestion. The muscles preferred by this author are the bilateral Pec. Claviculars.

The suspected foods may be brought in by the patient or the Doctor may prefer to use the Neuromuscular Hypersensitivity kit made available by Dr. Schmitt.

If the patient is sensitive to the food, it will not only weaken the bilateral pecs, but will also cause the patient to weaken to the cross crawl, identifying the cross K-27 form of N.D.. The food actually causes N.D.. Anything that abolishes or eliminates the N.D. will identify the deficiencies that keep the

body from metabolizing the food properly.

Determine the genealogy of the patient at least as far back as their parents, to determine if they are genetically predisposed to tolerate the food. Cultural diets may be found in the Encyclopedia.

MATERIALS AND METHODS

1. Have the patient insalivate the suspected food and test against strong bilateral pecs. If this creates a weakness have the patient perform the cross crawl for about 10 cycles. If they are sensitive to the food a strong indicator will now weaken to both gamma 1 and gamma 2 testing.

   Note: The food must be insalivated during this entire procedure, even when testing or insalivating nutrients

2. While the patient is weak to the cross crawl have him/her T.L. over the glabella (Hypothalamus/pituitary), thyroid/parathyroid, thymus, liver N.L., pancreas/spleen N.L. adrenal N.L., reproductive N.L., and any other organ system that may be involved according to the patients symptoms. One of the reflexes will abolish the weakness. This identifies the organ/gland system that the offensive food is effecting the most. Remove the T.L., so the indicator is again weak, and challenge it against different phases of respiration. When you find the phase of respiration that

abolishes the weakness, correct the cranial fault associated with it to abolish the generalized weakness created by the cross crawl. If phases of respiration do not abolish the weakness, check for a Frontal Fault involvement with T.L.. When the indicator weakness (generalized weakness) is abolished, evaluate the muscles associated with the organ/gland system that abolished the cross crawl weakness for a Gamma II weakness. For example, if therapy localizing over the Thyroid/Parathyroid glands abolishes the weakness evaluate both Teres Minor and Levator Scapulas respectively to determine which one is at fault. The Gamma II weakness may be bilateral or unilateral, but it will always be present.

Note: The Gamma II used, must be the one associated with the cross crawl weakness, and not just any Gamma II found on the T.S.Line.

3. Using the above Gamma II weakness evaluate it with the procedure described in this authors original paper on N.D., with the following addition. Between steps K and L insert the following: Have the patient Pre-Test Image (P.T.I.)[3] the Gamma II muscle test. If positive, determine the cranial fault associated with it (usually Nasosphenoidal or Frontals), and correct it at the end of the entire procedure.

If rebreathing is also positive, correct the above cranial fault while rebreathing.

Complete all of the steps, determine the nutrients involved and perform all of the structural corrections.

At this point, the bilateral pecs will still be weak and the patient will still weaken to the cross crawl, while insalivating the food. Have the patient insalivate all of the positive nutrients with the food and retest. The pecs. will now be strong and the patient will no longer weaken to the cross crawl. All of the positive nutrients must be insalivated simultaneously or the weakness will not be abolished. Remove only one, any one, and the weakness will remain. If the nutrients do not abolish the weakness, you have missed a step, and must go back over the previously negative steps and re-evaluate.

Supply the positive nutrients and retest in one week. The patient should no longer be sensitive to that food. Have the patient continue the supplements until they begin to weaken them.

RESULTS AND CONCLUSIONS

The sensitivity reaction is a very individualistic experience. Almost every patient tested, has been sensitive to whole wheat, but there seems to be no common organ/gland effected or nutrient involved. Wheat effects different systems in different people. It may effect one patients Liver, another's Hypothalamus and still another's Pancreas, etc... And even if it does effect the same organ/gland in two different patients, the nutrients or steps involved are often not the same.

This entire procedure must be done with each of the offensive foods, because wheat may effect the patients Liver, where as dairy may effect his Pancreas. Do only one food per office visit and schedule them one week apart.

This entire procedure, once familiar with it, should take no longer then 20-30 minutes. If your normal office visit is less then that, you may want to only do some of the steps and continue where you left off at the next visit, or schedule the patient for a double or triple visit and charge accordingly. The benefit the patient will receive in how they feel and function will greatly outweigh the additional cost.

Expect food sensitivities to effect just about every patient, but especially your problem patients. Often times, this author has found no objective findings to correlate with the patients subjective complaints until the offensive food is insalivated by the patient, or objective findings are recurrent until the sensitivity is eliminated.

Two dozen patients have been treated with this procedure and all have been desensitized to their offensive foods without any reoccurrence to date.

REFERENCES

1. Kubicek, David A., A Unique Approach to the Treatment of the Cross K27 Form of Neurologic Disorganization. I.C.A.K. Collected Papers, Summer 1988

2. Blaich, Robert M., Applied Kinesiology and Human Performance. I.C.A.K Collected Papers, Winter 1987

3. Schmitt, Walter H. Jr., Pre-Test Imaging, a Screening Test for Cranial Faults. I.C.A.K. Collected Papers, Summer 1987

APPLIED KINESIOLOGY HOLOGRAPHY TECHNIQUE

BY HERVE J. LAFLEUR D.C.

ABSTRACT: HOLOGRAPHIC FINDINGS AND CORRECTIONS.

RESEARCH: -The Change of tonus in a muscle function.

-E.I.D. and how it reveals coordination

-Weakness in muscles when a person is in a state of an open mouth laughing.

Isn't it a strange phenomena that an expression of happiness makes a person become weak with muscle testing.

OBSERVATION: What is a holographic subluxation? Subluxations which are hidden behind stress that the activity of the brain has constructed over a large number of years. Thes conditions of stress are giving the body wrong reactions of interpretation because of the brains' actions, also misinterpreting the real condition.

Three factors of stress:

Mental: A chock that has been forgotten

Chemical: Lack of memory of a chemical action and/or reaction.

Physical: A trauma that has healed and left scars.

Upon laughing we change these barriers of stress, and evidence then shows if there is or is not a holographic problem. Stress is a muscular reaction of contractions, that all incidents and accidents produce. The self

defense of the body in a muscular reaction creates an unease stress. Stress, due to a condition or an environment produces an imbalance mentally, chemically, and/or physically. This phenomena, true or false is stored in the brain as a reality. In the book of DVORAK & DVORAK a pathway of pain is illustrated and is interrelated with muscles in an abnormal state. When the full pathway of pain is not corrected the pain goes away leaving muscular scars creating holographic subluxations.

> One day having expressed a joke to a patient he started to laugh openly during muscle testing. During this period of laughter I was testing the psoas muscle. He could not hold it. When he stopped laughing, I decided to challenge the psoas again; it was strong. I was stunned and started questioning myself but with no answers. Again during his treatment he suddenly burst into laughter while I was testing the hamstring muscle. It was weak; when laughing stopped it was strong again. That was the end of my experience that day. He did leave me in a questioning state. The next day I repeated the experience with several patients. To my surprise some were weak, some were strong. I knew there had to be an answer. This is where my research started.

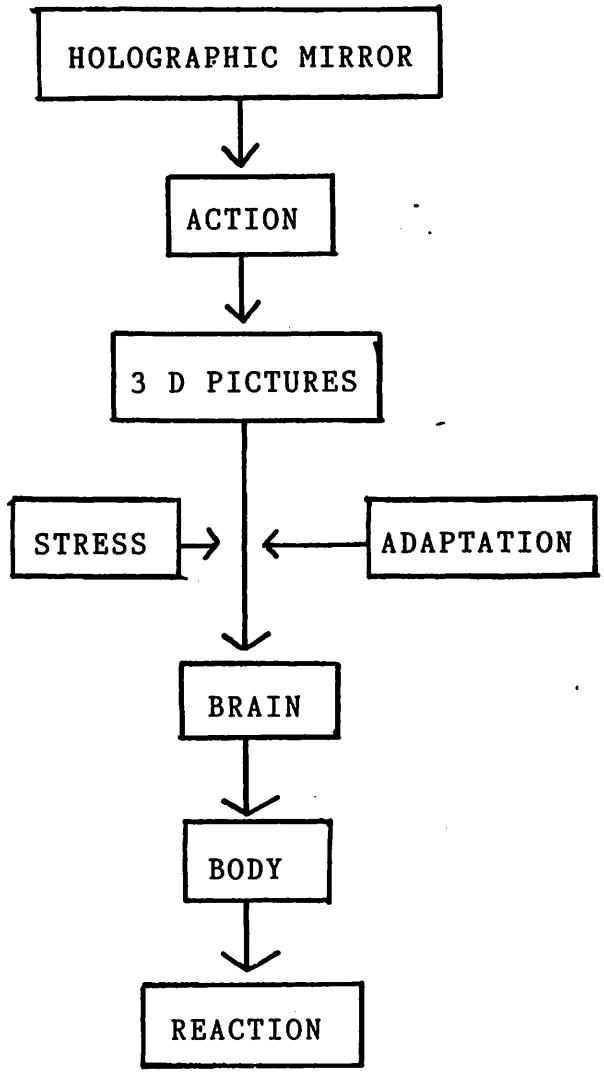
E.I.D. THE MIRROR IMAGE

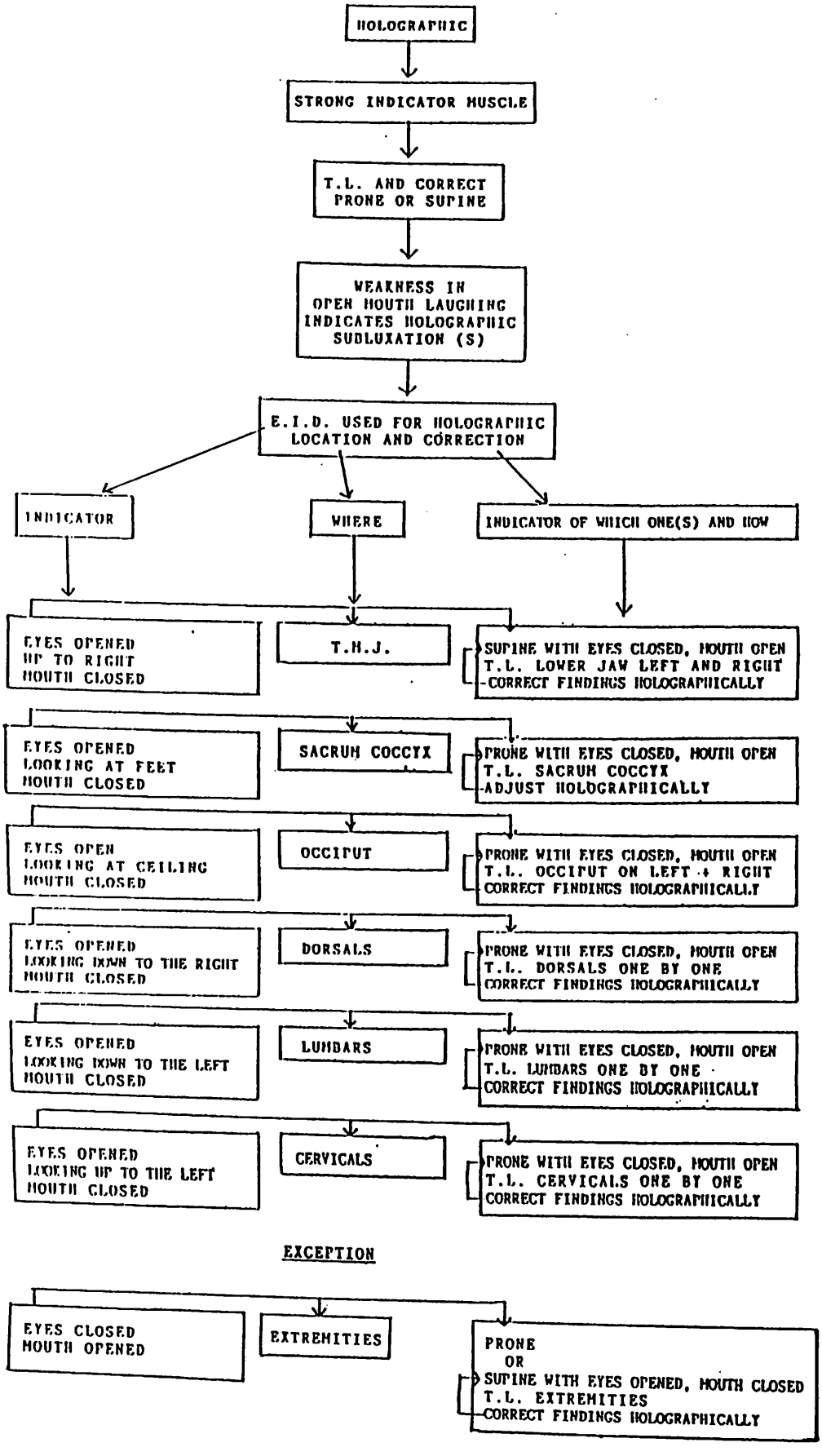
Eyes are seekers for the brain. Awakened they perceive in

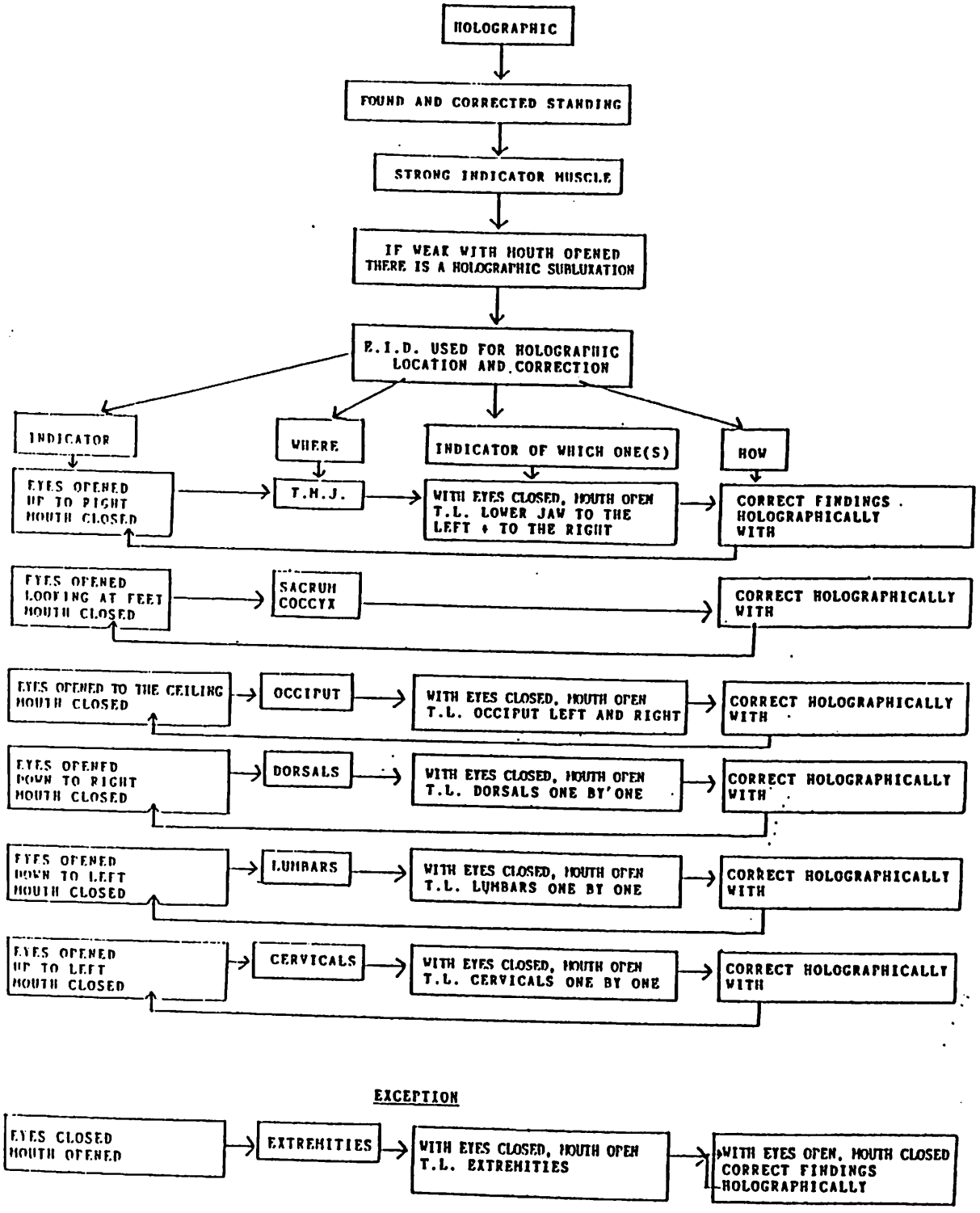
all directions and they become our heritage of what's real. For these reasons it creates hidden stress within the self. Learning the correlation between eye position and holographic subluxations will open the way for adjustment required in specific areas involved.

Correlation between stress and the eyes

My next experience came through from Neuro-Linguistic Programming (N.L.P.). During a consultation I asked a patient to think of a prior happening. I became aware that both of his eyes went straight up to the left. By inadvertance or hazard I already had his pectoralis muscle in position for testing. It tested weak. When both his eyes returned to normal I retested. It was strong. I voluntarily asked him to look up toward the left; the muscle tested weak again. I repeated the same experience with that patient shortly after and got the same results. This is how my research with the eyes started.


HOLOGRAPHIC MIRROR
ACTION
3 D PICTURES
STRESS
ADAPTATION
BRAIN
BODY
REACTION


HOLOGRAPHIC
STRONG INDICATOR MUSCLE
T.L. AND CORRECT PRONE OR SUPINE
WEAKNESS IN OPEN MOUTH LAUGHING INDICATES HOLOGRAPHIC SUBLUXATION (S)
E.I.D. USED FOR HOLOGRAPHIC LOCATION AND CORRECTION
INDICATOR
WHERE
INDICATOR OF WHICH ONE(S) AND HOW
EYES OPENED UP TO RIGHT MOUTH CLOSED
T.H.J.
SUPINE WITH EYES CLOSED, MOUTH OPEN
T.L. LOWER JAW LEFT AND RIGHT
-CORRECT FINDINGS HOLOGRAPHICALLY
EYES OPENED LOOKING AT FEET MOUTH CLOSED
SACRUM COCCYX
PRONE WITH EYES CLOSED, MOUTH OPEN
T.L. SACRUM COCCYX
-ADJUST HOLOGRAPHICALLY
EYES OPEN LOOKING AT CEILING MOUTH CLOSED
OCCIPUT
PRONE WITH EYES CLOSED, MOUTH OPEN
T.L. OCCIPUT ON LEFT + RIGHT
CORRECT FINDINGS HOLOGRAPHICALLY
EYES OPENED LOOKING DOWN TO THE RIGHT MOUTH CLOSED
DORSALS
PRONE WITH EYES CLOSED, MOUTH OPEN
T.L. DORSALS ONE BY ONE
CORRECT FINDINGS HOLOGRAPHICALLY
EYES OPENED LOOKING DOWN TO THE LEFT MOUTH CLOSED
LUMBARS
PRONE WITH EYES CLOSED, MOUTH OPEN
T.L. LUMBARS ONE BY ONE
CORRECT FINDINGS HOLOGRAPHICALLY
EYES OPENED LOOKING UP TO THE LEFT MOUTH CLOSED
CERVICALS
PRONE WITH EYES CLOSED, MOUTH OPEN
T.L. CERVICALS ONE BY ONE
CORRECT FINDINGS HOLOGRAPHICALLY
EXCEPTION
EYES CLOSED MOUTH OPENED
EXTREMITIES
PRONE
OR
SUPINE WITH EYES OPENED, MOUTH CLOSED
T.L. EXTREMITIES
CORRECT FINDINGS HOLOGRAPHICALLY


HOLOGRAPHIC
FOUND AND CORRECTED STANDING
STRONG INDICATOR MUSCLE
IF WEAK WITH MOUTH OPENED THERE IS A HOLOGRAPHIC SUBLUXATION
E.I.D. USED FOR HOLOGRAPHIC LOCATION AND CORRECTION
INDICATOR
WHERE
INDICATOR OF WHICH ONE(S)
HOW
EYES OPENED UP TO RIGHT MOUTH CLOSED
T.M.J.
WITH EYES CLOSED, MOUTH OPEN T.L. LOWER JAW TO THE LEFT + TO THE RIGHT
CORRECT FINDINGS HOLOGRAPHICALLY WITH
EYES OPENED LOOKING AT FEET MOUTH CLOSED
SACRUM COCCYX
CORRECT HOLOGRAPHICALLY WITH
EYES OPENED TO THE CEILING MOUTH CLOSED
OCCIPUT
WITH EYES CLOSED, MOUTH OPEN T.L. OCCIPUT LEFT AND RIGHT
CORRECT HOLOGRAPHICALLY WITH
EYES OPENED DOWN TO RIGHT MOUTH CLOSED
DORSALS
WITH EYES CLOSED, MOUTH OPEN T.L. DORSALS ONE BY ONE
CORRECT HOLOGRAPHICALLY WITH
EYES OPENED DOWN TO LEFT MOUTH CLOSED
LUMBARS
WITH EYES CLOSED, MOUTH OPEN T.L. LUMBARS ONE BY ONE
CORRECT HOLOGRAPHICALLY WITH
EYES OPENED UP TO LEFT MOUTH CLOSED
CERVICALS
WITH EYES CLOSED, MOUTH OPEN T.L. CERVICALS ONE BY ONE
CORRECT HOLOGRAPHICALLY WITH
EXCEPTION
EYES CLOSED MOUTH OPENED
EXTREMITIES
WITH EYES CLOSED, MOUTH OPEN T.L. EXTREMITIES
WITH EYES OPEN, MOUTH CLOSED CORRECT FINDINGS HOLOGRAPHICALLY

INTRODUCTION:

The brains' motor and sensory center, the connective tissue sheath of the muscles of the cervical spinous process and the hyoid placement when the body is healthy does not require consistency over a short period of time. When there is (are) holographic subluxation(s) involvement when we open our mouth laughing the hyoid will rotate and eliminate stress. The jaw opens slightly on inspiration and closes on expiration but when laughing the pattern of inspiration / expiration changes. This is the vector of the hyoid and its attaching muscles. Weak muscles in an area strengthen in the direction that the jaw is opened in, introducing the T.M.J. and its involvement. The knowledge that we now have on the T.M.J. plus the understanding of stress factors involved in human beings, is our premise point. This is why when we open our mouth laughing we eliminate the stress. Contraction of face and neck muscles releases the pathway described by DVORAK & DVORAK, thus giving an open track to see if there is a(an) holographic subluxation(s) involved.

SUMMARY:

It is evident that for every weak response in muscle testing something in the body is not healthy. Correcting what you find when muscle testing is evident. As Dr. Goodheart always says "WHY" does the treatement not hold. With the patients reflex of laughter I have found that by using the Holographic Tecnique there is positive indication in muscle testing response. Challenging, after holographic adjustment, what was hidden previously is now weak in the clear.

REFERENCES:

1. Muscle testing is a valuable clinical method of discovery; used for the majority of my research on holography. Over a year of trial and error, many years of experience, diverse Applied Kinisiology courses and collected papers of the members of I.C.A.K. have all advanced me toward holographic findings.

2. DVORAK, J. & DVORAK, V. Manual Medicine Diagnostics (Georg Thieme Verlag Stuttgart, New York)(Thieme-Stratton Inc. New York)

3. Gray, H. F.R.S. Gray's Anatomy Thirtieth American Edition 1985 (Lea & Febiger, Philadelphia)

4. Bandler, R. Using your brain for a change -Neuro-Linguistic Programming (Real People Press, Box F, Moab, Utah)

5. Grinder J., DeLozier J. & Bandler R. Patterns of the hypnotic techniques of Milton H. Erickson, M.D. (Meta Publications P.O. 565 Cupertino, California)

6. Goodheart, G.J. Research Tape 35 -Concepts of

7. Ekman P. & Friesen, Wv. Unmasking the Face, (Prentice-Hall, Inc. Englewood Cliffs, New Jersey)

8. Goodheart G.J. Research Tape 73 -General Concept of

9. Hammer, W. Tapping of the Temporal Mandibular Joint (A preliminary report) -General concept of-

THE THIRTY SECOND IMMUNE DEFICIENCY CURE

© Harvey Lang, D.C.

abstract

A NEW METHOD FOR STIMULATING THE IMMUNE SYSTEM OR POSSIBLY ANY DEFENSIVE SYSTEM IN THE BODY ALLOWING THE BRAIN TO STOP "IGNORING" THE PRESENCE OF FOREIGN INTRUDERS, PATHOGENS OR TUMORS. A WAY IN WHICH TO STIMULATE THE BODY TO CORRECT A PROBLEMATIC SITUATION, PREVIOUSLY IGNORED.

As developed and reported on by this author in "The Thirty Second Allergy Cure"1, a way to "erase" allergies and other aberrant responses has been used effectively by various practitioners of applied kinesiology. After the success of the above, I wondered if by a similar technique one might be able to induce the body to react to substancees, invaders, situations or even tumors. The following is an explanation of how I have successfully solved the above problem:

With this technique, combined with the previous "Thirty Second Allergy Cure" technique, we are able to turn on or turn off the immune or defense systems of the body. As mentioned before by this author, all responses were corrected using the upper left eye position.1 This use alone is proving not to be as effective as fixing all the eye positions which "strengthen" a gamma 1 muscle test (doctor initiated muscle test) weakness. To create an allergy or sensitivity response basically the opposite is done. All directions of gaze (in the presence of questionable substance) stays strong (or stays weak with a less powerful sensitivity response) are treated.* At this point an increased sensitivity is created. The upper right direction 10:30 is the direction in which the response works in every case. Also, somewhat less to the right or 9:00 is a very common sensitization direction. These correspond to the NLP (neural linguistic programming) (see figure 1) chart as visual constructed images (10:30) or sometimes auditory constructed (9:00). It's hypothesized that when the eyes are put in especially these two positions and manipulated while breathing in in the presence of a stimulus, a "file" is created in the brain for use later on. In the desensitization process the "visual remembered" images are "erased" (1:30).

* Treatment, as reported in previous paper, is pushing eye in indicated direction (4-5 times) while patient inspires.

To specifically do this technique when the substance tests strong in the clear in the neutral position and strong in the position of gaze, manipulating the 10:30 position will typically cause weakness in gaze at 10:30 (and sometimes 9:00 also), strengthen all the other positions and show weakness in the neutral position. The above is a typically reactive situation as in fact most allergic patients will have muscle weakness in the neutral position (eyes down at 6:00) muscle weakness at 10:30 and also sometimes 9:00 or 7:30. But, the rest will be strong. Another possibility is when the substance reaction is weak in the clear, that is in a neutral position, and also is weak in a particular position of gaze. Manipulation in that position of gaze causes increased sensitivity and the outcome is that all positions of gaze becomes strong and stays weak in the clear.

We are designing an experiment on terminally ill cancer patients with the cooperation of some local physicians. Some tumor cells, preferably from the patient's tumor, will be placed in the mouth with the afore-mentioned eye treatments. We will then do oncological follow-ups on the patient. It is hoped that in the same direction "pictures" are created by a person (upper right side 10:30), there will be creation of hostility of the body to invaders where none existed before. This procedure can also be applied to people who are showing upon therapy localization no or limited reaction to infectious lesions.

We invite practitioners to verify the efficacy of the above technique in different immune deficiency disorders.

conclusion A TECHNIQUE IS DEVELOPED THAT HAS THE POTENTIAL TO STIMULATE IMMUNE REACTIONS.

VISUAL ACCESSING FOR A RIGHT-HANDED PERSON, ("NORMALLY ORGANIZED").

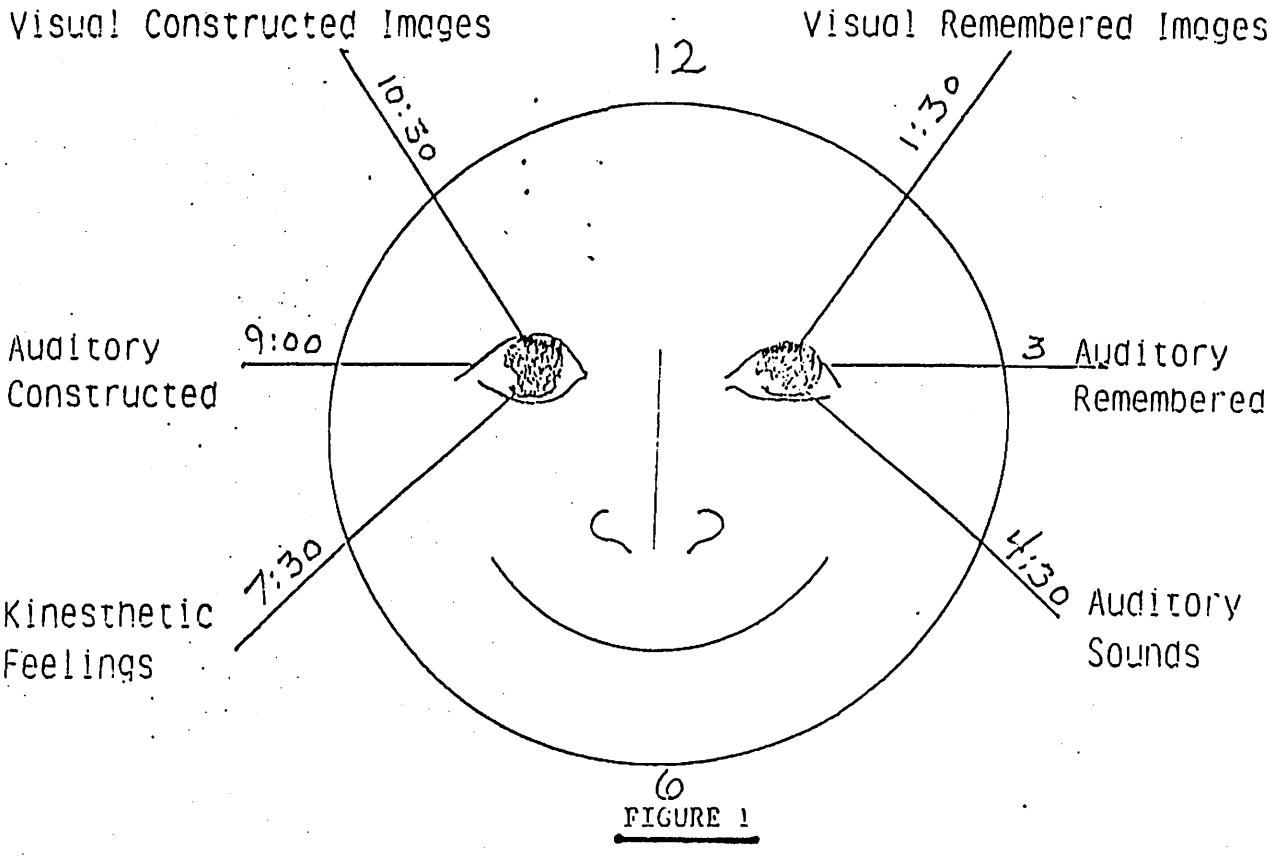


FIGURE 1

Vc Visual constructed images

Vr Visual remembered (eidetic) images.

(Eyes defocused and unmoving also indicates visual accessing.)

Ac Auditory constructed sounds or words.

Ar Auditory remembered sounds or words.

K Kinesthetic feelings (also smell and taste)

A Auditory sounds or words.

REFERENCES

1. Harvey Lang, The 30-Second Allergy Cure, the Collected Papers of the Members of the International College of Applied Kinesiology, Winter, 1988

2. George Goodheart, Research Tapes, 1988 and personal correspondence

CHEMICAL SENSITIVITIES: THE MISSING LINK TO SOLVING MANY OF YOUR TOUGHEST CASES

MICHAEL LEBOWITZ D.C.

ABSTRACT: Chemical sensitivities are as common as food allergies. Detection of specific sensitivities via tests outlined, plus correction via techniques described, can greatly enhance the health of even the most difficult cases.

INTRODUCTION

If you refer to the introduction of my food sensitivity paper[1], I talk about total load from a clinical ecology standpoint. When the body has more insults - psychological stress, chemical exposure, food sensitivities, pollen and mold sensitivities, structural imbalances, vitamin and mineral imbalances, etc. - than it can cope with, symptoms occur.

Antioxidant therapy and free radical pathology have been getting so much publicity these days. Reading health oriented journals, promotional materials put out by supplement companies, etc., it seems like it is almost universal.

One of the screening tests that I perform, the clorox test[2], is one I've used for years as a screen for free radical pathology. I have been happy with it, but recently wondered why with my practice specializing in chronic, difficult, allergic patients - I did not find it positive in that high a percentage of patients. Also when it did show up, it seemed simple to clear out.

Knowing that OCl^- is not the only free radical produced in the body (H_2O_2 and OH^- are 2 of many others), I began screening with H_2O_2 also.

The number of positive findings I had doubled, yet still seemed low to me.

I have been waking up feeling drugged for a number of years. It improves at times but would still recur fairly often. I had "fixed" my food senstivities[1] and was negative on H_2O_2, clorox, and just about everything else I could think of. One morning I awoke with my cervical pillow upside down and thought, "it must be due to my pillow - its height, shape, etc.". After testing awhile, I found out the reason: I had a chemical sensitivity to foam. Breathing in any foam that was a few years old or less, caused universal muscle weakness. I began testing all our cleansers, soaps, etc., and found 4-5 more products that caused weakness. I had major chemical sensitivities that had eluded my screening tests. All of these were negated by insalivation of selenium or vitamin E (2 free radical quenchers).

According to Levine[3], any form of stress, including chemical toxicity, is capable of causing an increase in endogenous free radical production. Dr. Milczarek[4] lists many causes of free radical production including chemical exposure.

I started testing my most difficult patients on products they clean with and use on their bodies. Many products caused universal muscle weakness. I started playing detective in my history taking. Did their fatigue come on upon moving, reupholstering, getting new carpet, changing laundry detergent? Often there were connections and you would find items like these to cause muscle weakness. The list of possible offenders is almost endless. The most common ones appear to be chlorine (in water, cleansers, bleach) gas or related products, cosmetics, foam, newsprint, carbonless carbon paper, tobacco, new paint, and synthetic carpet. Ask your patient if they get irritable in shopping malls.

If so, they are probably reacting to all the chemicals outgassing from new clothes, shoppers, etc.

One of the most interesting things is that common free radical quenchers, especially selenium and vitamin E, would often not strengthen a weak gamma-2 muscle in the clear. When inhaling the substance that causes the universal muscle weakness - simultaneous insalivation of the correct antioxidant (most often selenium or vitamin E) would negate the universal muscle weakness and strengthen the originally weak gamma-2 muscle.

A lot of us had been checking individual chemicals a while ago but, when screening tools like the clorox test came out, we streamlined our procedures. In some of the more difficult patients, our streamlining has made us miss some very important findings.

I have tried my best over the past few months to come up with a general screening tool for chemical sensitivities; to no avail. If anyone else does, it would be a blessing. Until then, play detective.

I have developed a technique to deal with chemical sensitivities that is similar to the one I developed for food allergies. On all patients that had chemical sensitivities, I checked to see if the weakness caused by inhaling the offending substance would be negated by any of the centering the spine torques[5] and/or hypothalamic set points[6]. In general (see statistics section for more details), I found the following: 1) Having the patient roll their eyes up would negate the weakness caused by chemical inhalation, 2) Therapy localization to the pectoralis minor neurolymphatic would negate the weakness, 3) Therapy localization to GV-27 would negate the weakness, 4) Selenium or vitamin E would negate the weakness (check different brands as patient need

is often specific). In the clear, none of these findings would usually be positive. Only when the stress is present would the body give us the solution.

The technique isn't always as long lasting as the one to fix food allergies. There is a higher recidivism rate. It takes time for the patient's antioxidant level to build up and during that time the person needs to avoid the offending substances. Otherwise, the weakness may recur and the technique may need repeating.

If the chemical is one in the patient's home or work place, changes in the person's lifestyle may need to occur for healing to take place. Most often, the changes are small and simple though occasionally a change in occupation or houses may be necessary.

Chemical sensitivities often go undetected for long periods of time. In many cases, a spreading phenomena occurs and the patient becomes hypersensitive to more and more substances. Ironically, I became sensitive to typewriter ribbon ink while typing my food sensitivity paper.

Case Histories

I'd like to give two brief case histories of fairly typical cases.

1) A patient of mine had been seeing another chiropractor three times a week for two years for a low back injury before coming to me. He operates a buffing machine at a chrome factory. I determined that he was sensitive to the buffing compound and had him stay home. Chemical sensitivities usually affect our weakest links; often the site of physical injuries. Without even adjusting him, his low back cleared after a week. He first went through three days of "withdrawal" with aggravated symptoms. He became symptom free despite the fact that he was doing hard manual labor on his farm. This is after two years of chronic pain.

Then suddenly it returned. He had his wife drive him over, his pain was so acute. She is a flagger for the state highway department and she smelled like hot tar. His symptoms returned right after she started working with the tar. After clearing his muscles, subluxations, etc., I had her come in and after having him take a few deep breaths of her tar laden clothes, his weakness and subluxations recurred. She now showers and changes clothes as soon as she gets home from work and he has been fine ever since. If he ever overexposes himself to hydrocarbons for prolonged time periods, his old symptoms flare up a little bit.

2) A woman with severe acne rosacea for ten years is ashamed to be seen in public because her face is so bad. We found her to be sensitive to chlorine. Supplementation, along with the structural fix, installation of a whole house water filter, and avoidance of bleach and cleansers containing chlorine brought 85% improvement in 2 weeks.

Oftentimes if multiple chemical sensitivities are present, having the patient decrease their daily exposure and doing the techniques described is enough to make dramatic changes in symptoms. For instance, in my case, switching to a cotton pillow, putting a water filter on our shower, and an air purifier in our treatment room (due to my sensitivity to perfume and tobacco on my patients) brought a 50% decrease in my symptoms. As my body is healing, my tolerance level to chemicals is increasing. There are many good books on the market by clinical ecologists to teach us how to implement what they call environmental control[7,8,9,10].

We are human guinea pigs. The number of chemicals in our environment is constantly increasing faster than our body's ability to handle them. Often

times a stressful incident, infection, increased chemical exposure (a new house for instance) is enough to start someone down the road of environmental illness. The symptoms can be circulatory, neurological, immunological, respiratory, etc., etc., and thus the cause often goes unnoticed.

Statistics

I compiled data on 50 patients. Anytime a chemical caused a weakness, it caused a free radical problem. Some substances like perfume also have the potential to cause an aldehyde problem due to a molybdenum deficiency - though they can also cause free radical generation. The nutritional and structural patterns helped me diagnose the type of problem. Items found to cause weakness included baby lotion, new carpet, foam, chlorine, newsprint, gasoline, natural gas, household cleaners, cosmetics, hydrogen peroxide, paint, tobacco, fluoride, perfume, carbonless carbon paper, phenol, white sugar, and milk.

Number of patients: 50

Weakness negated by eyes up or pectoralis minor NL: 47

Weakness negated by GV-27: 50

by Se: 40

by vitamin E: 14

Other things that negated the weakness (curve (lateral torque): 6

Copper: 2

Magnesium: 3

Glutathione: 2

Manganese: 1

As you can see, in all patients that tested for free radical pathology due to chemical inhalation, weakness was negated by simultaneously therapy localizing GV-27. Ninety-four percent had the weakness negated by therapy localizing the pectoralis minor neurolymphatic (result is same as rolling eyes up). After the first twenty patients the pattern became apparent and I stopped testing all the body torques and supplements unless the prevalent ones were negative.

Nutrientwise, the weakness was negated by selenium (the right brand is critical) in 40 out of 50 patients and by vitamin E in 14. As stated before, these nutrients most often did not strengthen a weak muscle in the clear. They are hidden weaknesses only brought out upon exposure to offending substances. The amount of selenium deficiencies I had previously missed astounded me. It appears that levels of the nutrients were adequate assuming no chemical exposures at all but, upon exposure, the reserve wasn't there and the weakness showed.

Summary of Procedures

1) Any person suffering from multiple food allergies, fatigue, headache, recurrent musculoskeletal problems, brain fog, irritability, etc. that you can't totally clear of symptoms should be suspected of having chemical sensitivities.

2) Question them carefully about their lifestyle at home and work. Have them gather suspected items for you to test. Also, suspect any substance that they find has a particularly attractive or offensive odor. Chlorine, foam, cleansers, and perfumes are a good start.

3) Find a gamma-2 weak muscle.

4) See which substances when inhaled cause universal muscle weakness.

5) Check to see if selenium or vitamin E negate the weakness (while the substance is inhaled).

6) Check to see if the pectoralis minor neurolymphatic and GV-27 negate the weakness (while substance is inhaled).

7) If none of the above 4 factors in steps 5 and 6 negate the weakness, check other nutrients, and/or centering the spine torques, and/or hypothalamic set points to see which do.

8) While inhaling the substance, tap GV-27 approximately 60x (or other appropriate point), then rub pectoralis minor neurolymphatic (or treat other appropriate torques) for 40 seconds.

9) The chemical should no longer create universal muscle weakness. The gamma-2 muscle (step 3) should be strong. All other chemicals that caused universal muscle weakness should now be negative.

10) Supplement with appropriate nutrients.

11) Counsel the patient about how to, and the importance of avoiding the offending substances for a few months.

12) WARNING - On rare occasions, having a patient breathe in the offending substances can cause a very acute reaction such as asthma, hysteria, tachycardia, etc.. If so, a dose of Buffered C (Nutricology), Tri-Salts (Cardiovascular Research) or Alka-Seltzer Gold will often negate the reaction. Have chemically sensitive patients keep one of these three in their house to deal with times of over exposure.

It is also possible that, due to testing of all the suspect chemicals, the patient may develop diarrhea, brain fog, fatigue, etc. later that day

or the following day. It will clear with avoidance. If the doctor suffers from it have yourself tested.

13) The rewards in fixing these patients that no one else can is worth the extra time and detective work.

Conclusion

Chemical sensitivities is an often overlooked problem in applied kinesiology Diagnosing and correcting them will help solve many perplexing cases.

References

1) Lebowitz, Michael, "A Technique to Abolish all Food Sensitivities", Collected Papers of I.C.A.K. Winter 1988.

2) Schmitt, Walter, "The Clorox Test: A Screening Test for Free Radical Pathology", Collected Papers of I.C.A.K. Summer 1985.

3) Levine, Stephen and Kidd, Parris, ANTIOXIDANT ADAPTATION, Published by Biocurrents Division, Allergy Research Group, 1986.

4) Milczarek, Mary, "Free Radicals, Rancidity, and Antioxidants: Implications in Food", Complimentary Medicine Magazine 3:3, 1988.

5) Schmitt, Walter, "Centering the Spine - Functional, Neurological, and Biochemical Considerations", Collected Papers of I.C.A.K. Summer 1987.

6) Lebowitz, Michael and Schmitt, Walter, "Hypothalamic Set Point Technique", Collected Papers of I.C.A.K. Summer 1988.

7) Dadd, Debra, NONTOXIC & NATURAL, Published by Jeremy P. Tarcher, Inc., 1984.

8) Rogers, Sherry, THE E.I. SYNDROME, Published by Prestige Publishers, 1986.

9) Zamm, Alfred and Gannon, Robert, WHY YOUR HOUSE MAY ENDANGER YOUR HEALTH, Published by Simon and Schuster, 1980.

10) Rosseau, David, YOUR HOME, YOUR HEALTH, AND WELL-BEING, Published by Hartley & Marks, 1988.

GAMMA-1 FOOD ALLERGIES

Michael Lebowitz D.C.

ABSTRACT: Certain foods create "allergic" responses on a localized level only. These reactions presently elude generalized applied kinesiological screening tests for food sensitivities. Diagnosis and treatment procedures are discussed.

For the past few years, working with severely allergic patients has brought near miraculous results at times. Some of these patients would stabilize at approximately 85-90% freedom from all allergic symptoms with no further progress. Being the perfectionist that I am, not being able to knock out the last 10-15% was frustrating. All systemic allergy patterns - histamine mediated[1], kinin mediated[2], and thymus mediated[3] - were negative on these patients. One day I had barley soup for lunch and had a moderate case of brain fog following it. I was negative on all these types of allergies but knew I was going through a food reaction. I was testing a PMS, which is prone to a gamma-2 muscle weakness on me, with no response. Because my symptom was brain fog, I decided to switch and test the supraspinatus (it was strong in the clear). When I tested celery, it weakened the supraspinatus on a gamma-1 level (right side only) but had no effect on any other muscles. I found that therapy localization to the supraspinatus NL negated the weakness. Treatment of the NL (with celery in the mouth) resulted in celery no longer weakening. Eating celery in the future no longer caused symptoms. In other patients I have also found certain foods will weaken just one muscle on a gamma-1 level. At different times I have seen the NL, hypothalamic set point[4], associated vertebra, etc., fix the weakness. There seems to be an infinite amount of food and muscle combinations to test. By knowing what

they ate within 24 hours of symptom appearance, it is usually fairly easy to find the gamma-1 allergy (or allergies).

It appears that in these cases the patient's system in general does not react negatively to the food, but one particular target organ is overcome. I have seen this to be the case with the colon, small intestine, brain, stomach, lungs, sinus and adrenals.

It has also become apparent that in some patients, chemical sensitivities, prostaglandin imbalances, and probably many other biochemical problems exist on a localized level only.

At this point I have found no pattern as to nutrients needed, etc..

SUMMARY OF PROCEDURES

1) All gamma-2 allergies should first be fixed (histamine mediated, kinin mediated, thymus mediated) and CCK, histamine, antronex, and copper, should neither strengthen a weak gamma-2 muscle nor cause an intact muscle to weaken on gamma-2 testing.

2) If patient is still symptomatic, suspected foods should be tested on gamma-1 muscles related to the symptoms (e.g. supraspinatus=brain fog, deltoid=lung congestion, etc.)

3) If a gamma-1 allergy is present it will often only weaken one muscle, often unilaterally (e.g. wheat weakening only the right supraspinatus).

4) While the positive food (food itself or powdered antigen, not homeopathic) is in the mouth, see if the NL, associated vertebra, set point, etc. will negate the weakness.

5) Treat whatever negates the weakness, while the food is in the mouth.

6) Retest. Food should no longer weaken that muscle.

CONCLUSION

Allergies affecting just one organ system are fairly commonplace among sensitive individuals. Diagnosing and treating these problems can greatly improve health in difficult cases.

REFERENCES

1) Schmitt, Walter, "The Use of Antronex and Histadine as Screening Tools", Collected Papers of I.C.A.K. Winter 1983.

2) Schmitt, Walter, "Kinin Mediated Allergies and Pancreatic Activity", Collected Papers of I.C.A.K. Winter 1986.

3) Lebowitz, Michael, "Thymus Related Allergies: Diagnosis and Correction", Collected Papers of I.C.A.K. Summer 1988

4) Lebowitz, Michael and Schmitt, Walter, "Hypothalamic Set Point Technique", Collected Papers of I.C.A.K. Summer 1988.

A TECHNIQUE TO ABOLISH ALL FOOD SENSITIVITIES

MICHAEL LEBOWITZ D.C.

ABSTRACT: All food sensitivities have certain structural faults in common. A technique to diagnose and treat these is discussed. The results are in most cases a cessation of food sensitivities after one treatment.

INTRODUCTION

Being a physician with ecologic illness is not always easy. Working with patients whose clothes are laden with tobacco smoke or who smell strongly from perfume, hairspray, aftershave, etc. can cause headaches, fatigue, confused thinking, irritability, depression, rash, and a host of other symptoms. Many patients with vague though very uncomfortable symptoms like this have spent literally thousands of dollars going through a barrage of medical tests. They come home with a diagnosis of stress or "it's all in your head", with no answers for relief. I have chosen to specialize in this type of patient partially as a quest to heal myself. Conventional applied kinesiology has made myself and my patients able to function, but we still have been a far cry from optimal health. It is incredible when you start asking patients how they feel around perfume, tobacco, gas fumes, shopping malls, etc., how many are bothered.

With the advent of gamma-2 muscle testing[1] further advances have occurred. I have still been forced to be very creative in my practice to try to offer these people help. Sensitivities would develop to many substances that there was frequent exposure to. Food sensitivities would be widespread and would change frequently. For example, a person was allergic to wheat and left it

off, and as a result ate more rice, a rice sensitivity would develop. Sensitivities to chlorine (in water, bleach, cleansers), natural gas (from heating systems), dust, etc., were frequently present. Normal living was difficult for these individuals.

As I "experimented" on myself and others, I found that each patient had combinations of histamine reactions[2], kinin reactions[3], thymus related allergies[4], free radical reactions[5], aldehyde sensitivities[6], leucotriene reactions[7], candida overgrowth, etc., etc.. Not every patient would exhibit each one of these but many would be present. I decided to start reading just about everything put out by clinical ecologists to try to turn it into something practical.

Clinical ecologists believe in the concept of "total load". To explain it simply, everyone due to genetics, past history, etc., has a certain capacity to handle environmental insults. Environmental insults are composed of stress, junk food, exposure to chemicals, allergens (foods, pollens, molds, etc.), bacteria, virus, toxic metals, structural imbalances, etc.. When a person goes past their individual threshold, symptoms occur. This concept I have found to be true in many cases. For instance, many patients I have when either eating a food they are allergic to, or breathing in tobaccoo smoke, or overloading stresswise - no matter which variable - always develop the same musculoskeletal and cerebral symptoms. These patients need very little extra stimuli to overload and produce symptoms. Clinical ecologists work at decreasing a patient's total load. They do this by avoidance of incriminating substances, major lifestyle changes, a rotation diet, desensitization injections, and nutritional supplementation. By decreasing the patient's total load, symptoms either lessen or disappear.

Being applied kinesiologists, we have some distinct advantages. Using muscle testing as functional neurology we can let the patient's body give us the answers to many problems. Our results can far surpass those of other practitioners. I have adapted structural techniques previously developed in applied kinesiology, along with nutritional supplementation to correct the body so that it does not react to foods, chemicals, aldehydes, toxic metals, etc. in a hypersensitive fashion.

This particular article discusses a technique that when applied properly, has so far abolished food sensitivities in two hundred patients of mine (plus many other patients of doctors on the East and West Coasts and in Australia that I have shared the technique with). Statistics compiled on the first 100 patients are presented later in this paper. There has been a 9% recidivism rate in which two treatments were needed instead of one. There has been less than 1% failure rate. The beauty of the technique is that it clears out all food allergies at once, instead of just one at a time. After the technique is used so many other previously positive findings are often negative - muscle weaknesses, subluxations, cranial faults, etc. My most difficult patients that were previously very difficult to stabilize due to their widespread and varied allergies have remained clear of food sensitivities since January 1988. Considering that we get patients from all over the East Coast and the Midwest, this has been truly remarkable and a real blessing.

FOOD SENSITIVITIES AND APPLIED KINESIOLOGY

There are three types of systemic food sensitivities easily identifiable with applied kinesiology. Patients are screened to see if histamine causes universal muscle weakness and antronex strengthens a weak gamma-2^2 muscle. If so, we

conclude that the patient has more histamine in their system than they can handle. Over the past few years, in each case in which histamine weakens, I have also been able to find at least one food that causes universal muscle weakness. Both the weakness caused by the food and by the histamine are almost always negated by simultaneous insalivation of vitamin B_6[4].

A second type food sensitivity is screened by seeing if cholecystokinin (CCK) causes universal muscle weakness[3]. If so, we assume the patient is producing more kinins that they can handle. I have been doing this test for almost three years and in all cases where it has been positive, I have found at least one food to cause universal muscle weakness too. Both the weaknesses caused by the food and the CCK have almost always been negated by simultaneous insalivation of the proper form of zinc (I presently prefer zinc citrate).

A third type of systemic food sensitivity is screened by seeing if copper strengthens a weak gamma-2 muscle[4]. Of the 200 or so patients I found strengthen on copper (between Dec. 1987 and May 1988), all have had at least one food cause universal muscle weakness. This weakness is negated by oral insalivation of copper.

I found that I had the ability to push food sensitivity mechanisms around. For example, a patient had a histamine reaction to milk and I would supplement with B_6. In many cases three weeks of B_6 and avoidance of milk would clear the allergy. In other cases, milk would again show positive within a few months as a kinin or thymus related allergy. The symptoms were often the same but the mechanism and treatment would change. My ability to "push around" allergies brought me to the conclusion that all three types of systemic allergies must have a common denominator and especially a common structural link. I shared this idea with Dr. Walter Schmitt in a phone

call on Dec. 1987 and he thought that it was very probable.

CENTERING THE SPINE AND HYPOTHALAMIC SET POINTS

I had been using the centering the spine technique[8], and hypothalamic set point technique[9] very successfully for quite sometime. At times I had found them to be negative in the clear on patients, but if the patient was therapy localizing a positive finding, a particular torque or set point would negate it. I considered whether the body had a torque (centering the spine) or set point problem that would only be apparent when they were exposed to a substance they were sensitive to. In other words, the insulting substance would bring the problem to the body's attention, and offer a solution.

After identifying a particular food sensitivity, I began keeping the food in the patient's mouth and seeing which torques and which hypothalamic set points would negate the weakness caused by the food (many clinical ecologists feel that "hypersensitivity may involve a disruption in hypothalamic functioning"[10]). Very soon a pattern emerged that was the same regardless of which of the three types of sensitivities were present. I found that when treating the torques and set points, while the food was in the mouth, a few things would occur afterwards: 1) the food would no longer cause universal muscle weakness, 2) the weak gamma-2 indicator muscle would now be strong, 3) all other foods that also caused universal muscle weakness would now test negative (each patient was screened on about seventy foods), thus doing the technique cleared all food sensitivities at once, 4) many other positive findings (subluxations, cranial faults, etc.) were now negative.

The fact that all other food sensitivities (to my surprise) were now negative made me feel we were getting to the cause. Treating each allergy individually,

as many techniques do, does not get down to the cause of the allergies in general and in many cases as some are fixed others develop.

I now knew I was onto something important but yet from a physiological standpoint did not understand what I was doing. I needed to take a closer look at it.

THE TECHNIQUE AND POSSIBLE EXPLANATION

In each case the food that caused universal muscle weakness was placed in the mouth. Flexing the right thigh would negate the weakness while testing a left arm flexor (this is called counter-clockwise torque or CCW torque). In the centering the spine technique this indicated a need for pituitary drive technique and more epinephrine in the system.

Also, if the patient therapy localized to the small intestine neurolymphatic on the lower anterior rib borders with one hand, and SI-19 with the other (small intestine hypothalamic set points), it would negate the muscle weakness (most often it would show bilaterally). Dr. Walter Schmitt had previously found that SI-19 is related to epinephrine[9]. In some cases additional set points needed to be treated (see statistics). These other set points seem to be related to the patient's main target organs (weak links, so to speak).

So, for instance, in a patient allergic to tomato, wheat, milk, eggs, and beef; tomato would be placed in the patient's mouth. A CCW torque and small intestine set point would both negate the weakness. While the tomato is in the mouth, the set point technique is performed, as is pituitary drive. (I usually perform the set point technique first, though I do not think the order is critical.) Tomato now tests negative, as does wheat, milk, eggs, and beef. It is important also to supplement the patient. Depending on what

type of allergy it is, either B_6, zinc, or copper is given. (On rare occasions, a different supplement is needed. See Statistics.) The patient is also asked to avoid all positive foods until they are rechecked (ideally a follow up visit is in 14-21 days). This allows nutrient levels to rebuild and decreases the recidivism rate.

Somehow this technique appears to increase the level of, or effectiveness of epinephrine in the system. We know that allergies are due in part to an overactive immune system and that epinephrine is an immune system suppressant. We seem to be toning down the immune response to substances it should not be reacting to. This makes sense when you consider severely allergic patients who carry around epinephrine to counteract the reactions to bee stings, etc.. Serotonin is released during allergic reactions[11]. People stuck in a clockwise torque have too much serotonin in relation to epinephrine. This technique could also perhaps be restoring the balance. B_6 and copper are both cofactors in epinephrine production. Zinc is necessary to "make B_6 work"[12]. Raising the body's epinephrine set point without adequate cofactors could lead to more problems. That is why supplementation is so crucial.

It is also possible that we are helping T suppressor cell function (often compromised in the ecologically ill).

STATISTICS

I compiled data on 100 patients with allergies. Of them 10 had two types of systemic allergies simultaneously and one patient had all three types, thus the totals equal 112.

HISTAMINE ALLERGIES

# of patients weakening on histadine: 36

food reaction negated by CCW torque: 36

food reaction negated by SI set points: 36

# of patients needing B_6: 33

one patient needed B_2, one needed zinc, one folic acid

other set points needed: St/pancreas NL: 8

LI/lung NL: 4

TW/adrenal NL: 2

TW/prostate NL: 1

GB/liver NL: 1

KININ MEDIATED ALLERGIES

# of patients weakening on CCK: 17

food reaction negated by CCW torque: 17

food reaction negated by SI set points: 15 (14 bilateral, 1 unilateral)

# of patients needing zinc: 17

other set points needed: St/pancreas NL: 6

LI/lung NL: 1

THYMUS RELATED ALLERGIES

# of patients strengthening on copper: 59

food reaction negated by CCW torque: 59

food reaction negated by SI set points: 58 (57 bilateral, 1 unilateral)

# of patients needing copper: 59

other set points needed: St/pancreas NL: 21

TW/adrenal NL: 4

TW/prostate NL: 1

TW/thymus NL: 1

As you can see, all the patients strengthened on CCW torque (need for pituitary drive), while 97% strengthened on SI set points. It is interesting to note that the 5 patients needing the lung set points in addition were all asthmatics. Pancreas set points were positive in about one third of the patients, showing how important enzyme production is in sensitivity prevention.

These 100 patients had a total of 715 individual food allergies (based on the 50-80 tests done on each patient). On 9 patients the treatment didn't hold and needed to be repeated a second time.

Of the 715 food sensitivities, the technique did not clear all the sensitivities on 5 of the patients. Each of these patients had one sensitivity remaining (they each had 9-16 to start with). On these patients, Dr. Lang's technique proved effective to clear the remaining sensitivity[13]. So all in all, the technique "fixed" 710 of 715 individual reactions.

SUMMARY OF PROCEDURES

1. All patients in whom allergies are suspected (including those with recurrent musculoskeletal problems) are screened for all three types of systemic allergies (histamine, kinin, thymus related)[2,3,4].

2. If any are positive, check appropriate nutrients to see if they negate the problem.

3. Screen the patient on all foods ingested in any form at least twice weekly (this usually comes out to fifty or more foods). You can use 6x homeopathic allergens to see if they strengthen a weak gamma-2 muscle, or see if the food itself causes universal muscle weakness. The author has found these two methods to correlate close to 100%.

4. One of the positive foods (now you must use the food, not the homeopathic)

is placed in the patient's mouth. Check to see if CCW torque and small intestine set points negates the weakness. Also check other set points based on patient's symptomology.

5. Fix torque and set points (bilaterally) while the food is in the mouth (in other words, perform the hypothalamic set point technique and pituitary drive)[8,9].

6. The food should now test negative and the gamma-2 muscle weakness will be gone.

7. Recheck other foods that were positive. They should now be negative.

8. Put patient on appropriate supplement and have them avoid the foods until the next visit (preferably 14-21 days).

9. At the next visit recheck the foods. If negative, reintroduce them one each day. If positive, the technique may need repeating or a higher dose of supplementation may be necessary.

Some doctors have put histamine or CCK in the mouth (instead of one of the positive foods) while performing the technique. The technique has worked successfully this way. One possible drawback of this method is a lack of identification and avoidance of positive foods; thus increasing the recidivism rate. Also, thymus mediated allergies do not have a screening substance that causes universal muscle weakness and could not be treated with the shortened technique.

REFERENCES

1. Schmitt, Walter, "Muscle Testing as Functional Neurology, Differentiating Functional Upper Motorneuron and Functional Lower Motorneuron Problems", Collected Papers of I.C.A.K. Winter 1985.

2. Schmitt, Walter, "The Use of Antronex and Histadine as Screening Tools",

Collected Papers of I.C.A.K. Winter 1983.

3. Schmitt, Walter, "Kinin Mediated Allergies and Pancreatic Activitity", Collected Papers of I.C.A.K. Winter 1986.

4. Lebowitz, Michael, "Thymus Related Allergies: Diagnosis and Correction", Collected Papers of I.C.A.K. Summer 1988.

5. Schmitt, Walter, "The Clorox Test: A Screening Test for Free Radical Pathology, Parts I and II", Digest of Chiropractic Economics 30:2, 1987 and 30:3, 1987.

6. Mowles, Richard, "A Clinical Study Investigating the Diagnostic Screening and Application of the Trace Mineral Molybdenum, in Applied Kinesiology", Collected Papers of I.C.A.K. Summer 1985.

7. Bland, Jeffrey, "Nutrition and Inflammatory Disease", Creative Audio, Seattle, WA March 1986.

8. Schmitt, Walter , "Centering the Spine: Functional, Neurological and Biochemical Considerations", Collected Papers of I.C.A.K. Summer 1987.

9. Lebowitz, Michael and Schmitt, Walter, "Hypothalamic Set Point Technique", Collected Papers of I.C.A.K. Summer 1988.

10. Miller, Claudia, "Chemical Susceptibility's Many Guises", The Human Ecologist June 1979.

11. Rogers, Sherry, THE E.I. SYNDROME: AN Rx FOR ENVIRONMENTAL ILLNESS, Prestige Publishing, Syracuse, NY 1986.

12. Schmitt, Walter, "Making B-6 Work (Activating Pyridoxine to Pyridoxal-5-Phosphate)", Collected Papers of I.C.A.K. Winter 1987.

13. Lang, Harvey, "The Thirty Second Allergy Cure", Collected Papers of I.C.A.K. Summer 1988.

MILD HYPERBARIA AND APPLIED KINESIOLOGY

A Preliminary Investigation

Dr. Philip Maffetone

Abstract: Three case histories are presented showing the clinical effects of mild hyperbaria on the human body. The use of a mild hyperbaric chamber, which increases the barometric pressure exerted on the subject inside, is shown to have dramatic effects on an individual's structure, chemistry, and psychology. These changes are apparently the result of a 10 - 15 percent increase in oxygen uptake by the subject, resulting solely from the increased pressure since no additional oxygen is added. Results include such changes as improvements in muscle function, range of motion, vital capacity, temperature, and performance.

INTRODUCTION

The body's requirement for oxygen is a simple and obvious one, yet the biochemical reasons are often forgotten.[1] Briefly, oxygen is necessary as the final electron and hydrogen acceptor at the end point of the respiratory chain, which, following the breakdown of food substrate, maintains ATP synthesis. Oxygen is also a vital part of beta-oxidation - the breakdown of fatty acid

to energy in the mitochondria - and must be available to accept hydrogen for this process to continue. The use of oxygen in both these biochemical events is a major part of our life support system from moment to moment.

The body's ability to take in and utilize oxygen is dependent on numerous factors, many of which can be enhanced by various techniques used in Applied Kinesiology. Some of these methods are associated with cranial faults, diaphragm muscle function, fatty acid and amino acid metabolism, and numerous other nutritional factors. Correction of these problems results in improvements in vital capacity, breath holding time, range of motion, blood pressure and temperature changes, and other measurable changes as well as a positive modification of the clinical symptom picture. Moreover, a healthy life style could maintain and further improve a higher oxygen uptake. This may take into account dietary habits, proper breathing, and exercise.

The use of hyperbaric chambers has been important for medical first aid purposes, past and present.[2] The most common applications include treatment of underwater divers, mountain climbers, and pilots for decompression sickness, which results from too rapid or extreme change in pressure exerted upon the body. Cardiac patients, and more commonly burn victims, have also been treated in hyperbaric chambers with added oxygen.

The higher pressure of a hyperbaric chamber mimics the pressure of low altitude, and to a lesser degree, high pressure weather systems. Low pressure, however, is experienced at higher altitudes and within low atmospheric pressure systems. For example, at sea level the atmospheric pressure is 760 mm. Hg*, and at an altitude of 3000 meters (9843 feet) the pressure decreases to 522 mm. Hg. Table 1 shows the relationship between altitude and pressure.[3]

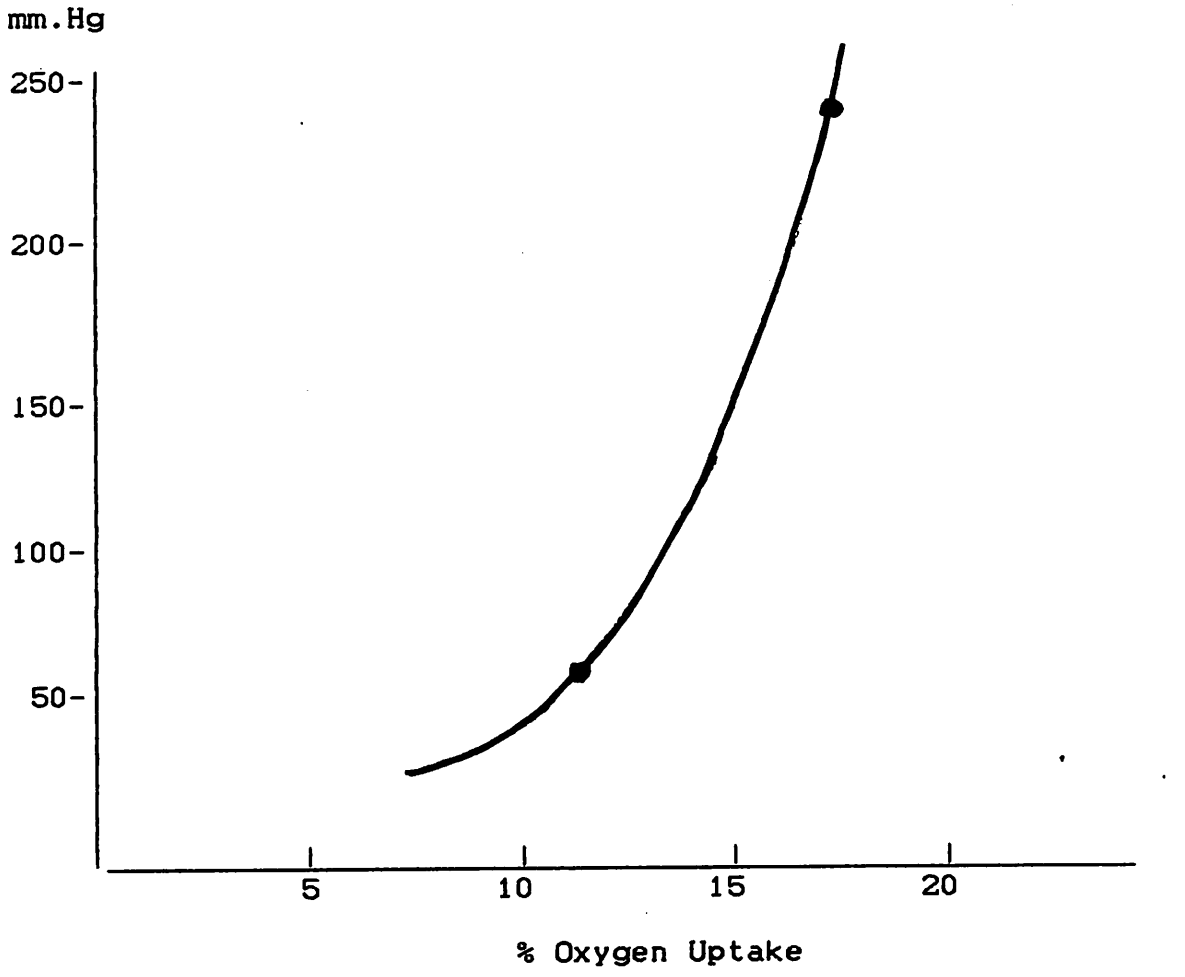
Table 1.

ALTITUDE in meters	(feet)	BAROMETRIC PRESSURE (mm.Hg)
sea level		760
1200	(3937)	654
3000	(9843)	522
4500	(14765)	433

* The proper nomenclature, in standard international units, for atmospheric pressure is the Pascal (N/m/m). However, since a sphigmomanometer is used to measure the pressures in this and other chambers, millimeters of mercury (mm. Hg) will be used here. Appendix A shows the relationship between the five units of measure in common use.

One of the most significant factors observed with changing pressures is the oxygen uptake.[4,5,6,7] In higher pressure environments, or low altitude, the oxygen uptake is increased, and in low pressure environments, or high altitude, the oxygen uptake decreases. Although both altitudes contain 20.9% oxygen, the lower pressure/higher altitude environment results in less oxygen uptake as a result of the decrease in partial pressure of oxygen. Therefore, high pressure increases oxygen uptake, and low pressure decreases oxygen uptake. Studies by Wyndham et al.[6] demonstrate that an increase in only 53 mm. Hg (i.e. descending from 1763 m. to sea level) increased oxygen uptake by 11.1 %, and further decent to 1270 m. below sea level resulted in a total oxygen uptake increase of 17.3 %. This increase is likened to those obtained through daily exercise after many months or even years of discipline. Figure 1 extrapolates the percent oxygen uptake versus pressure increases.

Figure 1.



SUBJECTS, METHODS AND PROCEDURES

The three cases presented here represent a sampling of over fifty trials using the mild hyperbaric chamber. Included in these three cases are a very active male and female and a relatively inactive female. Specifics are discussed under each case. All three individuals have been under the care of this author for varying periods of time and are relatively stable structurally, chemically, and mentally. All three cases had not been treated using AK within eight weeks before using the hyperbaric chamber.

The mild hyperbaric chamber (produced for research purposes by Hyperbaric Mountain Technologies, Inc.), a 17 cubic foot non-permeable nylon cylinder shaped "bag" with an air tight zipper, is a portable version of the more elaborate chambers presently used. It is inflated with an oil- free medical compressor, and the environment is regulated through pressure controlled vents. A continuous flow of incoming and outgoing air prevents carbon dioxide build up. The time spent in the chamber by the subjects was 45 minutes, unless otherwise indicated, at a pressure of 150 mm. Hg, as measured by a wall mercury sphigmomanometer (Baum Co. Inc.). All measurements were taken immediately before and after being in the chamber. Follow-up measurements were taken as indicated in each case.

Vital capacity measurements were taken with a Propper Spirometer. All subjects tested three times, with the average recorded. Oral pH was measured by pHydrion paper and oral temperature by Tempa-DOT disposable strips (Info-chem Inc.). The clorox test, as discussed by Schmitt[14] was employed for many subjects before and after chamber use to detect free radical problems. No such problems have been seen at this time.

All muscle testing was done in accordance with Kendall and McCreary,[9] and Walther.[10] The Applied Kinesiology procedures and nutritional testing used were those also described by Walther.[10,11]

Case 1.

T.E. is a 29 year old male, who works full time and is very active in cycling, running, and swimming. He presented the morning following a bicycle accident, having been screened the previous evening in a hospital emergency room. Shoulder ranges of motion were restricted as follows: Abduction - 110 degrees bilateral. Internal rotation - 30 degrees left, 20 degrees right. External rotation - 40 degrees left, 60 degrees right. The subject was in the hyperbaric chamber for 150 mm. Hg for 45 minutes. The following pre and post tests were recorded:

Test	pre	post
Vital capacity	5100	5900
Oral pH	6.3	7.2
Temperature (degrees F)	96.8	98.0
Clorox Test	neg	neg
Muscle weaknesses:		
Left supraspinatus	weak	weak
Right Latissimus	weak	strong
Tensor fascia Lata	weak bilateral	strong bilateral
Infraspinatus	weak bilateral	strong bilateral
R. Pectoralis Maj.Clav.	weak	strong.
Category II (right)	positive	negative

The resting heart rate in the chamber after 30 minutes was 35 beats per minutes as measured by a Heartwatch monitor (model 8799, Computer Instruments Corp.). T.E.'s average morning heart rate, as per his training diary, averaged 46 beats per minute (taken manually for one minute). Following the 45 minutes in the hyperbaric chamber, the ranges of motion normalized except for left shoulder abduction. The weak left supraspinatus, which did not respond to hyperbaria, was corrected by simple origin and insertion technique, which restored normal abduction. T.E. was able to successfully compete in a local swimming race the following morning. Measurements of vital capacity one week and two weeks later showed a steady level of 5800 each week.

Case 2.

B.D.is a 48 year old female. Although she has improved since her initial visit to the office five months ago, she still complains of some fatigue and an inability to lose weight. Two months ago she stopped taking the thyroid medication (Synthroid 1.5 mg. b.i.d.) she had been taking for 6 years. The following information was recorded both before and after she spent 50 minutes in the hyperbaric chamber at 150 mm Hg.

Test	pre	post
Temperature (degrees F)	96.2	99.2
Oral pH	6.7	6.7
Vital capacity	2700 (84%)	3300 (103%)
Muscle weaknesses:		
right sartorius	weak	strong
left psoas	weak	strong
diaphragm	weak	strong
left teres minor	weak	strong
Other AK indicators:		
Category II (right)	positive	negative
K-27/Cv-8 TL	positive	negative
Yaw pattern	positive	negative

One week later B.D.'s temperature was 98.6 F., and two weeks later, 98.2 F. She was placed in the hyperbaric chamber two more times, with a resulting loss of 6 pounds and a noticeable decrease in size, typically indicating fat loss.

Case 3.

G.M. is a 41 year old female who is a competitive runner, who also holds a full time job. Pre and post changes in vital capacity, temperature, and muscle weakness were similar to those in the first two cases, but a measured increase in athletic performance was shown in this case, similar to others who have been tested with this same procedure. This maximum aerobic pace (MAP), a method previously described by the author [12], in which the subject runs at a specified heart rate and records mile times, was used to measure athletic performance before and after 45 minutes in the hyperbaric chamber. Follow-up measurements were also taken. The information obtained is shown below:

Pre chamber MAP	Post chamber MAP
8:31	8:47

Post MAP (48 hrs)	Post MAP (72 hrs)	Post MAP (two weeks)
8:05	8:06	7:41

It has been a common phenomenon, in these procedures, to see performance increase, following an initial decrease, over the original level. Both Wyndham et al.[4] and Linnarsson et al.[7] demonstrated that performance levels are higher at lower altitudes and higher pressures. In those studies, performance increases were related to a higher oxygen uptake and decreased lactic acid production. It is well known [1,4,5] that lactic acid production occurs when the oxygen availability is decreased. Given the same exercise stress, with increased oxygen, less lactic acid is produced.

Lactic acid metabolism and hyperbaria may also be related to the mental/psychological aspect of certain subjects. It has been shown[17,18,19] that a relationship exists between lactic acid levels and depression, anxiety, and phobias. This relationship has also been discussed in a paper by this author.[13]

Clinically, a significant number of subjects using the hyperbaric chamber have reported beneficial effects on a mental/psychological level. Some of this subjective information may be placebo effects, but Rosen[8] has shown relationships between high pressure environments and mental states. The elimination of active emotional neuro-vascular points as well as meridian end points following the hyperbaric state also indicate mental/psychological benefits.

DISCUSSION

The changes obtained as a result of an increased oxygen uptake have been demonstrated in previous studies and texts.[1,2,4-7] However, the assumption that there is a change in beta-oxidation as a result of a hyperbaric state is an extrapolation from this information. At the time of this writing, two unique studies developed by this author and Professor R. Igor Gamow (developer of this hyperbaric chamber), are in progress at the University of Colorado in Boulder. In one study, we are measuring pre and post Respiratory Quotient (RQ), which is an indication, in part, of changes in beta-oxidation. This is done by measuring the oxygen and carbon dioxide ratio of the subject breathing into a gas analyzer. In another purely statistical study, we are comparing running race performances in various cities in North America with the barometric pressure on those race days to see if low pressure days produce slower race performances.

NUTRITIONAL FACTORS

The increased pressure in the chamber which results in a higher oxygen uptake will obviously not supersede such needs as primary nutrient requirements. For example: two cases not included above showed no change following a 45 minute period in the hyperbaric chamber. Both subjects (coincidentally) demonstrated a nutritional need for zinc. The relationship

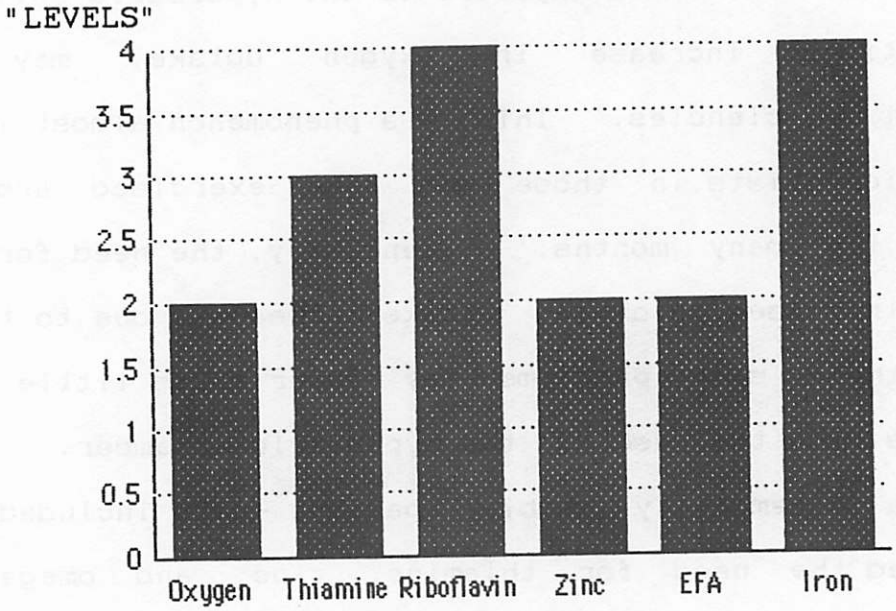
between zinc to breathing has been discussed by Goodheart [14] and Schmitt. [15]

More interesting is the relationship between other nutrients to hyperbaria. Continued exposure to the hyperbaric environment, hence continued increase in oxygen uptake, may produce nutritional deficiencies. This is a phenomenon almost identical to that which exists in those who have exercised and trained regularly for many months. Eventually, the need for certain nutrients increases as oxygen uptake increases due to training. However, these same problems may occur in as little as three weeks' time with the use of the hyperbaric chamber. One such patient (a chemically stable patient not included above) demonstrated the need for thiamine, zinc, and omega-6 fatty acid following five visits to the chamber over a four week period. Table 2 on page 14 shows how a rapid increase in oxygen uptake could theoretically affect the nutritional status of a person.

It has been argued that no difference exists between increased oxygen utilization from hyperbaria and increased oxygen utilization as a result of breathing pure oxygen without hyperbaria. Many, including this author, agree that hyperbaria (increased pressure) and hyperoxia (increased oxygen) are not the same. One important difference is in the oxygen and carbon

dioxide levels of cerebral blood flow. Hyperoxia actually lowers cerebral blood flow as well as arterial oxygen levels and cerebral oxygen consumption. [4]

Table 2



Of oxygen levels, originally at level 2, rise to level 4, relative deficiencies of thiamine, zinc, and EFA could appear, due to the increase in these nutrients required to utilize the higher level of oxygen.

CONCLUSION

Through the use of standard diagnostic tests, such as vital capacity, temperature, heart rate monitoring, and others, it can be shown that mild hyperbaria has a positive impact on human health. With the addition of Applied Kinesiology techniques, the extent of this impact on physical, chemical, and mental health becomes even more apparent. The use of a mild hyperbaric chamber in conjunction with the diagnostic capabilities of Applied Kinesiology is yet another means of bringing professionals together with the potential for forming a more wholistic scientific community. Three such areas are being pursued at the time of this writing: two at the University of Colorado, as discussed above, and one at the Center for Aerospace Sciences at the University of North Dakota. These projects will help introduce Applied Kinesiology to others who share our common goals.

References:

1. Martin, D. W., et al. Harper's Review of Biochemistry. Los Altos, CA: Lange Medical Publications, 1983.

2. Beeson, P. B., and McDermott, W., Textbook of Medicine. Philadelphia, PA: W. B. Saunders Co., 1975.

3. Gamow, R. I. Hyperbaric Mountain Bag Instruction Manual. Boulder, CO: 1988.

4. Mountcastle, V. B. Medical Physiology, Volume Two. St. Louis: C. V. Mosby Co., 1970.

5. McArdle, W. D. et al. Exercise Physiology. Philadelphia, PA: Lea and Febiger, 1986.

6. Wyndham, C. H. et al. "Physiological effects of acute changes in altitude in a deep mine". J. Appl. Physiol. 30: 552-555, 1971.

7. Linnarsson, D. et al. "Muscle metabolites and oxygen deficit with exercise in hypoxia and hyperoxia." J. Appl. Physiol. 36: 399-402, 1974.

8. Rosen, S. Weathering. New York: M. Evans and Co., 1979.

9. Kendall, F. P., and McCreary, E. K. Muscles, Testing and Function. Baltimore, MD: Williams and Wilkins, 1983.

10. Walther, D. S. Applied Kinesiology, Volumes One and Two. Pueblo, CO: Systems DC, 1981.

11. Walther, D. S. "Applied Kinesiology Nutritional Testing." Collected Papers of ICAK Members. Summer, 1988.

12. Maffetone, P. B. "Preliminary Observations of Exercise, Fitness and Training." Collected Papers of ICAK Members. Winter, 1983.

13. Maffetone, P. B. "The Aerobic Deficiency Syndrome." Collected Papers of ICAK Members. Winter, 1987.

14. Goodheart, G. J. Applied Kinesiology: 1986 Workshop Manual. Detroit, MI: Privately Published, 1986.

15. Schmitt, W. H. "Identifying the need for B vitamins, manganese, and coenzyme Q: An answer to recurrent hypoglycemia and hypoadrenia." Collected Papers of ICAK Members. Winter, 1987.

16. Schmitt, W. H. "The Clorox Test: A Screening Test for Free Radical Pathology." Collected Papers of ICAK Members.

17. Cohen, M. E. et al. "Blood Lactate Response Druing Moderate Exercise in Neurocirculatory Asthenia, Anxiety Neurosis, or Effort Syndrome." J. Clin. Invest: 339-342, 1947.

18. Gorman, J. M. et al. "Panic Disorder and Agoraphobia." Am. Psychiatric Assoc. Diagnostic and Statistical Manual of Mental Disorders, Third Ed. Washington, D.C. 1980.

19. Pitts, F. N. and McClure, J. N. "Lactate Metabolism in Anxiety Neurosis." N. Engl. J. Med: 1329-1336, 1967.

APPENDIX A.

Relationship between different units used to measure atmospheric pressure.

ATM (Atmospheres)	Pascal (N/m/m)	mm Hg (millimeters of mercury)	psi (pounds per square inch)	inch H2O (inches water)
1	1.01 x 105	760	14.969	406.8
9.89 x 10-6	1	7.5 x 10-3	1.45 x 10-4	4.0 x 10-3
1.31 x 10-3	1.33 x 102	1	1.93 x 10-2	5.4 x 10-1
6.81 x 10-2	6.89 x 103	51.7	1	27.68
2.46 x 10-3	249.08	1.87	3.61 x 10-2	1

SURROGATE TESTING:
DISCOVERY AND APPLICATION

Kerry M. McCord, D.C.

ABSTRACT: A guide to the understanding and utilization of surrogate testing gleaned from a video tape of Session 9, of the 10 Session Syllabus, taught by Goodheart and Schmitt in October of 1982.

Surrogate testing was discovered as a consequence of a serendipitous observation made by Goodheart while performing a brief and unscheduled examination on the mother of a brain-damaged, severely convulsive child being treated regularly in an attempt to improve the function of the child thus easing the burden of the mother. She requested assistance in resolving the aggravation of a problematic shoulder. Though very busy that morning, Goodheart offered to take care of her if the problem was simple, otherwise an appointment would be needed to provide adequate time for proper evaluation and treatment.

He began by testing the Teres Minor and Deltoid. Both seemed intact. Numerous interruptions made reassessment of muscle status appropriate. Upon retesting, and much to his surprise, the Teres Minor was found to be weak. Reviewing these seemingly contradictory responses in his minds eye he recalled that while previously testing the patient she was not holding her child. He therefore instructed her to put

the child down and proceeded to re-check the Teres Minor finding it, once again, intact. However, when the mother picked up or touched the child the Teres Minor was weak! Thinking he may have erred in judgment, Goodheart asked his associate to test and verify. His observation was confirmed. Thus began the concept of surrogate testing.

Goodheart recalled having read, in an Australian publication, that simultaneous electro-encephalographic tracings were done on nursing mothers in hospitals and their breast-fed children, forty feet down the hall. Spiking noted during REM activity in the dream state of the mother was paralleled in the electro-encephalographic tracing of the child done at exactly the same time, establishing a connection, beyond the usual, between mother and child. This relationship according to Goodheart has been well documented though the mechanism by which it occurs we really do not understand. But, he points out, we also do not understand the mechanism of Olfaction though there are many theories as to its mode of operation. One outstanding theory is that the nose is an infrared producer (on some it looks like it) and the infrared is projected like radar, the nose sensing the difference in the wave length sent out and the one that returns. Whatever the theoretical postulates may be, we still don't know how it works. We instead have theories yet to be established in fact but functional for purposes of explanation and understanding. In the same way, we do not know how surrogate testing works but we know (as

plain as the nose on our face) that it does work!

Although surrogate testing is extremely valuable in testing the comatosed and children, it is discouraged by Goodheart, even for demonstration purposes. However, if the clinician decides to use a surrogate, certain principles need to be kept in mind:

1. It is the doctor's first responsibility to keep him/herself as healthy as possible.
2. The surrogate should be a person in relatively good health so as to minimize potential for error.
3. Do not use surrogate testing unless it is impossible to test otherwise. Most patients including children (four and over, sometimes younger), paraplegics, and patients in severe pain, can be tested normally with the use of a muscle that is intact.
4. Test first to make sure surrogate is strong.
5. Upon the identification of weakness in the surrogate have the patient therapy localize a related reflex factor (e.g. neurolymphatic) and observe change. If weakness is suspected by not observed, therapy localize to uncover hidden fault. Treat and retest.
6. Remember: those who have only a casual relationship can rarely be used for testing. This is due to the fact that, on the average, the frequency at which we resonate varies. (The Brain, Restak)
7. The doctor will cause aberrant response in patient testing 1/2 to 1% of the time.

NEURAL ORGANIZATION TECHNIQUE:
AN OBSERVATION

Kerry M. McCord, D.C.

ABSTRACT: After a brief introduction and critical observation case studies are presented in support of the effectiveness of Neural Organization Technique in the treatment of the learning disabled and dyslexic child.

"Recent research by chiropractic kinesiologists has brought to light an astoundingly rapid solution to the abysmal load the learning disabled have had to bear.

Through rather simple structural and neurological analyses, the root of the problem is determined and the correction is made virtually then and there in an appreciable number of cases. The examination embraces many aspects of investigation that have been considered in past years by various learning institutions, but that is where the similarity ends.

Applied Kinesiology is then put into gear." [1]

Carl A. Ferreri, D.C., the founder and developer of Neural Organization Technique, has used principles of Applied Kinesiology in the resolution of the problems faced by the learning disabled and dyslexic. He postulates a system of evaluating and treating, in a sequential and predictable manner, this very difficult problem.

After careful study of the procedure recommended by Ferreri, I began to treat the learning disabled and dyslexic child. The results have been remarkably favorable. However, as one who is initiated in the nuances of Applied Kinesiology observes the testing and treatment procedures prescribed by Ferreri one immediately notices apparent ambiguities when the information learned is juxtaposed with already accepted AK practice. For example, two-hand therapy localization to the Temporomandibular Joint, according to Ferreri, implies an "imbrication" exists that can be corrected by manual manipulation of the mandible with the adjustive thrust being administered in a caudal direction moving the head of the mandible away from the temporal fossa.[2] This same pattern of therapy localization when previously observed was interpreted in a different manner.[3]

Not to belabor apparent disagreements, though further examination is appropriate and necessary, suffice it to say that in our hands the use of Neural Organization Technique has been both useful and consistent in clinical result.

Attached as appendices are testimonials and objective documentation accompanied by a brief outline of the clinical procedures used in each case study.

It appears, upon review of the data presented, that the use of N.O.T. as a tool for treating the learning "impaired" child is substantiated. This conclusion takes into account the facts that ancillary therapuetic modalities were employed and the patient sample was limited.

APPENDIX 1

Derek Medved: 18 years old

Male

Autistic

Diagnostic and Therapuetic Procedures:

Neural Organization Technique

Applied Kinesiology

Nutritional Supplementation

Physiotherapy

Cross Crawl

Diet - Eliminate refined carbohydrates, caffiene, fried foods, and increase fresh fruits, vegetables, and whole grains

Hair Analysis - revealed significant levels of Toxic Metals, especially Cadmium

Blood Work - revealed tendency to hypoglycemia

Supportive Documentation:

Laboratory Reports - Hair

Blood

Letter from teacher

Letter from mother

A. Q. JONES CENTER/EXCEPTIONAL STUDENT
SCHOOL BOARD OF ALACHUA COUNTY
1108 N. W. 7th Avenue
Gainesville, Florida 32601

Jefferson W. Jones, Jr.
Principal

Phone: (904) 377-5256
(904) 395-0725

December 18, 1987

TO WHOM IT MAY CONCERN:

DEREK MEDVED is a student in my classroom, and for the past few weeks I have noticed significant improvement in his interpersonal skills.

He is more verbal (appropriately), and initiates conversations with new people. Overall his mood is more cheerful and delightful. Several school staff members have mentioned how much more appropriate he is interacting .

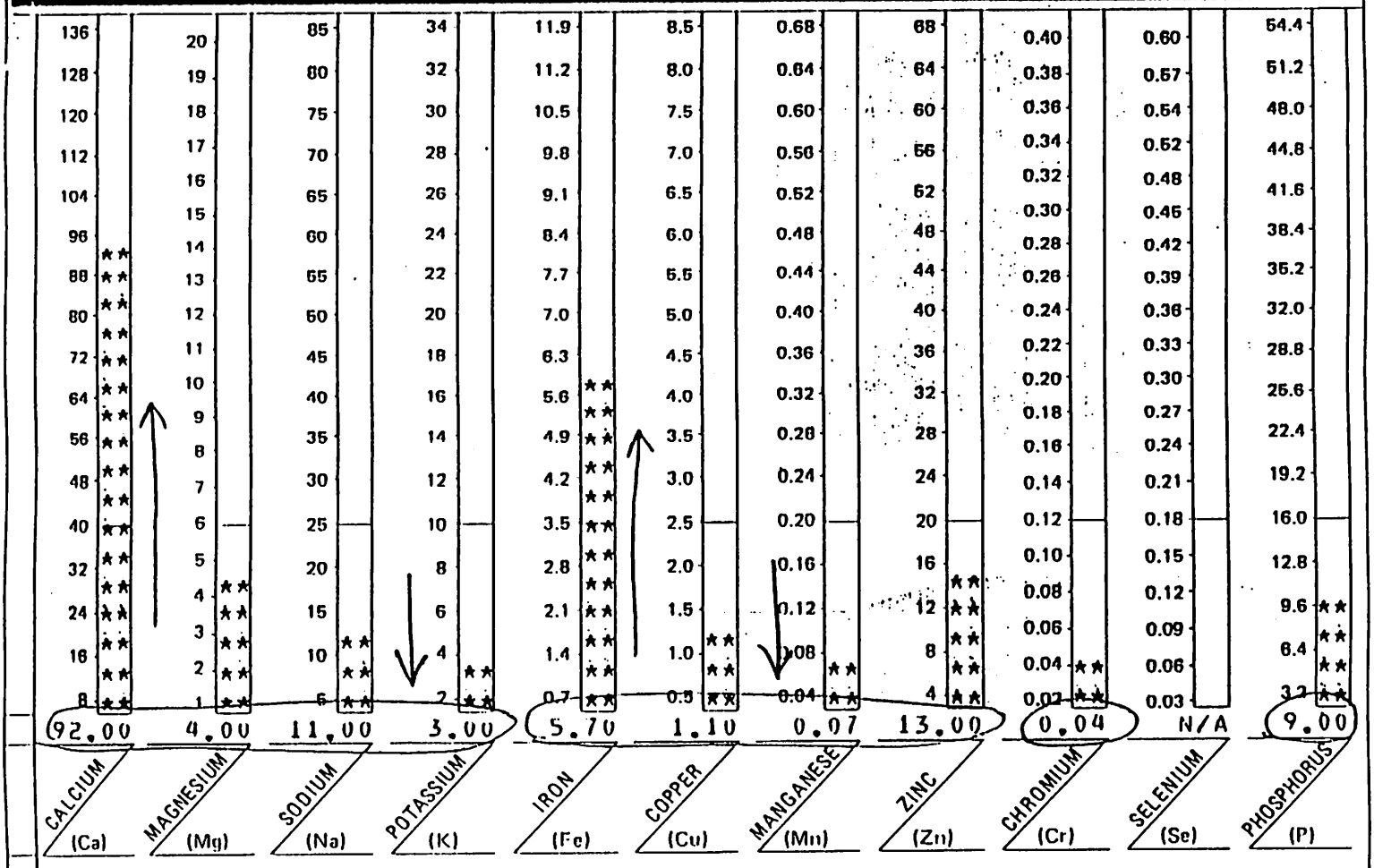
Linda Hallman

ARL ANALYTICAL Research Labs, Inc.
8650 N. 22nd Ave. • Phoenix, AZ 85021 • (602) 995-1581

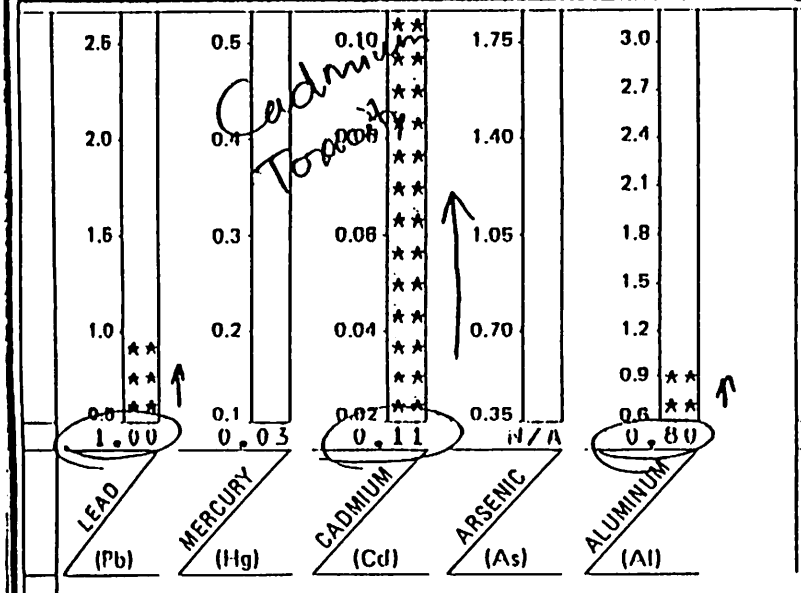
SEX: M	AGE: 18	DATE: 11/24/87	LAB NO.: 70842	CLIENT ACCT. NO.: 94308
PATIENT NAME: Derek Medved			REQUESTED BY: Kerry Mccord DC	

Testing By Accutrace Labs, CLIA # 02-1039 8152 N. 23rd. Ave Phoenix, Az. 85021

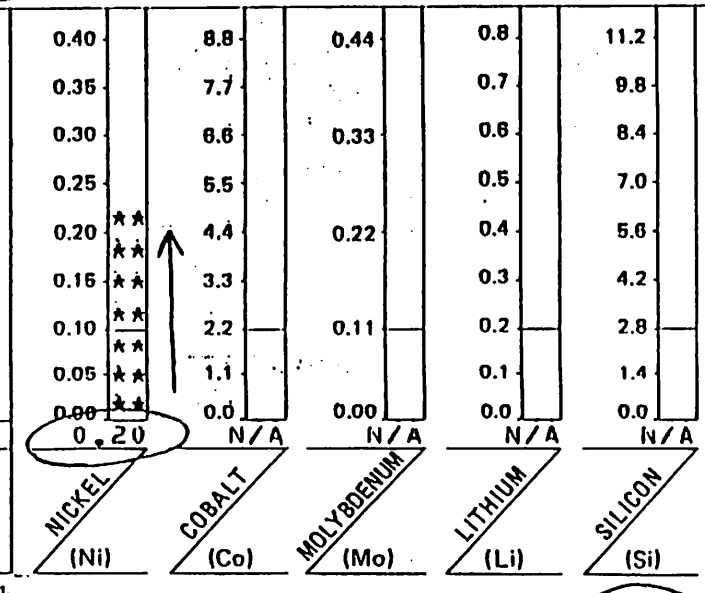
NUTRIENT MINERAL LEVELS



TOXIC METALS



ADDITIONAL MINERALS



MIXED OXIDIZER FAST OXIDIZER SLOW OXIDIZER

Neural Organization Technique/ McCord

266 DAMON CLINICAL LABORATORY
5415 LAUREL STREET
TAMPA, FLORIDA 33 7

53689,15471 [7]

MCCORD K M DC 11
6110 9 ST NORTH 4
ST PETERSBURG, FLORIDA 33703

PAT NAME				PATIENT ID
MEDVED, DEREK				
ACCESSION NO.	AGE	SEX	TOTAL VOL/SOURCE	DATE RECEIVED
4561767	18	M		12/29/87
REFERRING PHYSICIAN			CLIENT NO.	DATE REPORTED
			80135106	12/30/87
ORDER STATUS	COLLECTION DATE/TIME			CLIENT DATA
COMPLETE	12/29/87			

TEST	OUTSIDE RANGE	WITHIN RANGE	UNITS	REFERENCE RANGE
LAB SCAN				
GLUCOSE	53 ↓		mg/dl	70-115
BUN		16	mg/dl	10-26
CREATININE		0.9	mg/dl	0.7-1.4
B/C RATIO		17.8		
URIC ACID		4.8	mg/dl	3.9-9.0
CHOLESTEROL		122	mg/dl	120-170
TRIGLYCERIDE	25 ↓		mg/dl	40-170
TOTAL PROTEIN		6.6	Gm/dl	6.0-8.0
ALBUMIN		4.9	Gm/dl	3.4-5.2
GLOBULIN		1.7	Gm/dl	1.5-3.8
A/G RATIO	[redacted]	2.9 ↑		
TOTAL BILI		0.8	mg/dl	0.2-1.2
DIRECT BILI		0.1	mg/dl	0.0-0.3
INDIRECT BILI		0.7	mg/dl	
ALK PHOS		96	mu/ml	30-300
CALCIUM		9.7	MG/DL	8.5-10.5
PHOSPHORUS		3.3	mg/dl	2.5-4.5
LDH		201	MU/ML	60-215
SGOT	46 ↑		mU/ml	7-40
SGPT		18	mU/ml	0-45
SODIUM		140	meq/L	138-149
POTASSIUM		3.6	meq/L	3.5-5.3
CHLORIDE		102	mEq/L	96-110
CO2		25	mEq/L	24-32
CBC				
WBC		4.6 ↓	x10-3 CMM	4.5-12.5
RBC		4.75	x10-6 CMM	4.60-6.20
HGB		14.8	gm/dl	14.3-18.3
HCT		43.9	PER CENT	42.0-52.0
MCV		92 ↑	u-3	80-100
MCH		31.2	PER CENT	26.0-34.0
MCHC		33.8	PER CENT	31.0-37.0
RDW		10.8		9.0-16.0
PLATELET COUNT		251	x10-3 CUMM	144-440
POLYS		71.1		
LYMPHS		21.5		
MONOS		4.9		
EOS		1.5		
BASOS		0.4		
THYROID PROFILE GROUP 1				
T3		28.9	% UPTAKE	24.0-36.0
T4		9.6	MCG/DL	5.0-13.0
T7		2.77		1.20-4.68

Hypoglycemia

Protein metab

3c

12/22/87

My autistic son, Derek Medford, is being treated by Dr. Kerry McCord, of McCord Chiropractic in St. Petersburg, Florida. Though only 6 or 7 weeks have passed since Derek's initial visit to Dr. McCord, remarkable changes have already been noted in the home environment.

Derek (18) has always been hyper and subject to prolonged repetitions of actions and behavior (sharing objects, verbalizations words or sounds that made no sense, body motions, etc. Strange faces), all of which interfered with any kind of meaningful communication. His attention span has always been very short. If his sequential repetitious motion-sound behavior got interrupted for any reason he would "blow up" and start the sequence over. He spent little time in the real world.

Today, Derek is calmer, more appropriate, cheerful. His mannerisms are much less pronounced.

An example: On 12/18/87 we took Derek to a Christmas buffet dinner at the home of a friend. Normally we would have lost Derek at the shower stage, because in his mind showers are for

morning and taking a shower at 5:30 pm was not a part of his daily sequence; next we never would have gotten passed this stage - clothes. Derek only wears old, soft, cotton T-shirts of certain colors and any attempt to deviate usually results in several days of pronounced hostility, acting out and inappropriate behavior. I handed him a green plaid, 50-50 blend, long sleeve Dress shirt and he wore it. (green is not one of the colors)

Derek was totally appropriate throughout the evening. He sat in a chair with the other guests smiled answered questions, talked, drank juice, ate appropriately and appeared to have a good time.

Normally he would have been sidling along a wall, shaking something, foraging for sodas, trying to eat the desserts, hanging out in a far bedroom, sifting through trash cans + hiding items (straws, sticks, cups, cans etc, on his person or outside) making faces and strange unintelligible comments.

Christie Medved Perry

APPENDIX 2

Carrie Campbell: 8 years old

Female

Auditory Memory Disorder

Diagnostic and Therapuetic Procedures:

Neural Organization Technique

Applied Kinesiology

Physiotherapy

Nutritional Supplementation

Cross Crawl

Diet - eliminate refined carbohydrates, caffiene, fried foods, pork, milk, and increase fresh fruits, vegetables, and whole grains

Hair Analysis - revealed significant levels of Toxic Metals, especially Cadmium

Supportive Documentation:

Laboratory Reports - Hair

Psychological Evaluation

Report of School Psychologist, SLD Teacher, and Current Teacher

Diagnostic Teaching Report

Auditory Discrimination Test (Follow-up)

Report Cards

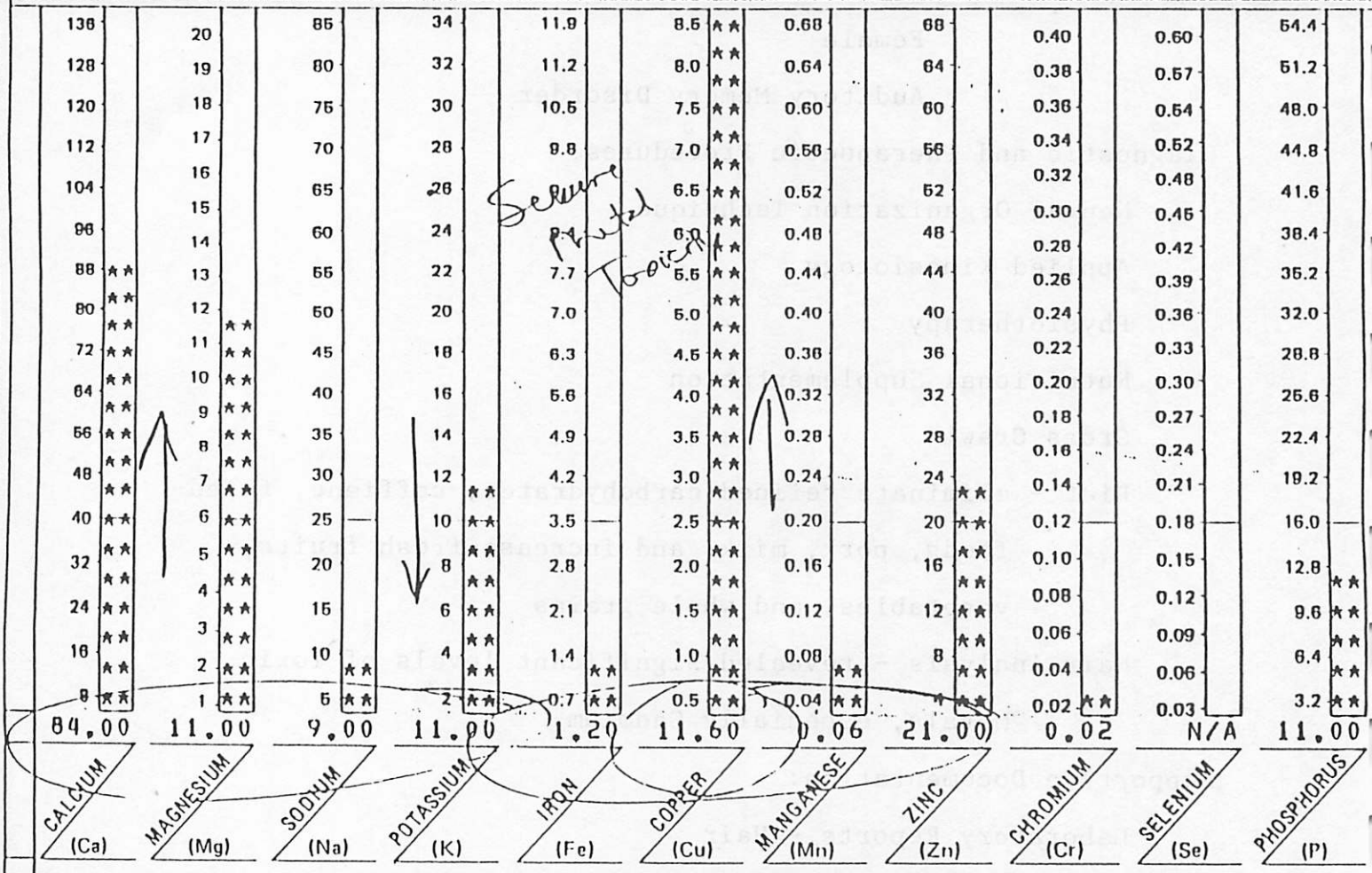
Letter from mother

ARL ANALYTICAL Research Labs, Inc.
8650 N. 22nd Ave • Phx, AZ 85021 • (602) 995-1581

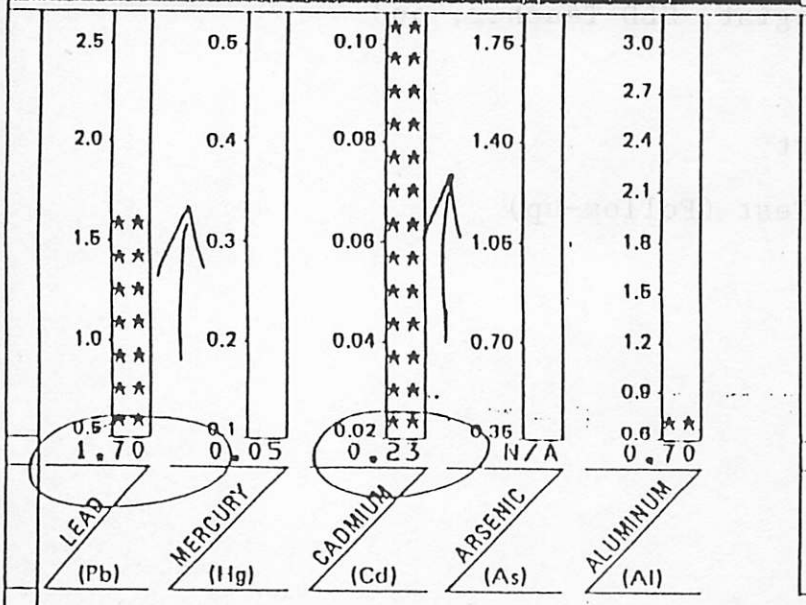
SEX: F	AGE: 8	DATE: 8/04/87	LAB NO.: 68355	CLIENT ACCT. NO.: 94308
PATIENT NAME: Carrie Campbell			REQUESTED BY: Kerry Mccord DC	

Testing By Accutrace Labs, CLIA # 02-1039 8152 N. 23rd. Ave Phoenix, Az. 85021

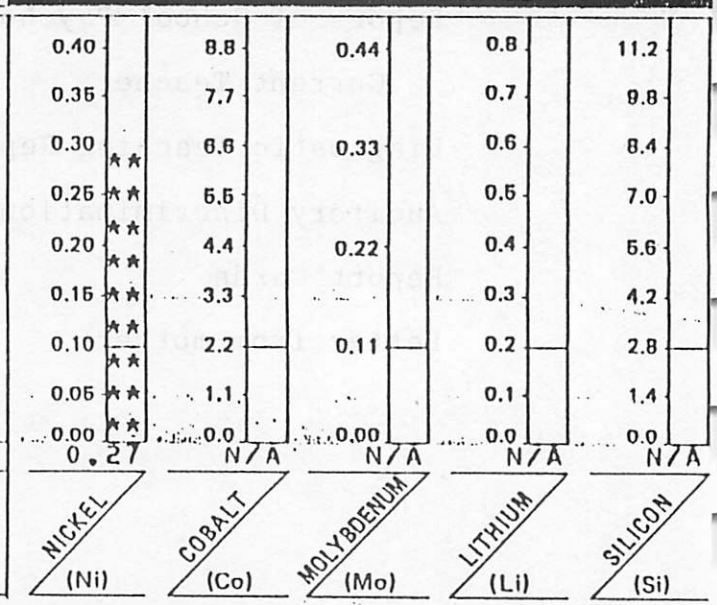
NUTRIENT MINERAL LEVELS



TOXIC METALS



ADDITIONAL MINERALS



4a

MIXED OXIDIZER ☐ FAST OXIDIZER ☐ SLOW OXIDIZER ★★★★

N/A - NOT AVAILABLE REPORTED IN mg%

Dorothy Thorne Lekarczyk, Ph.D.
Licensed Psychologist
Child, Adult and Family Services

728 East Meridian Avenue
Dade City, Florida 33525
P.O. Box 2424
Phone 904-567-6700

CONFIDENTIAL/FOR PROFESSIONAL USE ONLY

January 7, 1987

Psychological Evaluation.... Carrie Campbell

Name: Carrie Elizabeth Campbell
Date of Birth: July 28, 1979
Age at Evaluation: 7 years, 2 months
Place of Evaluation: Dade City, Florida
Referred by: Mrs. Pauline Campbell, mother
Dates of Evaluation: October 3, 1986 through November 11, 1986

Reason for Referral:

Carrie was referred by her mother, Mrs. Pauline Campbell, for evaluation and counseling. Object was to determine whether emotional factors or specific learning disabilities might be affecting Carrie's failure to complete her work and her general attitude toward schoolwork.

Evaluation Procedures:

Several clinical interviews, Wechsler Intellectual Scale for Children, revised (WISC-R), Wide Range Achievement Test, revised (WRAT-R), and Illinois Test of Psycholinguistic Abilities (ITPA). Additionally, Carrie was evaluated at school on the Gesell School Readiness Test. Finally, referral for audiological evaluation was made by this examiner to Tew A. Sak, M.D., otolaryngologist, to rule out hearing difficulties.

Desription:

Carrie is a neatly groomed, attractive seven-year-old girl with strawberry blonde hair. She dressed appropriately and neatly in casual school clothing. Carrie makes fairly good, though not consistent, eye contact, is friendly and smiles readily.

Brief History:

Carrie was born on July 28, 1979, and resides in Dade City with her mother, Pauline, and older sister, Amy. Carrie's father died approximately two years ago. Mother, and Carrie, both report good family relationships and experiences. Mother works full-time during school hours and spends a lot of time with her daughters outside of school hours.

Campbell

Observations:

Carrie was cooperative and friendly during our sessions. She tends to have a high activity level and a low attention span. Her restlessness and inattention occasionally interfered with Carrie's performance, but she generally was able to complete tasks. Carrie's behavior was appropriate throughout the testing sessions. Carrie did not indicate frustration when faced with difficult items and did not need significant urging in order to continue.

Test Results and Interpretation:

On the WISC-R, Carrie earned a Full Scale IQ score of 107, in the Average range of intellectual functioning. Her Verbal IQ was 95 (Average range), but her Performance IQ was substantially higher: 121 (Very Superior range). Carrie showed a great deal of scatter among various subtests, particularly in the Performance area. Her scaled subtest scores were as follows:

Verbal		Performance	
Information	12	Picture Completion	14
Similarities	12	Picture Arrangement	14
Arithmetic	8	Block Design	8
Vocabulary	8	Object Assembly	11
Comprehension	6	Coding	18
Digit Span	11		

Carrie's strengths were in the areas of Coding, Picture Completion, Picture arrangement, Information and Similarities. Good performance was also shown on Digit Span and Object Assembly. Carrie had substantial difficulty on the Arithmetic subtest. Auditory attention problems resulted in Carrie's needing repetitions of most of the items. Carrie showed impoverished responses to many of the Vocabulary items; constant urging was needed in order to elicit complete responses. Comprehension was another area yielding performance below average for Carrie's age.

In the Performance area, significant problems existed with Carrie's ability to copy designs. She was unable even to make a square with the blocks; rather she presented them scattered in an oddly-shaped pattern as her completed work products. Carrie completed only the first three designs and was unable to perform the next two at all.

Although Carrie did well on the Object Assembly subtest, she was poorly organized in her approach to problems, perseverated in incorrect approaches and paid excessive attention to nonessential details in completing the puzzles.

On the WRAT-R, Carrie's basic skills were good in reading (raw score 60, equivalent to beginning of 3rd grade). Her spelling was quite low and indicated lack of consistent knowledge of letter sounds: raw score was 13, equivalent to preschool level. In arithmetic, Carrie also was below her current grade level: raw scare of 17, equivalent to middle of first grade.

On the ITPA, Carrie again showed attention problems, on the Auditory Sequential Memory subtest. Problems were evident in Carrie's performance on: Auditory Reception, Auditory Memory, Visual Association and Grammatic Closure.

Campbell

Results of testing at school using the Gesell School Readiness Test were: Developmental Age = 6 to 6½ years
Chronological Age = 7 years
Range on various areas of the test = 5 years to 6½ years.
Dr. Sak, otolaryngologist tested Carrie on October 10, and reported normal hearing.

Some emotional factors may have and continue to affect Carrie's school performance, resulting from the accidental death of her father. We worked on this issue in counseling, using play therapy, art therapy and supportive counseling techniques. It is this examiner's opinion, however, that Carrie's Attention Deficit Disorder and some residual hyperactivity, along with very low spelling and math skills, are the major factors impinging upon Carrie's school performance at this time. If Carrie's deficits can be remediated through special instruction at school, she should make substantial gains in her motivation to complete schoolwork, without the attendant frustration she has been experiencing. After conversation with Mrs. Black, Carrie's teacher at Pasco Elementary, and with Mrs. Campbell, this examiner made the recommendation that Carrie by placed back in first grade. That was accomplished in November, 1986, with Carrie being moved simultaneously to Lacoochee Elementary, where her mother is employed.

Recommendations:

Further testing, if required, to determine Carrie's eligiblity for any special programs to remediate areas in which she shows deficits. Speech screening for inarticulation also is recommended. Carrie needs consistency, a great deal of structure and periodic access to a carrel in order to perform well in the classroom. She shows some emotional/behavioral immaturity and may show some substantial gains within the next year or two as she matures, as long as she has success experiences and is not unduly pressured.

Please do not hesitate to contact me if clarification of the above information is required.

Sincerely,

Dorothy Thorne Lekarczyk, Ph.D.
Florida License #2386

DTL/src

cc: Denise Wilkinson, School Psychologist ✓
Jan Ellsworth, Learning Disabilities Teacher

PASCO COUNTY SCHOOL SYSTEM
EVALUATION TEAM REPORT

NAME: Campbell, Carrie DATE: 2/2/87
BIRTHDATE: 7/28/79
SCHOOL: Lacoochee Elementary

We have reviewed all assessment data and other information concerning Carrie Campbell. We believe that this student meets eligibility criteria for placement in the Specific Learning Disabilities Program.

This decision is supported by the following:

1. There do not appear to be sensory, intellectual, emotional, or physical handicaps which would be interfering with this student's progress in school.

2. This student exhibits a process disorder in auditory memory as substantiated by selected subtests on the ITPA. This disorder is also substantiated by several depressed subtests on the WISC-R.

3. This student exhibits an academic disorder in spelling and reading which is substantiated by standard scores obtained on the WRAT-R which are at least 1 standard deviation below the intellectual standard score.

4. Diagnostic teaching of this student by the SLD teacher indicates that Carrie appears to be easily distracted. Weaknesses appear to be in auditory discrimination and some coping tasks.

There do not appear to be any relevant medical findings.

There do not appear to be economic, cultural, or environmental disadvantages which would significantly affect this student's performance in school.

There appears to be a severe discrepancy between achievement and ability which is not correctable without special education and related services. We recommend placement on Plan C with the focus of her time spent on auditory memory training and auditory discrimination. This recommendation is offered because she is currently performing at grade level academically in her classroom.

Denise Wilkinson
School Psychologist

Janice H. Ellsworth
SLD Teacher

Mary Anne Sherrod
Current Teacher

2/3/87
gcf
2/4/87

DISTRICT SCHOOL BOARD OF PASCO COUNTY

Diagnostic Teaching Report

Name Campbell, Cassie
Date of Birth 7/28/79
Type of Referral: Initial ✓
Temporary Placement
School LES Grade 1st
SLD Teacher J. Ellsworth
Diagnostic Hours 3
Date(s) Seen Jan. 1987
Date of Report

Suspected Areas of Difficulty to be Explored in this Report
(check or explain those that apply)
Math ✓
Reading ✓
Spelling ✓
Written Language
Process
Other
Strengths

Assessment Strategies

Activity/Area Brigance - Readiness
Results Carrie's readiness skills are very good. However, she did not recognize the color purple. Some rotations and distortions were noted on copying designs. She called lower case "l", "i". She also confused m + n when letters were printed.
Behavioral Observations Carrie was cooperative throughout testing. She appeared to try very hard to concentrate.

Activity/Area Brigance - Word Analysis -
Results Carrie was successful on 2 of 8 word pairs when words were said and she had to tell if the word pairs were the same or different. She knew all the vowel names + short vowel sounds a, e, i, o - long vowels known, u
Behavioral Observations Carrie was much more distractable during this segment of testing.

Activity/Area Brigance - Vocabulary
Results In all areas tested in this section, Carrie did very well. Her scores were well above grade level.

Behavioral Observations Carrie attended fairly well in this section, however, several times she asked to have the question repeated.

White: Cumulative Folder
Canary: Program Folder

Activity/Area Spelling - Brigance and Test of Written Spelling - 2

Results Approximate grade level on Brigance 1st. Total percentile on TWS-2 was 63%.

Behavioral Observations Carrie worked slowly and carefully. Several times she asked to have a word repeated.

Activity/Area Math - Key Math

Results Overall grade level approx 2.2 Strengths emerged in Numeration, Numerical Reasoning, Geometry + Symbols + Measurment. Weakest areas were in Mental Computation, Money, Word Problems, Addition + Subtraction.

Behavioral Observations Carrie gave up easily + needed to be encouraged. During + aid -, Carrie counted on her fingers.

Summary of Findings Carrie appears to be very easily distracted. Academic deficits appear in Auditory Discrimination and some copying tasks. Math weaknesses are in basic facts + Mental Computation as well as other areas mentioned previously. Word attack skills may also be weak.

Teacher Signature Janice H. Ellsworth

Date 11/30/87

White: Cumulative Folder
Canary: Program Folder

Neural Organization Technique/McCord

AUDITORY DISCRIMINATION TEST

Joseph M. Wepman
(Revised 1973)

Form I A

Name Carrie Campbell
Date 12/5/87
Age 8 years 4 months

Examples:			Different	Same
Man	–	Man		+
fist	–	fat	+	

Record response correct (+) or incorrect (–) in undecided box. Do not add examples to score.

			Different	Same
1.	tub	– tug	+	
2.	lack	– lack		+
3.	web	– wed	+	
4.	leg	– led	+	
5.	chap	– chap		+
6.	gum	– dumb	+	
7.	bale	– gale	+	
8.	sought	– fought	+	
9.	vow	– thou	+	
10.	shake	– shape	+	
11.	zest	– zest		+
12.	wretch	– wretch		+
13.	thread	– shred	+	
14.	jam	– jam		+
15.	base	– bath	+	
16.	tin	– pin	+	
17.	pat	– pack	+	
18.	dim	– din	+	
19.	coast	– toast	+	
20.	thimble	– symbol	+	
21.	cat	– cap	+	
22.	din	– bin	+	
23.	lath	– lash	+	
24.	bum	– bomb	+	
25.	clothe	– clove	+	
26.	moon	– noon	+	
27.	shack	– sack	+	
28.	sheaf	– sheath	+	
29.	ding	– king		+
30.	badge	– badge		+
31.	pork	– cork	+	
32.	tie	– thigh	–	
33.	shoal	– shawl	+	
34.	tall	– tall		+
35.	par	– par		–
36.	pat	– pet	+	
37.	muff	– muss	+	
38.	pose	– pose		–
39.	lease	– leash	+	
40.	pen	– pin	–	

Acidity= 8/10
Number correct in SAME columns

Score= 28/30
Number correct in DIFFERENT columns

Interpret Score turn to page 4 in the Manual.

Rating Scale Range: _____

DISTRICT SCHOOL BOARD OF PASCO COUNTY
Elementary Report Card
19 86 - 19 87 School Year

School: Pasco Elementary/Lacoochee Elementary

Student's Name	Grade Level
Carrie Campbell	2/1

Homeroom Teacher: Mrs. Black/Mrs. Sherrod
Reading Teacher:
Math Teacher:

GRADE CODING INFORMATION

S-Satisfactory U-Unsatisfactory N-Needs Improvement

Art, Music, Band, Chorus, Handwriting, Health, Conduct, Work Habits, Physical Education

SUBJECTS (Progress is reported in two ways)

Progress in relation to this child / Progress in relation to grade level

A - Excellent progress (94-100%) 1 - Above grade level
B - Above average progress (86-93%) 2 - Grade level
C - Average progress (78-85%) 3 - Below grade level
D - Below average progress (70-77%)
F - Unsatisfactory progress (0-69%)

PREP STRATEGY ASSIGNMENT (1-3 only)

Report Period	Preventive 1	2	3	4	Develop 1	2	3	4	Enrichment 1	2	3	4
Reading/LA	✓					✓	✓	✓				
Math	✓					✓		✓				
Health/Phys.	✓					✓	✓	✓				
Soc./Behavioral	✓					✓	✓	✓				

REPORT PERIOD	1	2	3	4
Days present	42	[illegible]	42	47
Days absent	1	1	2	0
Days tardy	0	0	0	0
Conduct	S	S	S	S
Work habits	N	N	S	S

PRIMARY READING KEY: R-Readiness P-Pre-Primer P-Primer

	1	2	3	4
Reading Grade Level	1	1	1	1
Reading Grade	C2	A2	A2	B2
Language	B2	A2	A2	A2
Spelling	A2	A2	A2	A2
Handwriting	N	S	S	S
Mathematics	C2	[illegible]2	B2	B2
Science	B2	A2	A2	A2
Social Studies	B2	A2	A2	A2
Health	S	S	S	S
Physical Education	S	S	S	S
Art	S	S	S	S
Music	S	S	S	S
Band				
Chorus				

Teacher Recommendation:
☒ Promoted ☐ Retained Summer School
☐ Retained ☐ Preventive Summer School
☐ Administrative Placement

I am proud of your improvement! [illegible] Mrs. Sedlack

Mrs. Sedlack

WOW Mrs. Sedlack

Mary Anne Sherrod
Teacher's Signature 41

TEACHER COMMENTS

Report 1 ( ) Parent Conference Requested
[illegible]

Report 2 ( ) Parent Conference Requested
[illegible]

Report 3 ( ) Parent Conference Requested
Carrie's work habits have improved tremendously. She has developed confidence and independence. Please encourage and praise her daily. I am very proud of Carrie.

Report 4 ( ) Parent Conference Requested
Carrie is a beautiful young lady. It has been a real privilege to be her teacher. Her work habits are terrific and she has a good attitude about school. She is ready for second grade.

Neural Organization Technique/McCord

DISTRICT SCHOOL BOARD OF PASCO COUNTY

Elementary Report Card

19 87 - 19 88 School Year

School Lacoochee Elementary

Student's Name	Grade Level
Carrie Campbell	2
Homeroom Teacher: Mrs. Sherrod	
Reading Teacher: //	
Math Teacher: //	

GRADE CODING INFORMATION

S-Satisfactory U-Unsatisfactory N-Needs Improvement

Art, Music, Band, Chorus, Handwriting, Health, Conduct, Work Habits, Physical Education

SUBJECTS (Progress is reported in two ways)

Progress in relation to this child	Progress in relation to grade level
A - Excellent progress (94-100%)	1 - Above grade level
B - Above average progress (85-93%)	2 - Grade level
C - Average progress (75-84%)	3 - Below grade level
D - Below average progress (65-74%)	
F - Unsatisfactory progress (0-64%)	

PREP STRATEGY ASSIGNMENT (1-3 only)

	Preventive				Develop				Enrichment			
Report Period	1	2	3	4	1	2	3	4	1	2	3	4
Reading/LA						✓						
Math						✓						
Health/Phys.						✓						
Soc./Behavioral						✓						

REPORT PERIOD	1	2	3	4
Days present	45	47		
Days absent	0	0		
Days tardy	0	0		
Conduct	S	S		
Work habits	S	S		

PRIMARY READING KEY: R-Readiness PP-Pre-Primer P-Primer

Reading Grade Level	2	2			
Reading Grade	A2	A2			
Language	A2	A2			
Spelling	A2	A2			
Handwriting	S	S			
Mathematics	A2	A2			
Science	A2	A2			
Social Studies	A2	A2			
Health	S	S			
Physical Education	S	S			
Art	S	S			
Music	S	S			
Band	/				
Chorus	/				

TEACHER COMMENTS

Report 1 ( ) Parent Conference Requested

Carrie is having a successful year in second grade. She is motivated, confident about herself and her learning. She seems to be very happy.

Report 2 ( ) Parent Conference Requested

Carrie is a delightful child. She is enthusiastic and loves learning. I am very pleased with her progress.

Report 3 ( ) Parent Conference Requested

Report 4 ( ) Parent Conference Requested

Teacher Recommendation
- ☐ Promoted
- ☐ Retained
- ☐ Administrative Placement
- ☐ 6 Retained Summer School
- ☐ 2 Preventive Summer School

Teacher's Signature

3/10/87

A year and a half ago I was a very frustrated mother. My younger daughter was having problems in second grade. She was not completing her assignments even though she was on grade level in all subjects. She wandered around the room, was very emotional, and was becoming a behavior problem. Evenings at home were full of tension as I tried to make her finish her work.

My first impression was that she was developmentally young. I had been asking her teachers if they thought she need to be retained since the middle of kindergarten. Her teachers all felt that the death of her father when she was five was contributing to her problem. Following a conference with my daughter's second grade teacher, I requested that Carrie be tested for SLD and also decided to take her to a psychologist for diagnosis and counseling. In November, 1986 Carrie was replaced in a first grade class at a different school.

Her grades improved immediately except for work habits. With the

help of a very positive and patient teacher, ever that grade improved. She still showed some difficulty with math concepts. She qualified for SLD with a deficit in auditory memory. I later learned that she scored at a very low level on the auditory discrimination test given. She also showed a weakness in math. The replacement in another grade had definitely helped, but there was still a definite problem.

A friend in the school system told me about Dr. McCord and his work. I had read an article about Chiropractors and dyslexia and had been discouraged to even consider it. But when your child has a problem, you're willing to try almost anything as long as it won't hurt her. And so, with mixed feelings, I took Carrie to Dr. McCord. I didn't understand everything he was doing with Carrie and I even thought the whole procedure was a little crazy. How could putting her thumbs in her mouth, fingers on her temples or in her ears help her learning problems? What correlation did her

response to Dr. McCord pushing on her leg have to do with her problem? I watched every manipulation carefully to make sure Carrie was not hurt. She expressed very little discomfort and never dreaded going back.

Carrie was also placed on a vitamin supplement program and a diet restricting preservatives, sugar, etc. and promoting more natural foods.

I know the replacement helped Carrie, but the NOT, supplements, and diet have carried her further. She is an extremely happy, confident child. She has made straight A's for two quarters this year. Her math is fantastic. She grasped regrouping very quickly and has often scored higher than most (sometimes all) of her classmates. She was retested on the auditory discrimination test and showed great improvement. The S.L.D. teacher also noticed that she was able to attend longer and was not as restless.

I still don't know how all of NOT works, but I know I don't want to discontinue it.

Polly Campbell

APPENDIX 3

John (Rusty) Wagner: 7 years old

Male

Dyslexic, Coordination Dysfunction,

Immaturity

Diagnostic and Therapuetic Procedures:

Neural Organization Technique

Applied Kinesiology

Physiotherapy

Nutritional Supplementation

Cross Crawl

Diet - eliminate refined carbohydrates and increase fresh fruits, vegetables and whole grains

Supportive Documentation:

Occupational Therapy Evaluation

Kaufman Assessment Battery for Children

Letter from Occupational Therapist (follow-up)

Report Cards

Letter from mother

OCCUPATIONAL THERAPY EVALUATION

NAME: Rusty Wagner

SCHOOL: Woodland Elementary

DATE OF BIRTH: 6-9-80

PROGRAM: K-3

PHYSICIAN: Dr. Sokol, M.D.

TEACHER: Mrs. Reed

MEDICATIONS: None

DIAGNOSIS: Undetermined

PRECAUTIONS: None

PERTINENT HISTORY: Rusty was born with crossed eyes which have been corrected surgically. The right eye is still slightly deviated in. Rusty was referred due to perseverative behavior and behavioral problems in the classroom. A definitive diagnosis has not been made at this time.

TESTS ADMINISTERED:

Bruininks - Oseretsky Test of Motor Proficiency
Draw-A-Man Test
Inventory of Basic Learning Abilities

POSITIONS OF POSTURE AND MOVEMENT: Rusty came willingly to the evaluation area. His appearance is that of a slightly built 6-1/2 year old boy who walks unassisted. His posture is such that the pelvis is tipped forward, internal rotation of the leg is seen at the hips and pigeon toe walking with more rotation seen on the right. There is fixation of the upper trunk with retraction of the shoulders observed and protrusion of the scapula. There is little movement of the upper trunk when walking but arm swing is noted to be WNL. Rusty's affect is flat with little facial expression. His eyes appear tiny with a far away look. Eye contact is poor.

GROSS MOTOR SKILLS:: Upon testing, Rusty exhibited fluctuating performance in this area. It appears that there is a generalized delay in motor-planning. He can perform most tasks but not without extra effort and thought on his part. He posture himself. he wants very much to please others and will verbalize the tasks he knows he does well often. He tries to avoid difficult tasks by stating he is tired.

When catching a ball, Rusty will sometimes cross his eyes. He postures his arms in anticipation of the ball throw. Upper-lower limb tasks were difficult and uncoordinated. Strength, however, was fairly good throughout but endurance was only fair. Range of motion was WNL. Rusty cannot skip.

PERCEPTUAL/FINE MOTOR SKILLS:: Rusty demonstrated that he is right handed and footed but left eyed. Motorically, Rusty can perform most tasks but exhibits immature adaptive hand skills in scissor cutting and fine-finger coordination. Speed and dexterity appear to be WNL for most tasks. No tremor is noted. Eye-tracking appears WNL in all planes but convergence is nearly absent. It was observed on several occasions that Rusty turned his head to adjust his vision using the right eye more than the left. He also holds his head sometimes only five inches from his work. Design copying was done well.

When drawing a person, Rusty starts with the feet and goes up towards the head. His person has 10 parts. The most outstanding deficit of this category is Rusty's perseverative behavior. For more than a year, he has been fascinated with stop signs, stop lights and green, red and yellow colors. He persistently draws these items and chooses these colors and becomes upset if one attempts to change to something else once he starts drawing. He freely verbalizes how much he likes these objects and sometimes carries little pictures of them with him. He perseverates on other things as well according to his mother.

Behavior in the classroom has been reported by the teacher as follows: a desire to be by himself, destroying other students work and pushing/shoving while standing in line.

SENSORY-MOTOR INTEGRATION: Rusty displayed difficulty standing on his preferred foot (right) for balance tasks. Equilibrium responses appeared to be in tack. Vestibular function appears in tack but immature. Rusty seldom chooses to move his head out of the horizontal plane or be in a position of stress in this area. Processing is taking place but it is delayed in gross-motor tasks especially.

DAILY LIVING TASKS:: Independent for age level.

RECOMMENDATIONS: It is recommended that Rusty receive occupational therapy servies due to a sensory-integration deficit. At this time, however, Rusty is not enrolled in another Exceptional Student Education (ESE) program and is not eligible for occupational therapy program at his school. Should he in the future be found eligible for another ESE program, he can become eligible for occupational therapy at his school.

Kay Ables, OTR/L

Kay Ables, OTR/L/Date: Jan. 5, 1987

KA/drb

Wagner, Rusty
Page Two

Kaufman Assessment Battery for Children (K-ABC) (Continued)

Achievement Subtests

Subtest	Percentile Rank	Standard Score
Faces & Places	2	70
Arithmetic	47	99
Riddles	37	95
Reading/Decoding	42	97

Global Scales

Scales	Percentile Rank	Standard Score
Sequential	45	98
Simultaneous	63	105
Mental Processing Composite (MPC)	55	102
Achievement	21	88

On the K-ABC Rusty obtained a Mental Processing Composite standard score of 102. This suggests that his level of intellectual functioning is within the average range. His abilities to problem-solve using step-by-step or gestalt-like methods are relatively equal. Analysis of mental processing subtests performance indicate a significant strength in the area of visual perceptual organization and a significant weakness in the area of sequencing pictures to tell a story. The strength in the area of perceptual organization is consistent with Rusty's father's report that he enjoys building and using his hands to create things.

On the Achievement subtests Rusty demonstrated adequate levels of achievement in arithmetic, vocabulary development, and reading. He demonstrated a significant weakness in factual knowledge.

SUMMARY AND RECOMMENDATIONS:

Currently Rusty is repeating kindergarten. He was cooperative during assessment. Results suggest that he is functioning within the average range with adequate levels of academic achievement. His ability in the area of perceptual organization appears to be adequate. Recommendations include the following:

1. A evaluation team and in-school staffing committee should review assessment results in order to assist with educational planning.

2. If factual knowledge is a weakness for Rusty, efforts should be made to increase his knowledge of his environment. For instance he could not identify pictures of well-known fairy tale characters. Reading a variety of stories to Rusty should improve his awareness.

Wagner, Rusty
Page Three

3. Since sequencing pictures to tell a story appears to have been difficult for Rusty, it is suggested that he practice this type of task.

Report from school psychologist supplied by Judith Wagner, (Rusty's mother).

District School Board of Pasco County

7227 U. S. Highway 41 Land O' Lakes, Florida 33539 813-996-3600

Thomas E. Weightman
Superintendent

Oma M. Pantridge, Director
Exceptional Student Education

October 6, 1987

McCord Chiropractic Center
6110 Ninth Street North
St. Petersburg, FL 33703

Dear Dr. McCord:

Enclosed are copies of the results of the initial evaluation and re-evaluation on Rusty Wagner.

To assist you in gaining additional information from my testing, I will elaborate on the results of the Bruininks test and others I did. The Bruininks in the only standardized test I administered.

Rusty has consistently demonstrated left eye dominance but hand and foot right dominance. I'm not sure if this mixed dominance is acquired or natural, but it may be the cause of some of his difficulties. I have never tried to switch eye dominance when it wasn't consistent with hand dominance. Do you have any research on this?

The results of the Bruininks re-evaluation show an overall increase in percentile rank. Although Rusty falls into the average category, I've always been aware that we were looking at the quality of his performance as well as could he or couldn't he perform a task. His progression in fine motor is best but he has not kept up with the population in gross motor. This test does not test frustration and certainly we see avoidance in many tasks that are difficult for him. I feel the results are fairly valid.

Page 2

I have used various other tests to evaluate perceptual performance. I am still seeing deficits in figure ground differentiation, directionality, right/left discrimination, laterality, auditory sequencing, visual acuity, visual coordination and pursuit, visual motor memory, visual motor speed of learning, and visual motor integration. I am involving Rusty in many different exercises (blackboard and paperwork) to address all these areas. He is showing improvement especially in right/left discrimination, laterality, and figure ground discrimination.

In gross motor we have done a number of things. I used an exercise program during the summer where I saw Rusty for 2 hours a week for 5 weeks, to strengthen the trunk, pelvis, and shoulder girdle. Then I have gone to some ball skills, balancing, jumping, scooterboard and other assorted tasks to continue this into functional tasks. These are a few of the things I see and am still concerned about:

Walking - due to toe-in gait, Rusty has difficulty walking a heel-toe stepping pattern on a tape line or balance beam. He can't keep hands on hips.

Running - Rusty has difficulty changing directions on command, and doesn't appear to use his vision well when running.

Ball catching - 10" ball from 10 feet - best
6" ball from 10 feet - often trapped
tennis ball from 10 feet - often missed

We work on ball catching, target throwing, and dribbling tasks. He has trouble coordinating hands, feet, and vision for these tasks. He is showing that he can learn this skills, however.

Balance - Rusty persists in assuming various abnormal postures to accomplish balance tasks. Has to use his arms a lot to maintain balance.

Broad jump - Okay.

Hopping - Very uncoordinated. He shows how much he has to think to accomplish one foot hopping tasks.

Page 3

Body posture Imitation - This is especially difficult and needs continued practice and reassurance.

Muscle strength - improvement noted proximately.

I hope this will help you. Rusty's general affect is changing to be more relaxed. His confidence is improving. He is more receptive to teaching now, also. We're not through with him, but I'm sure we're on the right track and I appreciate what you have done on your part. I can tell the difference in his organizational ability. Let me know if you have further questions or concerns. I would welcome input from you also.

Sincerely,

Kay Ables, OTR/L

Kay Ables, OTR/L

KA/dw

Enclosure

Student's Name John Wagner

Teacher Mrs. June Reed

S-Satisfactory N-Needs Improvement U-Unsatisfactory
H Skill has not been evaluated
*Achievement is considered above grade level

SOCIAL DEVELOPMENT	1	2	3	4
Knows address	N	S	S	S
Knows telephone number	S	S	S	S
Knows birthdate	S	S	S	S
Practices self-control	S	S	S	S
Respects rights and property of others	N	S	S	S
Participates in group activities	S	S	S	S
Displays self-confidence	S	S	S	S
Is willing to take turns and share	N	S	S	S
Follows classroom rules	N	S	S	S
Respects authority	S	S	S	S
Rests quietly	N	S	S	S
WORK SKILLS				
Works independently	N	S	S	S
Completes work in reasonable amount of time	N	S	S	S
Listens attentively and follows directions	N	S	N	S
Works without disturbing others	N	S	N	S
LANGUAGE ARTS				
Recognizes own name	S	S	S	S
Writes name correctly	N	S	S	S
Recognizes and names 8 basic colors	S	S	S	S
Categorizes objects	S	S	S	S
Matches objects as to size, shape and color	S	S	S	S
Matches like letters and words	S	S	S	S
Participates in group discussions	N	S	N	S
Communicates experiences verbally	S	S	S	S
Uses complete sentences	S	S	S	S
Identifies words that rhyme	-	-	-	S
Retells events in sequence	-	-	-	S
Identifies beginning sounds	S	S	S	S
Demonstrates clearly left to right progression	-	-	-	S
Identifies ending sounds	-	-	-	S
Recognizes sight words	-	-	-	S

Book 1 Level County Reading Series

MOTOR SKILLS
M Skill has been introduced
∞ Skill has been mastered

LARGE MUSCLE
- ∞ Balance beam
- ∞ Jumps
- ∞ Hops
- ∞ Skips
- ∞ Catches and Throws

SMALL MUSCLE
- ∞ Holds pencil/crayon correctly
- ∞ Traces w/reasonable accuracy
- ∞ Cuts w/reasonable accuracy
- ∞ Buttons
- ∞ Ties shoes

M Letter has been taught but not yet mastered
∞ Letter has been mastered
I I Letter has not been evaluated

Identifies letters of the alphabet

Aa Bb Cc Dd Ee Ff Gg Hh Ii Jj Kk Ll Mm
∞ ∞ ∞ ∞ ∞ ∞ ∞ ∞ ∞ ∞ ∞ ∞ ∞
Nn Oo Pp Qq Rr Ss Tt Uu Vv Ww Xx Yy Zz
∞ ∞ ∞ ∞ ∞ ∞ ∞ ∞ ∞ ∞ ∞ ∞ ∞

Identifies phonetic sounds of the alphabet

A B C D E F G H I J K L M N O P Q R S T U V W X Y Z
∞ ∞

TEACHER RECOMMENDATION:
- [X] Promoted
- [ ] Retained
- [ ] Administrative Placement
- [ ] Retained Summer School
- [ ] PREP Preventive Summer School
- [ ] Advance to ____

Mrs. June Reed
Teacher's Signature

DISTRICT SCHOOL BOARD OF PASCO COUNTY

Kindergarten Report Card
1986 - 1987 School Year

MIS # 529
Rev. 7/85

School Woodland Elem

MATHEMATICS (circled indicates mastery)	1	2	3	4
Names geometric shapes ○□△◇	S	S	S	S
Matches objects one to one	S	S	S	S
Counts orally 1 2 3 4 5 6 7 8 9 10	S	S	S	S
Counts objects 1 2 3 4 5 6 7 8 9 10	S	S	S	S
Names numerals 0 1 2 3 4 5 6 7 8 9 10	S	S	S	S
Matches numerals to sets 0 1 2 3 4 5 6 7 8 9 10	S	S	S	S
Writes numerals 1 2 3 4 5 6 7 8 9 10	S	S	S	S
Names penny, nickel, dime, quarter	S	S	N	S
Ordinal numbers (first, second, third, fourth, fifth)	S	S	S	S
Demonstrates an understanding of quantity (e.g., whole, half, more than, less than)	-	S	S	S
Identifies spatial relationships: up/down, front/back, over/under, near/far, inside/outside	S	S	S	S
Classifies objects as: short/tall, light/heavy, big/little, small/large	-	-	S	S
Demonstrates an understanding of addition and subtraction processes *	-	-	-	S
SCIENCE	S	S	S	S
HEALTH	S	S	S	S
SOCIAL SCIENCE	S	S	S	S
MUSIC	S	S	S	N
ART	S	S	S	S
PHYSICAL EDUCATION	S	S	S	S

P.R.E.P. STRATEGY ASSIGNMENT

	Preventive				Developmental				Enrichment			
REPORT PERIOD	1	2	3	4	1	2	3	4	1	2	3	4
Reading/LA	—	—	—	—	✓	✓	✓	✓	—	—	—	—
Math	—	—	—	—	✓	✓	✓	✓	—	—	—	—
Health/Phys.	—	—	—	—	✓	✓	✓	✓	—	—	—	—
Soc./Behavioral	—	—	—	—	✓	✓	✓	✓	—	—	—	—

ATTENDANCE

REPORT PERIOD	1	2	3	4
Days Present	42	46	34	33
Days Absent	1	0	5	2
Days Tardy	0	0	0	0

TEACHER COMMENTS

Report 1 ( ) Parent Conference Requested

Rusty needs to improve his classroom behavior. He continues to do well with his class work.

Report 2 ( ) Parent Conference Requested

Rusty's behavior is improving. He is doing really well with his class work.

Report 3 ( ) Parent Conference Requested

Rusty continues to do well with his classwork.

Report 4 ( ) Parent Conference Requested

Have a nice summer and good luck next year.

DISTRICT SCHOOL BOARD OF PASCO COUNTY

Elementary Report Card

19 87 - 19 88 School Year

School _____

GRADE CODING INFORMATION

S-Satisfactory	U-Unsatisfactory	N-Needs Improvement
Art	Chorus	Conduct
Music	Handwriting	Work Habits
Band	Health	Physical Education

SUBJECTS (Progress is reported in two ways)

Progress in relation to this child	Progress in relation to grade level
A - Excellent progress (94-100%)	1 - Above grade level
B - Above average progress (85-93%)	2 - Grade level
C - Average progress (75-84%)	3 - Below grade level
D - Below average progress (65-74%)	
F - Unsatisfactory progress (0-64%)	

Student's Name WAGNER, Rusty Grade Level

Homeroom Teacher / A Smith

Reading Teacher / B. Pruett

Math Teacher / Wade

PREP STRATEGY ASSIGNMENT

(1-3 only)	Preventive				Develop				Enrichment			
Report Period	1	2	3	4	1	2	3	4	1	2	3	4
Reading/LA					✓	✓						
Math					✓	✓						
Health/Phys.					✓	✓						
Soc./Behavioral					✓	✓						

REPORT PERIOD	1	2	3	4
Days present	44	46		
Days absent	1	1		
Days tardy	0	0		
Conduct	S	S		
Work habits	S	S		

PRIMARY READING KEY: R-Readiness PP-Pre-Primer P-Primer

Reading Grade Level	PP	P		
Reading Grade	1.2	1.2		
Language	1.2	1.2		
Spelling	1.2	1.2		
Handwriting	S	S		
Mathematics	1.2	1.2		
Science	1.2	1.2		
Social Studies	1.2	1.2		
Health	S	S		
Physical Education	S	S		
Art	S	S		
Music	S	S		
Band	/	/		
Chorus	/	/		

TEACHER COMMENTS

Report 1 ( ) Parent Conference Requested

Report 2 ( ) Parent Conference Requested

Rusty does excellent work in language arts. LB

Report 3 ( ) Parent Conference Requested

Report 4 ( ) Parent Conference Requested

Teacher Recommendation:

[ ] Promoted
[ ] Retained
[ ] Administrative Placement
[ ] 1-6 Retained Summer School
[ ] 1-2 Preventive Summer School

5i

Rusty is 7 years old and is in the first grade. He spent 2 years in kindergarten. After the first year, his teacher recommended he repeat, and she felt it was due mainly to immaturity. During this second year, the same problems persisted. They were:

1. Not being able to complete assignments within the designated time.
2. Preferring to lay down instead of sitting
3. Reversing letters
4. Not participating with the other kids
5. Could not skip or catch a ball
6. He would spend all his time doing one thing for days at a time.

After consulting with his teacher, the school psychologist, an occupational therapist and the placement specialist, Dr. McCord was suggested. That was in March. He has now been working with Dr McCord for 9 months, and she has also been working with the school occupational therapist. She has been working on strengthening his upper body, balancing, catching, and other coordination activities.

Rusty is now able to sit through class. He no longer reverses letters. He plays with other children more often. He is able to skip and catch a ball, however he still needs work in that area. We are also still working on completing assignments and not spending quite so much time doing the same thing, even though these two areas have improved. I sincerely feel Dr. McCord was an answer to prayer.

Judith B. Wagner
3/11/88

APPENDIX 4

Jamie Anderson: 5 years old

Female

Dyslexic, Auditory Memory Disorder, Visual Memory Disorder

Diagnostic and Therapuetic Procedures:

Neural Organization Technique

Applied Kinesiology

Physiotherapy

Nutritional Supplementation

Cross Crawl

Diet - eliminate refined carbohydrates and increase fresh fruits, vegetables and whole grains

Supportive Documentation:

Letter from mother (School Board Staffing Specialist)

March 14, 1988

To: Dr. Kerry McCord

From: Joanne W. Anderson

RE: NOT for Jaime Nicole Anderson

I am writing this letter to document results of the neurological organization test for Jaime Nicole Anderson (4/4/81). Please check medical file located in the office of Dr. Kerry McCord, for specific dates.

My daughter, Jaime, entered kindergarten in the Fall of 1987. She was subsequently evaluated both formally and informally by her classroom teacher, Mrs. Peggy Riggs, and the following results were evidenced:

1. Jaime evidenced numerous letter reversals in writing and also perceived them visually in that manner.

2. Jaime evidenced significant auditory memory problems. This manifested itself in her difficulty in associating the written symbol with a spoken sound.

3. Jaime evidenced a visual memory problem which manifested itself in the inability to retain knowledge of written words. In other words- she would learn materials and appear to have mastered it, then would "forget" what she had learned.

4. Jaime lacked confidence in her ability to perform academically as well as physically. She would not take "risks" that she needed to take physically in order for the next stage to develop.

5. Jaime appeared "hyper" at times, especially when required to remain on task for several minutes. When reading her a story, she would often lose interest at mid-point and refuse to listen to the conclusion.

In spite of these difficulties, Jaime had many strengths and was considered to be a very intelligent person. She evidenced excellent long term memory in her ability to recall times, places, and things. She had a vocabulary well advanced for her age, and social skills were excellent. (I am speaking in the past tense, due to documentatio

of the past. This is still true of her, however.)

Jaime was referred to Dr. McCord for her classroom difficulties because she was in the lowest reading group in her class and she was suffering in terms of frustration and self-concept. She was evaluated and found to be evidencing physiologically what was being manifested in the classroom. Jaime's teacher was not informed of this intervention.

After Jaime's second visit with Dr. McCord, her teacher called me in for a conference. Her words were, "I don't know what you have done with Jaime, but whatever it is, keep doing it." "It's like a light has turned on in her head." Jaime began to readily grasp the materials presented in the classroom and by the third quarter in the school year, she was placed in the top reading group in her class.

After the third evaluation- Jaime did something really exciting. We had been working with her on learning to ride her bicycle. On Sunday we spent some time in that endeavor. She was completely unable to balance. On Thursday she went for her NOT. When we returned home, Jaime "disappeared" for a time. When she came into the house, she asked me and her dad to come outside because she had something to show us. She then proceeded to ride her bicycle halfway down her street. When asked for an explanation of how she had been able to do this, she said, "I have my balance now."

Jaime is now in the first grade. She has made the honor roll both grading periods and is in the top reading and math group. She has proven to be an excellent student. Her attention span is better than average and we notice this at home as well. Her self-concept has improved dramatically since her NOT and she is now readily taking "risks" that she needs to take in order to continue her mental, intellectual, and physical growth. Evidence of having a learning disability has been removed.

I have referred several students for this evaluation. Learning disabilities are extremely common to find in school children today. I believe that this intervention is extremely helpful in diagnosing and remediating this dysfunction, and I wish all children in need had access to it.

Sincerely,

Joanne W. Anderson

Joanne W. Anderson

BIBLIOGRAPHY

1 Carl A. Ferreri, D.C., Ph.C., and Richard B. Wainwright, D.C., Ph.C., Breakthrough For Dyslexia and Learning Disabilities, Exposition Press of Florida, Inc. 1984, p. 4

2 Carl A. Ferreri, D.C., Ph.C., N.O.T. Seminar, Tampa, Florida, October 1986

3 George J. Goodheart, Jr., D.C., Research Tape #77, Side 2, 370, Privately Published, 1985 (approx)

NOTE: All patient and physician reports included in this paper are published with permission from each of the individuals cited.

"COP-OUT" - TREATING THE COMMON PROBLEM OF EXCESS COPPER

Thomas Rogowskey, D.C. and Walter H. Schmitt, Jr., D.C.

There has been a lot of attention given recently to proper copper levels in the body. Copper deficiency has been discussed by Liebowitz (1) and Brea (2) especially regarding its importance allergies. Copper excess has been discussed by Mowles and Schmitt. (3,4,5) Excess copper patients are common in rural areas where well water is found to be high in copper content and in urban or rural areas of acidic water and copper pipes. Urban areas may be populated by emigrated patients from areas of high copper. Vegetarians can also show copper excess due to the high copper content of vegetarian diets. We all have patients in our practices who have hidden copper excesses.

Patients with excess copper are quite commonly encountered as can be seen by the wide variety of typical excess copper symptoms which include: premenstrual syndrome and menstrual irregularities, anemia, idiopathic scoliosis, other spinal instabilities, Candida albicans syndrome, and functional liver problems including blood sugar handling problems.

You should begin to think of excess copper as a problem in a patient who shows a need for copper's antagonists: zinc, manganese, molybdenum, and/or iron. Also, excess copper patients often show a need for folic acid and/or pantothenic acid, presumably caused by the presence of the copper. (4) Suspect excess copper in patients who demonstrate needs for more than one or two of these nutrients, or who continue to show a need

for these nutrients even though they are being supplemented. Laboratory verification of high copper can be attained by hair analysis, red blood cell analysis, and 24 hour urine analysis.

COP-OUT was designed as a product to aid in the body's elimination of excess copper. It is composed of zinc, manganese, molybdenum, iron, folic acid, and pantothenic acid. These are the nutrients which are frequently found necessary when there is excess copper. To avoid having the patient taking as many as six different pills at a time, COP-OUT was designed.

COPPER EXCESS AND MENSTRUAL RELATED PROBLEMS

Mowles performed an excellent study of copper excess in women with premenstrual syndrome and all of its associated symptoms. (3) He found a high correlation with not only premenstrual syndrome symptoms and elevated copper, but a parallel between symptom remission and decreased body copper levels.

Females with excess copper and associated endocrine imbalances also often develop cold-virus symptoms at their menses. These symptoms are usually indistinguishable from a flu or the common cold. Sinus congestion which lasts four to six days frequently accompanies these other symptoms at the onset of menses. Patients often miss the correlation of these other symptoms with their menses unless they are prodded by the doctor's questioning. These symptoms may be part of the patient's initial complaints or they may arise during the process of copper detoxification.

EXCESS COPPER AND COMMON SPINAL PROBLEMS

Schmitt and Tolen (6, 7) discussed a superior-inferior tipping of the vertebral column from top to bottom which they

called a cranial-spinal torque. This pattern often exists in the presence of a copper, manganese, zinc imbalance. In the presence of excess copper, the relative imbalances between copper and manganese and copper and zinc result in the vertebrae being torqued in two opposite directions, both up on the right / down on the left and vice versa. This causes a great deal of spinal instability and a number of commonly encountered problems in the chiropractic practice.

The consequences of this type of continued stress to an adolescent spine are pressures which are felt to cause vertebral deformity and the development of scoliosis. Excess copper as identified by hair analysis has been associated with idiopathic scoliosis in children. (8)

The same type of continued torque pressures on the adult spine is felt to lead to unusual disc pressures which can lead to disc degeneration over time. Manganese has long been accepted as necessary for many disc patients. Some patients taking large amounts of manganese for a disc problem get caught in a trap where the imbalance of low zinc and high manganese results in a cranial-spinal torque problem which keeps the disc under constant mechanical stress.

In patients with either idiopathic scoliosis and/or disc problems, oral insalivation of copper will often cause generalized muscle weakness. COP-OUT will negate these cranial-spinal torque patterns. These patients are much better treated with COP--OUT than with just manganese alone which has often been the case in the past.

Other excess copper symptoms include hair loss, low thyroid symptoms, dizziness, depression, "spaciness" or other mental perceptual symptoms, or any other nagging symptoms including difficult musculoskeletal problems which will not stabilize.

COPPER AND WILSON'S DISEASE

Pathologically high copper levels are associated with Wilson's Disease. This disease is characterized by portal cirrhosis, degeneration of the lenticular nuclei of the basal ganglia, and Kayser-Fleischer rings which are pigmented rings in the periphery of the eye. The disease is thought to be a recessive Mendelian defect resulting in a deficiency of a copper handling enzyme. Nervous system features are muscular rigidity, a Parkinsonian type tremor, speech articulation difficulty, and marked emotionalism. The hepatic system is also disturbed resulting in fatigue, ascites, and other signs of liver cirrhosis. Both the liver and the affected brain areas are found to contain pathological deposition of copper. (12)

Until recently, Wilson's disease has been treated with the drug, D-penicillamide (9). However, penicillamide is toxic in many patients. Zinc supplementation has successfully replaced penicillamide in limiting copper uptake and in increasing excretion of excess copper. (9, 10) Use of penicillinamide with zinc supplementation (or COP-OUT) can facilitate the recovery of patients with Wilson's Disease. (11)

COPPER AND ALLERGIES

Michael Lebowitz, D.C. has identified a relationship between copper need and certain allergy patients. He has called this type of allergy pattern a "thymus mediated allergy" since

muscle weakness induced by oral challenge with allergenic foods is negated by therapy localization to the thymus reflexes. (1) Preliminary clinical investigations in one of our offices (WS) has shown a high degree of correlation between Lebowitz's thymus mediated allergies and positive blood tests for IgG and IgG immune complexes related to specific foods.

Dr. Lebowitz has found that most patients who show a thymus mediated allergy have weak muscles which strengthen upon tasting copper. He also recommends copper supplementation to many of these patients.

In an effort to enhance the diagnostic approach to these patients, it was reasoned that if the thymus mediated allergy patients needed copper, that using a copper antagonist like COP-OUT might expose some hidden weakness which would be of diagnostic importance. Such is the case.

Thymus mediated allergy patients will show a weakening of any strong indicator muscle when they simultaneously hold COP-OUT in the mouth and therapy localize (TL) to the thymus reflex (over the angle of Louis on the upper sternum). These patients may or may not show a weak muscle strengthening on thymus TL. That factor is apparently inconsequential.

There will be no weakening of a strong indicator muscle by either thymus TL or insalivation of COP-OUT alone, only when the insalivation and TL are performed simultaneously. Weakening of a strong muscle with simultaneous thymus TL and COP-OUT insalivation is an indication to perform provocative testing of foods by oral neuromuscular food hypersensitivity testing. This

screening test is best performed whether or not the patient responds to copper.

Many problems we encounter in our practices on a daily basis can be related to excess body burden of copper. Some of them have been discussed here. The following procedures will serve as screening tests for copper excess and identifying the need for copper antagonists as found in COP-OUT.

PROCEDURE

1. Upon finding a weak pectoralis major, sternal (PMS) or supraspinatus muscle, have the patient chew up a COP-OUT and observe for a strengthening of the weak muscle. This is probably a sign of excess copper which is affecting the liver and/or the brain. Test a strong muscle against insalivation of copper. If it weakens, the patient most likely has an excess copper body burden.

2. Test a strong muscle against insalivation of copper (preferably a PMS or supraspinatus). If a strong muscle weakens, check COP-OUT against a weak muscle. If copper weakens a strong muscle and COP-OUT strengthens a weak muscle, there is a good chance that the patient has excess copper and will be benefitted by COP-OUT. COP-OUT should be recommended at a rate of one to three tablets per day. Continue this dosage for six to twelve weeks when the patient's need for COP-OUT should be reevaluated. COP-OUT may be used as a preventive supplement in cases of drinking water with high copper content or if the patient has a propensity for copper retention. High estrogen patients or vegetarians, for example, might fall into this category.

3. With the patient prone or supine, simultaneously challenge

superiorly on one side of the spine (over the TPs) and inferiorly over the other side of the spine (TPs). Perform this challenge of the spine at several areas - i.e., up on one side and down on the other, then vice versa. Chewing COP-OUT will negate either of these challenges if the patient is copper toxic. Check this pattern in idiopathic scoliosis patients, difficult disc patients, and any other patients with spinal instabilities.

4. When oral COP-OUT and thymus TL together cause a strong muscle to weaken, check the patient for thymus mediated (IgG or IgG immune complex) allergies.

REFERENCES

1. Liebowitz, M., Thymus related allergies: diagnosis and correction. ICAK-USA collected papers, 1988-89, volume 1. p.204.
2. Brea, T., Copper status: the other side of the coin. ICAK collected papers, winter, 1987.
3. Mowles, R. A., A clinical investigation of pre-menstrual syndrome and the correlation with copper. ICAK collected papers, Summer, 1983.
4. Mowles, R. A., An applied kinesiology study of copper interaction with iron and folic acid. ICAK collected papers, Summer, 1983.
5. Mowles, R. A., and Schmitt, Walter H., Jr., An applied kinesiological approach to candida albicans allergy. Digest of chiropractic economics 28:4 Jan-Feb, 1986. p.133.
6. Schmitt, Walter H., Jr., and Tolen, Stephen C., The cranial-spinal torque pattern. ICAK collected papers, Summer, 1983.
7. Schmitt, Walter H., Jr., Further observations on the links between the nervous system and the body chemistry. ICAK collected papers, Summer, 1983.
8. Pratt, W. B., and Phippen, W.G., Elevated hair copper in idiopathic scoliosis. Spine 5:3, 1977.
9. Brewer, G.J., Hill, G.M., Juni, J.E., Prasad, A.S., Dick, R.D., Treatment of Wilson's disease with zinc. II validation of oral 64 copper with copper balance. Am J Med Sci (U.S.), Dec. 1986, 292(6). p. 344-9
10. Hoogenraad, T.V., Koevoet, R, du Ruyter Korver, E.G., Oral zinc sulphate in long term treatment of Wilson's disease. Eur Neurol 1979, 18(3), p. 205-11
11. Personal conversations with John Bandy, D.C., Blowing Rock, NC
12. Boyd, Wm., A textbook of pathology 8th ed., 1970 Lea and Febiger:Philadelphia. p. 884, 1257-8.

An Essay on the Clinical Practice of Applied Kinesiology

Marc S. Rosen, M.S., D.C.

A B S T R A C T: This essay presents a clinical theory for the etiology and physiopathology of "Functional Illness". The theory relates to the neurobiologic consequences of biped posture and gait, (The Janse - Illi Concept), and to the neuroendocrine, immune, and digestive manifestations of Hans Selye's well known General Adaptation Syndrome. From this physiologic perspective, the clinical signs and symptoms of "Functional Illness" are directly associated with (biped) structural imbalance, and the physiologic demands of biologic adaptation. The diagnostic value of manual muscle testing, in clinical chiropractic and osteopathic practice, is discussed in relation to the functional assessment of posture, gait, range of motion, and spinal-pelvic biomechanics. The "Functional Neurology" of manual muscle testing may be of value in identifying both the presence and nature of the various physiologic dysfunctions that are related to biologic adaptation. The paper outlines a theoretical etiology for "muscle weakness", and offers a review of the practical aspects of muscle testing. Common (functional) syndromes and subjective complaints are listed in an effort to define the nature of "Functional Illness". In the terms of this essay, manual muscle tests have the clinical potential to monitor the physiologic response to physical, chemical, and mental stimuli. The clinical assessment of function is proposed to be a correlation of that observed response, with a functional interpretation of the history, physical and laboratory exam, and instrumentation.

ACKNOWLEDGEMENTS

The essay that follows, is a synthesis of concepts set forth by the following individuals:

Joseph Janse - Fred Illi - Biped posture and Gait (Clinical Consequences)

Hans Selye - General Adaptation Syndrome - Physiologic Effects of Adaptation

George Goodheart - Structural Imbalance/Gait Impairment/Altered ROM -Secondary to Unequal Pull of Antagonist Muscles

Clinical Utilization of Manual Muscle to Evaluate Function

Clinical Utilization of Physical Exam, Lab, Instrumentation

Irvin Korr - Robert Denslow - Segmental Facilitation - Relation to Spinal Lesions

David Walther - Neurologic Disorganization - Practical Aspects of Muscle testing Evaluating Response to Stimulous - Concept of Clinical Correlation

Walter Schmitt - Functional Neurology - Eccentric/ Concentric Contraction - Neural Mechanisms - GAS and Applied Kinesiology Findings

Please consult the attached bibliography for each individuals published work.

INTRODUCTION

Applied Kinesiology is an approach to clinical practice that was developed within the chiropractic profession. The methodology is concerned primarily with the clinical assessment and subsequent restoration of function in the human body. This clinical purpose is predicated upon the concept that physiologic functions can become physiologic dysfunctions, and thereby undermine biologic adaptation (to environmental stressors). The proposition set forth in this statement reflects a basic chiropractic and osteopathic premise. Very simply, the idea is that unsuccessful adaptive responses are major etiological factors in the disease process (Janse 1976, Levine 1964, Hoag,Cole, and Bradford 1969, Homewood 1962). In theory, a dysfunction during biologic adaptation is productive of a constellation of signs and symptoms that are encountered in any clinical practice. The term "Functional Illness" classifies clinical signs and symptoms as occuring in a causal relationship to physiologic dysfunction, but in the absence of pathologic states or processes. Most of applied kinesiology revolves around this conceptual differentiation between "functional" and "pathological" illness. The former is thought to be the forerunner of the later. In that, a close investigation of the nature of physiologic dysfunction reveals theoretical etiologic relationships to the major components of the degenerative disease processes, i.e. atherosclerotic, arthritic, malignant, metabolic, and inflamatory. Since pharmacologic and surgical intervention are characteristically productive of physiologic dysfunction, the manifestations of functional health problems may be more responsive to a non-allopathic or physiologic approach, i.e. one that considers clinical nutrition, biomechanics, etc. It is important to note not only a differentiation between functional and pathological therapeutics, but that the diagnostic methodology differs as well. Allopathic assessment via physical examination, laboratory tests, and radiologic and other imaging techniques may be exclusively sensitive to the presence of pathology. The pathologic interpretation of such procedures is of course indicated to "rule-out pathology", and should therefore be of routine application. In fact, in terms of this discussion, a differential diagnosis, represents this rule-out process or the differentiation between functional and pathological signs and symptoms. In applied kinesiology, traditional (allopathic)

aspects of the history, physical examination, and laboratory evaluation have been modified in application and in interpretation. This is in order that the diagnostic values might be more predictive of physiologic dysfunction. Likewise, many functional signs and symptoms are unique considerations in the practice of applied kinesiology.

The issues that now require clarification,are: (a) what is the nature of physiologic dysfunction, (b) what are the related signs and symptoms, (c) by what means might the clinican assess functional aspects of health related problems?

DISCUSSION

That the human condition is a biped one, is a biologic concept that has been elaborated upon by many chiropractic and osteopathic educators. Notably, Jospeh Janse and Fred Illi. The clinical implications of biped posture and locomotion (gait) are many and for the most part neurologic in consequence. Considering the human frame, we see that the lumbosacral junction and sacroiliac joints are responsible for the upright (postural) support of the primary curves of the spine. A very mobile and proportionately small atlantoaxial region must support a twelve pound cranium. The sacroiliac joints are unstable by their very design (not symetrical and wider at their anterior aspect). The sacral base angle and the primary spinal curves are thus increased and/or decreased (or forced into lateral flexion) by postural distortion. It appears that a biped posture is predisposed to distortion. Postural distortion of the sacral base and spinal curves predisposes transitional areas to intersegmental subluxation and/or fixation. Vertebral subluxation/fixation are thought to be characterized by a lowered threshold for motor responses (Korr - Denslow Concept of Segmental Facilitation). Postural distortion also contributes to abdominal visceroptosis, with the potential for vascular and lymphatic stasis in the pelvic floor and lower extremities.

Musculoskeletal distortion is a factor that relates directly to an impairment of a very specialized biped gait. The resultant articular "wear and tear", from abnormal weight bearing and range of motion, might be responsible for aberent stimulation of joint receptors. Proprioceptive dysfunction is also a component

of myofascial dysfunction, which may be both an etiology and a consequence of postural distortion (structural imbalance).

The fundamental concept is that biped posture and gait predispose the human nervous system to proprioceptive dysfunction and segmental facilitation. As a physiologic entity, and in applied kinesiology terminology, this pattern of neural dysfunction is referred to as "Neurologic Disorganization" (Walther 1981). In this state, abnormal patterns of facilitation and inhibition may over or under-stimulate vasomotor, secretatory, lymphatic, immune, digestive, and other physiologic functions. In addition, abnormal neural activity perpetuates postural distortion and abnormalities in gait (the proverbial vicious cycle).

In applied kinesiology, an operating clinical concept is that postural (structural) balance is dependent upon an equal pull of antagonist muscles. Loss of pull or tone may contribute to postural distortion, altered range of motion, and abnormalities in gait. The primary curves of the spine, as previously mentioned, are predisposed to distortion, abnormal weight bearing, and articular mobility. The proposed etiologies for loss of muscular tone are:

- myofascial dysfunction (microavulsion and proprioceptive dysfunction)
- peripheral nerve entrapment
- segmental facilitation / neurologic disorganization
- viscerosomatic relationships (aberent autonomic reflexes)
- nutritional deficiency
- toxic chemical influences
- dysfunction in the production and flow of cerebral spinal fluid
- dysfunction in the meridian system
- lymphatic and vascular impairment

Consider now, the clinical assessment of posture and functional (spinal) biomechanics. Manual tests of muscle function are immensely useful in a correlative clinical process that invloves aspects of the history, postural analysis, myofascial palpation, and observations of gait and range of motion (spinal, femoral, etc.).

In applied kinesiology musculoskeletal symptoms and findings are interpreted according to their relationship to posture and gait. Postural attitudes that

either ameliorate or exacerbate subjective or objective findings, will define the position of the patient for individual muscle tests. These same factors will also define which structures/functions will be examined/evaluated via applied kinesiology procedures (Goodheart 1986 , Walther 1981).

Manual muscle tests can become a dual source of clinical information. In terms of the preceding discussion, the functional capacity of a specific muscle can be evaluated,to determine a causal relationship to a postural distortion and/or gait abnormality. The same applies to altered ranges of motion.

From a slightly different perspective, manual muscle tests have the clinical potential for use in monitoring the neural mechanisms that mediate muscle function during a manual test. These mechanisms are presumed to be a monosynaptic stretch reflex (for eccentric contraction), and a cortical influenced gamma efferent for concentric contraction of the test muscle, (Schmitt, 1986).

If a manual muscle test does, in fact, reflect the functional neurology of muscle proprioception, and upper and lower motor neuron responses, then it follows that manual muscle tests should also be capable of monitoring the physiologic factors that influence muscle function, i.e. lymphatic and vascular supply, related nutrient's, etc.)

To develop accurate clinical impressions, manual muscle tests must be applied according to a very precise testing protocol. This is the art or practical aspect of this particular clinical assessment tool. Walther outlines the "art" of muscle testing as follows:

- proper positioning (isolation of test muscle)
- adequate stabilization of regional anatomy
- observation of the mannerin which the patient assumes and maintains the test position
- observation of the manner in which the subject performs the test
- consistent timing, pressure, and position
- avoidance of preconceived impressions as to the outcome of the test
- non-painful contacts and actual test
- contraindications due to age, debilitating disease, acute pain, local pathology or inflammation

The biologic response to the environment is such that exposure to physical, chemical, mental, and thermal stressors, evokes neuroendocrine mediated changes in vascular, immune, and digestive functions. Repeated exposure to environmental stressors (which is the pattern of contemporary society, perhaps even during intrauterine life) results in facilitation of the sympathetic nervous system and a release of adrenal catecholamines and glucocorticoids. Prolonged neural stimulation and hormonal action might over stimulate physiologic processes out of phase with time and need. Physiologic functions may now become physiologic dysfunctions (Selye 1978, Schmitt 1981).

According to this concept, environmental stressors and the resultant adaptive (neuroendocrine) response, might predispose the human condition to neuroendocrine, immune, digestive, and circulatory dysfunction. These physiologic dysfunctions are thought to be productive of many of the signs and symptoms that are "functional" in origin and in character. Collectively, functional signs and symptoms may occur as a "syndrome". Examples of functional syndromes, that might occur in the absence of pathology, but may be related to biped posture/gait and biologic adaptation, are:

Low Back and Leg Pain
Cervical Brachial Syndromes
Headaches
Allergies, Asthma, Chronic Bronchitis
Digestive Disturbances
Fatigue
Chronic Infections
Premenstrual Syndrome
Learning Disabilities
Depression

In the clinical practice of applied kinesiology, a physiologic rationale is usually formulated, to draw causal relationships between subjective complaints and objective findings, and the various patterns of physiologic dysfunction that may in turn be causally related to structural imbalance and biologic adaptation (Selye's General Adaptation Syndrome).

Many patients and many health care providers may not attach any significance to functional symptomatology. This is simply because there is no physiologic concept of etiology. Symptoms that do not signal a medical emergency, or that are not acute enough to limit a particular activity, are especially overlooked. However, when questioned during the case history, patients may relate a history of:

- photophobia
- postural vertigo
- dry mouth, metallic taste in mouth
- difficult swallowing, lump in throat
- TMJ pain, bite cheek/tongue while chewing
- mid-afternoon fatigue
- chronic fatigue
- difficulty falling and/or staying asleep
- frequent upper respiratory symptoms
- heartburn, abdominal distention after meals, sleepy after meals
- flatus, chronic constipation, diahrea
- hunger shortly after eating, hunger despite eating
- varicose veins, hemmoroids
- difficulty remembering what was read, difficulty reading
- difficult concentration
- poor coordination, confuse directions
- depression, crying, irritability
- poor response to exercise
- stiffness in morning or after sitting
- cracking joints (crepitus on range of motion)
- dry skin, hair loss, brittle or soft nails

The clinical assessment of neuroendocrine and related functions, involves a correlation and functional interpretation of findings from several sources.

continued next page

The clinical assessment of "function:, involves:

- the history
- the physical exam
- laboratory tests
- instrumentation
- analysis of posture, gait, and range of motion
- static and motion palpation
- manual muscle testing and related procedures (therapy localization challenge)

Individual procedures might be referred to as a physiologic measure, which is a clinical value or response that is indicative of a physiologic process or response. As an example: A spirometer measures vital capacity as a forced volume of expelled air. The function of the diaphragm is fundamental to this measurement. A second example: The sympathetic nervous system maintains systolic blood pressure during changes in posture. Measuring systolic blood pressure in multiple positions may reflect the functional capacity of catecholamine release.

In addition to vital capacity and postural blood pressure, the clinical assessment of function, might include:

- observation of the pupil response to light
- measurement of axillary temperature
- measurement of salivary and urinary ph
- urinary analysis of chlorides and calcium
- interpretation of WBC and RBC counts and MCV values
- assessment of calf pressure tolerance
- phonocardiograph tracings

The list is by no means complete, but it does serve to illustrate the use of "physiologic measures" in the clinical practice of applied kinesiology. The reader should also note that a discussion of each "measure" exceeds the scope of this paper.

Manual muscle tests appear to reflect facilitation and inhibition of the neural mechanisms that mediate eccentric and concentric contraction. In both clinical and research settings, manual muscle tests have been found to be responsive to physical, chemical, and in some instances, mental stimuli. This ability to monitor the physiologic response to an applied stimulous, is of potential clinical value during the interpretation of the aforementioned physiologic measures. Certain "functional aspects" of the case history, might also be interpreted in terms of the physiologic response of manual muscle testing.

In the clinical practice of applied kinesiology, manual muscle tests are used to monitor the following physical, chemical, and mental stmuli:

- digital contact of the skin (therapy localization)
- transient directional vevtors of force, applied to the articular aspects of the spine, pelvis, cranium, and extremities
- stretch (muscle and joint)
- repetitive contraction of muscle or motion of a joint
- inhilation of the fumes of a chemical substance
- tasting of a source of nutritional material
- a phase of diaphragmatic respiration
- the visualization of a mental stressor

The neural response to the above stimuli, is of value,only in so far as it relates to facilitation or inhibition of neural mechanisms. Specific diagnostic claims, based solely on the results of a manual muscle test, are incorrect.

In an applied kinesiology assessment of functional spinal biomechanics, the generation of a monosynaptic reflex (mediator of eccentric contraction) has been reported to be influenced by transient vectors of force applied to suspect areas of subluxation (segmental facilitation). The phenomenon has also been observed when vectors (of force) are applied to the pelvis, cranium, and extremities. This is a prime example of the utilization of manual muscle testing to monitor the neural response to an applied stmulous (Scmitt's "Functional Neurology"). This is also an example of a correlative clinical process that involves postural analysis, static/motion palpation, and range of motion.

In terms of this discussion, the utilization of manual muscle testing in clinical practice, is limited to:

- The evaluation of eccentric and concentric contraction (to establish an etiology for a postural imbalance, impaired gait, and/or altered range of motion).

- The evaluation of factors that influence the neural mechanisms that mediate eccentric and concentric contraction (physiologic response to a stmulous, used with physical exam, lab, etc. to identify physiologic dysfunction)

Infectious and malignant processes will of course alter function in the human body. However, the findings generated through the use of manual muscle testing, are not valid indicators of a specific infectious or malignant state or process,

Using a manual muscle test to evaluate the patient's functional response to the examiners mental thought or image, is a questionable clinical practice, and probably unreliable.

SUMMARY

Applied Kinesiology is literally "writing the book" on "functional illness". Although, at this point, the concept and the terminology, are not to well defined. To define the concept of functional illness, we must establish an etiology. The preceeding essay proposes that biped posture and gait, and biologic adaptation, are significant etiological factors in the production of functional health problems. The essay also proposes that manual muscle testing can be a significant clinical means of assessing physiologic dysfunction, when correlated with certain (functional) aspects of the history, physical exam, laboratory tests, and instrumentation.

BIBLIOGRAPHY

Joseph Janse, Principles and Practice of Chiropractic, An Anthology, Edited by R.W. Hildebrandt, (Lombard, Ill.,National College of Chiropractic, 1976)

Hans Selye, The Stress of Life, (Montreal, McGraw-Hill, 1978)

George J. Goodheart, Collection of Reprints, (Detroit, Published by Author)

George J. Goodherat, You'll Be Better, The Story of Applied Kinesiology, (Detroit, Published Privately by the Author)

Irvin M. Korr, The Collected Papers of Irvin Korr (The American Academy of Osteopathy, 1979)

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

Walter H. Schmitt, Common Glandular Dysfunctions in the General Practice, (Chapel Hill, NC, Applied Kinesiology Study Program, 1981)

Hoag, Cole, & Bradford, Osteopathic Medicine, (McGraw - Hill: 1978)

A.E. Homewood, Neurodynamics of the Vertebral Subluxation, (Toronto: Canadian Memorial Chiropractic College, 1962)

Mortimer Levine, The Structural Approach to Chiropractic,(New York: The Comet Press, Inc, 1964)

Walter H. Schmitt,Muscle Testing as Functional Neurology, Differentiating Functional Upper Motorneuron and Functional Lowe Motorneuron Problems, ICAK Selected Papers, 1986

THE AMMONIA SNIFF TEST

Walter H. Schmitt, Jr., D.C.

ABSTRACT: Applied kinesiology muscle testing procedures are employed to evaluate patient responses to nasal sniffing of ammonia, which should cause no change in muscle strength. Muscle weakening or strengthening which results from sniffing ammonia indicates a problem with some metabolic pathway associated with ammonia or its metabolites. Abnormal ammonia responses lead to evaluation of nutrient status of vitamin B-6 (and pyridoxal-5-phosphate), molybdenum, and iron. Alpha-ketoglutaric acid, which is produced in the citric acid cycle (CAC) is able to accept ammonia and become glutamic acid in the body's major transamination pathway. Faulty CAC activity leads to inadequate alpha-ketoglutaric acid production and an inability to handle ammonia. Likewise, deficiencies of the nutritional substances necessary for urea cycle activity cause ammonia to back up in the system producing hyperammonemia. The clinical effects of impaired ammonia metabolism are discussed in relation to neurotransmitter levels and the wide ranging symptoms associated with these imbalances.

INTRODUCTION

The availability of ammonia groups and the ability of the body to regulate the transfer of ammonia groups to and from various molecules is at the heart of amino acid metabolism, and is essential for the growth, repair, and degradation of most

tissues in the body. When there is an excess, deficiency, or inability to process ammonia, amino acid metabolism is affected throughout the body.

Frequently patients have disturbances with their abilities to process ammonia. These are most often nutritional in nature and are encountered on a daily basis in a busy practice. Symptoms associated with ammonia metabolism include fatigue, emotional symptoms such as irritability, mental sluggishness and confusion, water retention, premenstrual syndrome, headaches, and inflammation anywhere in the body. The ability to identify these problems helps greatly to enhance the rapid resolutions of patients' symptoms but is dependent on an effective screening test for problems of ammonia metabolism and amino acid metabolism. Such a test is the ammonia sniff test.

A normal muscle response to the sniffing of ammonia is no change in muscle strength. That is, sniffing ammonia should cause no strong muscles to weaken nor any weak muscles to strengthen. Changes in muscle testing patterns which occur when patients sniff ammonia have been of great benefit in helping to understand the metabolism of ammonia and helping to improve patients' health problems. The symptoms from altered ammonia metabolism may affect any tissues of the body since amino acid metabolisms effect virtually all cells.

Patients who strengthen upon sniffing ammonia may do so due to a generalized amino acid deficiency, either due to improperly practiced vegetarianism, starvation, or improper digestion and/or

assimilation of protein materials. These are the minority of patients we see, but it is important to consider this in patients who have a strengthening response to ammonia.

The vitamin and mineral factors which affect ammonia metabolism and which may be involved in altered ammonia sniff tests are the following: vitamin B-6, molybdenum, iron, alpha-ketoglutaric acid and the citric acid cycle (CAC), and the urea cycle. This paper will discuss each of these factors individually and summarize them with a flow chart at the end of the paper.

Based on muscle testing reactions to sniffing ammonia, problems with ammonia metabolism are commonly encountered. These problems, being systemic in nature, result in gamma 2 type muscle weakness patterns. Having a patient take a short sniff of ammonia should cause neither strengthening nor weakening of any muscle. One of the common factors we have observed in metabolic problems which cause gamma 2 weaknesses in patients is a problem with this ammonia metabolism, with the ammonia sniff either strengthening or weakening a gamma 2 muscle test. When ammonia either strengthens or weakens the patient we then follow procedures for testing the above nutrients one by one, as will be described.

B-6 AND AMMONIA METABOLISM

Vitamin B-6 in its activated pyridoxal-5-phosphate form (P5P) form is the essential activating molecule for all the transamination (i.e., transporting amines) reactions in the body.

Inadequate vitamin B-6 in its P5P form will result in inadequate transamination and the ammonia sniff test will cause either a strengthening or a weakening of muscles. In either case of ammonia strengthening or weakening, the first thing we want to check is the patient's vitamin B-6 status.

When testing vitamin B-6 and/or P5P, we test against a muscle which shows both gamma 1 and gamma 2 weakness patterns. Once the muscle is identified, we proceed using gamma 1 testing for simplicity's sake. We generally screen with P5P first, followed by plain B-6. In the case that P5P strengthens a gamma 2 weakness, and B-6 does not, we have to test the associated co-factors (zinc, magnesium, phosphorus and B-2) as previously discussed. [1]

In patients who weaken on ammonia sniff due to a B-6 problem, placing the appropriate nutrient, (i.e., B-6, P5P, or one of the co-factors) in the mouth will negate the weakening effect induced by sniffing ammonia. In the case where ammonia strengthens the patient, one of the above nutrients will also strengthen the patient's gamma 2 weak muscles.

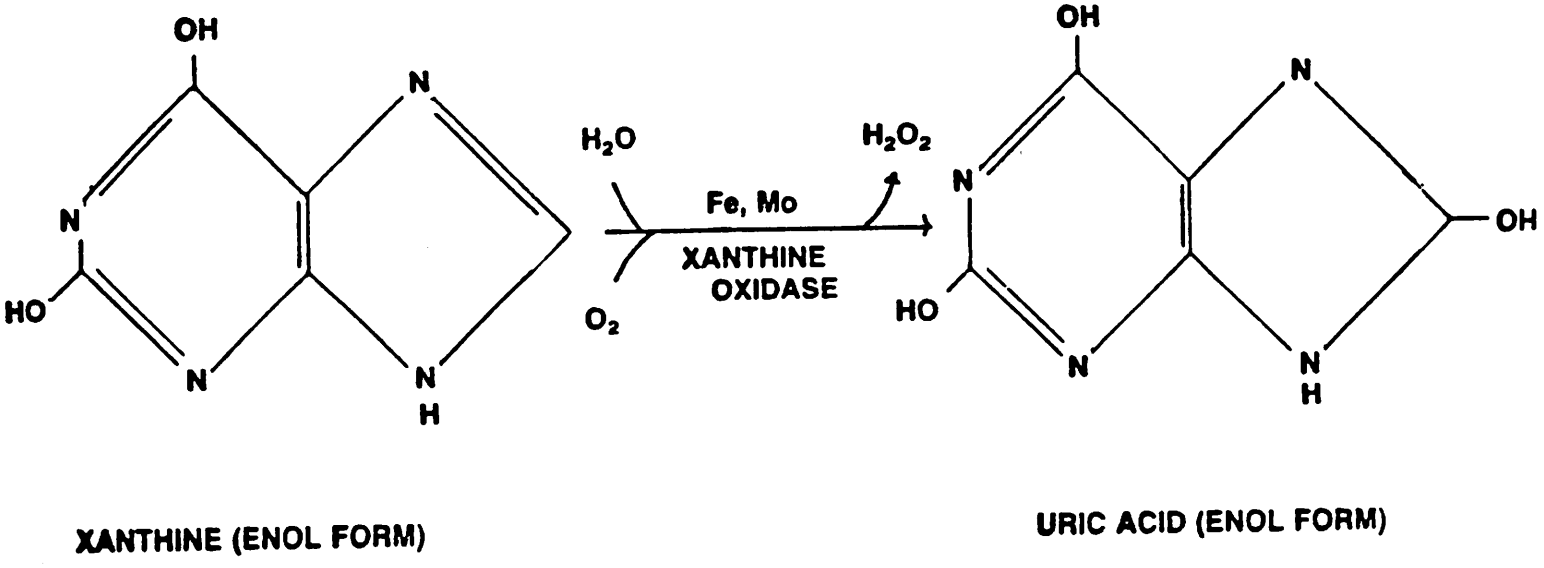
Upon ruling in or ruling out the need for B-6, we then continue on to identify other problems in ammonia metabolism.

AMMONIA, MOLYBDENUM, IRON, AND XANTHINE OXIDASE

Ammonia is used in the synthesis of purines. Purines are essential substances which are synthesized by the body and inevitably degraded down into uric acid and spilled into the urine. Each molecule of uric acid contains four nitrogen

molecules which are originally derived from four ammonia molecules. See figure 1. This route of elimination of uric acid from the purines into hypoxanthine and into xanthine and on into uric acid is one route of elimination of ammonia from the body. See figure 2.

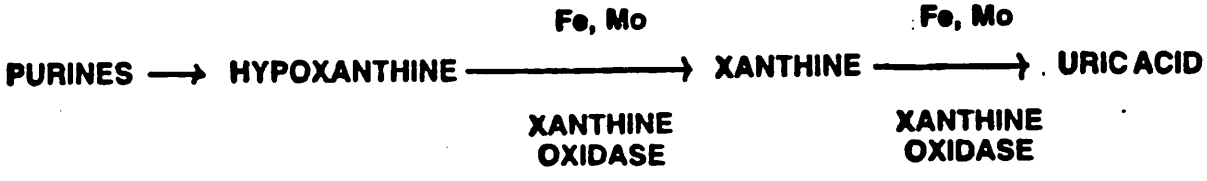
FIGURE 1



XANTHINE (ENOL FORM)

URIC ACID (ENOL FORM)

FIGURE 2



For the production of uric acid from xanthine and hypoxanthine, the enzyme xanthine oxidase is used. Note in figure 1 that the enzyme xanthine oxidase is dependent on iron and molybdenum. The work of Dr. Richard Mowles has led us to understand that some patients who weaken on ammonia sniff will be

weakening due to a deficiency of iron or molybdenum and a backing up of this xanthine oxidase pathway. [2]

Since the degradation of purines depends on iron and molybdenum and xanthine oxidase activity, dysfunction in this pathway can apparently result in a backing up of purine metabolism. Since purine metabolism is one of the destinations of the ammonia molecule, this ostensibly results in a back up of ammonia into the system and inhibits one of the normal routes of elimination of ammonia.

A number of patients who weaken on sniffing ammonia will also show a gamma 2 muscle weakness which will be strengthened by insalivation of iron and/or molybdenum. Holding the iron and/or molybdenum in the mouth will negate the weakening effect of ammonia in these patients. They should be supplemented with adequate quantities of iron and/or molybdenum in order to maintain normal ammonia metabolism in the body.

AMMONIA TRANSPORT AND ALPHA-KETOGLUTARIC ACID

The major transamination reaction in the body involves ammonia combining with alpha-ketoglutaric acid to become glutamic acid as shown in figure 3. This pathway is bi-directional with

FIGURE 3

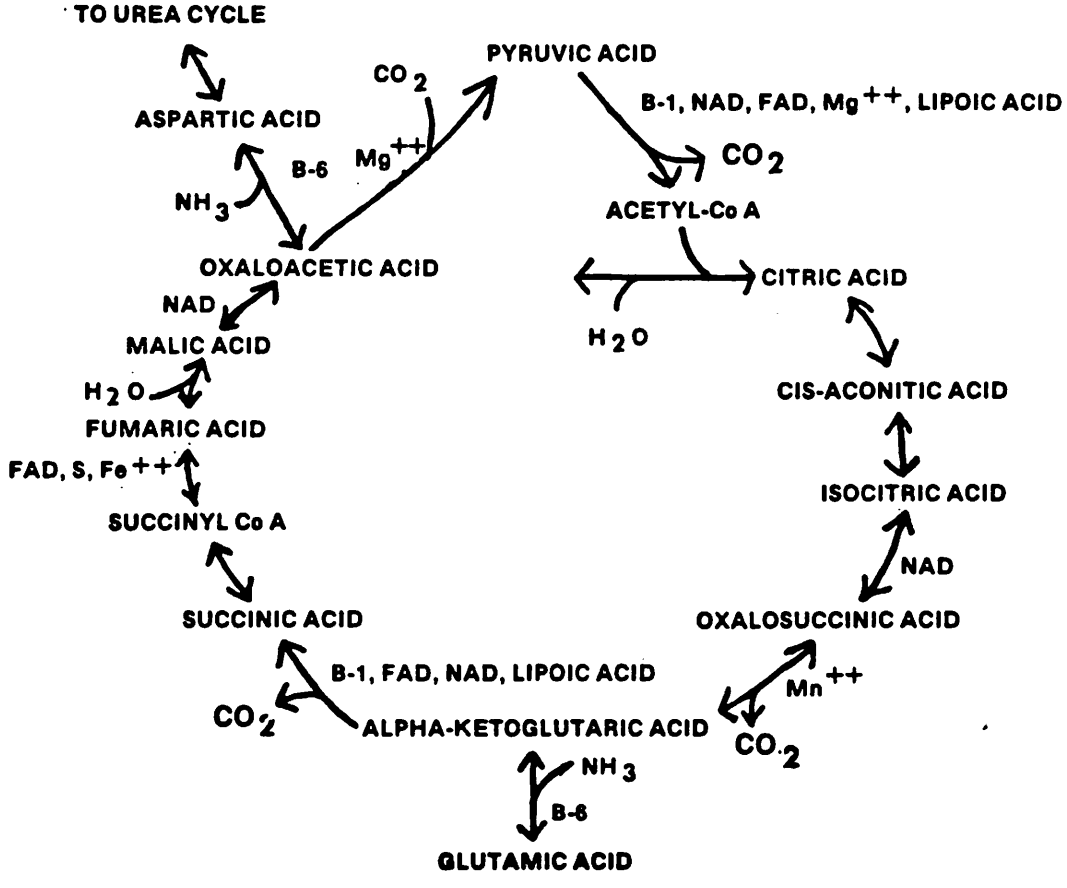
glutamic acid releasing an ammonia molecule and becoming alpha-ketoglutaric acid. This transamination reaction is the major

pathway for give and take of ammonia groups in the body. Glutamic acid, a non-essential amino acid, is synthesized primarily from the amination of alpha-ketoglutaric acid.

Alpha-ketoglutaric acid is produced in the body in the citric acid cycle and is a major constituent of that cycle. See figures 4a and 4b. Patients who have inadequate abilities to produce alpha-ketoglutaric acid have faults with their CACs.

FIGURE 4a

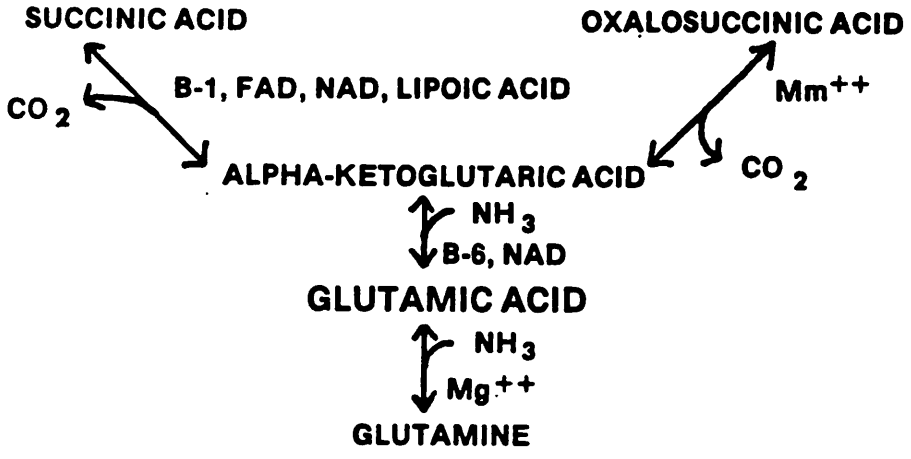
CITRIC ACID CYCLE



It is common knowledge that water and carbon dioxide (CO_2) are released during energy production. As can be seen in figure

4a, there are four places in the CAC where a CO_2 molecule is released. When problems occur with the normal function of the CAC, the result is usually inadequate production of CO_2. Patients with CAC problems will have a strengthening response from increasing their CO_2 levels by rebreathing their own air in a paper bag.

FIGURE 4b: BLOW UP OF LOWER PART OF FIGURE 4a



Patients who strengthen on rebreathing or on tasting alpha-ketoglutaric acid are further analyzed by testing with citric acid. Depending on the reaction to citric acid, various nutrients are tested to identify the location of the problem in the CAC. [3] Supplying the appropriate nutrient or nutrients to stimulate CAC function will enable the body to produce its own alpha-ketoglutaric acid.

In cases of severe toxicity where hyperammonemia has been chronically present and is creating serious acute symptoms, the use of alpha-ketoglutaric acid as a supplement is recommended for short terms of time. Alpha-ketoglutaric acid supplementation is

maintained until such time as the other CAC nutrients have time to catch the body's own production of alpha-ketoglutaric acid up with its needs. The nutrients which affect the CAC, and hence the production of alpha-ketoglutaric acid, are listed in Fig. 5 and include B-1, B2, B-3, pantothenic acid, manganese, "B", "G", and lipoic acid. Thorough evaluation of the CAC is discussed in another paper. [3]

FIGURE 5

CITRIC ACID CYCLE FACTORS

B-1
"B" (S.P.L.)
MANGANESE
PANTOTHENIC ACID
B-2
NIACINAMIDE
"G" (S.P.L., E.P.)
LIPOIC ACID

AMMONIA AND THE UREA CYCLE

The major route of elimination of ammonia waste products in the body is via the urea cycle as shown in figure 6. Problems involving the function of the urea cycle interfere with normal processing of ammonia and result in patients weakening on the ammonia sniff test. The nutrient factors for the urea cycle are summarized in figure 7.

Note in figure 6 that the urea cycle that it is generated by the combination of an ammonia molecule with a carbon dioxide

FIGURE 6

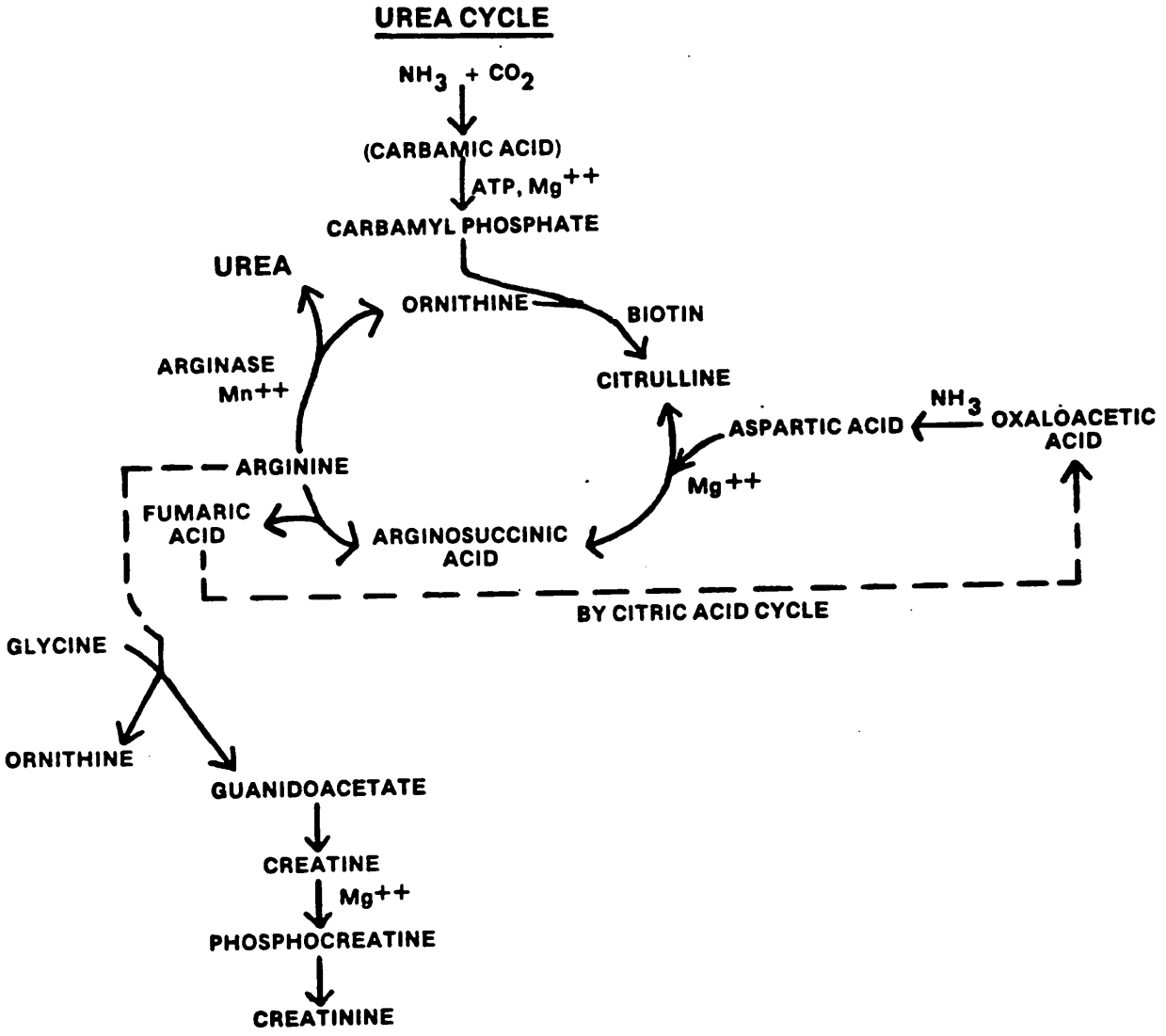


FIGURE 7

UREA CYCLE FACTORS

B-6
MAGNESIUM
BIOTIN
ASPARTIC ACID (CITRIC ACID CYCLE)
MANGANESE
ARGINEX (S.P.L.)

in the presence of B-6 and magnesium to form carbamic acid. The availability of carbon dioxide from the CAC as previously discussed is essential for initiating the urea cycle. Also B-6 (P5P) and magnesium are essential for this first step.

The patient who has inadequate urea cycle function is most easily identified by first eliminating the previously mentioned factors: B-6, molybdenum, iron and alpha-ketoglutaric acid (CAC problems). By routinely checking B-6 and alpha-ketoglutaric acid, we have already screened for two of the important urea cycle factors as shown in figure 7.

After ruling in or ruling out these factors, the patient should then be tested for arginine. Urea is derived from arginine as you can see in figure 6. Urea is produced and eliminated from the body and the body is left with ornithine. Arginine has three ammonia molecules. Two of them are eliminated in the urea molecule, and one is recycled in the form of ornithine.

Following the pathways shown in figure 6, we see that ornithine then combines with carbamyl phosphate under the influence of biotin to form citrulline. Citrulline combines with aspartic acid which is derived directly from oxaloacetic acid from the CAC. (See figure 4a.) Aspartic acid and citrulline combine to form arginosuccinic acid. This step requires magnesium. Arginosuccinic acid becomes arginine by releasing a fumaric acid molecule.

Note that the fumaric acid molecule is recycled back to the

CAC, and the pathway of fumaric acid to oxaloacetic acid to aspartic acid and on through arginosuccinic acid forms its own little cycle. It is just like a gear between the CAC and the urea cycle, intimately tying the two functions together.

When we test arginine and it weakens a patient, we assume that the problem is with the body's inability to break down arginine. Arginine breakdown requires the arginase enzyme (which is available in the product "Arginex" by Standard Process Labs), as well as manganese. When this step is impaired, the whole cycle backs up and ammonia is not removed from the body resulting in a positive ammonia sniff test.

Many musculoskeletal patients have manganese deficiency in relation to ligamentous problems which complicates their symptoms. The need for manganese also results in faulty urea cycle function. This leads to excess ammonia buildup which increases swelling and water retention. Many patients who hold water do so because of urea cycle dysfunction and this must be ruled out.

When musculoskeletal injuries occur, the inflammatory response sometimes goes overboard and actually slows the healing process while increasing the pain. A time tested remedy for hastening the healing of everything from sprained ankles to disc protrusions has been to supplement the patient with a product containing purified, food grade urea. The urea induces diuresis and decreases swelling quite effectively. It has worked wonders in the past, but more recently, we asked, "why isn't the body

making its own urea?"

When the urea product strengthens a patient, the ammonia sniff test is always positive. Likewise, there will be a weakening (or sometimes strengthening) upon insalivating arginine indicating poor urea cycle functioning. It is better to correct the patient's urea cycle than to blindly supplement with urea as a diuretic.

Patients who weaken on arginine will always strengthen on either manganese or Arginex supplementation. Insalivation of these nutrients will strengthen a gamma 2 weak muscle and neutralize the weakening effect of sniffing ammonia as long as the appropriate nutrient is in the mouth.

Other patients show a strengthening of arginine and a weakening on ammonia. These patients' urea cycle is dysfunctioning and they are unable to produce adequate arginine to eliminate ammonia from the body. There are two different possibilities when arginine strengthens and ammonia weakens. The most common occurrence is that there is a blockage in the urea cycle at a previous step along the way. The other cause is a shunting of arginine out of the urea cycle into a combination with glycine to form phosphocreatine. See figure 6.

To identify the blockage in the urea cycle when arginine strengthens the patient, we back up along the cycle, testing each individual amino acid. First we test citrulline. If citrulline strengthens the patient we assume there is inadequate citrulline.

If citrulline has no effect or weakens the patient, we

assume there is adequate or excess citrulline, respectively. In this case, when arginine strengthens, it means that there is a blockage between citrulline and arginine. This blockage is due to either inadequate aspartic acid from the CAC and/or inadequate magnesium for this reaction to take place.

Aspartic acid is then tested and if aspartic acid strengthens, we then analyze the CAC for a problem, using the nutrients discussed in figure 5 and/or biotin. If aspartic acid also weakens and citrulline weakens, it implies a deficiency of magnesium which is inhibiting any forward progress at this point of the cycle, hence a build up of these two amino acids. Or if aspartic acid has no effect and citrulline weakens, it also implies a deficiency of magnesium.

If citrulline has no effect, neither strengthening or weakening, we then back up to ornithine and test ornithine orally. If ornithine strengthens the patient, it implies a deficiency of ornithine, and we must backtrack farther.

If, however, ornithine weakens the patient or has no effect, and citrulline strengthens, it means there is a block between ornithine and citrulline. This block is usually overcome by supplying biotin.

Remember that biotin is produced in our body from normal intestinal flora activity. Many patients who show a deficiency of biotin also have toxic bowel, which further complicates the ammonia toxicity. You can see how many of these patients who weaken on ammonia have multiple levels of toxicity.

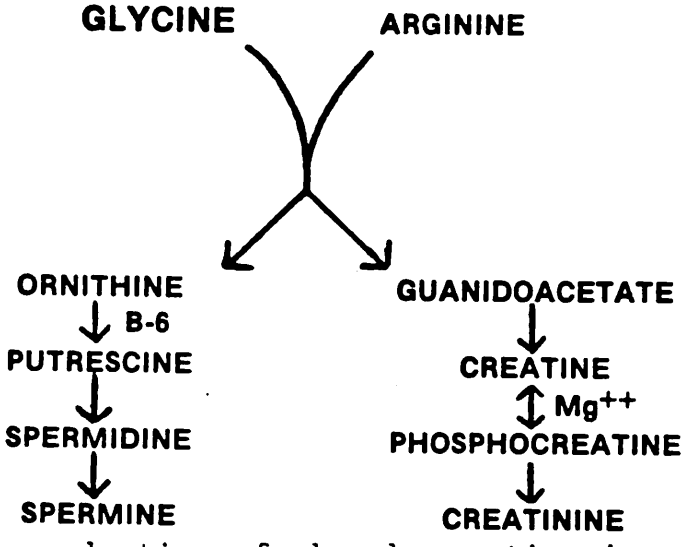
In patients who test positive for biotin, we usually supplement with biotin, but then further investigate the large intestine. Gastrointestinal flora imbalances are often the cause of the biotin deficiency. In difficult cases, we perform a comprehensive digestive stool laboratory analysis.

Supplying biotin will then allow ornithine to be converted into citrulline and the ornithine will no longer weaken, nor will ammonia weaken the patient with the biotin in the mouth. If ornithine strengthens the patient, it implies that there is inadequate ornithine, and the preceding steps in the urea cycle must be investigated, specifically the need for B-6 and magnesium, and CO2 from the CAC to generate it in the first place.

Ornithine is an essential nutrient in some patients who have memory lapse problems or senility type problems, especially disorientation. [4] Patients with memory loss and disorientation are many times greatly aided by the supplementation of ornithine. As with other non-essential amino acids, when we supplement patients with a specific amino acid, we also attempt to identify missing nutrient cofactors so that the body can produce its own. We rarely supplement non-essential amino acids without also investigating the potential needs for other nutrient cofactors and supplementing them as well. Supplementing the appropriate nutrient in the urea cycle will cause strengthening of the weak gamma 2 muscle and also neutralization of the weakening effect induced by sniffing ammonia.

Another factor associated with arginine strengthening the patient (and occasionally ammonia weakening) is in the over-trained patient. The over-trained or exhausted athlete, such as one who has over-extended the body in a marathon, will often re-route arginine from the urea cycle to combine with glycine and produce phosphocreatine. See figures 6 and 8. In these

FIGURE 8: ARGININE PLUS GLYCINE METABOLISM



patients, the production of phosphocreatine is a secondary energy source which becomes recruited when ATP stores become depleted from the excessive muscular activity. The damage to the body from the trauma of marathon racing also results in the breakdown of protein containing tissues and this further adds to the body's ammonia load. This pattern can also occur in other extremely stressed patients such as those with chronic disease or following major trauma or surgery.

In some patients the urea cycle is short-circuited to the point that the body is unable to totally rid itself of ammonia waste products. These patients have a very difficult time

recovering from their overactivity or stress, especially when confronted with both depleted ATP and phosphocreatine stores as well as ammonia toxicity. There is often a need for inorganic phosphorus supplementation in the form of ortho-phosphoric acid or disodium phosphate.

Supplying inorganic phosphorus to this patient is often sufficient to decrease the body's need to produce phosphocreatine for energy. Arginine can then be released from its alternate pathway back into completing the urea cycle and ammonia will be eliminated via that route. The production of phosphocreatine is a normal process, but when over-stressed, sometimes leads to a back-up of ammonia. This occurs even though the body can still further reduce the ammonia content by the conversion of phosphocreatine to creatinine which spills in the urine. Combined with the extra ammonia load from tissue damage, some patients have a great many symptoms and swear they will never compete again. Fortunately, few patients put themselves into such a predicament. But with proper diagnosis and nutritional therapy, they can rapidly recover.

The majority of the time when arginine strengthens a patient, it is due to a short-circuit in the urea cycle which can be analyzed step-by-step with the appropriate amino acids. Supplementing the appropriate amino acids for the short-term, and the appropriate nutrient cofactors for the long-term, leads to gratifying improvements in symptoms. This normalization of the urea cycle, elimination of ammonia waste, and consequent

detoxification of the body leads to impressive changes in neurological and neuromusculoskeletal function and improvement in many symptoms from many tissues.

IMPORTANT CLINICAL IMPLICATIONS OF HYPERAMMONEMIA

With excess available ammonia in the system, many neurotransmitters are affected, hence the transmission along many neurological pathways is affected. Changes in neurological transmission results in sensory, motor, emotional, and mental symptoms. Excess ammonia blocks the production of some neurotransmitters, enhances the production of others, and converts some neuroactive substances into other neurotransmitters with substantially different actions.

Glutamic acid, for example, which depends on ammonia for its synthesis from alpha-ketoglutaric acid from the CAC, is a facilitory neurotransmitter. Likewise, aspartic acid is a neurotransmitter which is synthesized when an ammonia molecule is attached to oxaloacetic acid. See figures 6 and 9. Oxaloacetic acid is part of the CAC and this reaction parallels alpha-ketoglutaric acid from the CAC being converted to glutamic acid. as can be seen by comparing figures 3 and 9.

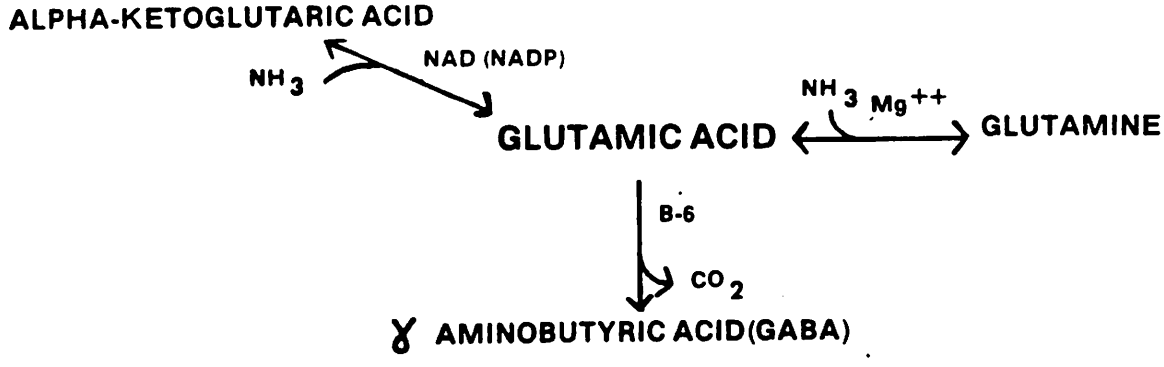
FIGURE 9

In the presence of excess ammonia, glutamic acid will be further converted to glutamine, and aspartic acid will be further converted to asparagine. See figures 6 and 9. The ammonia

problem essentially short-circuits the neurological pathways which are dependent on glutamic acid and aspartic acid.

Likewise, glutamic acid's conversion to glutamine inhibits its ability to be converted to gamma-aminobutyric acid (GABA), the most important inhibitory neurotransmitter in the brain. See figure 10. One-third of the cells in the brain use GABA. Some of today's most commonly prescribed medications, the benzodiazepine tranquilizer group, function by increasing GABA activity. These drugs include valium, xanax, tranxene, librium, ativan, dalmane, serax, halcion, restoril, and others. Correcting the functional metabolic faults associated with ammonia will allow many patients to produce their own GABA and decrease their needs for these medications.

FIGURE 10



Epileptic seizures and convulsive disorders are also often associated with decreased GABA activity. This includes from petit mal to grand mal type seizures and in any age group from newborns to geriatrics. Correcting GABA activity can help to control seizure activity and often allow patients to keep their

medication levels at a minimum. We do not withdraw these medications from patients without consulting with the prescribing physician. But many patients must take such high levels of these medications that they encounter side effects. Improving GABA activity will usually allow medications to be maintained below levels to induce side effects.

Glycine is another amino acid with important inhibitory neurotransmitter activity. Glycine is the neurotransmitter for the recurrent inhibition of Renshaw cells in the spinal cord. This pathway inhibits anterior horn motorneuron activity once the motorneuron has fired. The smooth contraction and relaxation of muscles is maintained at the spinal cord level by glycine activating this pathway. Deficiency of glycine causes the patient to experience much muscle tightness. We have seen many patients symptoms of muscle cramps, during the day and/or night, relieved by improving glycine activity.

Patients who have a deficiency of ammonia and who strengthen on the ammonia sniff test may also have faulty neurotransmitter production due to the inability of the body to convert alpha-ketoglutaric acid into glutamic acid, hence glutamine, hence GABA. The production of aspartic acid from oxaloacetic acid may be similarly affected.

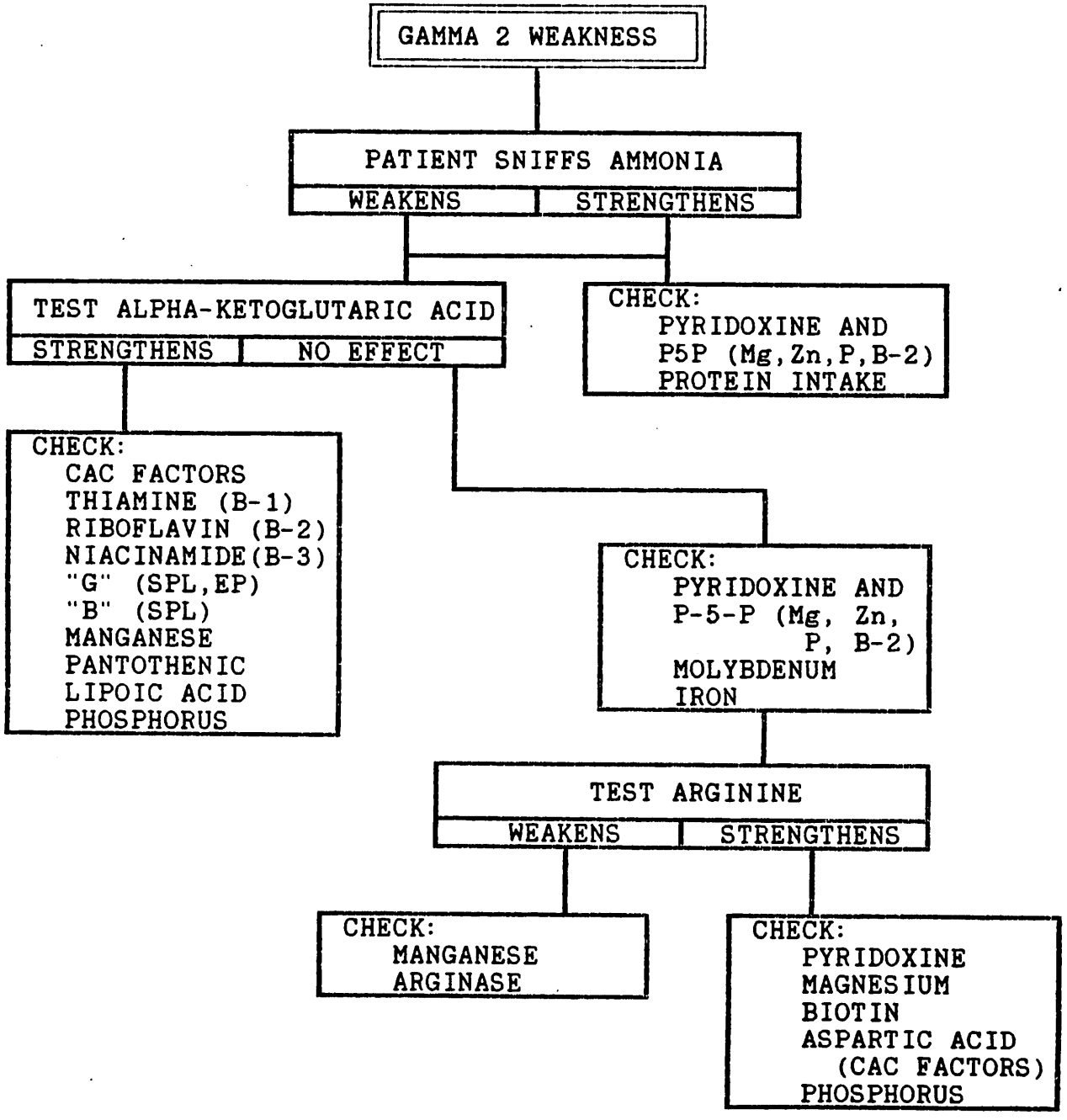
All major neurotransmitters (with the exception of acetylcholine) are either amino acids or are derived from amino acids. The more complicated neuropeptides (polypeptide molecules with neurological activity) are also dependent on adequate amino

acid metabolism. This amino acid metabolism is dependent on adequate ammonia transport which can be simply tested by having the patient sniff ammonia and observe for strengthening or weakening of muscles. The implications of faulty ammonia metabolism includes enzymes, hormones, and other peptide-related molecules and the list could go on and on. The clinical applications of these principles to neurological function, particularly the neuromuscular system, and hence the effects on musculoskeletal patients is certainly widespread. The vast variety of symptoms which we see as secondary complaints are also often explained by altered ammonia metabolism.

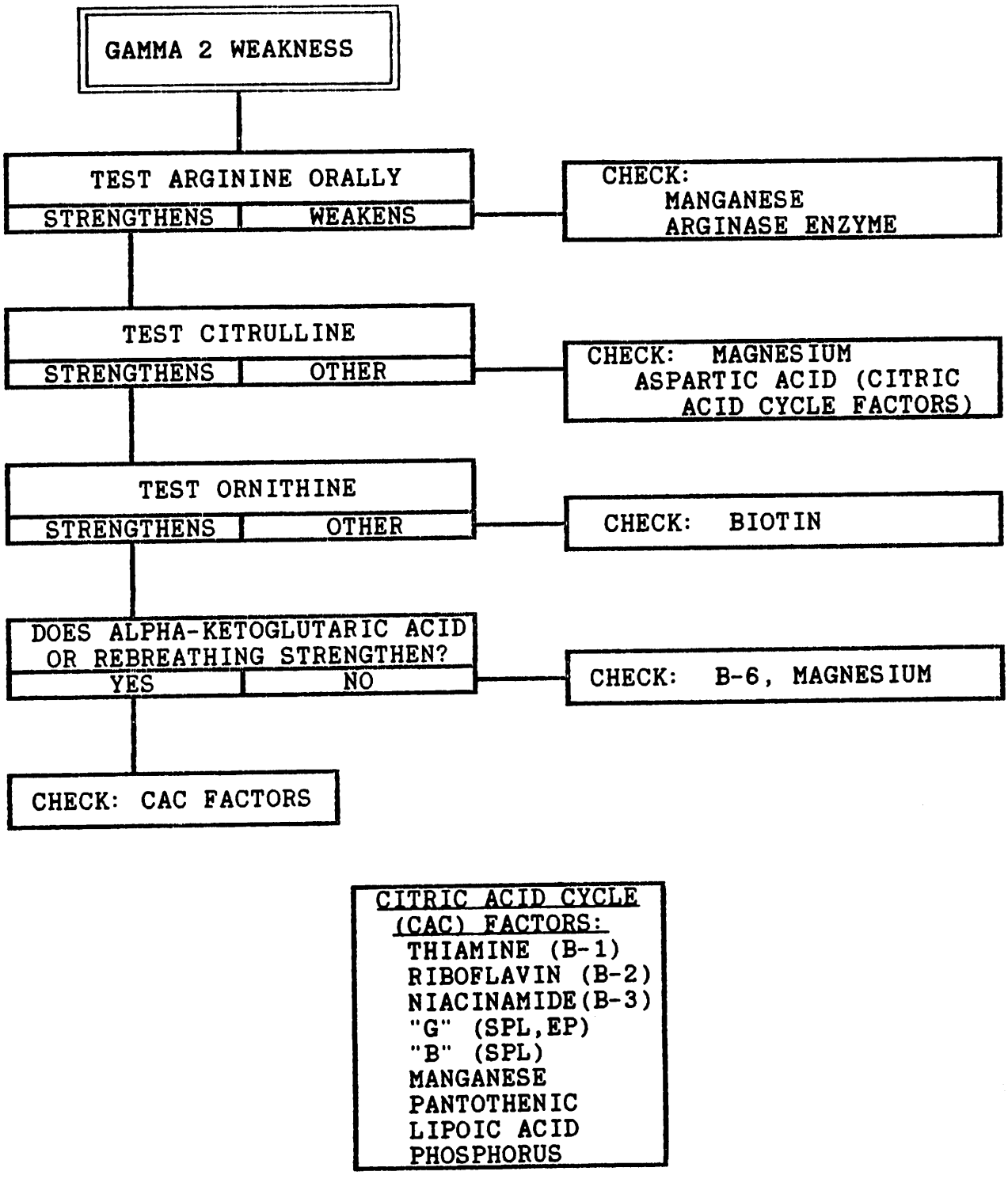
CONCLUSIONS

The identification of problems with ammonia metabolism as ascertained by ammonia sniff test allows us to penetrate deeply into the intermediary metabolism of our patients. Strengthening or weakening of muscles on sniffing ammonia implies problems which have far-reaching effects in our metabolism and in the repair and degradation of tissues.

FLOW CHART: THE AMMONIA SNIFF TEST



FLOW CHART: UREA CYCLE ANALYSIS



CITRIC ACID CYCLE (CAC) FACTORS:
- THIAMINE (B-1)
- RIBOFLAVIN (B-2)
- NIACINAMIDE(B-3)
- "G" (SPL,EP)
- "B" (SPL)
- MANGANESE
- PANTOTHENIC
- LIPOIC ACID
- PHOSPHORUS

REFERENCES

1. Schmitt, Walter H., Jr. Making B-6 work. I.C.A.K. collected papers, Winter, 1987.

2. Mowles, Richard A., A clinical study investigating the diagnostic screening and application of the trace mineral, molybdenum, in applied kinesiology. I.C.A.K. collected papers, Summer, 1985.

3. Schmitt, Walter H., Jr. Identifying the need for B vitamins, manganese, and coenzyme Q: an answer to recurrent hypoglycemia and hypoadrenia. I.C.A.K. collected papers, Winter, 1987.

4. Goodheart, George J. Applied Kinesiology 1987 Research Manual. Detroit: privately published, 1987. pp. 77-79.

GLUTATHIONE: THE MOST IMPORTANT MOLECULE IN THE CELL

Walter H. Schmitt, Jr., D.C.

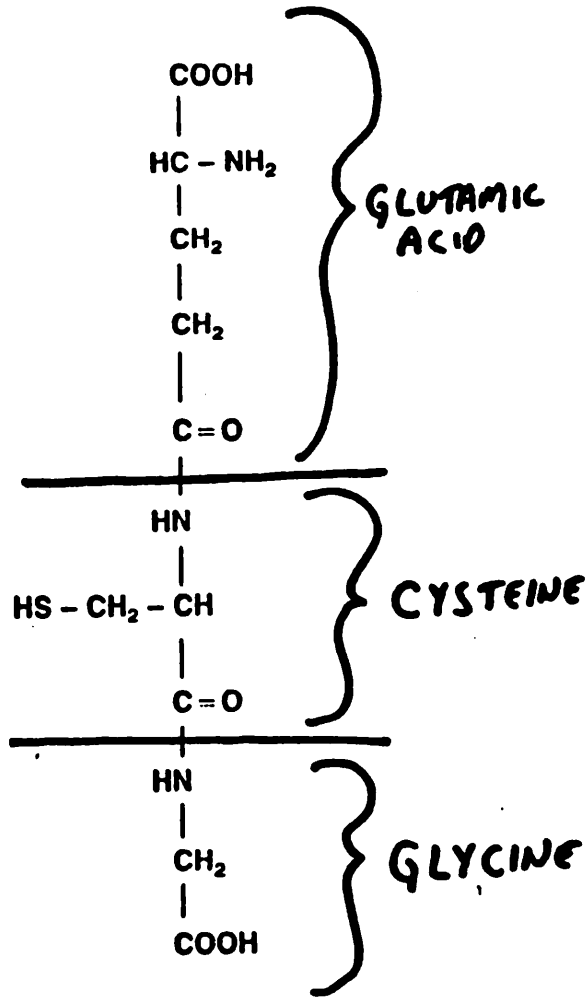
ABSTRACT: The tripeptide glutathione is at the center of the electron poising system for regulating oxidation and reduction in cells. Restrictions of any of its precursors, the amino acids cysteine, glutamic acid, or glycine, can result in metabolic problems which may be monitored by muscle testing. Procedures are described for identifying needs for the vitamin and mineral cofactors for the production of these three amino acids, and hence glutathione itself. These procedures penetrate of the metabolism of sulfur amino acids, the citric acid cycle, the urea cycle and energy metabolism.

INTRODUCTION

The molecule glutathione is a tripeptide which consists of three amino acids: cysteine, glutamic acid, and glycine. See figure 1. It is a molecule which is ubiquitous in nature and is of the utmost importance to the functioning of the cells in the human body, as well as elsewhere in nature. The importance of glutathione lies in its ability to maintain the electron poising system as an oxidation - reduction balancing act in the cell. In the absence of adequate glutathione, the cellular chemistry is unable to self-regulate via the cellular electron poising system.[1] See figure 2.

We have learned that it is relatively easy using muscle testing to evaluate the status of glutathione in the body, at least in a general sense. When there are inadequate amounts of

FIGURE 1

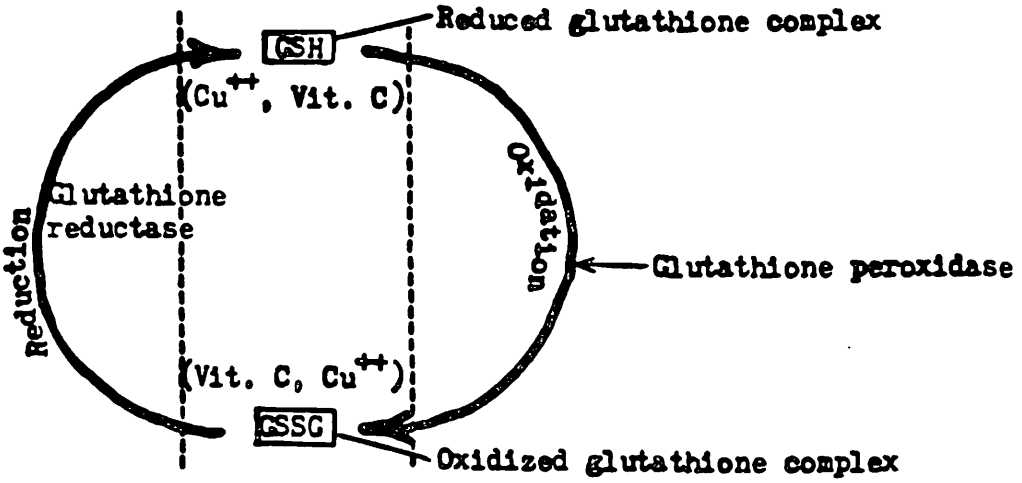


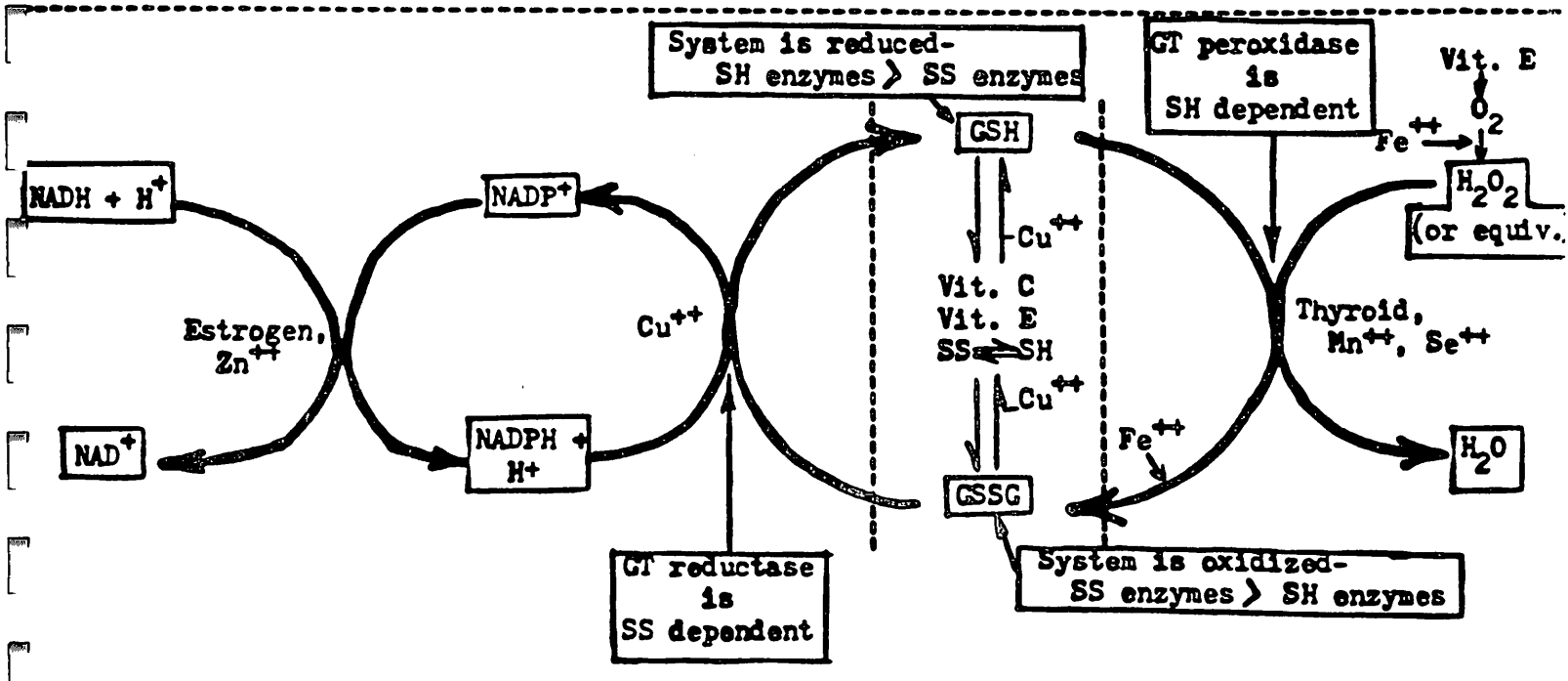
the three amino acid precursors to glutathione, cellular chemistry will go awry and the symptoms can be any at all, anywhere in the body. Therefore, we have found it useful to screen for problems with glutathione in all patients with problems which are not easily resolved. This paper reviews the clinical procedures involved with the nutritional evaluation of

the glutathione status of the patient and its three amino acid precursors. Simplified flow charts of the step-by-step procedure for evaluating glutathione status will be included.

FIGURE 2: THE ELECTRON POISING SYSTEM

Copper catalyzes the coupling of glutathione and ascorbic acid. This Cu^{++}, glutathione, ascorbic acid complex functions to "poise" electrons for the oxidation and reduction potentials of the cell.





Adapted from "A Precis on Cellular Electron Poising, Ergodization, and Molecular Quantization" by James Pershing Isaacs and John C. Lamb, 6th Annual Trace Mineral Conference, University of Missouri, 1974.

First we will discuss the synthesis of glutathione and potential problems therein. Next we will discuss the availability of each of the individual amino acids for the production of glutathione, and the nutritional factors which are associated with each.

GLUTATHIONE SYNTHESIS

The initial screening procedure for a glutathione need is performed using a muscle which shows both gamma 1 and gamma 2 type weaknesses. We ask the patient taste a substance which contains all three amino acids, cysteine, glutamic acid, and glycine, in equal amounts and retest the weak muscle. We have learned that using a mixture of amino acids is a better screening test than using the glutathione complex itself. This is different from what you might expect.

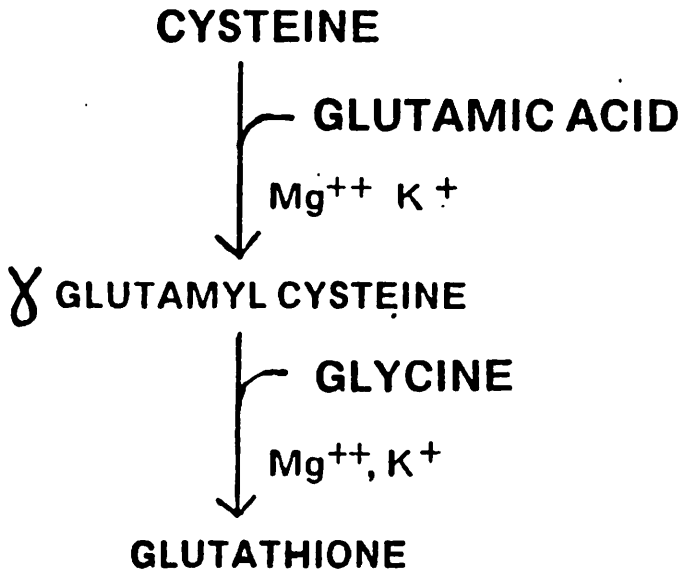
Glutathione complex, in its tripeptide form, is very expensive, and is used by some practitioners to supply glutathione in its complete form. However, the body is supposed to be able to synthesize glutathione from the three precursor amino acids. We have not seen any value in supplementing or testing with glutathione complex in the patients we have seen. In fact, use of the glutathione complex for screening will not cause strengthening of a weak muscle in many patients who do strengthen with the three amino acid mixture.

When a patient strengthens on the glutathione three amino acid mixture, we then orally test each of the amino acids individually. Our order of preference is to first test cysteine

against a gamma 2 weakness, then glutamic acid, then glycine. We note the response of these individual amino acids.

If glutathione mixture strengthens a patient, one (or more) of the three amino acid precursors will also strengthens the patient. If two or more of these three amino acids strengthen the patient, then we consider a possibility that the body is not properly synthesizing glutathione. If one uses the glutathione complex, rather than the amino acid mixture, and this strengthens the patient, we also need to rule out the possibility of inadequate glutathione synthesis.

FIGURE 3: SYNTHESIS OF GLUTATHIONE



The synthesis of glutathione is in two steps. See figure 3. The first step combines cysteine with glutamic acid. Note in figure 3 that potassium and magnesium are co-factors for this reaction. Then this molecule, gamma-glutamyl cysteine, combines

with glycine to form the tripeptide glutathione (gamma-glutamylcysteinylglycine). Note in figure 3 that this step also requires magnesium and potassium. When two or three of these amino acids strengthen the patient, we consider the possibility that the body is not properly synthesizing glutathione, possibly due to a deficiency of potassium or magnesium.

Parathyroid activity is also essential in this synthesis. If a patient strengthens on two or three of the amino acids, we may test for magnesium, potassium, or parathyroid activity in the patient. Recalling that magnesium and potassium are two important alkaline-ash minerals, we also must consider that this patient may be overly acidic and require alkaline ash mineral supplementation and/or more alkaline-ash material in the diet.

GLUTATHIONE PRECURSORS

An unavailability of cysteine, glutamic acid, and/or glycine will also disrupt the production of glutathione due to the restriction of these amino acids for building the glutathione molecule. Since each of these amino acids are synthesized by the body, they are considered non-essential. But there are various essential vitamin and mineral cofactors which are necessary for their production. A deficiency of any vitamin or mineral cofactor will produce a limited availability of the amino acid which is dependent upon that nutrient.

Cysteine, glutamic acid, and glycine have several nutrient cofactors in common. If two or three of the individual glutathione precursors strengthen the patient, it may be due to a

deficiency of one of the nutrient cofactors that they each have in common. These include B-6, folic acid, and B-2.

The synthesis of each of these three amino acids is dependent on adequate vitamin B-6 activity. Also, for two of these amino acids, cysteine and glycine, folic acid is necessary. For glutamic acid and glycine, B-2 is necessary. So, depending on which two or three amino acids strengthen the patient, we may also find a nutritional deficiency of one of the nutrients which they have in common, rather than a synthesis problem involving potassium, magnesium, or parathyroid activity.

We have found it useful upon identifying a need for one of these nutrients to leave the nutrient in the patient's mouth while we continue treatment. Without the oral activation of the nutrient, many spurious findings remain present which will throw us off-course in treating the patient. Leaving the essential nutrient in the mouth will allow the nervous system to believe that it is going to be having adequate glutathione activity for regulation of cellular chemistry and therefore will make the treatment of the patient much more straight-forward and clear.

After ruling out involvement of two or three of the amino acids, we then evaluate each of the amino acids individually.

Because glutathione and each of its precursor amino acids are normally synthesized by the body, and none of them is an essential amino acid, their importance has often been overlooked. Their levels have simply, but erroneously, been assumed to be adequate. The concern with the essential amino acids is of minor

importance in our civilized society, and yet these have always been emphasized in nutrition courses.

Most people in our society have more than adequate amino acid intake. Some people have inadequate digestion and absorption. But it is the synthesis of the non-essential amino acids, (and hence glutathione) which depends on numerous vitamin and mineral co-factors, that can be found at fault most commonly in our experience. The resultant problems with the glutathione regulatory system can lead to symptoms in any tissues in the body which will not resolve until this system is normalized.

In most patients who strengthen on the glutathione mixture, only one of the precursor amino acids will also strengthen. We will discuss the important factors and cofactors for the synthesis of each of these amino acids separately.

CYSTEINE

Cysteine is a sulfur-containing amino acid which is synthesized by the body. The synthesis of cysteine is involved with a number of nutrients which are summarized in figure 4. Its formula along with the formulae of the other sulfur containing amino acids is found in figure 5. It is derived from the essential amino acid methionine. See figure 6.

Methionine is a major methyl donor in the body chemistry. It is recirculated after donating a methyl group in a pathway which involves folic acid, vitamin B-12, and another methyl donor such as choline or betaine. Methionine recirculates and gives and takes methyl groups in many normal metabolic pathways.

FIGURE 4: CYSTEINE FACTORS

CYSTEINE

MAGNESIUM
B-6
FOLIC ACID
B-12
METHYL DONOR

IF CYSTINE WEAKENS:
B-2
NIACINAMIDE
G (S.P.L.)
COPPER

FIGURE 5: SULFUR CONTAINING AMINO ACIDS

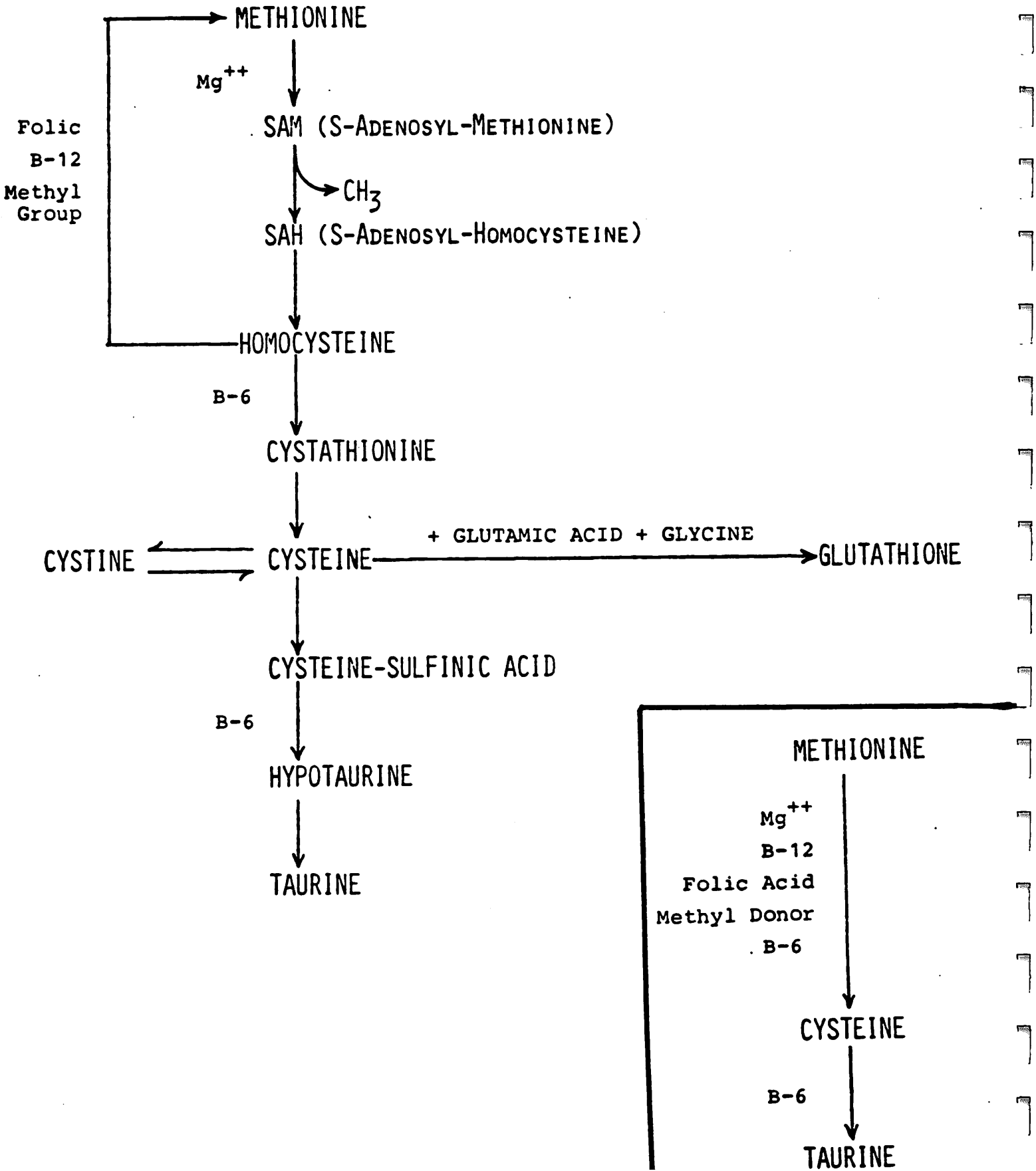
METHIONINE: $CH_3-S-CH_2-CH_2-CH(NH_2)-COOH$

CYSTEINE: $HS-CH_2-CH(NH_2)-COOH$

CYSTINE: $CH_2(NH_2)-CH-COOH$ — $S-S$ — $CH_2(NH_2)-CH-COOH$

TAURINE: $SO_3H-CH_2-CH_2(NH_2)$

FIGURE 6: SULFUR AMINO ACID METABOLISM



Methionine can also be converted through homocysteine into cysteine and further into taurine. Taurine is an essential anti-oxidant substance for the neutralization of hypochlorite free radical (OCl^-), which is screened for by the Clorox sniff test. [2] But before taurine can be produced, the body must first be able to produce cysteine.

When cysteine strengthens a patient, we next test for methionine. If methionine strengthens the patient, there are two possibilities. The first is that the patient has deficient methionine. The second is that the patient is not adequately recycling methionine. If the patient is deficient in methionine, there will usually be some clinical indication, such as vegetarianism, poor diet poor digestive process, which will become clear from the patient's history. The use of methionine is important in these cases, but they are the exception rather than the rule.

More often we find patients with deficiencies of vitamin B-12 or folic acid or the need for a methyl donor, such as choline or betaine, in order to allow methionine to recirculate. In figure 6 you will note that methionine gives off its methyl group in the presence of magnesium. In some patients a magnesium deficiency will limit the demethylation of methionine, and will cause a back-up of methionine. In these patients, we observe a weakening on insalivation of methionine. We have also observed methionine weakening in some patients who have a molybdenum need. If methionine weakens, magnesium and molybdenum both must be

tested for the patient.

If methionine strengthens, this implies either a need for methionine or the factors which help keep the methionine demethylation - methylation cycle going: vitamin B-12, folic acid, and methyl donor, such as choline, or betaine. If cysteine strengthens and methionine has no effect, it implies that there is a problem somewhere between methionine and cysteine. Observing the pathway in figure 6, you will see that this would indicate a need for vitamin B-6. In these patients, muscle testing usually indicates a vitamin B-6 need. We support the patient with whichever nutrient (or nutrients) tests positive.

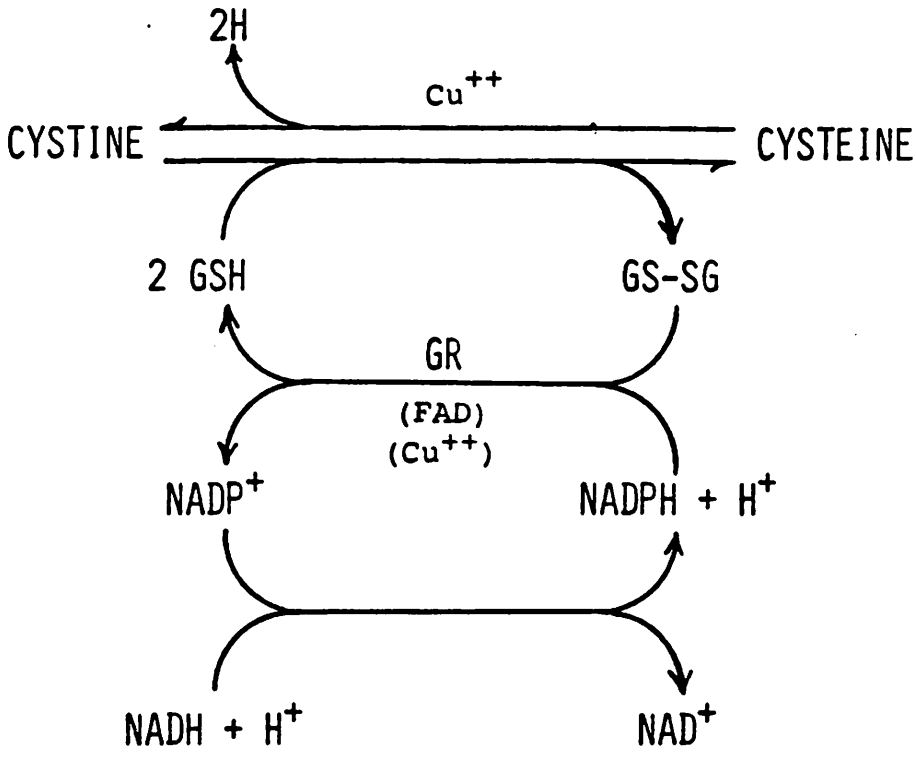
Some patients whose tissues are very over-oxidized (such as occurs in free radical pathology) will be caught in a vicious cycle of over-oxidation with the inability of adequate glutathione production for regulation of the excess oxidation at the cellular level. In these patients we will often find that glutathione strengthens the patient while none of the precursors previously mentioned cause a response.

The amino acid, cystine, is produced by the oxidation of two cysteine molecules (figures 5 and 7) which causes them to bind together. If we orally test cystine on over-oxidized patients, we will find that these patients will usually weaken on cystine and strengthen on cysteine. This implies that as fast as these patients produce cysteine, it is oxidized to cystine.

In these patients we must test the nutrients which help to convert cystine into cysteine, and these include riboflavin,

niacinamide and also monitoring the copper status. See figure 7.

FIGURE 7



GSH = REDUCED GLUTATHIONE

GS-SG = OXIDIZED GLUTATHIONE

GR = GLUTATHIONE REDUCTASE
(RIBOFLAVIN (FAD) DEPENDENT)
(COPPER (Cu^{++}) DEPENDENT)

Too much or too little copper can interfere with this reaction. We also must screen these patients for other forms of over-oxidation, such as the clorox sniff test [2] and for the possible need for other anti-oxidants which can help unload this particular anti-oxidant mechanism.

The vicious cycle in these patients is often compounded when the short-circuiting of cystine into cysteine blocks the ability

of the body to produce taurine. Since taurine acts as an anti-oxidant to neutralize hypochlorite free radicals, this aggravates the over-oxidized state of the tissues and stimulates still more conversion of cysteine into cystine, further short-circuiting the production of taurine. This patient gets in a very serious tail-spin downward and oftentimes needs aggressive application of the nutritional measures mentioned to pull them out.

GLUTAMIC ACID

Glutamic acid is produced from alpha-ketoglutaric acid by the addition of an ammonia group as shown in figure 8. Alpha-ketoglutaric acid is an important keto-acid normally synthesized in the citric acid cycle. See figures 9a and 9b. When we identify a patient who strengthens on glutamic acid, our next step is to test alpha-ketoglutaric acid so we may determine the reason for the glutamic acid need.

FIGURE 8

If alpha-ketoglutaric acid does not strengthen the patient, and glutamic acid does strengthen, this implies a problem with the amination (adding of an ammonia group) of alpha-ketoglutaric acid to produce glutamic acid. See figure 9b. This alpha-ketoglutaric acid to glutamic acid pathway is the major transamination mechanism (i.e., transport for ammonia groups) in the body. When transamination pathways are faulty, the body can build up excess ammonia. Excess ammonia in the blood is called

FIGURE 9a

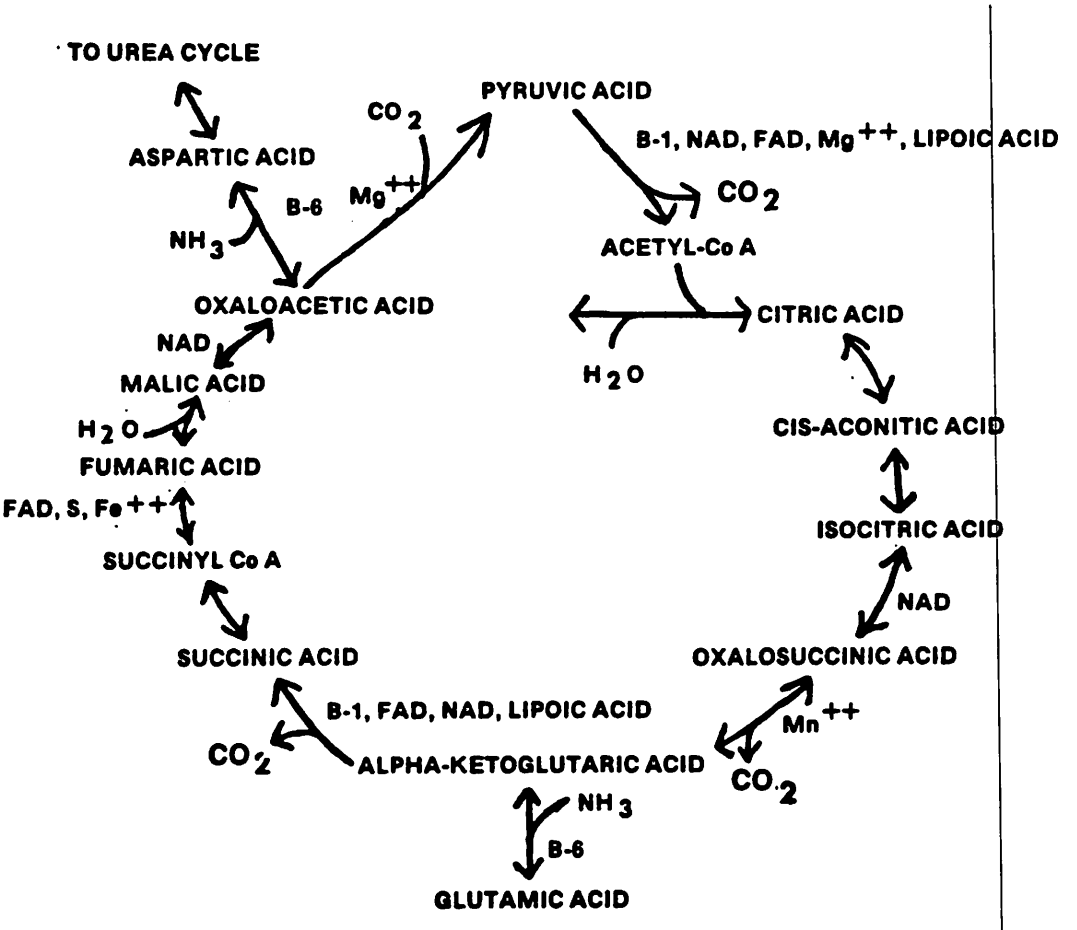
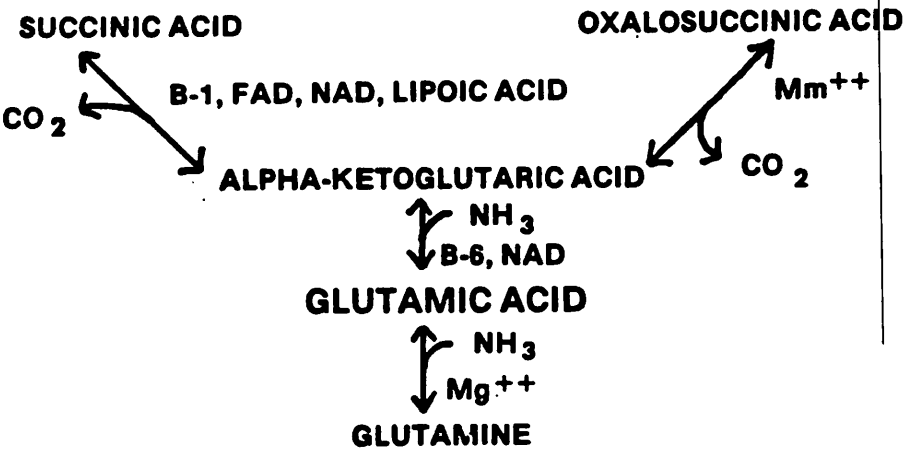


FIGURE 9b: PORTION OF CITRIC ACID CYCLE



hyperammonemia.

If the patient is unable to produce glutamic acid from alpha-ketoglutaric acid, many other transaminations throughout the body will be affected. Problems in transamination have far-reaching negative impacts including interference with the growth and repair of tissues, the synthesis of protein enzymes, hormones, neurotransmitters, and also the degradation of these substances. The importance of this pathway for the movement of ammonia around the body cannot be over-emphasized.

If alpha-ketoglutaric acid has no effect and glutamic acid strengthens, the first thing we do is have the patient sniff ammonia. If the patient strengthens or weakens on sniffing ammonia, we can assume that this transamination pathway is not working properly. We must make the appropriate nutritional corrections in these patients if we ever hope to achieve any repair or healing of tissues and regulation of cellular chemistry at the levels of enzymes, hormones, and neurotransmitters.

Glutamic acid is synthesized from alpha-ketoglutaric acid using vitamin B-6 and niacinamide. Most commonly if alpha-ketoglutaric acid has no effect and/or weakens the patient (and glutamic acid strengthens) the problem is with the amino acid transport and requires B-6. In some rare individuals there may be a total amino acid deficiency due to improperly practiced vegetarianism or starvation, and these patients will tend to strengthen to ammonia. Upon supplying adequate protein in the diet and/or improving protein digestion, these patients will

improve. The majority of patients, however, require vitamin B-6 or niacinamide to correct this pathway.

Vitamin B-6 in its activated form, pyridoxal-5-phosphate (P5P), is a major activator of all the transamination reactions in the body. Vitamin B-6 must often be tested in its activated, P5P form to identify its need. (See paper entitled "Making B-6 Work". 3)

If glutamic acid strengthens the patient and alpha-ketoglutaric acid also strengthens the patient, the problem lies in the ability of the body to produce alpha-ketoglutaric acid. Note in figures 9a and 9b that the production of alpha-ketoglutaric acid involves the release of a carbon dioxide (CO_2) molecule. When alpha-ketoglutaric acid strengthens a patient, having the patient rebreathe in a paper bag for eight to ten times will also strengthen a weak muscle. Rebreathing increases CO_2 levels in the body and parallels the finding of alpha-ketoglutaric acid strengthening.

Upon finding alpha-ketoglutaric acid or rebreathing to strengthen, we assume a problem with the citric acid cycle. We would then test the patient keeping the pathways of the citric acid cycle in mind. See figure 9a. When we identify a problem in the citric acid cycle, we proceed to test the various nutrients which are involved in the promulgation of this cycle. These include B-1, B-2, B-3. pantothenic acid, lipoic acid, "B", "G" and manganese, as discussed in a previous paper. [4]

The nutrient factors which are concerned with the production

are summarized in figure 10. Applying the appropriate nutrient will not only correct citric acid cycle function in these patients but also correct the major transamination reaction in the body. As you can see, glutamic acid is a non-essential amino acid and yet it has great importance in the body. It is also the precursor to gamma-aminobutyric acid (GABA - see figure 11) and glutamine.

FIGURE 10: GLUTAMIC ACID FACTORS

GLUTAMIC ACID

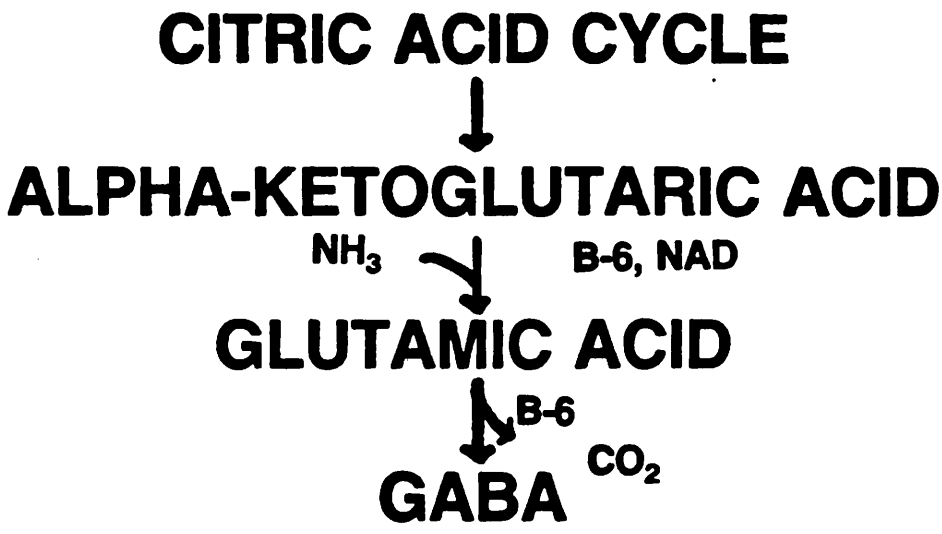
B-6 (NH_3)

IF REBREATHING AND/OR ALPHA-KETOGLUTARIC ACID STRENGTHEN:

B-1
"B"
MANGANESE
PANTOTHENIC ACID
B-2
NIACINAMIDE
"G'

GABA is the most important inhibitory neurotransmitter in our brains and a full one-third of the cells in the brain use GABA. To give you an idea of how many people have GABA problems, the major class of tranquilizer drugs, the benzodiazepines, have their effects by increasing GABA activity in the central nervous

FIGURE 11: GABA METABOLISM



system. These tranquilizers are some of the most commonly prescribed drugs and include: valium, librium, tranxene, ativan, xanax, dalmane, serax, halcion, restoril, and others.

GABA's precursor, glutamic acid, is a facilitory neurotransmitter in its own right. Therefore the neurological implications of a deficiency of glutamic acid, are far-reaching.

The inability of the body to process ammonia and hence, produce adequate glutamic acid, may also be associated with a problem in production of glutamine. Glutamine is nothing more than glutamic acid plus an ammonia group as can be seen in figures 8 and 9b. In patients with hyperammonemia, some of the symptoms are low GABA symptoms from the short-circuiting of glutamic acid away from GABA and into glutamine.

Some patients show no response (i.e., normal levels) to alpha-ketoglutaric acid and strengthen (i.e., a need for) on glutamic acid. In these patients, if you measure glutamine, you find that glutamine weakens the patient. Ammonia sniff will

always induce weakness in these patients as well.

There is a lot of popular misinformation about glutamine circulating in the public lately. The major falsehood being advanced is involved with the fact that glutamine is the major route of elimination of ammonia from the brain. This, of course, occurs due to ammonia attaching itself first to alpha-ketoglutaric acid, and then to glutamic acid to form glutamine, as just discussed. Glutamine can cross the blood-brain barrier and hence is the chief route of getting ammonia out of the brain.

Based on a loose interpretation of this metabolic fact, many misinformed lay people are taking glutamine to try to help their brains get rid of excess ammonia which they fear they might have there. They say "glutamine carries ammonia out of the brain and so I am taking it to help that process." If these poor people do have a excess ammonia problem, glutamine will only contribute to it. Glutamine contains two ammonia molecules, and to supplement more ammonia in the hyperammonemia patient is not just foolhardy, it is dangerous. What these excess ammonia patients should take is alpha-ketoglutaric acid, or glutamic acid, and/or the appropriate other vitamin and mineral cofactors such as B-6 and the citric acid cycle factors. Glutamine usually weakens them as does sniffing ammonia. This is a perfect example of a little knowledge being a dangerous thing.

The handling of ammonia involves a number of pathways other than those just discussed. The urea cycle is discussed in the next section and other methods which affect ammonia metabolism

are the subject of an accompanying paper. [5]

GLYCINE

Glycine is the simplest of all amino acids. It is a simple two carbon amino acid which is easily synthesized through several pathways in the which require adequate folic acid, vitamin B-6, manganese, and vitamin B-2. See figure 12. Sometimes in the presence of a deficiency of one of these nutrients, glycine is inadequately synthesized. However, oftentimes the problem with glycine is that it is being used up faster than it can be produced.

FIGURE 12: GLYCINE METABOLISM

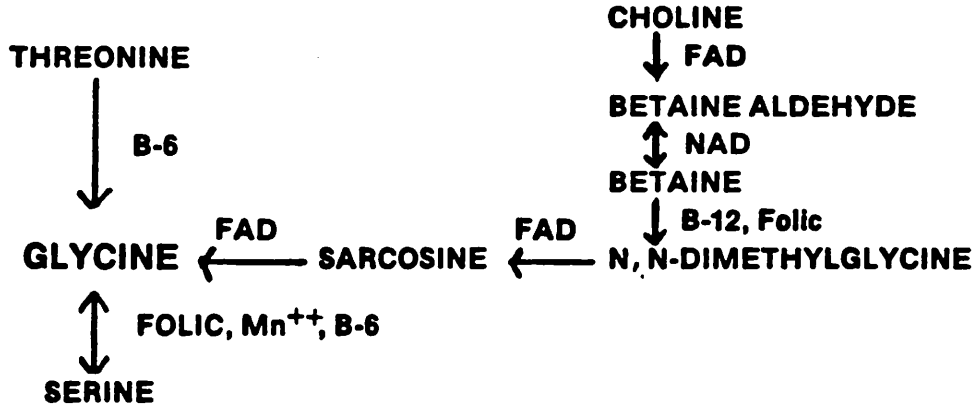
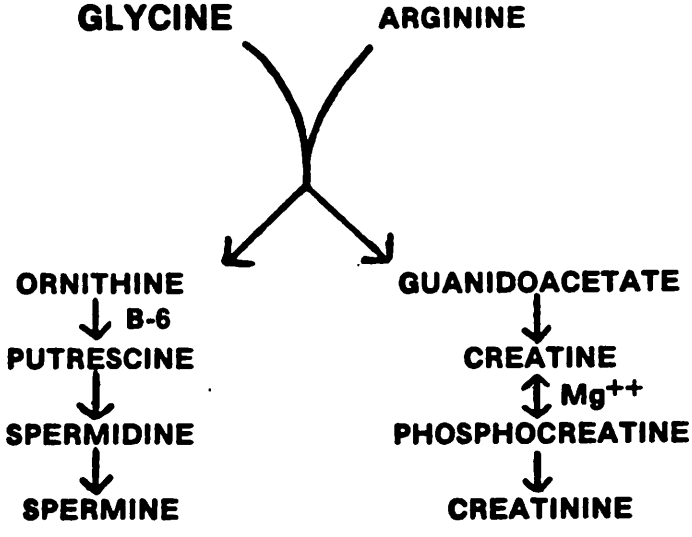


FIGURE 13: GLYCINE AND ARGININE METABOLISM



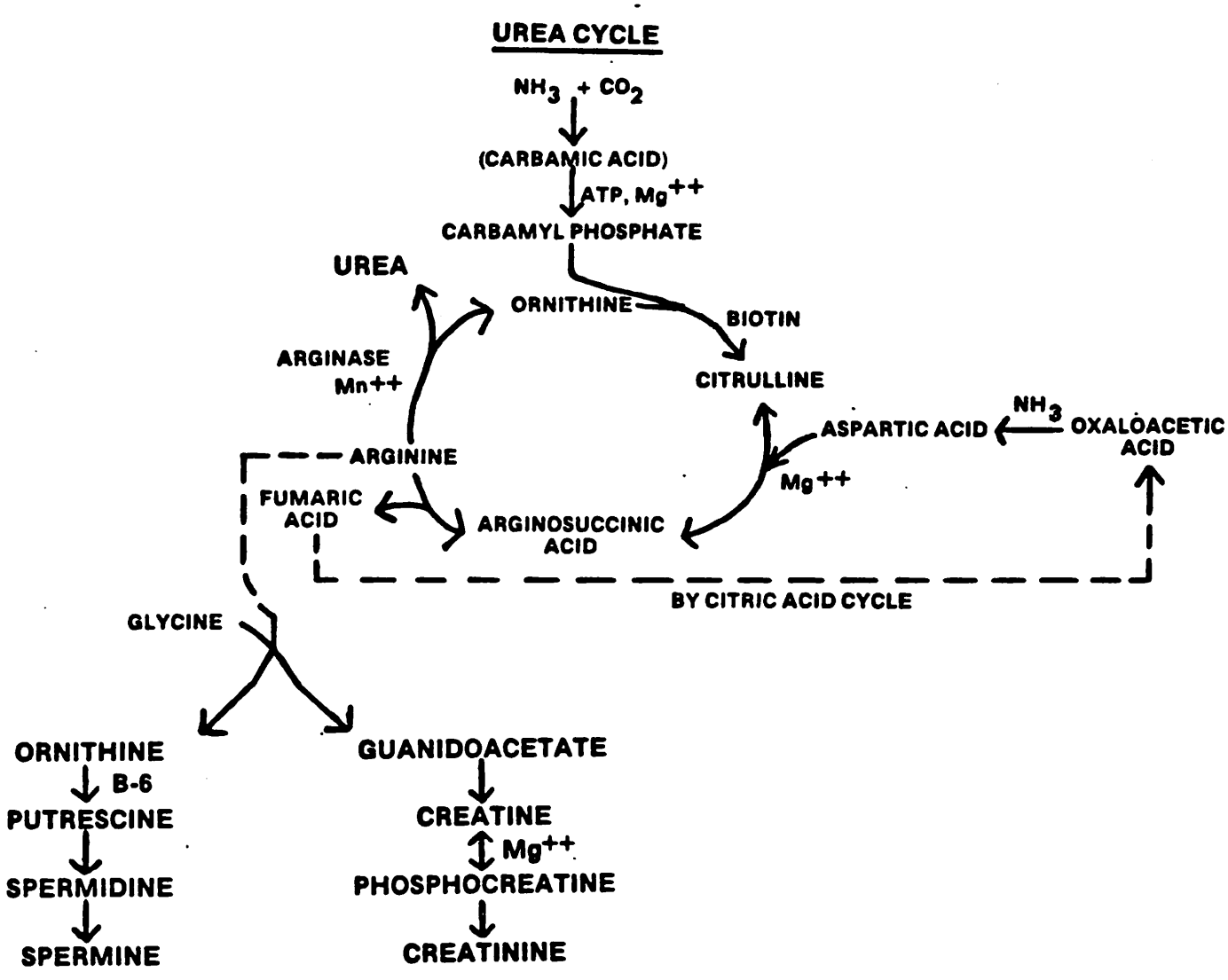
In another pathway, glycine combines with arginine and through two steps produces phosphocreatine (creatine phosphate) for muscle energy. Phosphocreatine is further converted to creatinine which spills in the urine and acts as a secondary route of elimination for ammonia metabolism. See figure 13.

There are two factors about this pathway which can result in a depletion of glycine: 1. Excess levels of arginine can combine with all the available glycine stealing if from its other functions. 2. Depletion of muscular phosphocreatine reserves, such as in overtraining or marathon racing, can also shunt glycine into this pathway leaving a lack of it for other functions.

Arginine is a normal constituent of the urea cycle, the major route of elimination of ammonia waste for our bodies. See figure 14. Each molecule of arginine contains three ammonia molecules. Each molecule of urea carries two ammonia molecules out of the body as it is eliminated. It is arginine, under the influence of arginase, a manganese-dependent enzyme, which is broken down into urea and removed by the kidneys. What is left is ornithine which keeps the urea cycle going.

Deficiencies of manganese or other factors which interfere with arginase enzyme activity will interrupt the urea cycle with consequent buildup of arginine. If arginine cannot be broken down into urea, the body will find an alternate pathway to rid itself of the excess ammonia. This alternate pathway involves arginine combining with available glycine and eliminating ammonia

FIGURE 14



via the formation of creatinine which spills in the urine as shown in figure 13.

Because of this close relationship of glycine and arginine, when we find glycine strengthens a patient we also find it useful to test arginine. If arginine has no effect on muscle testing, neither strengthening nor weakening, we then assume that the problem with glycine is in its synthesis and test the four

synthesis related nutrients as previously mentioned.

When glycine strengthens, if arginine either strengthens or weakens a muscle, we make the assumption that the need for glycine is due to glycine's combination with arginine. The most common problem we see here is that arginine weakens the patient because the body is unable to rid itself of arginine due to sluggish arginase enzyme activity. The urea cycle becomes short-circuited and ammonia sniffing will also weaken the patient. In this case, we test the patient for two substances which are necessary for the break-down of arginine, arginase enzyme ("Arginex", Standard Process Labs), and its activating mineral, manganese. One or both of these will strengthen the patient and should be supplemented. The glycine factors which have been discussed so far are summarized in figure 15.

FIGURE 15: GLYCINE FACTORS

FOLIC ACID
MANGANESE
B-6
B-2

IF ARGININE WEAKENS:

ARGINEX (S.P.L.)
MANGANESE

If glycine strengthens the patient and arginine also strengthens the patient, it implies that the body needs both of them. This is not a very common occurrence in the average

patient. However, it is seen commonly in over-trained athletes or marathoners who have over-extended themselves. As muscle energy is depleted, ATP stores are depleted and the body unloads high-energy phosphate bonds from phosphocreatine to maintain energy cycles.

The excess need for phosphocreatine production in these patients depletes arginine and glycine stores and we see both arginine and glycine strengthening. Phosphorus can also be depleted in these patients and they must be tested for possible phosphoric acid supplementation. It is also necessary to supply the nutritional substances which help the body to produce arginine. These include B-6, magnesium, biotin, and aspartic acid which from the citric acid cycle as can be seen in figure 14. If aspartic acid strengthens, the citric acid cycle nutrient factors must be tested as well. These nutrients can be tested for individually or by a thorough analysis of the urea cycle, which is discussed in another paper. [5]

Arginine can also cause strengthening in patients who are not overtrained. This implies a need for better urea cycle function and is often accompanied by the ammonia sniff weakening. When arginine strengthens and ammonia weakens, the factors summarized in Figure 16 must be tested.

In this patient, the depletion of these nutrients further complicates the patient's condition due to the hyperammonemia from the faulty urea cycle. This especially complicates the healing process in the over-trained individual.

FIGURE 16

IF ARGININE STRENGTHENS AND AMMONIA WEAKENS:

B-6
Magnesium
Biotin
Aspartic Acid (Citric Acid Cycle)
or
Phosphorus (eg. over-training)

Supplying the appropriate nutrients for arginine production will lead to correction in most of these patients. Occasionally, however, it is also necessary to supply nutrient cofactors for glycine production to insure adequate quantities of both amino acids for phosphocreatine replacement. With the possible addition of phosphoric acid as well, restoring these reserves helps the patient more rapidly replete ATP reserves and recover much faster.

Although we have most commonly observed this pattern in patients who have had difficulty recovering from a marathon, you might also find it in patients who are slow to recover from accidents, surgery, or other reserve depleting incidents. Supplying the appropriate nutrients enhances rapid recovery and the patient can soon resume previous activity levels.

CONCLUSIONS

Glutathione is the most important molecule in the cellular chemistry due to its role in the maintenance of oxidation - reduction reactions. This oxidation - reduction regulation is

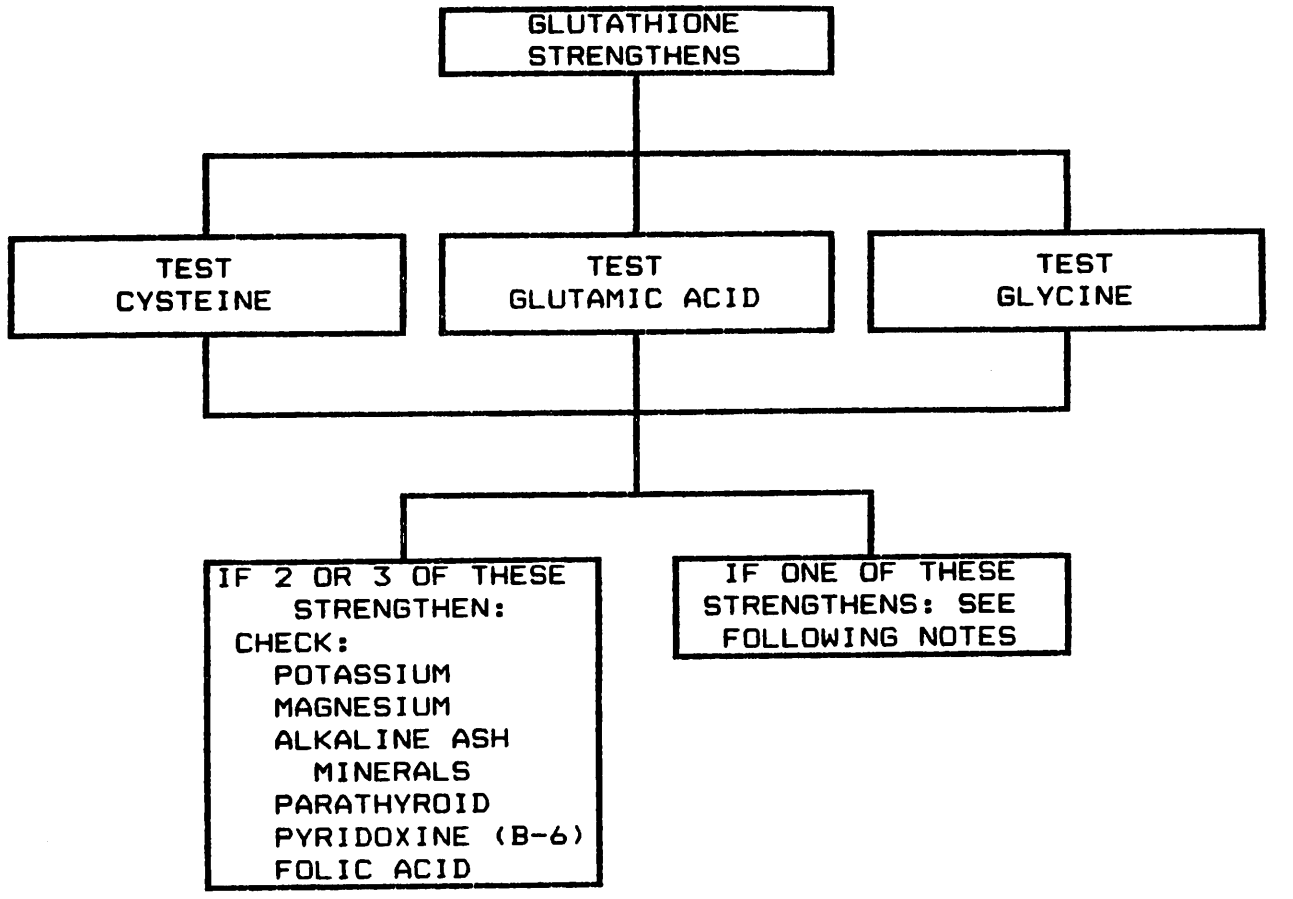
essential for cells to perform their functions, whether they be muscles producing energy, glands producing hormones, nerves producing neurotransmitters, or any other body functions. Without glutathione, the cellular chemistry of any or all tissues in the body is unable to self-regulate. Homeostasis cannot be achieved, and any symptom can appear due to the disregulation of cellular chemistry.

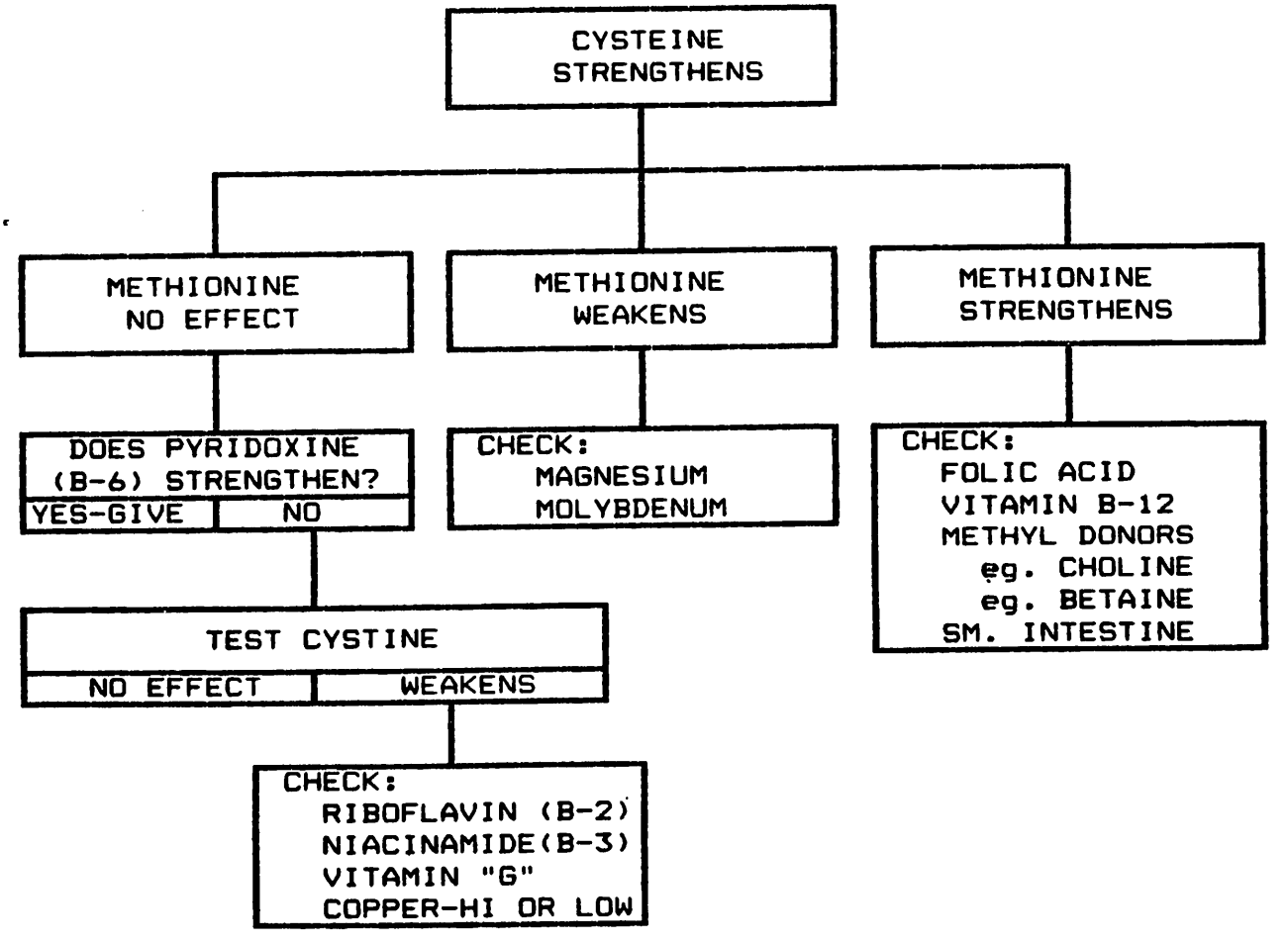
Following relatively simple muscle testing screening procedures using oral testing of glutathione mixture and each of the individual amino acids, we are able to supply patients with nutritional factors which allow the body to produce adequate glutathione. In doing so, we penetrate deeply into the body's intermediary metabolism. We can see first hand the metabolism of the sulfur amino acids, the citric acid cycle, the urea cycle and energy metabolism. Our patients body's become our laboratories for qualitative analysis which can be cost effectively monitored from day to day. These muscle testing procedures can be correlated at appropriate intervals with blood and urine tests for quantitative analysis.

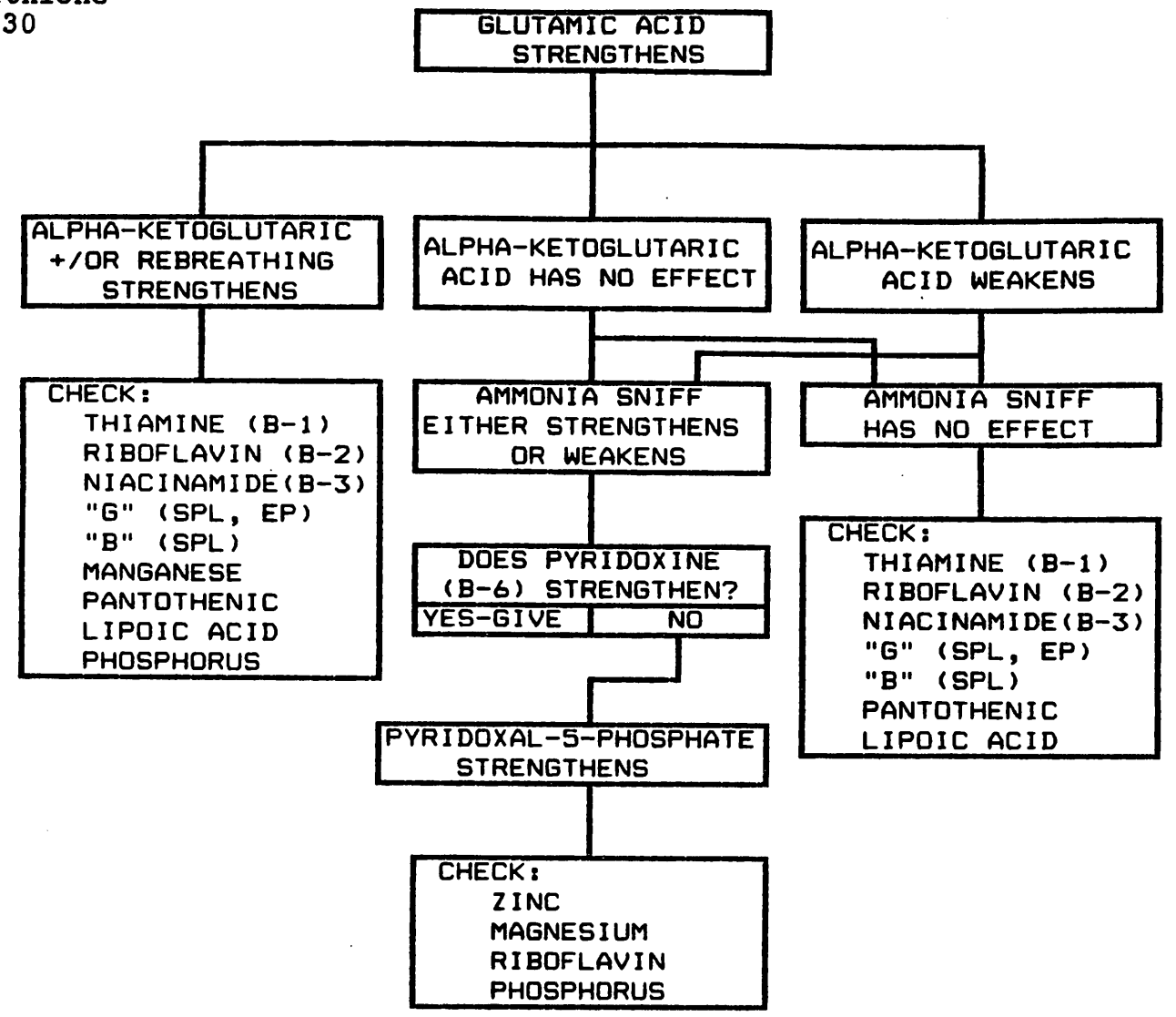
By making simple nutritional corrections, we can allow the body to regulate its cellular chemistry and return to homeostasis. The far-reaching implications of these procedures include not only more rapid recovery from problems which we regularly encounter. But far more importantly, with these muscle testing tools and with recent developments in laboratory tests to augment them, we can begin to understand the <u>processes</u> which go

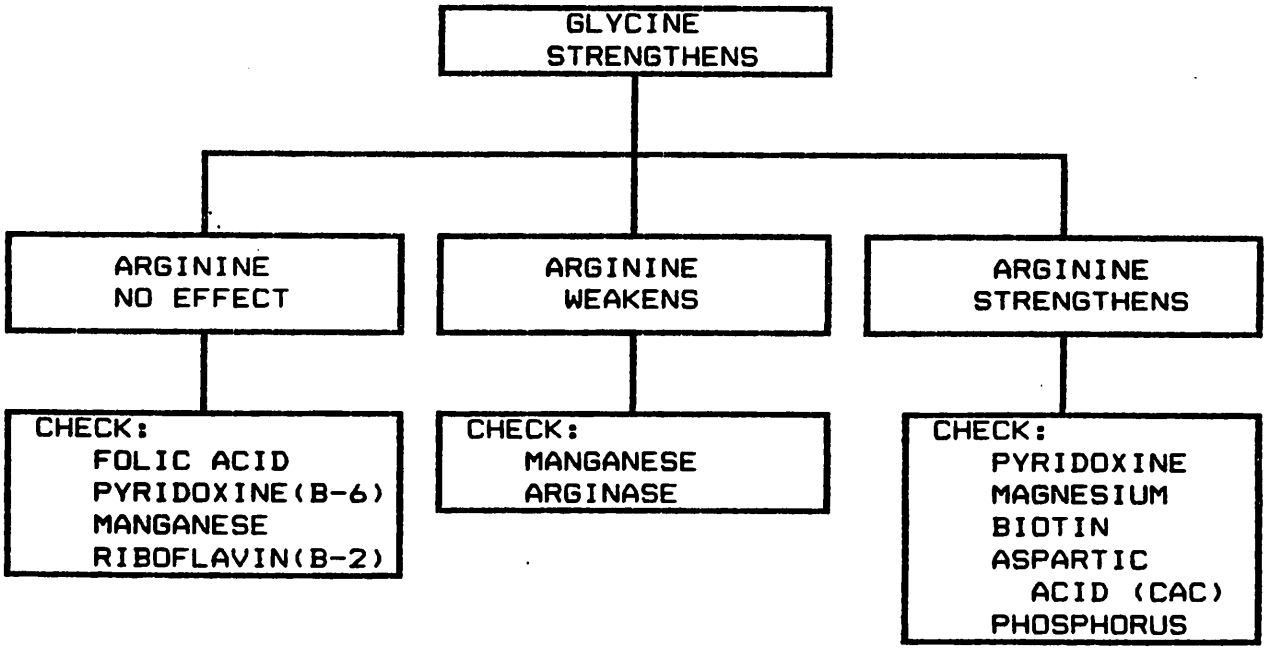
awry at the cellular level. We now have quick, simple tools for piercing the mysteries of these processes which result in diseases, both named and unnamed. We can now evaluate and correct many patients in light of their physiology rather than classify and categorize them according to their symptoms. We are pioneers at the edge of the physiological frontier.

FLOW CHART FOR GLUTATHIONE









Courtesy to Evan Mladennoff, D.C. for original flow charts

REFERENCES

1. Isaacs, James P., and John C. Lamb. A precis on cellular electron poising, ergodization, and molecular quantization. Conference on trace substances in environmental health, University of Missouri 1970; volume 8: 313-321.

2. Schmitt, Walter H., Jr. The clorox test: a screening test for free radical pathology, parts I and II. Digest of chiropractic economics 30:2, September-October, 1987, and 30:3, November-December, 1987.

3. Schmitt, Walter H., Jr. Making B-6 work. I.C.A.K. collected papers, Winter, 1987.

4. Schmitt, Walter H., Jr. Identifying the need for B vitamins, manganese, and coenzyme Q: an answer to recurrent hypoglycemia and hypoadrenia. I.C.A.K. collected papers, Winter, 1987.

5. Schmitt, Walter H., Jr. The ammonia sniff test. I.C.A.K. collected papers, 1988-89, volume II.

NOTE: "Amino Acid Flow Chart" by Jon Pangborn, Ph.D. is available from Bionostics, Inc., P.O. Drawer 400, Lisle, Illinois 60532.

GLUTAMIC ACID AND ARGININE AS SCREENING TOOLS FOR FUNCTIONAL ENDOCRINE PROBLEMS

Walter H. Schmitt, Jr., D.C.

ABSTRACT: A method of identifying hidden functional endocrine problems is discussed. When insalivation of glutamic acid causes general muscle weakness, the weakness pattern is negated by treating adrenal (or gonadal) neurolymphatic reflexes (NLs). When insalivation of arginine produces general muscle weakness, this weakness is negated by treating thyroid NLs. The relationship of electrolyte cations (calcium, potassium, sodium, and magnesium) to adrenal and thyroid patterns is discussed. These concepts are related to the holographic patterns of body chemistry (electron poising system) being reflected through body structure which can be monitored by muscle testing procedures.

INTRODUCTION

Arginine and glutamic acid both have reputed neurotransmitter activity. These amino acids are readily synthesized in the body using vitamin and mineral cofactors. We have used these substances as testing substances for various metabolic pathways (eg. glutamic acid for the citric acid cycle; arginine for the urea cycle) for a number of years. [1,2,3] We have stumbled across a new finding which involves the use of these two amino acids in the identification of seemingly hidden endocrine problems, particularly functional hypothyroidism and functional hypoadrenia.

Some patients show a universal weakness upon insalivating

glutamic acid while other patients show a similar weakness on insalivation of arginine. Patients who show universal weakening on insalivation of glutamic acid have been found to show a need for increasing adrenal (or occasionally gonadal) activity. Patients who weaken on arginine are found to respond to stimulating thyroid activity.

We find many patients who have evidence of functional thyroid and/or adrenal problems such as lowered temperature, positive Ragland effect (postural hypertension), or other clinical indications [4], yet who also display no evidence of associated muscle weakness. That is, in thyroid problems, we would expect to find weakness of the teres minor and in adrenal problems, weakness of the sartorius, gracilis, and/or posterior tibialis. When we cannot find the expected weakness, we now employ the use of glutamic acid or arginine to uncover hidden problems.

PROCEDURE

The procedure is quite simple. Following correction of higher order functional neurological problems such as cranial faults, tonic labyrinthine reflexes, and major switching factors, we test a strong indicator muscle against the insalivation of glutamic acid, and later, arginine. In hidden adrenal (or gonadal) problems, insalivation of glutamic acid will induce generalized weakness which will only be neutralized by therapy localization (TL) to an adrenal neurolymphatic (NL) or occasionally an ovarian/testicular NL.

In hidden thyroid problems, placing arginine in the mouth will create dramatic weakness which will only be negated by TL to a thyroid NL. Treatment of the indicated NL reflexes with the appropriate amino acid in the mouth negates the generalized weakening effect. This procedure has helped to identify difficult hidden problems in some patients which were otherwise concealed from our view.

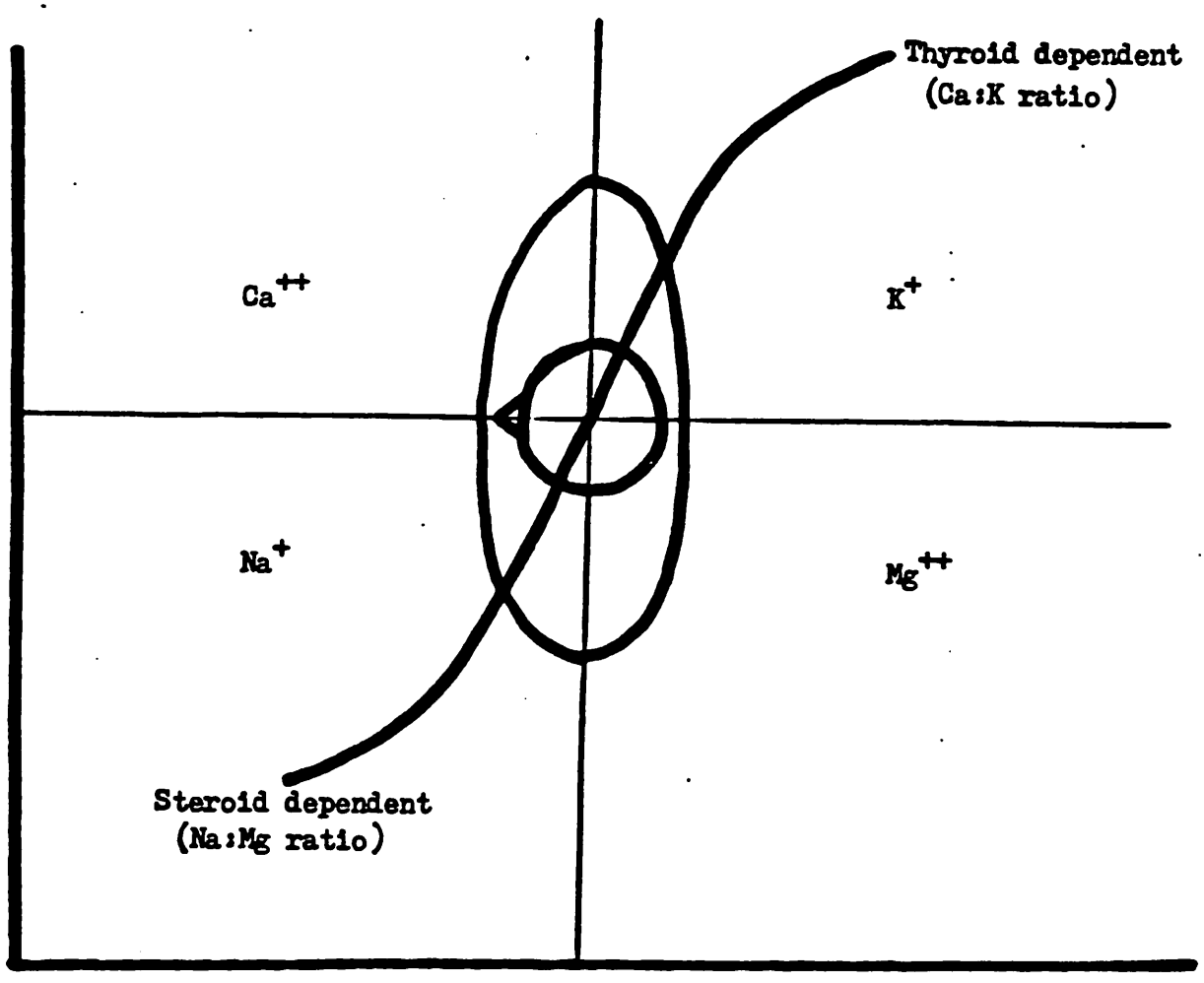
RELATIONSHIP TO ELECTROLYTE CATIONS

Patients who weaken on glutamic acid very often also weaken on insalivation of both calcium and potassium together. Patients who weaken on arginine will usually also weaken on insalivation of sodium (such as table salt) and magnesium together. The implications of these electrolyte cation pairs weakening were discussed in a previous paper by this author. [5] In that paper it was discussed that each of the four body quadrants was associated with a different cation: calcium, the right anterior flexors; potassium, the right posterior extensor muscles; sodium, the left anterior flexor muscles; and magnesium, the left posterior extensor muscles. These ideas are based on the concepts of the holographic representation of man's structure on the electron poising curve. [5] (See Fig. 1)

We attempted to place these minerals in the mouth to identify if we could manipulate the patient's body position in relation to these holographic chemical concepts. Putting calcium and potassium in the mouth together tends to pull the body up and to the right on the curve, towards the thyroid end of the curve

and away from the adrenal portion of the curve. The upper, right half of the electron poising curve is the thyroid dependent portion of the curve. Often, calcium and potassium in the mouth together weakens the patient. This is neutralized by TL and treatment of the adrenal gland (or gonadal) NL(s). Improving steroid function by treating the adrenals (or gonads) pulls the patient back down and to the left on the electron poising curve, back down toward the reduced state.

FIGURE 1: BODY QUADRANTS AND RELATED CATIONS



Likewise when sodium and magnesium weaken a patient, the

patient tends to be pulled down and to the left on the curve toward an over-reduced state. This is negated and corrected by treating the thyroid NL which pulls them back up towards the top, right half of the curve, that is, towards the oxidized state.

We tested many patients with these two combinations of cations until it was decided that there must be a neurological equivalent. By trial and error observations we tested patients with every potential neurotransmitter and neurotransmitter precursor that we were aware of. It was found that calcium/potassium causing weakness was most often paralleled by glutamic acid weakening.

Therefore, the principles of calcium/potassium weakness relating to adrenal (or gonadal) was equated to glutamic acid weakening. Screening with glutamic acid is usually simpler than placing the two separate mineral pills in the mouth and this procedure became preferred.

Likewise it was found that the sodium/magnesium weakening effect was also most often paralleled by arginine weakening. Instead of testing with sodium and magnesium, we found arginine usually creates an equivalent effect. Although the cation - amino acid patterns do not correspond one hundred percent of the time, they are highly correlated.

There are reasons other than endocrine problems why glutamic acid or arginine may induce weakness. These are discussed in other papers. [2,3] But when other high level neurological patterns have first been corrected, testing with glutamic acid

and arginine does give us methods of readily identifying otherwise hidden endocrine problems. When we have some diagnostic criterion indicating functional endocrine involvement, such as lowered temperature, postural hypotension, and other classical signs of lowered thyroid and/or lowered adrenal function, and yet cannot identify associated muscle weakness, testing with these two amino acids is employed.

The use of glutamic acid and arginine as a screening test to help identify the need for the treatment of endocrine activity when the expected individual muscle weakness is not present has been of value in solving problems of several difficult patients. This is not presented as an all-inclusive technique, but simply as an adjunct in screening patients for functional endocrine problems which may previously have been missed.

SUMMARY OF PROCEDURES

1. Correct high order functional neurological problems (i.e., cranial faults, tonic labyrinthine reflexes, major switching).
2. Patient insalivates glutamic acid.
3. If weakens, weakness negated by adrenal or gonadal NL.
4. Treat indicated NL with glutamic acid in mouth.
5. Patient insalivates arginine.
6. If weakens, weakness negated by thyroid NL.
7. Treat thyroid NL with arginine in mouth.

REFERENCES

1. Schmitt, Walter H., Jr. Identifying the need for B vitamins, manganese, and coenzyme Q: an answer to recurrent hypoglycemia and hypoadrenia. I.C.A.K. collected papers, Winter, 1987.

2. Schmitt, Walter H., Jr. The ammonia sniff test. I.C.A.K. collected papers, 1988-89, volume II.

3. Schmitt, Walter H., Jr. Glutathione-the most important molecule in the cell. I.C.A.K. collected papers, 1988-89, volume II.

4. Schmitt, Walter H., Jr. Common glandular dysfunctions in the general practice. Chapel Hill, NC: privately published, 1981.

5. Schmitt, Walter H., Jr. The body quadrants, polarities, electrolyte levels, electron poising, and body torque. I.C.A.K. collected papers. Winter, 1983.

A PILOT STUDY SHOWING EFFICACY FOR APPLIED KINESIOLOGY MUSCLE TESTING PROCEDURES AS A SCREENING TOOL FOR IMMUNE SYSTEM MEDIATED FOOD ALLERGY PATTERNS

Walter H. Schmitt, Jr., D.C.

Abstract: Seventeen patients were found positive on applied kinesiology (A.K.) muscle testing screening procedures indicating food hypersensitivity (allergy) reactions. Each patient showed muscle weakening (inhibition) reactions to oral provocative testing of one or two foods for a total of 21 positive food reactions. IgE (RAST), IgG (RAST), IgE immune complex, and IgG immune complex assays were performed for all 21 of the A.K. positive testing foods. 19 of the 21 foods (90.5%) positive for hypersensitivity response on muscle testing showed one or more positive blood tests.

INTRODUCTION

The four classic hypersensitivity reactions which describe allergic reactions to foods, airbornes, and other antigens are called the Gell-Coombs Types I, II, III, and IV reactions. [1] (See Figure 1.) In hypersensitivity reactions of the Gell-Coombs types I and II, higher than normal amounts of IgE (type I) or IgG (type II) are produced by plasma cells when they encounter antigens. (IgM or IgA can also be produced in a type II reaction.) Immune complexes which are produced and are not adequately broken down (by the liver and/or the spleen) will be elevated in type III reactions. A typical type IV reaction is the tuberculin skin test. Type IV reactions will not be

discussed further in this paper.

FIGURE 1

4 TYPES OF HYPERSENSITIVITY RESPONSE (GELL - COOMBS)

ANTIBODY MEDIATED IMMUNITY

TYPE I - IgE - ANAPHYLACTIC TYPE

TYPE II - IgG (and IgM) - CYTOTOXIC RESPONSE

TYPE III - IMMUNE COMPLEXES

CELL MEDIATED IMMUNITY (T CELLS AND MACROPHAGES)

TYPE IV - DELAYED HYPERSENSITIVITY

IgG (or IgE) combines with antigen to form an antigen-antibody complex. One antigen can bind at least two IgG molecules together. This sets up the potential for chains of these antigen-antibody molecules which are called immune complexes. IgG is converted to IgG immune complex which should be cleared by the liver and/or the spleen. When these immune complexes build up, this is the basis for Type III reactions.

The rate of formation and clearing of IgG immune complexes affects both the levels of IgG and its immune complex. In other words, IgG may be rapidly produced, but just as rapidly converted to immune complex form. This can result in low IgG while IgG immune complexes may be severely elevated. Likewise, IgG may be elevated while IgG immune complexes may be adequately cleared. This explains the necessity of measuring as many parameters as possible before ruling out immune hypersensitivity reactions.

The type I reactions result in rapid (anaphylactoid) type

reactions. Histamine release by mast cells is the major symptom producing factor. The half-life of IgE is 2 1/2 days. The type II IgG type reaction is complement mediated and results in a slower onset of symptoms (a type of delayed hypersensitivity). IgG has a half-life of 21 days, hence its effects are much longer lasting than an IgE reaction.

Immune complex formation is potentially the most tissue destructive as these complexes settle in tissues and cause microthrombi formation, complement cascade which can result in tissue damage, and leukocyte chemotaxis with the subsequent release of inflammatory mediators. Immune complexes have been implicated in autoimmune disease processes.

Applied kinesiology procedures involve muscle testing as a functional evaluation of patterns of inhibition and facilitation in the nervous system. Many clinical factors have been found to effect neuromuscular function and result in patterns of inhibition which induces reversible weakness of muscles to standard testing procedures.

One factor which is said to affect changes in muscle strength is the oral insalivation of allergic foods. A.K. procedures involve a particular type of provocative testing for food hypersensitivity which is based on the patient insalivating a food substance and the doctor performing muscle testing to various muscles. A weakening reaction of the muscle induced by the patient's insalivation of the food is suggested to be indicative of a neuromuscular hypersensitivity (allergic)

reaction to that food.

Although this type of provocative testing procedure to identify food allergies or hypersensitivities is widely employed by A.K. doctors, only one study has been performed to test this hypothesis. [2] This project was designed as a pilot study to identify if, in fact, the reported weakness on provocative oral neuromuscular hypersensitivity to foods is due to food allergy or hypersensitivity as identified by measurements of standard immune system blood assays.

MATERIALS AND METHODS

Patients were tested using food allergy screening tests developed by Dr. Michael Lebowitz [3] and this author. [4] These included 1) a weak muscle strengthening on insalivation of the natural anti-histamine, yakriton, 2) a strengthening response on insalivation of copper, 3) a positive therapy localization (T.L. - causing a weak muscle to strengthen) to the thymus area over the angle of Louis on the sternum, 4) a positive T.L. to thymus with copper in the mouth, and/or 5) a strong muscle weakening during simultaneous T.L. to the thymus while a copper antagonist supplement (Cop Out) is in the mouth. These screening tests are listed in Table 1 under "pre-test findings".

Patients who were positive on one or more of these tests were further tested for muscle testing reactions to common food allergens. These included whole wheat flour, cornmeal, soy flour, brewer's yeast, baker's yeast, cow's milk powder, powdered egg, potato flour, and others.

While the food was held in the patient's mouth, various strong muscles were tested to observe for changes in strength. A weakening of strong muscles to oral challenge with a food is an A.K. finding suggestive of food sensitivity to that food.

With the weakening food in the patients mouth, several additional factors were tested to identify possible negation of the weakening response. These factors were 1) placing yakriton in the mouth (with the food), 2) T.L. to the thymus area, 3) T.L. to the liver neurolymphatic reflex (NL), and 4) placing the spine in a right foot gait torque pattern (called a CCW torque) by placing orthopedic wedges under the right hip and the left shoulder. If a patient was negative to the CCW torque of the spine, other spinal positions were checked for negating the food induced muscle weakness. One patient (#14) was found to have the weakness negated by a left convex lateral flexion of the spine. These findings are listed in Table 1 under "with food in mouth".

When a weakening response to oral food challenge is observed, blood was drawn prior to further treatment. The patient's serum was sent to Immuno Nutritional Clinical Laboratories in Van Nuys, California [5] where it was analyzed for levels of IgE (RAST test), IgG (RAST test), IgE immune complexes, and IgG immune complexes for the suspected food(s). For several foods, only IgE and IgG are available. Patients were included in the study only when all four tests were available for the food(s) to which they showed sensitivity by neuromuscular hypersensitivity testing.

The laboratory reports results as either as reactive in one of three categories: equivocal, moderate, or severe, or non-reactive. These results are included in Table 1 under "laboratory results".

RESULTS

17 patients with positive muscle testing findings had their blood tested for all four immune parameters. 15 patients showed positive blood tests which paralleled their muscle testing findings. Four patients had two positive foods by muscle testing findings which were compared with blood testing. Therefore, there were a total of 21 foods which were muscle tested and blood tested. 19 of the foods which were positive to neuromuscular hypersensitivity provocative testing also showed positive blood tests. The results are shown in Table 1.

Of the 21 foods tested, the following positive reactions were found: IgE - 5, IgG - 14, IgE immune complexes - 0, IgG immune complexes - 10. The total number of positive blood reactions is 29 because a number of patients had multiple positive reactions. The severity of the reactions was as follows: equivocal - 8, moderate - 9, severe - 12. These findings are summarized in Table 2.

The pre-test findings and findings with the food in the mouth were included in the study to attempt to identify any diagnostic trends. None were observed, but the data is included in Table 1 also. Due to the nature of this study being compiled based on regular patients in our office during regular office

TABLE 1

PRE-TESTING FINDINGS							WITH FOOD IN MOUTH				LABORATORY RESULTS			
Pt	Yak.	Cu	Thy-mus	Cu+ Thym	Cop Out	Food	Yak	Thym	Liv	CTS	IgE	IgE	E-IC	G-IC
1.	S	O	O	O	O	SOYBEAN	+	O	+	CCW	E	E	O	O
2.	O	W	O	S	O	MILK	O	+	O	CCW	S	S	O	O
3.	S	S	O	S	W	BR. YEAST	+	O	O	CCW	O	S	O	E
4A.	O	O	O	S	W	WHEAT	+	O	+	CCW	O	M	O	E
4B.	"	"	"	"	"	MILK	+	O	+	CCW	S	M	O	O
5.	O	S	O	-	W	MILK	O	+	+	O	O	M	O	M
6.	O	O	S	-	W	BR. YEAST	O	+	+	CCW	O	S	O	M
7.	O	S	O	-	O	BR. YEAST	O	+	+	CCW	O	S	O	O
8.	S	S	O	-	W	BR. YEAST	+	O	O	CCW	O	S	O	O
9A.	O	O	O	S	W	CORN	O	+	O	CCW	O	M	O	O
9B.	"	"	"	"	"	MILK	O	+	+	CCW	M	S	O	E
10.	O	O	O	S	W	BR. YEAST	O	+	+	CCW	O	S	O	E
11.	O	NA	NA	NA	NA	CORN	O	+	+	NA	O	O	O	S
12.	O	O	O	S	NA	*CORN	NA	+	NA	NA	O	O	O	O
13.	O	O	O	S	W	CORN	NA	+	NA	CCW	O	O	O	O
14A.	O	S	S	-	W	MILK	NA	+	NA	)	E	O	O	O
14B.	"	"	"	"	"	CORN	NA	+	O	CCW	O	E	O	O
15.	O	O	O	S	W	WHEAT	NA	+	NA	CCW	O	M	O	O
16.	NA	S	NA	NA	NA	BR. YEAST	NA	+	NA	CCW	O	O	O	M
17A.	S	O	O	S	NA	CORN	NA	O	NA	NA	O**	O**	O	S
17B.	"	"	"	"	"	WHEAT	NA	+	NA	NA	O**	O**	O	S
TOT	16	16	15	15	13		13	21	14	17	21	21	21	21
	S=4	S=6 W=1	S=2	S=9	W=10		+=5	+=15	+=9	CCW=15)=1	5	14	0	10

* Corn oil only weakened; cornmeal tested OK

** General screening test for 6 grains including corn and wheat

PRE-TESTING FINDINGS

Yak. = Antronex in mouth strengthens a weak muscle

Cu = Copper in mouth strengthens a weak muscle

Thymus = T.L. to thymus (angle of Louis) strengthens a weak muscle

Cu+Thymus = Copper in mouth plus T.L. to thymus

Cop Out = Cop Out in mouth plus thymus T.L.

S = Strengthens

W = Weakens

O = No effect on muscle strength

NA = Not tested

- = Not applicable since copper strengthened weak muscle

WEAKENING FOOD IN MOUTH FINDINGS

Yak = Antronex in mouth

Thym = Thymus T.L.

Liv = Liver NL T.L.

CTS = Centering the spine-spinal position which negates food induced weakness

+ = Negated weakening effect of food in mouth

CCW = Counterclockwise pelvic torque negated weakening effect of food in mouth

) = Lateral flexion convex to left negated weakening effect of food in mouth

LABORATORY FINDINGS

IgE = IgE RAST test

IgG = IgG RAST test

E-IC = IgE Food Immune Complex Assay

G-IC = IgG Food Immune Complex Assay

O = Non-reactive

E = Equivocal positive reaction

M = Moderate positive reaction

S = Severe positive reaction

hours, not all pre-test findings and findings with the food in the mouth were recorded for all patients.

TABLE 2

SEVERITY OF REACTION	TYPE OF REACTION IgE	IgG	E-IC	G-IC	TOTALS
EQUIVOCAL	2	2	0	4	8
MODERATE	1	5	0	3	9
SEVERE	2	7	0	3	12
TOTALS	5	14	0	10	29

DISCUSSION

The results suggest that applied kinesiology muscle testing procedures are an excellent screening test for positive IgE (Type I), IgG (Type II), and IgG immune complex (Type III) mediated hypersensitivity reactions. The percentage of positive laboratory findings was 90.5% (19 of 21) of the foods which showed positive to provocative muscle testing procedures. A 95% confidence interval of (.777, 1.00) was calculated for the data. The formula employed was :

$$\frac{y}{n} \pm 2\sqrt{\frac{y}{n}\left(\frac{n-y}{n}\right)\frac{1}{n}}$$

where y = number of laboratory positives (19)

n = number of foods tested (21).

Further research is definitely indicated. Three specific directions are recommended. First, a self-controlled pilot study

using muscle testing to identify both positive and negative hypersensitivity testing foods needs to be performed. This can tell us whether muscle testing is predicting only positives or if it can be used to identify non-reactive foods as well. Secondly, a multi-center study needs to follow up on this study and the one just proposed. Thirdly, follow up studies on patients who have already been tested as positive with both muscle testing and blood testing should be performed after applied kinesiology desensitization techniques have been administered.

The pilot study for comparing both positive and negative muscle testing findings with positive and negative blood test reactions is being formulated in our office as of the writing of this paper. We are also planning follow-ups on as many patients in this study as possible following desensitization techniques.

REFERENCES

1. Roitt, Ivan M., Brostoff, Jonathon, Male, David K. Immunology. St. Louis: C.V. Mosby, 1985.

2. Scopp, Al, An experimental evaluation of kinesiology in allergy and deficiency disease diagnosis. Journal of orthomolecular psychiatry 7:2, 1978. pp. 137-8.

3. Lebowitz, Michael, A technique to abolish all food sensitivities. I.C.A.K. collected papers, 1988-89, volume II.

4. Schmitt, Walter H., Jr. Applied kinesiological observations of allergic patients - Parts I and II. Digest of chiropractic economics 27:1, July-August, 1984 and 27:2, September-October, 1984.

5. Immuno Nutritional Clinical Laboratory, 6700 Valijean Avenue, Van Nuys, California 91406. (818) 780-4720.

A STUDY ON FREQUENCY OF FUNCTION HYPOADRENIA

DR. KURT A. VREELAND, D.C.,D.I.C.A.K.

ABSTRACT

This study was performed on 30 new patients entering my clinic for treatment of various conditions.

The significance of this very brief study is that out of a possible 120 objective signs for functional hypoadrenia 92 were positive. "Adrenial problems" is a much abused term, in my opinion. It would be beneficial if A.K. practicioners would review adrenal function, and I feel that it is essential that certified teachers become fluent in adrenal physiology.

I will not attempt to reiterate the excellent writings of David Walter, D.C. in several of his texts. What I will cover is lesser talked about adrenal physiology. This should give the reader a better understanding of why the adrenal gland is so important, and why it is necessary to support muscle testing with objectifiable tests, and why having the patient ensalivate potassium, uncovers hidden adrenal weakness. Wally Schmitts protocol for evaluation and treatment of adrenal function is excellent. Dr. Goodhearts work on amino acids and B&E points on the face has also been invaluable in changing biochemistry at a much higher level. Standard muscle testing (Gamma I Type) procedures can leave many unanswered questions.

THE ADRENAL CORTEX

In 1916 Marshall & Davis suggested that kidney function was influenced by the adrenal. Soon after that it was shown that in adrenalectomized cats there was an elevated serum potassium and a lowered serum sodium level. Several researchers in the years that followed demonstrated that a high sodium, low potassium diet would benefit Addisons patients, and kept adrenalectomized

dogs alive for indefinate periods.

The early 20th century also gave rise to research demonstrating the adrenal cortical relationship to glucose metabolism, pituatary functions, and a relationship to the gonads.

The histology of the adrenal cortex becomes important in that it relates functional to the glandular secretions. The adrenal cortex is compossed of three distinct zones. The outer most zone is the zone Glomerulosa. This zone is responsible for the secretionof the mineralocorticoid aldosterone. The middle zone is the Zona Fasciculata. This zone is responsible for the secretion of glucocorticoids e.g. (cortisol). The inner most zone is responsible for the secretion of glucocorticoids and androgens, e.g. cortisol and testosterone. See Diagram I.

Several studies have shown that alteration of sodium/potassium ratio in animals result in changes in the zona glomerulosa with little or no change in the zona reticularis or zona fasciculata.

Hypophysectomy in animals produce atrophy in the inner zones and no change in the zona glonerulosa.

Renin and angiotension produced changes in the zona glonerulosa and no change in the inner two zones.

The conclusion has been made that the inner two zones are under A.C.T.H. control. While the outer zone is controlled by NA+/K+ ratios and the renin/ angiotensin levels.

This may seem like acedenic trivia, but it is significant when a patient has a problem with glucose metabolism or lack of lilido. As opposed to the patient who has changes in electrolyte balance. The first patient may need a pituatay pump, the later may only need a high sodium low potassium diet.

The renin angiotensin mechinism. Renin is secreted into the blood from

the kidney. Renin is than converted to angiotensin I. Angiotensin I is converted to angiotensin II in the lung. Angiotensin II has two effects which can elevate arterial presure. (1) elevates arteriole presure by vasoconstriction. (2) causes increase in aldoesterone from the adrenal cortex, and inturn causes salt and water retension. See diagram II. Of all the stimuli that causes renin release from the kidney the sodium/potassium ratio have the greatest influence. A 5 percent decrease in NA+ concentration doubles aldoesterone secretion. A 10 percent elevation in K+ also doubles the rate of aldosterone secretion. One must remember that a 10 percent change in K+ concentration is only 0.4 meg./liter that translates to a very slight change in K+ concentration.

It is not hard to see how on having the patient ensalavate K+ would cause trenendous stress on the adrenal if that adrenal were not able to respond with aldosterone production. One must also consider to test a source of sodium against a weak adrenal indicator muscle.

The glucocorticoid (cortisol) is involved directly or indirectly with many biochemical reactions in the body. The following is a list of what the glucocortcoids regulate or modulate with other hormones.

1. Carbohydrate Metabolism
2. Protein Metabolism
3. Lipid Metabolism
4. Bone and Cartilage Metabolism
5. Maintenance of Cardiovascular Systems
6. Maintenance of Hemopoitic System
7. Maintenance of the Skin, Connective Tissue, and Mesenchymal Tissue
8. Maintenance of C.N.S. Responsiveness
9. Maintenance of the Immune System

10. Maintenance of the Striated Muscle

11. Growth Hormone

12. The Inflamatory Response to Inqury or Disease

13. Maintenance of the Gastrointestinal System

14. The Response to Olfaction, Audition, and Gustation

The glucocorticoids as the name implies increase the production of glucose. Muscle glycogn is also under cortisol control.

Thymus and Lymphoid tissue retrogress under excessive cortisol loads.

Tissue histamine levels are lowered with cortisol production.

Cortisol modulates calcium absorption from the gut by action on the parathyroid hormone and vitamin D.

Cortisol slows ossification and cartilaginous synthesis.

Glucocorticoids help control HCL secretion by the parietal cells. The integrity of the mucosal cells of the gut are also controlled by cortisol.

Glucocorticoids inhibit the transmission rates of signals coming from the ears, tongue, and olfactory sensed. Adrenal corticoid insignificant patients have increased sensitivity to smell, taste, and hearing. Could this account for many allergic patients being ultra sensitive to fumes and smoke. many of my hypersensitive patients also complain of a bad taaste in their mouths. There is a complex hypothalmic-pituatary adrenal cortical neuroandocrine axis. The amount of corticotrophic releasing factors (CFR's) secreted by the hypothatmus that stimulates adrenocorticotrophic hormone from the pituatary fluctuates according to circadian rythum.

Melatonim, 5-hydroxytryptamine (serotonin) and acetylcholine are neurotrasmitters that have been shown to have an effect on CFR. It has to become clear here of the importance of trytophan, tyrosine metabolism, electron poising, choline and B & E technics and the importance of the accupuncture

points on the head and face, that influences hypothalmic function. To try to balance the many aspects of adrenal function on a lower level could become frustrating.

ADRENAL MEDULLA

The adrenal medulla consists of only 10 percent of the whole gland. The catecholamines are the primary secretions of the adrenal medulla. Norepinephrine and Epinephrine are the principle hormones secreted. Tyrosine is converted to norepinephrine. There are enzymes and cofactors necessary for this conversion mainly copper, vitamin C and pyroidoxal phosphate (B6). The sympathetic nervous system also converts tyrosine to catecholamines in exactly the same manor. Again the importance of Dr. Goodhearts work on tyrosine, trytophan balance becomes obvious.

The hypothamus is a major site of nervous system control of adrenal medullary function, ie the "Flight or Fight Response".

The secretions of catacholamines from the medulla can also be elicited by stimulation of the splanchnic nerves. Acetylcholine is released from preganglionic sympathetic nerve sending which depolarize the adrenal medulla cells and lead to catecholamine secretion. Depolarzation causes NA+ and CA+ ions to move into the medullary cells. The secretion of catecholamines has been shown to be in direct proportion to the calcium ion concentrartion. A.T.P. and magnesium substantially reduces the loss of catecholamines.

In addition to the emotional flight or fight response, other factors that increase adrenal medullary response are, hypoglycenia, hypoxia, and hypercapnia. Many other stimuli including nicotine, caffine, histanime, glucyon, and others stimulate medullary secreation.

(6)

In general adrenal medulla function is regulated by hypothacamic pathways. there are certain rate limiting factors such as vitamin C, copper, B6, calcium, magnesium, A.T.P., Tyrosine, Trytophan, & choline.

Epinephrine affects the metabolism of protein, fats, and carbohydrates. It prevents hypoglycemia by causing gluconeogenisis. Epinephrine increase serum cholestrol and low density lipids. Epinephrine also causes increase cardiac work and O_2 consumption. It is a powerful cardiac stimulation. Epinephrine produces vasoconstriction of the skin, mucosa and kidneys, and vasodilation of the skeletal musculature. Epinephrine causes relaxatiion of the gastrointestinal musculature except for the pyloric and ileocecal sphineters. It also relaxes broncjial smooth muscle.

SUMMARY

This study shows the frequency of adrenal deficiences and there physiologic corrolation to adrenal function. It is meant as a reference rather than a technic paper.

The reference is very brief and the reader, if interested in refered to the reading for further details.

There are so many aspects of adrenal function that can be acurately measure and monitered in your office. It becomes quite apparent that to try identifing adrenal involvement is easy. According to this study 100 percent of new patients entering my office had some type of adrenal involvement, according to the criteria used. Of the combined total female patients there was a total of 76 possible positive signs, 62 were positive which translates to 82 percent. The male group had a possible 44 positive signs with 30 being positive that becomes 68 percent. The combined total 30 patients with 120 possible positive signs of adrenal deficiency, 92 being positive

translates to 77 percent total.

In general the adrenal cortex is divided into zones. Part is under hypothalamic control by C.F.R.'s and A.C.T.H.. Part is mainly under control of NA+/K+ ratios.

In general the medulla is under control of the hypothalmus. Secretion can also be elicited by stimulation of the splanchnic nerves which secrete acelylcholine to depolarize the medulla.

I did this study just to satisfy my own couriosity since so many of my patients seem to have some kind of adrenal involvement. With much greater numbers and proper experimental design and research protocol many aveness can be explored.

This study will have a follow up. Corrolating muscle testing and nutrient testing.

REFERENCES

1. George H. Gass, Harold M. Kaplan., Handbook of Endocrinology. CRC Press.

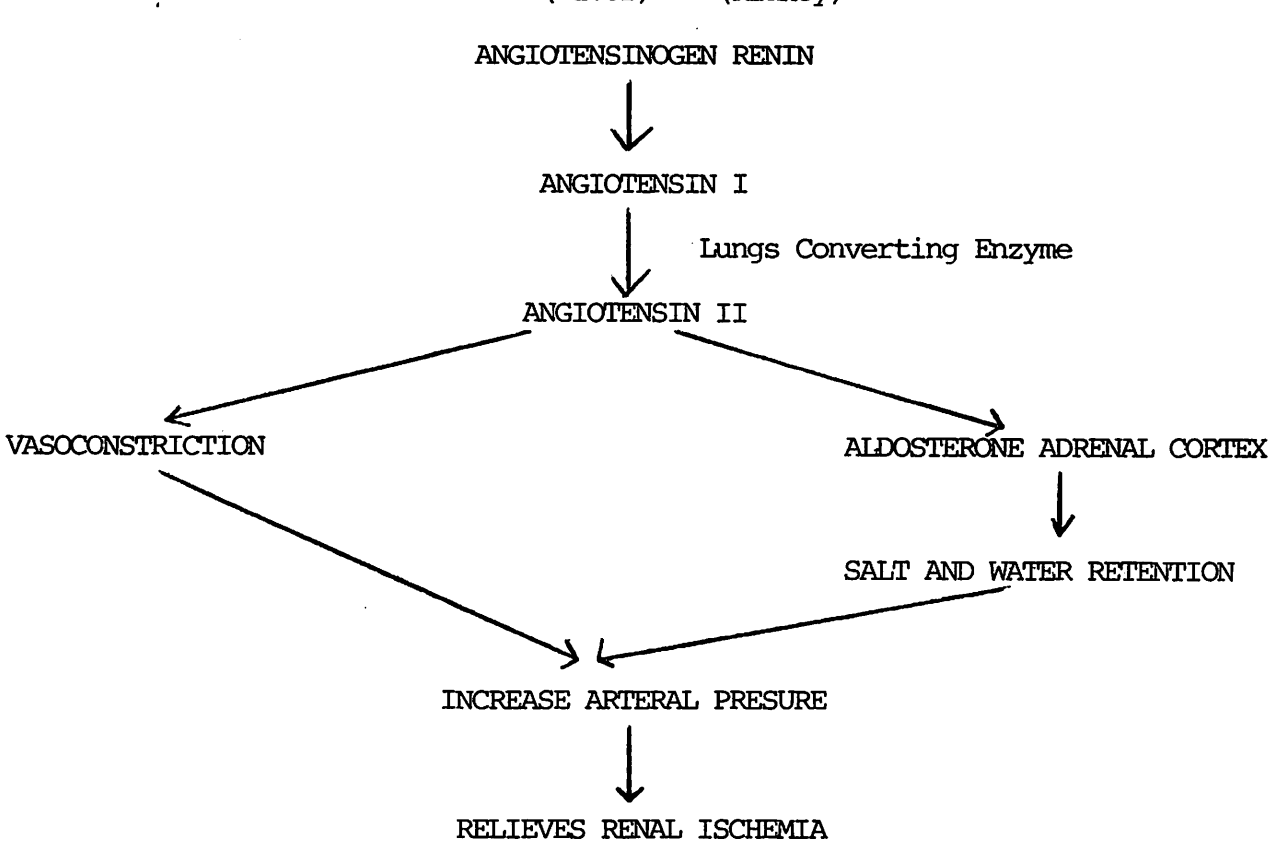
2. R.K. Murray., D.K. Granner., P.A. MAyers., V.W. Rodwell., Harpers Biochemistry Appleton & Lange, pub.

3. Guyton. Textbook of Medicine Physiology. Saunders, pub.

DIAGRAM I

ADRENAL CORTEX	Chemical Secreted
CAPSULE	
ZONA GLOMERULOSA	15% Mineral-corticord e.g. Aldosterone
ZONA FASCIULATOR	50% Glucocorticoid e.g. Cortisol
ZONA RATICULARIS	7% Glucocorticoid Androgens e.g. Cortisol Testosterone
MEDULLA	28% Catecholamine

DIAGRAM II


(Liver) (Kidney)
ANGIOTENSINOGEN RENIN
ANGIOTENSIN I
Lungs Converting Enzyme
ANGIOTENSIN II
VASOCONSTRICTION
ALDOSTERONE ADRENAL CORTEX
SALT AND WATER RETENTION
INCREASE ARTERAL PRESURE
RELIEVES RENAL ISCHEMIA

(1)

Criteria Koenisburg $\geq$ 30 or $\leq$ 10

Postural Blood Presure less than a 4 mm rise in systolic BP from lying to sitting or sitting to standing.

Pulmonary endocardiograph tracing: increase in second heart sound, equal second heart sound.

Pupil paradox: if pupil fails to constrict and stay constricted while shining a light into it.

Female

1. 3 of 4 signs (+) PET Normal
2. 3 of 4 signs (+) P.P. Normal
3. 4 of 4 Signs (+)
4. 4 of 4 signs (+)
5. 3 of 4 signs (+) Koen Normal
6. 4 of 4 signs (+)
7. 3 of 4 Signs (+) Koen Normal
8. 4 of 4 signs (+)
9. 4 of 4 signs (+)
10. 4 of 4 signs (+)
11. 2 of 4 signs (+) P.P.(-), Koen Normal
12. 2 of 4 signs (+) B.P. Normal, Koen Normal
13. 3 of 4 signs (+) Koen Normal
14. 4 of 4 signs (+)
15. 4 of 4 signs (+)
16. 3 of 4 signs (+) Koen Normal
17. 1 of 4 signs (+) Koen Normal, B.P. Normal. PET Normal
18. 3 of 4 signs (+) Koen Normal

19. 4 of 4 signs (+)

Male

1. 2 of 4 signs (+) B.P. Normal, PET Normal
2. 3 of 4 signs (+) Koen Normal
3. 3 of 4 signs (+) Koen Normal
4. 3 of 4 signs (+) P.P. Normal
5. 2 of 4 signs (+) Koen Normal, PET Normal
6. 2 of 4 signs (+) PET Normal, P.P. Normal
7. 2 of 4 signs (+) Koen Normal, PET Normal
8. 4 of 4 signs (+)
9. 3 of 4 signs (+) P.P. Normal
10. 3 of 4 signs (+) B.P. Normal
11. 3 of 4 signs (+) Koen Normal

Female			Male		
76 possible signs			44 possible signs		
62 positive signs = 82%			30 positive signs = 68%		
Koen	8x Normal	10.5%	Koen	5x Normal	11.4%
P.P.	2x Normal	2.5%	P.P.	3x Normal	6.8%
PET	2x Normal	2.5%	PET	4x Normal	9.0%
B.P.	2x Normal	2.5%	B.P.	2x Normal	4.5%

Combined Female and Male Patients

30 patients

120 possible (+) signs of adrenal def

92 positive signs of adrenal def = 77%

Function Hypoadrenia-Vreeland

(3)

Koen 13x Normal 11.0%

P.P. 5x Normal 4.0%

PET 6x Normal 5.0%

B.P. 4x Normal 3.0%

(1) Female age 33

Koen 100+

P.P. (+)

PET Normal

B.P. o 92/70 o 110/90 o 100/80

(1) Male age 14

Koen 41

P.P. (+)

PET Normal

B.P. o 110/70 o 110/80 o 120/80

(2) Female age 32

Koen 15

P.P. (+)

PET 2nd

B.P. o 102/70 o 110/80 o 102/80

(2) Male age 49

Koen 20

P.P. (+)

PET 1st & 2nd

B.P. o 110/60 o 110/70 o 110/20

(3) Female age 51

Koen 40

P.P. (+)

PET 2nd

B.P. o 98/70 o 100/70 o 100/70

(3) Male age 63

Koen 19

P.P. (+)

PRT 2nd

B.P. o 130/90 0 140/90 o 140/90

(4) Female age 23

Koen 40

P.P. (+)

(4) Male age 36

Koen 75

P.P. (-)

PET 2nd

B.P. o 100/70 o 100/70 o 100/80

(5) Female age 40

Koen 20

P.P. (+)

PET 2nd sound

B.P. o 110/80 o 120/90 o 110/80

(6) Female age 39

Koen 58

P.P. (+)

PET 2nd sound

B.P. o 130/90 o 120/90 o 120/90

(7) Female age 30

Koen 25

P.P. (+)

PET 2nd

B.P. o 110/60 o 120/70 o 100/90

(8) Female age 28

Koen 70

P.P. (+)

PET 1st and 2nd

B.P. o 110/70 o 110/70 o 110/80

PET 2nd

B.P. o 130/90 o 100/80 o 120/88

(5) Male age 52

Koen 20

P.P. (+)

PET Normal

B.P. o 230/130 o 250/132 o 220/140

(6) Male age

Koen 110

P.P. (-)

PET Normal

B.P. o 132/90 o 122/100 o 122/100

(7) Male age 40

Koen 15

P.P. (+)

PET Normal

B.P. o 100/70 o 100/70 o 98/70

(8) Male age 23

Koen 35

P.P. (+)

PET 1st and 2nd

B.P. o 140/100 o 140/106 o 140/100

(5)

(9) Female age 45

Koen 30

P.P. (+)

PET 2nd

B.P. o 100/70 o 110/80 o 100/80

(9) Male age 58

Koen 100

P.P. (+)

PET 2nd

B.P. o 120/80 o 130/80 o 140/90

(10) Female age 40

Koen 44

P.P. (+)

PET 2nd

B.P. o 110/78 o 120/80 o 120/80

(10) Male age 31

Koen 15

P.P. (+)

PET 2nd

B.P. o 130/70 o 130/80 o 140/80

(11) Female age 35

Koen 15

P.P. (-)

PET 2nd

B.P. o 100/70 o 110/80 o 110/80

(11) Male age 28

Koen 24

P.P. (+)

PET 2nd

B.P. o 120/80 o 130/90 o 120/90

(12) Female age 24

Koen 13

P.P. (+)

PET 2nd sound

B.P. o 110/80 o 110/80 o 120/80

(13) Female age 42

Koen 28

P.P. (+)

(6)

PET 1st and 2nd

B.P. o 130/80 o 130/80 o 110/80

(14) Female age 31

Koen 70

P.P. (+)

PET 2nd

B.P. o 110/60 o 110/80 o 110/80

(15) Female age 62

Koen 45

P.P. (+)

PET 2nd

B.P. o 100/80 o 110/80 o 110/80

(16) Female age 64

Koen 33

P.P. (+)

PET 2nd

B.P. o 140/90 o 120/80 o 150/100

(17) Female age 61

Koen 20

P.P. (+) slight flicker

PET Normal

B.P. o 110/80 o 110/80 o 120/80

(7)

(18) Female age 35

Koen 17

P.P. (+)

PET 2nd

B.P. o 110/70 o 90/60 o 90/60

(19) Female age 23

Koen 84

P.P. (+)

PET 2nd

B.P. o 100/80 o 112/80 o 100/80

APPLIED KINESIOLOGY NUTRITIONAL TESTING

by David S. Walther, D.C.

Abstract

This paper critically reviews a recent study that purports to evaluate AK nutrition testing. Faults in the study are pointed out and recommendations are given for further studies and methods of evaluation using muscle testing.

A system of observing the effect of nutrition on body function has been developed in applied kinesiology that adds to the available clinical methods of evaluating the body's nutritional needs. It is emphasized that this type of nutritional testing is only an adjunct to the other methods available for determining nutritional needs. The method consists of stimulating the nervous system with the substance being tested and observing for change in function by manual muscle testing. Presently the International College of Applied Kinesiology (ICAK) recognizes only stimulation of the gustatory or, under some conditions, the olfactory nerve receptors as proper methods of stimulating the nervous system for this type of testing.

Evaluation of nutritional needs or allergic and hypersensitive reactions of the body by manual muscle testing, as done in applied kinesiology, must be put into proper perspective. Since Goodheart[6,7] described how chewing nutrition changes the results of a manual muscle test under certain circumstances, there have been numerous modifications by others to his original method of testing; many are not viable from the ICAK's viewpoint.

As noted, the ICAK's method consists of having the nutrition stimulate the gustatory or olfactory nerve receptors. Another method has come into use by some in which the nutrition or other substance being tested is hand-held or laid on the abdominal area, and a muscle is tested for weakening. Those using this method have the substance directly contacting the skin; sometimes they even have the substance contained within a vial. These methods sometimes cause a change in the results of a manual muscle test, but the ICAK does not endorse these methods because of inconsistency.

To study the validity of such methods, the ICAK Executive Board sponsored a double-blind study with John Triano as the principal investigator.[20] It was found that hand-holding nutrition or chewing it failed to significantly change the strength of a specifically associated muscle. As a result of this study the ICAK adopted and published the "ICAK Status Statement,"[8,9] a part of which states: "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." In addition to this caveat being cited in applied kinesiology literature, it has been widely circulated throughout the chiropractic profession;[8,9] it was updated in 1988.[10]

Recently a study was published that indicates applied

kinesiology is unreliable for assessing nutrient status.[13] Although the Executive Board of the ICAK strongly favors research on applied kinesiology methods, exception must be taken to studies that are improperly conducted. The study by Kenney et al.[13] purports to investigate applied kinesiology testing of nutritional supplementation, but it studies nothing that the ICAK supports or teaches.

The study centered on two lay people and one chiropractor who evaluated nutrition by touching "Ridler" points or by somehow evaluating acupuncture meridians; neither method is described in the literature accepted by the ICAK as reference material for the applied kinesiology diplomate examination. The method used in the study to evaluate nutrition via acupuncture meridians is not presented in the paper's description of methods. This writer, who is a charter member of the ICAK and has been practicing applied kinesiology for twenty-four years, is not aware of any method of evaluating the acupuncture meridians with manual muscle testing to evaluate nutritional status. The paper describes the three muscle testers as being "...recognized and experienced in applied kinesiology techniques." The two lay persons are not even eligible to attend ICAK-conducted applied kinesiology educational programs; one must be licensed as a primary health care provider to attend ICAK educational programs. The reason for this is that the ICAK recognizes the need for standard diagnostic procedures to be combined with applied kinesiology methods. It is necessary that one have expertise in standard diagnosis prior to studying applied kinesiology. The chiropractor in the study is apparently not recognized by the ICAK, because the methods used in the study

are outside those recognized by the organization.

A full-page chart shown in the paper is described as "...a typical 'nutritional test chart' used by applied kinesiologists." Again, as a long-term applied kinesiologist and a major author on the subject, I have never seen this chart before. It shows numerous points to touch on the skin to indicate various nutritional deficiencies. This is NOT part of applied kinesiology, nor has it ever been printed in applied kinesiology literature. The statement is made on the chart, "Muscle response testing for possible nutritional deficiencies is an intuitive science." This writer wonders what an "intuitive science" is.

The results of these examiners' muscle testing to evaluate nutritional status in the described manner were almost random. The first deficiency in this study is that there is no evidence of quality muscle testing. The study failed to determine intra- and interexaminer reliability between the three individuals. Members and diplomates of the ICAK have, both independently and in cooperation with colleges, done intra- and interexaminer reliability studies showing that statistical agreement is a product of proper training and discipline. When properly done, there is good interexaminer reliability.[1,11,14,19] Attempting to study the reliability of testing nutrition by manual muscle testing without determining the muscle testers' reliability is akin to evaluating the effects of dietary change on serum cholesterol levels when it is unknown whether the laboratory can reproduce its results in quantitating serum cholesterol. Because of the study's failure to first establish the muscle testers' intra- and interexaminer reliability, there would ordinarily be

no point in discussing this study further because the validity of any results must be questioned. There is value, however, in continuing to evaluate the study to help prevent its other errors in future, better-designed studies.

Numerous studies that have been done in an attempt to quantitate manual muscle tests have had only limited success to this date. It is well-established that results of manual muscle testing cannot be duplicated by dynamometers, such as the Cybex II.[1,2,5] Members of the ICAK continue efforts to develop methods to quantitate the manual muscle test.[14,17]

The blood of the subjects tested in the study by Kenney et al. was analyzed for thiamine, zinc, and vitamins A and C, which were the substances the muscle testers were attempting to evaluate in the subjects. The laboratory results compared to the deficiency finding of the three muscle testers were random, as would be expected by anyone knowledgeable in applied kinesiology methods. If the other aspects of this study had actually followed applied kinesiology methods, this portion of the study would have been valuable. When the results of a muscle test change from chewing a nutritional substance, it is unknown whether it is as a result of actual vitamin or mineral deficiency, or if stimulation to the gustatory receptors causes change in various aspects of nervous system function. There are numerous studies cited in applied kinesiology literature[21,22] showing immediate change in nervous system function upon stimulation of the gustatory receptors. For example, Kare[12] describes studies where dogs were prepared with both gastric and intestinal fistulae so food could be diverted from the body at the stomach or intestine. Previous

experiments had shown that food directly entering the stomach had no influence on pancreatic secretion. When the dogs were fed kaolin (hydrated aluminum silicate) there was no pancreatic response, whereas sugar was effective in increasing both enzyme content and flow. A high-fat diet resulted in a greater lipase flow, but a high-carbohydrate diet had no effect on amylase flow. It was concluded that taste and the substance tasted are involved in modifying the exocrine function of the pancreas. This and other studies show that gustatory receptor stimulation causes almost immediate change in many types of body function.

Kenney et al.[13] cite the study of Rybeck and Swenson[18] as failing "...to find any correlation between mechanical measurements and manual ones by experienced applied kinesiologists." They go on to state, "Those researchers did report that sugar produced a weakening of muscles which was detected by applied kinesiology but not measurable by the mechanical device. However, Nicholas et al.[16] found that tactile stimulation itself causes a temporary weakening of the muscle when it is measured by a mechanical device similar to the one used in this study." This is an improper citation of Rybeck and Swenson's study, since they did not use tactile stimulation such as used in the Kenney et al. study.

The study compares the three examiners' manual muscle test findings with the subjects' strength produced against a Cybex II dynamometer. The results were slightly below those of random guessing. This may be a product of poor manual muscle testing, or the fact that producing power against a dynamometer does not give the same results as manual muscle testing. Manual muscle testing

is NOT a test of "strength." The timing and application of force against the subject's resistance is the key factor that influences the examiner's perception of "strength."[15] The function of concentric or isometric contraction in a Cybex dynamometer test, as opposed to eccentric contraction of a manual muscle test, employs different neurologic factors.

The authors cite Nicholas et al. to make a point about touching the skin, but they fail to mention that in the same study[16] there was a failure to duplicate manual muscle testing with Cybex II isometric testing in a strong population of subjects. The extensive studies of Nicholas et al. show that dynamometer muscle testing, such as the Cybex II, cannot reproduce the results of manual testing. They state, "What is measured manually cannot be measured by the Cybex alone."

It is interesting that the authors quote from and "strongly support" the ICAK's Status Statement, which indicates that the methods these testers used are not acceptable, but they still repeatedly throughout the article call the testers applied kinesiologists, which they are not. Those of us in the ICAK appreciate the authors' strong support of the ICAK's "Status Statement," but we wonder how anyone could assume that the methods used by these three testers fall into the realm of applied kinesiology. Applied kinesiology is a discipline that many practice in an exemplary manner, combining the functional evaluations it yields with their standard diagnostic and therapeutic approaches for improved understanding of the patient's health problem.

The ICAK accepts applications for grants to study applied

kinesiology methods. Interest ranges from studying questionable procedures, such as were done in the Triano study previously cited, to the reproducibility of procedures, quantifying manual muscle testing, and understanding the neuromuscular mechanisms involved. There is an active ICAK Research Advisory Committee that can refer potential investigators to studies that have been done and make them aware of accepted ICAK methods of practicing applied kinesiology. Proper research design takes all factors into consideration. Stimulating the nerve receptors with nutrition is only one factor that changes muscle function. Under certain circumstances, there are many other factors that might be involved in muscle dysfunction as perceived by the manual muscle test. These must be ruled out or controlled in proper research design. It is mandatory that those designing research on applied kinesiology be totally familiar with the subject. It is unfortunate that sometimes research efforts and assets are expended on studies that, because of improper design, produce questionable results and fail to broaden the data base of knowledge on a new and developing discipline to improve health care.

REFERENCES

1. Blaich, R.M., "Manual and machine muscle testing before and after correction of 'respiratory faults,'" in **Selected Papers of the International College of Applied Kinesiology**, ed B.C. Markham, (Detroit, MI: ICAK, 1981).

2. Blaich, R.M., & E.I. Mendenhall, "Manual muscle testing and Cybex machine muscle testing, a search for a correlation," in **1984 Selected Papers of the International College of Applied Kinesiology**, ed W.H. Schmitt, Jr. (Park City, UT: ICAK, 1984).

3. Conable, K.M., & B.T. Hanicke, "Inter-examiner agreement in

applied kinesiology manual muscle testing," in **1987 Selected Papers of the International College of Applied Kinesiology**, ed D.H. Schusterman (Shawnee Mission, KS: ICAK, 1987).

4. Corneal, J.M., & R. Dick, "An attempt to quantify muscle testing using meridian therapy/acupuncture techniques," in **1988 Selected Papers of the International College of Applied Kinesiology-USA**, ed D.H. Schusterman (Shawnee Mission, KS: ICAK, 1988).

5. Cybex, a division of Lumex, Inc., 2100 Smithtown Ave., Ronkonkoma, NY 11779-0903.

6. Goodheart, G.J., Jr., **The Cranial Sacral and Nutritional Reflexes and Their Relationship to Muscle Balancing** (Detroit: privately published, 1968).

7. Goodheart, G.J., Jr., "Structural imbalance and nutritional absorption (a new route to the brain)," **Chiro Econ**, Vol 13, No 3 (Nov/Dec 1970).

8. International College of Applied Kinesiology, "Applied Kinesiology - Adopted Status Statement of the ICAK," **Chiro Econ**, Vol 26, No 3 (Nov/Dec 1983).

9. International College of Applied Kinesiology, "Status Statement-International College of Applied Kinesiology," **Am Chiro** (Jan/Feb 1984).

10. International College of Applied Kinesiology Status Statement, updated and approved by the Executive Board of the ICAK January, 1988. Printed in **The Collected Papers of the Members of the International College of Applied Kinesiology-USA**, Vol I (Shawnee Mission, KS: ICAK, 1988-89).

11. Jacobs, G.E., "Applied kinesiology: An experimental evaluation by double blind methodology," **J Manip Physiol Ther**, Vol 4, No 3 (Sep 1981).

12. Kare, M.R., "Some functions of the sense of taste," **J Agr Food Chem**, Vol 17, No 4 (Jul/Aug 1969).

13. Kenney, J.J., R. Clemens, & K.D. Forsythe, "Applied kinesiology unreliable for assessing nutrient status," **J Am Diet Assoc**, Vol 88, No 6 (Jun 1988).

14. Maitland, K.H., M. Davids, & L. Ruggiero, "Inter-practitioner reliability study. Agreement between examiners on muscle strength and weakness." Proceedings of Summer Meeting, ICAK, Washington, DC, 1987.

15. Nicholas, J.A. et al., "Factors influencing manual muscle tests in physical therapy - the magnitude and duration of force applied," **J Bone Joint Surg**, Vol 60A, No 2 (Mar 1978).

16. Nicholas, J.A., M. Melvin, & A.J. Saraniti, "Neurophysiologic inhibition of strength following tactile stimulation of the skin," **Am J Sports Med**, Vol 8, No 3 (1980).

17. Raffelock, D., "An investigation of applied kinesiology's manual muscle testing by three dimensional computerized force-plate analysis," in **1988 Selected Papers of the International College of Applied Kinesiology-USA**, ed D.H. Schusterman (Shawnee Mission, KS: ICAK, 1988).

18. Rybeck, C.H., & R. Swenson, "The effect of oral administration of refined sugar on muscle strength," **J Manip Physiol Ther**, Vol 3, No 3 (Sep 1980).

19. Sabella, M.A., B. Decker, & T. Krawchuk, "An inter-examiner reliability study of manual muscle testing." Proceedings of Summer Meeting, ICAK, Washington, DC, 1987.

20. Triano, J.J., "Muscle strength testing as a diagnostic screen for supplemental nutrition therapy: A blind study," **J Manip Physiol Ther**, Vol 5, No 4 (Dec 1982).

21. Walther, D.S., "Applied kinesiology nutritional testing," in **1988 Selected Papers of the International College of Applied Kinesiology-USA**, ed D.H. Schusterman (Shawnee Mission, KS: ICAK, 1988).

22. Walther, D.S., **Applied Kinesiology - Synopsis** (Pueblo, CO: Systems DC, 1988).

ADDENDUM

This addendum/summary is being placed after the references so the preceding paper can be copied without these observations; thus, the paper can be given to someone who has questions about the reviewed paper of Kenney et al.

Applied kinesiology has experienced great growth in its use by doctors and in techniques of examination and treatment since Goodheart first wrote about it in 1964. Unfortunately, not all of the examination methods proposed have been found viable. The International College of Applied Kinesiology made its first stand on proper applied kinesiology procedure by adopting the "Status Statement" in 1983 and updating it in 1988. Among other factors, the "Status Statement" indicates that nutrition testing should be done by stimulating the gustatory or olfactory receptors, and therapy localization should be combined with a patient's history and other diagnostic factors to arrive at a final conclusion.

Unfortunately, there are many who use muscle testing to evaluate nutrition and other factors in a manner similar to that studied by Kenney et al. and reviewed in the foregoing material. The regulatory agencies that govern the licensing of health care professionals have the responsibility to ensure that examination and treatment methods applied to the public are viable and appropriate to a patient's needs. There have been occasions in which a regulatory agency, such as a board of chiropractic examiners, has held a disciplinary hearing against doctors using muscle testing to evaluate nutrition. The incident being reviewed is usually having the patient hand-hold nutrition while the

doctor tests the patient's muscle. In some cases the doctor tests a surrogate while the patient hand-holds the nutrition or has it in a bottle lying on his/her belly.

Strong support can be made for applied kinesiology nutrition evaluation when it is combined with a proper case history and clinical examination with supporting laboratory tests (if applicable). The findings from the standard examination and applied kinesiology findings are then analyzed with known nutritional information and physiology to determine the optimal therapeutic approach for the patient's condition. There is considerable literature in the applied kinesiology data base that ties nutrition testing with standard physiology to be applied to the patient's other diagnostic work-up. There are several papers in this issue of the "Collected Papers of Members of the International College of Applied Kinesiology-USA" that add to one's ability to tie nutrition in with the patient's physiologic deficit.

It is imperative that the ICAK continue to support research to study the mechanisms underlying the effects of applied kinesiology testing procedures. Failure of the ICAK and its members to support AK procedures with proper clinical or basic research studies will allow others to judge it as an inexact science. We must continue to distinguish AK from the methods of those who test patients with pills on the belly and make diagnoses by therapy localization with no substantiating data.

THE EFFICACY OF THERAPY LOCALIZATION AS A DIAGNOSTIC TOOL

Jeffrey Weber

ABSTRACT

The purpose of this study was to examine the efficacy of therapy localization as a diagnostic tool. A computerized pneumatic muscle tester was used as an objective evaluator of the middle deltoid muscle during pre-and active therapy localization. The results document an extremely high degree of significance for therapy localization as a diagnostic tool.

In any given number of patients, it is not uncommon to find a sizeable group that experiences a disproportionate distribution of muscle strength from one side of the body to the other. In personal discussions with physiatrists and orthopedists, both chiropractic and medical, there seemed to be a general agreement that the dominant side on muscle testing should be approximately 10% stronger. However, on trying to document this imbalance, I could not but have accepted this premise as common knowledge. Where it is generally accepted that there should be a 10% difference between the dominant and non-dominant sides, theoretically with the dominant side being stronger, our testing has revealed a much greater variance of weakness between sides in many patients--often as high as 25-30% greater than expected, frequently with the non-dominant side being stronger. While the question of "why" this variance occurs is certainly an interesting one, we are more concerned with "how": how these weaknesses can be corrected. The purpose of this study is to determine if therapy localization can be used as a diagnostic tool to isolate neurolymphatic reflexes to be utilized for treatment of specific muscle weakness.

page 2

Therapy localization (T.L.) is a procedure where the patient places his hand over a suspected area of involvement, and then uses muscle testing to determine whether there is any change in muscle strength. When an involved area is touched by the patient, either a strong indicator muscle will weaken, or a weak muscle will strengthen.

For example, if a subluxation of the spine is touched, a muscle which is weak because of that subluxation will test strong on manual muscle testing while a previously strong muscle will weaken. Either of these situations is known as positive therapy localization. After the subluxation has been corrected, touching the area will no longer cause a change of muscle strength as observed by manual muscle testing. It should be noted that therapy localization is strictly a diagnostic tool and is not a means of treatment.

For our study, therapy localization was used in conjunction with a system of reflexes originally developed in the 1930's by an Osteopath named Frank Chapman for the improvement of lymphatic drainage. Chapman correlated these reflexes with specific organs and glands and with different types of health problems, finding that each organ or gland was influenced by a related reflex point.

Apparently Chapman used these reflexes to diagnose a condition and then massaged the reflex which he had found on an empiric basis. His research appears to have been for the purpose of correlating palpatory findings with the patient's condition. Although his original writings are difficult to trace, there are numerous references to the Chapman Reflexes, primarily those texts of the Osteopathic profession. Most written works relate to the empiric use of the reflex in different conditions. Diagnostic techniques in Applied Kinesiology have given clinical support to effects of the reflexes, revealing doubtless reproducibility of their influence on muscle function.

In 1965 Chapman's system of reflexes was taken a step further and used in a study by Goodheart involving manual muscle testing. Whereas Chapman related the reflexes to organs and glands, Goodheart found that specific muscles, registering weak on manual muscle testing, would strengthen dramatically when a certain Chapman reflex was stimulated by massage. This was the beginning of correlating various therapeutic measures , with muscle testing being the diagnostic tool. These reflexes have become known in Applied Kinesiology as "neurolymphatic reflexes".

METHODS

Throughout the years there have been several devices used to test relative strength and weakness of muscles groups. The dynomometer, used in orthopedics to evaluate the gripping capability of the hand, has been used with the examiner placing the device between his testing hand and the patient's extremity. This is often clumsy and cumbersome and the metallic contact can cause pain to the patient. It has not been found very satisfactory as a testing device.

The Cybex II dynamometer by Lumex has been used by many as a method of correlating muscle activity with Applied Kinesiology techniques. The device evaluates muscle contraction as isometric or isokinetic. When the test is isokinetic, the Cybex II is capable of controlling the speed of muscle contraction fro 0-300 degrees per second. In isometric contractions, though, the unit fails to reveal the weakness that is exhibited in manual muscle testing. It will, in fact, sometimes show the muscle to be actually producing greater power than the contralateral muscle which tests normal with manual muscle testing.

As another option, mechanical spring gauges and transducers have been attached to various forms of framework so that the testing device is held stationary and the patient's extremity is attached to the measurement device. The patient pulls against the device to record the amount of power generated by the muscle. This method removes the operator from the exam, eliminating one variable which could cause error. The factor of timing is not taken into consideration by this method, and the examiner must watch very closely for change in patient position and consequent substitution of synergistic muscles for the prime mover being tested. This is known as recruitment.

Also commonly used is a simple device which contains the bladder of a sphygmomanometer. The bladder is moderately inflated, but not enough to register pressure on the pneumatic gauge. The partially inflated bag is then held between the examiner's hand and the patient's tested extremity, similar to the hand dynamometer previously mentioned. As testing is accomplished, the millimeters of mercury pressure are observed by the examiner. The device requires very accurate positioning of the bladder so that it is centered in the testing activity for any reproducibility. It is also bulky and adds additional problems because the measurement must be read during testing.

Finding the aforementioned pieces of equipment plagued with problems and often inaccuracies, we opted for a relatively new piece of equipment, one that we feel reveals computer generated results closest to those that we got directly from our patients. The Comparative Muscle Tester (CMT-1000) has a surface broad enough so that when pressure is exerted by the patient, alterations and vector changes are eliminated at the source of airpressure change. The timing starts the reset switch to initiate the timing of the test and it can be run in 5, 10, 20, 30, 40, or 60 second duration in order to obtain reliable monitoring.

The patient can be shown simple testing positions to be done seated or standing and an operator is not required. The CMT-1000 has been cited in studies performed at the New York College of Podiatric Medicine and Columbia University Teacher's College Department of Movement Science and Education, as having been found extremely useful in objective and quantitative evaluation of muscle testing. A 1-2% margin of error in repeated testing on patients is cited in The Applied Kinesiology Research Manual of 1986 by Dr. Goodheart.

STUDY

After examining numerous patients with a variety of muscle dysfunctions, 19 subjects (12 male, 7 female) aged 19-77, with a common MIDDLE DELTOID weakness, were chosen for the study. Patients were tested on the CMT-1000 before and during active therapy localization of neurolymphatic points for the middle deltoid as cited in Walther's Applied Kinesiology text. A reading was taken for both dominant and non-dominant sides. Based on the theory stated earlier that the dominant side ideally should be no less than 10% stronger than the non-dominant side, the ideal value for the dominant side based on the non-dominant side was then calculated. This will be denoted I and has the value: $I_D = (1.1)N$ where N=reading on the non-dominant side.

The Deviation from Ideal, X_I, is then calculated: $X_I = \frac{I_D - D}{I_D}$ where D=actual value of dominant hand.

page 6

The same procedure is carried out after therapy localization, and a new Deviation from Ideal, X_2 is now obtained in the same fashion.

Functional recovery, Y, is now defined using the percentage change from Ideal as measured by pre versus active therapy localization. Specifically, define

$$Y = \frac{X_1 - X_2}{X_1}$$

In certain instances, the actual reading on the dominant side, D, is higher than I_D. In such cases, we determine that the non-dominant side is too weak, and the ideal value for the non-dominant side based on the dominant side is then calculated. Based on the "10%" theory: $I_N = (\frac{100}{110}).D$

Here, deviation from Ideal will be calculation as $X_i = \frac{I_N - N}{I_N}$

(where i=1 for deviation before therapy localization

and i=2 for deviation during active therapy localization)

Notation (Table of Variables)

N=reading on non-dominant side
D=reading on dominant side
I_N =ideal value for non-dominant side
I_D =ideal value for dominant side
X_1 =deviation from ideal pre-therapy localization
X_2 =deviation from ideal active therapy localization
Y=functional recovery

page 7

PRE-THERAPY LOCALIZATION
(note that "L" denotes lefthanded)

Patient	N	D	I (110% N)	I (100/110D)	% Dev. from X_1
1	6.19	2.36	6.81		65.3
2	11.68	9.3	12.85		27.6
3	11.76	10.03	12.94		22.5
4	6.27	4.42	6.9		35.9
5	5.61	2.92	6.17		52.7
6	5.02	7.23	N/A	6.57	23.6
7	5.27	5.05	5.8		12.9
8	5.55	8.26	N/A	7.51	26.1
9	14.23	11.28	15.65		27.9
10	8.43	7.24	9.27		21.9
11	4	3.93	4.4		10.7
12	13.68	9.24	15.05		38.6
13 (L)	9.48	11.07	N/A	10.06	5.8
14	3.94	5.34	N/A	4.85	18.8
15	8.4	6.17	9.24		33.2
16	11.76	8.84	12.94		31.7
17 (L)	10.21	9.32	11.23		17
18	9.14	7.17	10.05		28.7
19	9.06	4.4	9.97		55.9

ACTIVE THERAPY LOCALIZATION

Patient	N	D	I (110%N)	I (100/110D)	% Dev. from X_2
1	5.47	3.47	6.31		45
2	6.53	7.72	N/A	7.02	7
3	9.46	11	N/A	10	5.4
4	7.07	5.38	7.78		9.1
5	3.72	2.96	4.09		27.6
6	6.39	7.44	N/A	6.76	5.5
7	6.3	6.03	6.93		13
8	6.76	11.74	N/A		36.6
9	14.54	13.22	15.99	10.67	17.3
10	8.88	7.96	9.77		18.5
11	9.53	9.56	10.48		8.8
12	12.85	9.88	14.14		30.1
13 (L)	10.45	11.05	11.5		3.9
14	5.41	6.23	N/A	5.66	4.4
15	7.64	6.62	8.4		21.2
16	11.13	10.67	12.24		12.1
17 (L)	10.99	10.37	12.09		14.2
18	10.6	9.78	11.66		16.1
19	9.79	7.77	10.77		27.9

PATIENT (SEX/AGE)	WEAKER SIDE (N/D)	X_1 DEV. FROM IDEAL (PRE-T.L.)	X_2 DEV. FROM IDEAL (ACTIVE T.L.)	$Y=(X_1-X_2)/X_1$ FUNCTIONAL RECOVERY
1. F/32	D	65.3%	4.5%	31.1%
2. M/33	D	27.6%	7.0%	74.6%
3. M/29	D	22.5%	5.4%	76.0%
4. M/31	D	35.9%	9.1%	74.7%
5. F/77	D	52.7%	27.6%	47.6%
6. M/59	N	23.6%	5.5%	76.7%
7. F/19	D	12.9%	13.0%	-0.8%
8. M/30	N	26.1%	36.6%	-40.2%
9 M/31	D	27.9%	17.3%	38.0%
10. F/63	D	21.9%	18.5%	15.5%
11. F/22	D	10.7%	8.8%	17.8%
12. M/53	D	38.6%	30.1%	22.0%
13. M/31	N	5.8%	3.9%	32.8%
14. F/36	N	18.8%	4.4%	76.6%
15 M/24	D	33.2%	21.2%	36.1%
16. M/38	D	31.7%	12.1%	61.8%
17. M/24	D	17.0%	14.2%	16.5%
18. M/21	D	28.7%	16.1%	43.9%
19. F/49	D	55.9%	27.9%	50.1%
Sample Mean:		$\overline{X}=29.3$	$\overline{X}=17.0$	$\overline{Y}=39.5\%$
Standard Deviation		$S_{X_1}=15.0$	$S_{X_2}=11.4$	$S_Y=30.1$

RESULTS

Active T.L. values reflect significance on t-testing that the procedure of therapy localization is valuable diagnostically as opposed to purely coincidental. We feel that the findings of this small sample are of such significance that a much larger group should be used in subsequent studies. The CMT-1000 is recommended for its low margin of error and its ability to test muscle groups necessary to graphically display the validity of the the localization/neurolymphatic reflex hypothesis.

The unmistakeable reproducibility of our results leads us to believe that therapy localization, is, at the very least, an excellent diagnostic tool for evaluating neurolymphatic reflexes. We are of the opinion that the results of this study document facts about the use of therapy localization which have never been objectively brought to light prior to this. Due to the outstanding difference between pre- and active T.L. findings we are able to demonstrate something which we have seen in our patients for years: therapy localization is a justifiable and accurate means of detecting active N.L.reflexes and its documentation has been long overdue.

As we advocate the CMT-1000 as a truly exceptional piece of equipment for clinical and legal documentation, we would also like to take a moment to acknowledge the pitfall of manual muscle testing, due to inaccurate forms of testing and inexperienced testers. The most significant assets that one can possess are thorough knowledge of anatomy and the artful use of manual muscle testing, both of which take time to acquire. We would also like to acknowledge Jay Martin for his assistance in compiling the statistics for this study.

REFERENCES

Basmajian, J.V. and Bazant, F.J. "Factors Preventing Downward Dislocation of the Adducted Shoulder Joint: An Electromyographic and morphological Study". Journal of Bone and Joint Surgery, (1959), p. 41-A.

Basso, Michele D. and Gentile, A.M. and McCarthy, Susan and Souzis, Leanne. "Test-Re-test Reliability of a new Isometric Strength Testing Device". Department of Movement Sciences and Education, Teacher's College-Columbia University, Dec. 1985.

Chapman, Frank. Introduction to An Endocrine Interpretation of Chapman's Reflexes by Charles Ow ens (n.p.: privately published, n.d.)

Cole, Donald and Hodse, William and Trainor, Frieda S. "Reliability and Reproducibility of the Comparative Muscle Tester: CMT-1000". New York College of Podiatric Medicine, Feb. 5, 1987.

Cybex II Dynamometer, manufactured by Lumex, Inc. 100 Spence Street, Bay Shore, N.Y. 11706.

Diamond, John. Behavioral Kinesiology. (New York: Harper and Row), 1979.

Goodheart, George Jr. Applied Kinesiology 1986 Research Manual. (Ohio: AK Printing), 1986, pp. 39-40.

Goodheart, George Jr. Applied Kinesiology, 3rd Edition. (Detroit: privately published), 1965.

Leaf, David W. "A Validation Study on the Effects of Music on the Muscle Strength of the Body." Proceedings of Summer Meeting, International College of Applied Kinesiology, Detroit, 1979.

Morgan, Peter. "CMT-1000: A Clinical Evaluation of Muscle Testing". The Chiropractic Professional. (Lincoln: Nebraska Litho), March-April 1987, p. 31.

Small, H.R. A report presented in An Endocrine Interpretation of Chapman's Reflexes by Charles Owens, (n.p.: privately published, n.d.)

Walther, David S. Applied Kinesiology Volume I: Basic Procedures and Muscle Testing. (Colorado: Systems DC), 1981, pp. 9-11, 26, 220-221, 243, 247, 253, 459-460.

RESPONSIBILITY IN PRACTICING APPLIED KINESIOLOGY

by David S. Walther, D.C.

ABSTRACT

Another viewpoint on the practice of applied kinesiology is presented regarding the paper of Richard L. Cook, which appears elsewhere in this edition of the **Collected Papers.**

Richard L. Cook presents some interesting and thought-provoking ideas in his paper, **An Insight Into the Subtleties of Bio-Energetics.** He helps put into perspective the patient's and physician's mental role in health care. I take no exception to the importance of the mental aspect with which we deal in treating our patients on both a physician and patient level. I do, however, believe that another and vitally important aspect must be considered regarding Cook's subtitle, "If It Works for Me - Is It Valid?" There is an overtone in the paper that if the patient's and physician's belief in the system being used is strong enough, the type of therapy makes little difference.

Cook practices in England where there is no chiropractic regulating agency. As I understand it, anyone can practice whatever s/he wishes as long as there is no law against it. Also, in England and some other countries one has little concern for malpractice suits since that adversarial aspect of doctor/patient relationship is seldom encountered.

Teaching diplomates of the ICAK must be aware that doctors using the procedures they teach may some day have to support the efficacy and reproducibility of the method in a court of law or

before a health care regulatory agency. There are many ways this can come about, and I have been an expert witness or consultant in almost all situations. To date, when applied kinesiology methods as supported by the ICAK Executive Board have been challenged, applied kinesiology has been found to be a proper method of practice. In fact, to my knowledge, when applied kinesiology has been challenged, it has been due to practitioners using methods not supported by the ICAK.

Doctors who use applied kinesiology as endorsed by the ICAK rarely, if ever, find it necessary to support their methods before a regulatory agency. Proper methods are easy to support when necessary. Unfortunately, it is doctors using questionable procedures who find themselves called before a board, or having to support the procedure in a legal setting. Doctors who are prepared to effectively support their procedures generally haven't used questionable methods in the first place, and they are seldom called before a board.

One should be aware of how regulatory hearings, malpractice cases, and adversarial court appearances come about. These examples are all anecdotes from actual cases, but there is no point in mentioning names or places. One of the most common problems develops from nutrition testing. Doctors are able to support their procedures when nutrition testing is done by gustatory or olfactory stimulation and is part of a total work-up. A total work-up includes a proper case history, clinical examination, and laboratory tests when indicated. The problem develops when the patient hand-holds the nutrition or it is laid on his belly, either in or out of a vial. The problem is

compounded when a large quantity of nutrition is prescribed. The patient, upset about the cost or the unusual procedure, may describe the tests to a regulatory agency, such as a state board of chiropractic examiners, or to another physician. The method is recognized as unusual and a hearing may result. Although these are not recognized ICAK procedures, the physician in question often states he is practicing applied kinesiology. If members of the regulatory agency are not familiar with AK procedures, there may be attempts to put the entire practice of applied kinesiology on the list of techniques unapproved for that state. This attempt has been made in two states under these circumstances. Fortunately, testimony on behalf of the ICAK about its accepted procedures was successful in avoiding the boards' censure of applied kinesiology.

Arriving at a diagnosis without adequate work-up is another area that has resulted in regulatory agency hearings. This is usually done by the inappropriate use of therapy localization. It has been emphasized over and over in applied kinesiology that therapy localization tells only that something is involved -- NOT what is involved. When a diagnosis often treated by medical means is made, the patient may seek a second opinion, frequently from a doctor not knowledgeable in applied kinesiology procedures. When the second physician examines the patient and asks how the diagnosis was made, s/he often becomes livid about the "weirdo" who made that diagnosis. A regulatory hearing, a malpractice suit, or even a civil fraud suit may develop. There are many examples of how this can happen. An AK practitioner may find a weak pectoralis major (sternal division) muscle and positive

therapy localization over reflexes for the liver and simply state to the patient, "You have a liver condition." A liver condition, to most patients, means a liver disease. That's ominous in the patient's mind, and with no further explanation by the examining doctor, it is only reasonable to obtain a second opinion.

We all care for patients involved with personal injury litigation at one time or another. Those whose practices are not geared to this type of patient and the resulting depositions and court appearances may be ill-prepared to support their records and methods in the adversarial system. During a deposition or court appearance every word in the record is open for scrutiny. It is very difficult to explain and support entries in the record regarding finger modes, a diagnosis based only on therapy localization, or to explain the patient's description of pills on the belly muscle testing.

Methods of evaluating nutritional needs such as described by Schmitt in **Glutathione: The most important molecule in the cell** which appears elsewhere in this edition are based on standard physiology and are reasonable. Note that he states, "These muscle testing procedures can be correlated at appropriate intervals with blood and urine tests for quantitative analysis." His final statements are, "We can now evaluate and correct many patients in light of their physiology rather than classify and categorize according to their symptoms. We are pioneers at the edge of the physiological frontier." Even though these techniques are at the "edge of the physiological frontier" they are supportable because they tie in with standard practice methods. They still must be tested, and if successful they may someday be elevated to theory.

If the hypothesis fails proper testing, then the hypotheses must be revised and the testing process continued. We shouldn't feel badly when a hypothesis fails. Beveridge[1] states ". . . the vast majority of hypotheses prove to be wrong." Additionally "Hypotheses should be used as tools to uncover new facts rather than as ends in themselves."

What about the methods used in applied kinesiology that at this time are unexplainable? Therapy localization is an example. Chiropractic has passed the stage of being able to say that it works and that's what counts. We must understand the physiology and be able to show reproducibility by anyone properly trained in the method. Papers such as those of Sprieser,[2,3,4] and in this edition **A New View of the Pathways of Therapy Localization,** present hypotheses of the mechanisms at work. This is an important first step, but until the hypotheses are tested it is important that the physician use therapy localization in combination with other standard methods of diagnosis. Therapy localization and other AK procedures should enhance the physician's diagnostic ability, not replace it. Techniques that do not fit within the guidelines of ICAK should not be used in the name of applied kinesiology.

Summary

Some factors that have created false impressions of what the ICAK stands for in the practice of applied kinesiology are discussed. The general body of applied kinesiology is sometimes judged by what a few practitioners do, even if it is not in the generally accepted method of practice. If one wants to practice in a parapsychological manner, s/he should call it that and not applied kinesiology.

REFERENCES

1. Beveridge, W.I.B., **The Art of Scientific Investigation** (New York: W.W. Norton & Co Inc., 1957).

1. Sprieser, P.T., "Holographic memory and therapy localization." Proceedings of Summer Meeting, ICAK, Detroit, 1983.

2. Sprieser, P.T., "Pathways of therapy localization," **Chiro Econ**, Vol 26, No 3 (Nov/Dec 1983).

3. Sprieser, P.T., "Further explanation of surrogate testing and therapy localization," **Chiro Econ**, Vol 29, No 4 (Jan/Feb 1987).

IMPROVING ABILITY TO REPRODUCE APPLIED KINESIOLOGY STUDIES

by David S. Walther, D.C.

ABSTRACT

This commentary on "The Efficacy of Therapy Localization as a Diagnostic Tool" by Jeff Weber is directed toward improving ICAK papers for acceptance by refereed journals.

The study on "The Efficacy of Therapy Localization as a Diagnostic Tool" by Jeff Weber in this edition of the ICAK **Collected Papers** is an important addition to the objective study of applied kinesiology. According to Weber,[8] this is a pilot study directed toward ultimately being submitted to a refereed journal for publication.

One of the most important factors in any scientific study is to elaborately describe the control of variables applicable to the study. When one knowledgeable about a subject reads a study on it that fails to explain the control of known variables, the study immediately loses credibility. An excellent example of this is Kenney et al.'s[2] study "Applied Kinesiology Unreliable for Assessing Nutrient Status," which I review in this edition of the **Collected Papers**. Before I completed the second page of that paper, the study was completely discredited in my mind for lack of variable control.

Another reason for a complete description of study methods is to enable another research group to reproduce the study

exactly. When research is reproduced by independent investigators, the validity of the initial study is enhanced. If an attempt to reproduce the study fails, the initial study becomes suspect. There is greater chance for failure to reproduce the research when the exact methods are unknown.

There are several variables that could influence the outcome of Weber's study that are left to the reader's speculation. The subjects of the study are from a patient population. It is unknown whether they are aware of the purpose of the study, or whether they have previously experienced therapy localization to neurolymphatic centers. It has been shown that psychological motivation is an important factor in muscle testing. Markham[3] found that when a subject was told he was getting weaker and encouraged to try harder, there was an average of 20% increase in force produced against the Cybex II dynamometer. Is it possible that an individual knowledgeable about therapy localization to the neurolymphatic center may expect to be stronger with the therapy localization than without? There might be a difference in findings between a naive population and a patient population that has experienced neurolymphatic therapy localization.

The subjects were chosen from a patient population that exhibited "a common middle deltoid weakness." It is not explained whether the middle deltoid weakness was determined by manual muscle testing or by testing against the CMT-1000. If determined by manual muscle testing, was the test patient- or doctor-induced?

Weber states of the CMT-1000, "The patient can be shown simple testing positions to be done seated or standing and an

operator is not required." In reality, an operator is not required for judgment of force produced in the test, but one is required for patient instructions. Was there control of how the instruction was given? Johansson et al.[1] have demonstrated an average of 8% increase in muscle contraction strength when instructions are given in a louder voice. In proprioceptive neuromuscular facilitation (PNF), training to properly use the voice and other clues to enhance patient performance are emphasized.[6]

Weber is to be congratulated for applying statistical analysis to his study. This is a much needed and lacking factor in applied kinesiology research. This writer does not have the ability to analyze the methods of statistical analysis used. I am accustomed to reading the results of t-testing as a probability factor, e.g., $p < 0.001$ rather than simply seeing the statement, "Active TL values reflect significance on t-testing..."

Those of us in applied kinesiology sometimes have a tendency to be overenthusiastic about positive research results. Following is a quote from Weber's paper; then it is re-phrased in a manner that I believe puts the author on much safer ground.

Original version: "The unmistakable reproducibility of our results leads us to believe that therapy localization, is, at the very least, an excellent diagnostic tool for evaluating neurolymphatic reflexes. We are of the opinion that the results of this study document facts about the use of therapy localization which have not been objectively brought to light prior to this."

Revised version: The significance of our results indicates

that therapy localization adds to the clinical evaluation of neurolymphatic reflexes. This study objectively supports the use of therapy localization, which has previously had minimal objective study.

What is noted as "references" at the end of the paper is actually a bibliography. Most refereed journals do not accept a bibliography and require references that are identified in the paper by number. This helps the reader find more information about a subject or identify the source of the author's statement. For example, if one does not know what "therapy localization[7]" is, a reference ([7]) directly refers the reader to a text with the information.

Finally, an error that is made by many writing on chiropractic and applied kinesiology is to consistently capitalize the words "chiropractor," "chiropractic," and "applied kinesiology." Stoner's book, **The Eclectic Approach to Chiropractic**[5], was criticized by Schafer[4], in a book review. Such capitalization, Schafer stated, "...is typical of cultist literature." In any event, it is both improper and unnecessary.

Again, Jeff Weber is to be congratulated for undertaking this project and producing an excellent paper. These comments are in no way made to degrade his effort; in fact, I chose to comment on his paper as an introduction to the new commentary section of the **Collected Papers** because of its excellence. We need to begin looking at our papers as the scientific community does, because they **are** looking at them.

REFERENCES

1. Johansson, C.A., B.E. Kent, & K.F. Shepard, "Relationship between verbal command volume and magnitude of muscle contraction," **Phys Ther**, Vol 63, No 8 (Aug 1983).

2. Kenney, J.J., R. Clemens, & K.D. Forsythe, "Applied kinesiology unreliable for assessing nutrient status," **J Am Diet Assoc**, Vol 88, No 6 (Jun 1988).

3. Markham, B.C., "Various EMG and force of contraction studies during hamstring contraction." Proceedings of Winter Meeting, ICAK, San Diego, 1977.

4. Schafer, R.C., "Book review of The Eclectic Approach to Chiropractic," **ACA J Chiro**, Vol 13, No 5 (May 1976).

5. Stoner, F., **The Eclectic Approach to Chiropractic** (Las Vegas: F.L.S. Publishing Co, 1975).

6. Voss, D.E., M.K. Ionta, & B. Myers, **Proprioceptive Neuromuscular Facilitation Patterns and Techniques**, 3rd ed (Philadelphia: Harper & Row, 1985).

7. Walther, D.S., **Applied Kinesiology: Basic Procedures and Muscle testing** (Pueblo, CO: Systems DC, 1981).

8. Weber, J., 2037 Ocean Ave., Brooklyn, NY 11230 - personal communication.

INDEX

Index
Cont'd