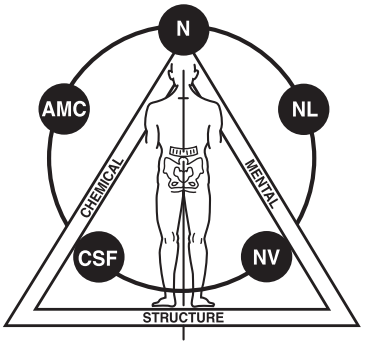
International College of Applied Kinesiology®-U.S.A.

Experimental Observations of Members of the ICAK

Volume I, 2005 – 2006

Proceedings of the Annual Meeting



International College of Applied Kinesiology®-U.S.A.

Experimental Observations of Members of the ICAK

Volume I, 2005 – 2006

Proceedings of the Annual Meeting

Presented

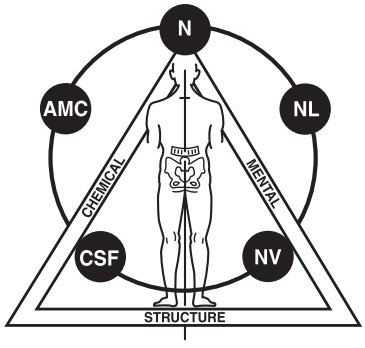
June 9 – 12, 2005

Marina del Rey, CA

Publications Staff:

Terry K. Underwood

Andria Dibbern



Message From the Chairman

David Leaf, D.C., DIBAK

For 29 years, the members of the International College of Applied Kinesiology®-U.S.A. have shared their insights, outcomes, case histories and research through the papers presented in the *Proceedings*. The ICAK-U.S.A. continues to thrive as an "Arena of Ideas" through which members have the opportunity to present their observations and research results. These published works document the first steps toward the furtherance and development of the authors' hypotheses, concepts and procedural techniques which can culminate in their material becoming part of the accepted body of knowledge we know as applied kinesiology. We invite all members to participate in this endeavor in the future.

Past history shows that the observations of one doctor stimulate the minds of others and the end result can be, as Dr. Goodheart credits Dr. Deal as saying, "and now we have another piece of the puzzle."

I am pleased to again have the opportunity to read and share with the members the advances and successes of this year.

Thank you and congratulations to all of our contributors. And a special thanks to Drs. Rebecca Hartle, David Engel, Jan Calhoon, Alan Zatkin, and Denise Natale for all of their help during the review process. We look forward to seeing you at the Annual Meeting, June 9-12, 2005 in Marina del Rey, CA.

Introduction

This forty-seventh collection of papers from members of the International College of Applied Kinesiology®-U.S.A. contains 29 papers (including 9 case histories) by 21 authors. The authors welcome comments and further ideas on their findings. You may talk with them at the meeting or write them directly; addresses are given in the Table of Contents.

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

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

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

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

Instructions to Authors Proceedings of the ICAK-U.S.A.

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

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

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

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

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

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

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.

All manuscripts (meaning any material submitted for consideration to publish) must be accompanied by a properly completed *RELEASE FORM*, signed by all authors and by employer if submission represents a "work for hire." Upon such submission, it is to be accepted by all authors that no further dissemination of any part of the material contained in the manuscript is permitted, in any manner, without prior approval from the editor; nonobservance of this copyright holder stipulation may result in withdrawal of submission for consideration to publish.

Continuing call for papers includes:

Research Studies (Investigations)—reports of new research findings into the enhancement factors of health, causal aspects of disease, and the establishment of clinical efficacies of related diagnostic and therapeutic procedures.

Hypotheses—preliminary studies that may establish a solid basis for further in-depth investigations.

Literature reviews—critical assessments of current knowledge of a particular subject of interest, with emphasis on better correlation, the pointing up of ambiguities, and the delineation of areas that may constitute hypotheses for further study. Meta-analysis is included here.

Clinical procedures—succinct, informative, didactic papers on diagnostic and therapeutic procedures, based heavily on authoritative current knowledge.

Case reports—accounts of the diagnosis and treatment of unusual, difficult, or otherwise interesting cases that may have independent educational value or may contribute to better standardization of care for a particular health problem when correlated with similar reports of others.

Case reviews—a retrospective comparative assessment of the diagnosis and treatment of several cases of a similar condition, ie, the comparative evaluation of two or more (perhaps hundreds) of case reports.

Clinical Observations (Technical reports)—the reporting and evaluation of new or improved equipment or procedures, or the critical evaluation of old equipment or procedures that have not previously been critically evaluated.

Commentary—editorial-like, more in-depth essays on matters relating to the clinical, professional, educational, and/or politicolegal aspects of health care principles and practice.

Critical Review (Letters to the editor)—communications that are directed specifically to the editor that critically assess some aspect of the ICAK, particularly as such assessment may add to, clarify, or point up a deficiency in a recently published paper; authors are afforded the privilege of a counter-response.

The following editorial policies will apply:

Informed consent—Manuscripts that report the results of experimental investigations with human subjects must include a statement that informed consent was obtained, in writing, from the subject or legal guardian, after the procedure(s) had been fully explained.

Patient anonymity—Ethical and legal considerations require careful attention to the protection of the patient's anonymity in case reports and elsewhere. Identifying information such as names, initials, actual case numbers, and specific dates must be avoided; identifying information about a patient's personal history and characteristics should be disguised. Photographs or artistic likenesses of subjects are publishable only with their written consent or the consent of legal guardian; the signed consent form, giving any special conditions (ie, eyes blocked off), must accompany manuscript.

Authorship—All authors of papers submitted to ICAK-U.S.A. must have an intellectual stake in the material presented for publication. All must be willing to answer for the content of the work. Authors should be willing to certify participation in the work, vouch for its validity, acknowledge reviewing and approving the final version of the paper, acknowledge that the work has not been previously published elsewhere, and be able to produce raw data if requested.

Conflict of Interest—In recognition that it may at times be difficult to judge material from authors where proprietary interests are concerned, authors should be ready to answer requests from the editor regarding

potential conflicts of interest. The editor makes the final determination concerning the extent of information released to the public.

Acknowledgments—Illustrations from other publications must be submitted with written approval from the publisher (and author if required) and must be appropriately acknowledged in the manuscript.

Author responsibility—Manuscripts accepted for publication are subject to such editorial modification and revision as may be necessary to ensure clarity, conciseness, correct usage, and conformance to approved style. However, insofar as authors are responsible for all information contained in their published work, they will be consulted if substantive changes are required and will have further opportunity to make any necessary corrections on the proofs.

Reproductions—The entire contents of the *Proceedings of the ICAK-U.S.A.*is protected by copyright, and no part may be reproduced by any means without prior permission from the publisher. In particular, this policy applies to the reprinting of an original article in another publication and the use of any illustrations or text to create a new work.

Manuscript preparation

Authors are encouraged to submit final manuscripts on computer diskette along with the printed revised copy. Authors accept responsibility for the submitted diskette exactly matching the printout of the final version of the manuscript. Manuscripts produced on IBM or compatible computers are preferred. Macintosh files should not be saved using the Fast Save option. Identify the diskette with journal name, manuscript number, name of first author, manuscript title, name of manuscript file, type of hardware, operating system and version number, and software and version number. Each article should be on a separate diskette. Please put all manuscript parts (text, references and figure legends) in one file.

The ICAK-U.S.A. does not assume responsibility for errors in conversion of customized software, newly released software and special characters. Mathematics and tabular material will be processed in the traditional manner.

Approved manuscript style

Manuscripts submitted for consideration to publish in *The Proceedings of the ICAK-U.S.A.* must be compiled in accordance with the following instructions, and manuscripts not so compiled are subject to return to the author for revision.

Summary of requirements

Type the manuscript double-spaced, including title page, abstract, text, acknowledgments, references, tables, and figure legends. (Note: footnotes should be avoided by including any necessary explanatory information within the text in parentheses). Do not break any words (hyphenate) at the end of any line; move to the next line if entire word does not fit.

Each manuscript component should begin on a new page, in the following sequence:

- Title page (page 1)
- Abstract and key word page (page 2)
- Text pages (starting on page 3)
- Acknowledgment page

- Reference page(s)
- Table page(s)
- Legends for illustrations pages(s).

Detailed preparation procedure

Begin each of the following sections on separate pages: title (including author name[s], address and phone number of principal author, running head, etc), abstract and key words, text, acknowledgments, references, individual tables, and figure legends.

Units of Measurement—In most countries the International System of Units (SI) is standard, or is becoming so, and bioscientific journals in general are in the process of requiring the reporting of data in these metric units. However, insofar as this practice is not yet universal, particularly in the United States, it is permissible for the time being to report data in the units in which calculations were originally made, followed by the opposite unit equivalents in parentheses; ie, English units (SI units) or SI units (English units). Nevertheless, researchers and authors considering submission of manuscripts to the ICAK-U.S.A. should begin to adopt SI as their primary system of measurement as quickly as it is feasible.

Abbreviations and symbols—Use only standard abbreviations for units of measurement, statistical terms, biological references, journal names, etc. Avoid abbreviations in titles and abstracts. The full term for which an abbreviation stands should precede its first use in the manuscript, unless it is a standard unit of measurement.

Title page

The title page should carry (1) the title of the article, which should be concise but informative; (2) a short running head or footline of no more than 40 characters (count letters and spaces) placed at the foot of the title page and identified; (3) first name, middle initial, and last name of each author, with highest academic degree(s); (4) names of department(s) and institution(s) to which work should be attributed; (5) disclaimers, if any; (6) name, address, phone, and fax number of author responsible for correspondence, proofreading of galleys, and reprint requests (usually principal author); (7) the source(s) of support in the form of grants, equipment, drugs, or all of these.

Abstract and key word page

The second page should carry an abstract of no more than 150 words, 250 if using a structured abstract. The structured abstract is now required for all original data reports, reviews of the literature and case reports; prose abstracts will be accepted for use in only certain original papers not reporting data (ie, position papers, historical treatises).

Please visit the following link online for helpful information on structured abstracts:
http://www.soto-usa.org/Newsletter/DCInternetEdition/dc_internet_ed_vol_3_no3Abstrak/StructuredAbstracts.htm

Below the abstract, provide, and identify as such, 3 to 10 key indexing terms or short phrases that will assist indexers in cross-indexing your article and that may be published with the abstract. Use terms from the Index Medicus Medical Subject Headings (MeSH) as much as possible.

Text pages

The text of observational and experimental articles is usually—but not necessarily—divided into sections with the headings Introduction, Materials and Methods, Results, Discussion, and Conclusions. Long articles may need subheadings within some sections to clarify or break up content. Other types of articles such as case reports, reviews, editorials, and commentaries may need other formats.

Please visit the following link online for helpful information on writing patient case reports:
http://www.soto-usa.org/Newsletter/DCInternetEdition/dc_internet_ed_vol_3_no3Abstrak/Green%20Johnson%20Case%20Reports.pdf

Reference: Green BN, Johnson CD, Writing Patient Case Reports for Peer-Reviewed Journals: Secrets of the Trade Journal of Sports Chiropractic & Rehabilitation. 2000 Sep; 14(3): 51-9.

Introduction

Clearly state the purpose of the article. Summarize the rationale for the study or observation. Give only strictly pertinent references and do not review the subject extensively; the introduction should serve only to introduce what was done and why it was done.

Materials and methods

Describe your selection of the observational or experimental subjects (patients or experimental animals, including controls) clearly. Identify the methods, apparatus (manufacturer's name and address in parentheses) and procedures in sufficient detail to allow others to reproduce the work for comparison of results. Give references to establish methods, provide references and brief descriptions for methods that have been published but may not be well known, describe new or substantially modified methods and give reasons for using them and evaluate their limitations.

When reporting experiments on or with human subjects, indicate whether the procedures used were in accordance with the ethical standards of the Committee on Human Experimentation of the institution in which the research was conducted and/or were done in accordance with the Helsinki Declaration of 1975. When reporting experiments on animals, indicate whether the institution's or the National Research Council's guide for the care and use of laboratory animals was followed. Identify precisely all drugs and chemicals used, including generic name(s), dosage(s), and route(s) of administration. Do not use patient names, initials, or hospital numbers or in any manner give information by which the individuals can be identified.

Include numbers of observations and the statistical significance of the findings when appropriate. Detailed statistical analyses, mathematical derivations, and the like may sometimes be suitably presented in the form of one or more appendixes.

Results

Present your results in logical sequence in the text, tables, and illustrations. Do not repeat in the text all the data in the tables, illustrations, or both; emphasize or summarize only important observations.

Discussion

Emphasize the new and important aspects of the study and conclusions that follow from them. Do not repeat in detail data given in the Results section. Include in the Discussion the implications of the findings and their

limitations and relate the observations to other relevant studies. Conclusions that may be drawn from the study may be included in this discussion section; however, in some cases, they may be more succinctly presented in a separate section.

Conclusions

The principal conclusions should be directly linked to the goals of the study. Unqualified statements and conclusions not completely supported by your data should be avoided. Avoid claiming priority and alluding to work that has not been completed. State new hypotheses when warranted but clearly label them as such. Recommendations (for further study, etc), when appropriate, may be included.

Acknowledgments

Acknowledge only persons who have made substantive contributions to the study itself; this would ordinarily include support personnel such as statistical or manuscript review consultants, but not subjects used in the study or clerical staff. Authors are responsible for obtaining written permission from persons being acknowledged by name because readers may infer their endorsement of the data and conclusions.

Reference pages

References are to be numbered consecutively as they are first used in the text (placed in line in parentheses) and listed in that order (not alphabetically) beginning on a separate sheet following the text pages. The style (including abbreviation of journal names) must be in accordance with that specified by the US National Library of Medicine: see recent January issue of Index Medicus for a complete listing of indexed journals.

Only those references that actually provide support for a particular statement in the text, tables, and/or figures should be used. Excessive use of references should be avoided; normally, 1 or 2 authoritative references to support a particular point are sufficient. A short article of up to 5 or 6 manuscript pages may be adequately supported by 5 to 10 references; longer articles of up to 20 pages by 15 to 25.

References must be verified by the author(s) against the original document. Abstracts, "unpublished observations" and "personal communications" may not be used as references, although reference to written (not verbal) communications may be inserted (in parentheses) in the text. Information from manuscripts submitted but not yet accepted may be referred to (in parentheses) in the text. Manuscripts accepted but not yet published may be included in the references with the designation "In press." When a previously cited reference is used again, it is designated in the text (in parentheses) by the number originally assigned to it by its first use: do not assign it another number or again list it in the references as "op cit."

For the most part, sources of information and reference support for a bioscientific paper should be limited to journals (rather than books) because that knowledge is generally considered more recent and (in the case of refereed journals) more accurate. Consequently, the basic form for approved reference style is established by journal listings; others (books, etc) are modified from journal listings as may be required. A summary of journal reference style is as follows:

Last name of author(s) and their initials in capitals separated by a space with a comma separating each author. (List all authors when 6 or fewer; when 7 or more, list only the first 6 and add et al.)

Title of article with first word capitalized and all other words in lower case, except names of persons, places, etc.

Name of journal, abbreviated according to Index Medicus; year of publication (followed by a semicolon); volume number (followed by a colon); and inclusive pages of article (with redundant number dropped, ie, 105-10).

Specific examples of correct reference form for journals and their modifications to other publications are as follows:

Journals

1. Standard article You CH, Lee KY, Chey RY, Menguy R. Electrogastrographic study of patients with unexplained nausea, bloating and vomiting. Gastroenterology 1980;79:311-4.
2. Corporate author The Royal Marsden Hospital Bone-Marrow Transplantation Team. Failure of synergeneic bone-marrow graft without preconditioning in post-hepatitis marrow aplasia. Lancet 1977;2:242-4.
3. No author given Coffee drinking and cancer of the pancreas [editorial]. Br Med J 1981;283:628.
4. Journal supplement Magni F. Rossoni G, Berti F. BN-52021 protects guinea-pig from heart anaphylaxis. Pharmacol Res Commun 1988;20 Suppl 5:75-8.
5. Journal paginated by issue rather than volume Seaman WB. The case of pancreatic pseudocyst. Hosp Pract 1981;16(Sep):24-5.

Books and other monographs

6. Personal author(s) Eisen HN. Immunology: an introduction to molecular and cellular principles of the immune response. 5th ed. New York: Harper and Row; 1974. p. 406.
7. Editor, compiler, chairman as author Dausset J, Colombani J, editors. Histocompatibility testing 1972. Copenhagen: Munksgaard; 1973. p. 12-8.
8. Chapter in a book Weinstein L, Swartz MN. Pathogenic properties of invading microorganisms. In: Sodeman WA Jr, Sodeman WA, editors. Pathologic physiology: mechanisms of disease. Philadelphia: WB Saunders; 1974. p. 457-72.
9. Published proceedings paper DuPont B. Bone marrow transplantation in severe combined immunodeficiency with unrelated MLC compatible donor. In: White HJ, Smith R, editors. Proceedings of the 3rd Annual Meeting of the International Society for Experimental Hematology. Houston: International Society for Experimental Hematology; 1974. p. 44-6.
10. Agency publication Ranofsky AL. Surgical operations in short-stay hospitals: United States—1975. Hyattsville (MD): National Center for Health Statistics; 1978. DHEW publication no (PHS) 78-1785. (Vital and health statistics; series 13; no 34).
11. Dissertation or thesis Cairns RB. Infrared spectroscopic studies of solid oxygen [dissertation]. Berkeley (CA): University of California; 1965.

Other articles

12. Newspaper article Lee G. Hospitalizations tied to ozone pollution: study estimates 50,000 admissions annually. The Washington Post 1996 Jun 21; Sect. A:3 (col. 5).
13. Magazine article Roueche B. Annals of medicine: the Santa Claus culture. The New Yorker 1971 Sep 4:66-81.

Table pages

Type each table on a separate sheet; remember to double-space all data. If applicable, identify statistical measures of variation, such as standard deviation and standard error of mean. If data are used from another published or unpublished source, obtain permission and acknowledge fully.

Using arabic numerals, number each table consecutively (in the order in which they were listed in the text in parentheses) and supply a brief title to appear at the top of the table above a horizontal line; place any necessary explanatory matter in footnotes at the bottom of the table below a horizontal line and identify with footnote symbols *, †, ‡, §, , ¶, **, ††, ‡‡, etc.

Illustration legend pages

Type legends for illustrations double-spaced, starting on a separate page, following the table pages. Identify each legend with arabic numerals in the same manner and sequence as they were indicated in the text in parentheses (ie, Figure 1). Do not type legends on artwork copy or on pages to which illustrations may have been mounted; they must be typed on separate pages from the illustrations themselves.

When symbols, arrows, numbers or letters are used to identify parts of the illustrations, identify and explain each one clearly (if necessary) in the legend. Explain internal scale and method of staining in photomicrographs, if applicable.

Illustration preparation

Illustrations (including lettering, numbering and/or symbols) must be of professional quality and of sufficient size so that when reduced for publication all details will be clearly discernible; rough sketches with freehand or typed lettering are not encouraged. All illustrations (including x-rays) are best submitted as professional-quality, unmounted, black and white glossy prints at least 127 by 173 mm (5 by 7 in) but no larger than 203 by 254 mm (8 by 10 in). Do not place titles or detailed explanations on the illustration; such information should be given in the figure legends. Do not send x-ray film.

Each figure should have a label on its back indicating the number of the figure, author name(s), and top of the figure indicated with an arrow. Do not write on the back of the illustrations themselves; do not mount them on other sheets; do not bend, scratch or mar them with paper clips.

If photographs of persons are used, either the subjects must not be identifiable or their pictures must be accompanied by written permission to publish the photographs.

Cite each figure in the text (generally in parentheses) in consecutive order. If a figure has been published, acknowledge the original source and submit written permission from the copyright holder to reproduce the material. Permission is required, regardless of authorship or publisher, except for documents in the public domain. Articles may appear both in print and online versions, and wording of the letter should specify permission in all forms and media. Failure to get electronic permission rights may result in the images not appearing in the online version.

Electronic illustration submission

Figures may be submitted in electronic format. All images should be at least 5 in wide. Images should be provided in EPS or TIF format on Zip disk, CD, floppy, Jaz, or 3.5 MO. Macintosh or PC is acceptable. Graphics software such as Photoshop and Illustrator, not presentation software such as PowerPoint, CorelDraw, or Harvard Graphics, should be used in the creation of the art. Color images need to be CMYK, at least 300 DPI, with a digital color proof, not a color laser print or color photocopy. Gray scale images should be at least 300 DPI and accompanied by a proof. Combinations of gray scale and line art should be at least 1200 DPI with a proof. Line art (black and white or color) should be at least 1200 DPI with a proof. Please include hardware and software information, in addition to the file names, with the disk.

Manuscript submission summary

Manuscript components

In terms of completeness of submission, the "manuscript" includes the following components:

- Manuscript (the original and 2 clear photocopies). The author should be sure to retain an additional copy in case of loss of the submission copies in transit.
- Illustrations (1 set for each manuscript).
- *RELEASE FORM* (signed by all authors, and by employer if study was a work for hire).
- Letter(s) of permission to use previously published material in all forms and media (if applicable).
- Consent form(s) to publish photographs in which subjects may be identifiable (if applicable).
- Cover letter from principal author (or author specified as correspondent) providing any special information regarding the submission which may be helpful in its consideration for publication.
- Computer disk with manuscript(s).

Mailing instructions

The manuscript should be securely packaged in a heavy-weight envelope (or carton if bulky) with illustrations placed between cardboard to prevent bending; do not use paper clips or in any manner fasten illustrations to cardboard that could scratch or mar them.

The manuscript package should be mailed (first class or express, insured, return receipt requested, if desired) to:

ICAK-U.S.A. Central Office
6405 Metcalf Ave., Suite 503
Shawnee Mission, KS 66202
PH. 913-384-5336, FAX 913-384-5112, EMAIL ICAK@dci-kansascity.com

Applied Kinesiology Status Statement

International College of Applied Kinesiology®-U.S.A.

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

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

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

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

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

- Myofascial dysfunction (micro avulsion and proprioceptive dysfunction)
- Peripheral nerve entrapment
- Spinal segmental facilitation and deafferentation
- Neurologic disorganization
- Viscerosomatic relationships (aberrant autonomic reflexes)

- Nutritional inadequacy
- Toxic chemical influences
- Dysfunction in the production and circulation of cerebrospinal fluid
- Adverse mechanical tension in the meningeal membranes
- Meridian system imbalance
- Lymphatic and vascular impairment

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

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

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

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

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

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

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

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

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

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

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

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

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

Table of Contents

Division I – Informative Papers

Division II – Critical Review

Division I

Informative Papers

Functional Systems Approach to Central Nervous System Evaluation

Richard Belli, D.C., D.A.C.N.B.

Abstract

A functional systems approach to evaluation of the central nervous system is presented. The value of functional systems evaluation vs. an attempt to evaluate individual nuclei etc. is discussed. Twelve functional systems are presented with suggestions for evaluation with manual muscle testing or applied kinesiology. Additionally, typical clinical signs and symptoms associated to dysfunction of central systems are presented.

Key Indexing Terms: Manual muscle test, applied kinesiology.

Introduction

Health care practitioners that use manual muscle testing, such as applied kinesiology, typically approach the patient seeking to test muscles that are neurologically or conditionally inhibited without provocation. However, many patients only suffer symptomology, chronic as it may be, intermittently or while involved in activity. Presented here is a novel paradigm of approaching conditionally inhibited muscle patterns after provocation. The systems and patterns presented here are primarily related to central nervous system dysfunction. Dysfunction of these systems and the associated symptomology are often only present when challenged by provocation or activity. Therefore, it is necessary to clinically challenge these systems to observe dysfunction and afford the patient correction.

Discrete or localized areas of the central nervous system are very difficult or impossible to evaluate with manual muscle testing. However, manual muscle testing provides the perfect tool for evaluating the CNS as functional systems. A functional system may be thought of as an integrated area or areas of the CNS that have specific or in some cases multiple functions. For example, all of the patterns presented here are modulated by the basal ganglia and cerebellum, so they are major players in all movement, but don't have dedicated movement patterns of their own.

It is a well accepted paradigm that nearly all functional systems have a related motor activity that results in distinct inhibition and facilitation patterns. Therefore, functional systems can simply be evaluated by provoking the system into action and observing the motor response with manual muscle testing. Complex reflexes (e.g. scratching, yawning, chewing, respiration etc.) and movement patterns (walking, running, etc.) are controlled by pattern generators that in turn are controlled by midbrain locomotor centers acting via the reticular formation and reticulospinal tracts. For example, the mesencephalic locomotor center has to be cortically stimulated to evoke walking movements.[1] Walking then ensues and normally programmed inhibition and facilitation patterns take place.

A simple example of a functional system is the so called "deep tendon reflex," which facilitates the muscle spindle bundle. The reflex is initiated by a sudden stretch of the muscle spindle, which sends a signal into the spinal cord affecting the inhibitory and facilitatory interneurons. These interneurons cause the appropriate inhibitory and facilitatory motor pattern. The pattern generator, or the inhibitory and facilitatory interneu-

rons, are modulated by segmental input from proprioceptors, viscera, and descending supraspinal projections, all of which is combined into a functional system.

Functional systems are modulated by activity at all levels of the neuraxis, resulting in a multitude of opportunities for causes of dysfunction as well as treatment. For example, an old injury may be causing a withdrawal reflex that would override nearly any other pattern generator. Therefore, with the withdrawal reflex present, other functional systems being tested would probably not result in the appropriate motor response. Therefore the system tested is dysfunctional as a result of an old injury. Appropriate analysis would lead to the injury as the cause, correction of the injury withdrawal, and the functional system upon the next perturbation demonstrating the appropriate motor response indicating that it is now functioning normally.

The development of the functional systems approach to CNS analysis is the result of ideas derived from extensive literature search and clinical testing of hundreds of normal and dysfunctional patients. All twelve systems presented here have a known and predictable motor response. If upon provocation the response is not congruent with the predicted outcome, it can be assumed that dysfunction is ultimately affecting the outcome. Since the resultant effect is on the central nervous system, the health consequence can be significant.

In the context of this text, each functional system has been named for ease of understanding and clinical use. The nomenclature is based on the knowledge of the time and conjecture, but in absolute terms, in some cases may not be accurate. No doubt it is flawed, partial, and open to debate, therefore, more accurate and appropriate nomenclature will most likely be assigned in the future. What ever the case, the names may change, but the systems and the tests will not.

With twelve systems presented here, and most likely many more in existence, the analysis for the clinician from a purely muscle testing stand point can be impractical. Therefore, it is necessary to become familiar with the general symptomology of dysfunction of each of the systems. Mindful of that knowledge, combined with clinical evidence and history, the clinician can narrow the field of systems to be tested dramatically. For example, in the case of insidious onset low back pain and muscle spasm, the clinician may want to evaluate spinal cord interneuron dysfunction. If these pattern generators are not functioning correctly, muscles that should be normally inhibited and facilitated are not, resulting in a sudden onset of pain and spasm upon a simple movement. The patient will express that they simply started to bend over and got stuck. Another example is related to function of the vagus system, of which is both motor and sensory in function. If the patient complains of digestive dysfunction the vagal motor system would warrant evaluation. If the patient complains of constant hunger and weight gain, due to suspected decreased sensory feedback from the stomach, the vagal sensory system would warrant evaluation.

Correction techniques for the dysfunctional systems presented here is not within the scope of this presentation. However, keeping in mind that the domain of the cause of the dysfunction is either segmental, extra-pyramidal, or pyramidal in nature, correction can be effectively achieved by standardized applied kinesiology approaches.

Discussion

Each test evokes a typical inhibition and facilitation pattern. When the system being tested is dysfunctional the pattern of facilitation and inhibition will be reversed. Or, the normal pattern of inhibition will be facilitated and the pattern of facilitation will be inhibited.

Clinical experience indicates that coming from a treatment stand point, and all the necessary tests, that it is easier to work from the side of the pattern that is normally facilitated, but is now functionally inhibited after provocation.

The dysfunctional pattern or system is determined by the normally facilitated muscle (after provocation) being conditionally inhibited in all three muscle test types as described by Walter Schmitt.[2] It is postulated here that the inhibition as described by Schmitt, under these parameters, may be related to pyramidal, extra pyramidal, and segmental systems.

Once the conditionally inhibited muscle pattern has been evoked by provocation, the primary location of dysfunction, such as pyramidal, extrapyramidal, or segmental areas can be determined. The determination is done by evoking facilitation of the inhibited muscle and testing all three types.[3] Once the muscle type evaluation has been done standard applied kinesiology protocols can be applied for correction.

Technique

A reliable screening tool for the need of CNS evaluation is simultaneous therapy localization of GV-27 and CV-24 using a left leg forward gait position. The normally inhibited right latissimus is tested while the patient does the double therapy localization (typically using the index and middle fingers). If CNS evaluation is needed the TL will functionally facilitate or disrupt the normal gait pattern.

Inhibition and Facilitation Patterns for CNS Dysfunction

1.) Spinal cord:
- Withdrawal reflex: "mass limb" reflex (cord and multi-segmental interaction)[4]
- IML
  - — One eye closed
    - – Facilitates ipsilateral flexors and inhibits contralateral flexors
- Interneurons:
  - — Stretch or approximate muscle spindle, or pinch tendon organ[5, 6]
    - – Check appropriate reciprocal facilitation and inhibition patterns

2.) Myelencephalon/reticular formation:
- Head rotation
  - — Opposite pectoralis is facilitated and opposite latissimus is inhibited

3.) Vagal system:
- Stimulate Vagal sensory with visceral pressure or pressure to nodose ganglion under SCM muscle
  - — Facilitates flexors and inhibits extensors
  - — Solitary nucleus
  - — Barroceptor challenge or pressure applied to carotid
    - – Decreases heart rate and inhibits subscapularis
    - – Solitary nucleus to Vagal motor nucleus
  - — Swallow/think about food
    - – Facilitates flexors and inhibits extensors (nucleus ambiguous)
    - – Inhibits subscapularis bilaterally (dorsal motor nucleus)

4.) Trigeminal motor system-muscles of mastication:
- Occlusion
  - — Facilitates flexors and inhibits extensors

5.) Vestibulospinal system, bulbo reticular facilitatory area:
- Both feet on table (stance position)
  - — Upper body flexors are inhibited and extensors are facilitated

6.) Reticular formation:
- Neck flexion
  — Facilitates flexors globally and inhibits extensors globally
  — Neck extension does the opposite

7.) Diencephalon and gait locomotion system:
- One foot on table
  — Typical gait pattern of inhibition and facilitation

8.) Mesencephalon:
- Accommodation
  — Follow object from far to near
  — Facilitates extensors and inhibits flexors

9.) Cardiac sympathetic autonomic system:
- Single IML effect
  — Cover one eye
  — Ipsilateral subscapularis is facilitated and contralateral subscapularis is inhibited

10.) Pyramidal system:
- Finger to thumb activity
  — Facilitates ipsilateral flexors and inhibits contralateral flexors (related to IML)

11.) Limbic system:
- Sad thought
  — Facilitates global flexors and inhibits global extensors
- Thought of outrage
  — Facilitates global extensors and inhibits global flexors
- Eyes directly lateral
  — Facilitate contralateral flexor and inhibit contralateral extensor

12.) Sensory system:
- Ipsilateral sensory input to auditory, somatic or visual system
- Facilitate contralateral flexor and inhibit contralateral extensor

Conclusion

Presented in this paper is a system of evaluation and treatment of common dysfunction of CNS functional systems. This method of testing presents a new paradigm of approaching the patient from conditionally inhibited muscles after provocation of functional systems. Dysfunction of these systems often only present symptoms when in use, therefore presented is an opportunity of a new window into the function of the central nervous system. The practitioner must be mindful that this system is designed to locate evaluate and treat dysfunction. If the patient does not appropriately respond it would be prudent to consider pathology and evaluate accordingly. Finally, the beauty of evaluating functional systems is the ability to find and correct CNS dysfunction and afford the patient relief from what are often chronic and far reaching symptoms that no other treatment system other than manual muscle testing and applied kinesiology can effectively approach.

References

1. Lambert, John. Lecture Notes in Neurophysiology: Control of Motor Function. Princeton University, 2003.

2. Schmitt, Walter H., Jr. A Nneurological model for the three types of manual muscle testing. Proceedings of the I.C.A.K.–U.S.A. Volume 1 1996–97. p 79**.**

3. Schmitt, Walter H., Jr. A Nneurological model for the three types of manual muscle testing. Proceedings of the I.C.A.K.–U.S.A. Volume 1 1996–97. p 79**.**

4. Lin, Robert S. The Neurological Control Systems for Normal Gait. JPO Vol 2, 1990, Num 1, pp2.

5. Belli, Richard. Deep Tendon Reflexes. I.C.A.K. News Update, 1994.

6. Belli, Richard. Autogenic Inhibition. Proceedings of the I.C.A.K. – U.S.A., Volume I, 1995–1996. pp15–18.

General Resources

Hendelman, Walter J. Atlas of Functional Neuroanatomy. New York: CRC Press; 2000.

Brodal, Per. The Central Nervous System: Structure And Function. 3rd ed. New York: Oxford University Press; 2004.

Robinson, David. Primer On The Autonomic Nervous System. 2nd. ed. San Diego: Elsevier Academic Press; 2004

Kalavis, Peter W. and Barnes, Charles D. Limbic Motor Circuits and Neuropsychiatry. New York: CRC Press; 1993.

Stein, Paul S.G. Neurons, Networks, and Motor Behavior. Massachusetts: MIT Press; 1999.

The Role of The Anterior Fifth Lumbar in Hammer Toes and Disequilibrium – A Case Study

Harlan Browning, D.C., C.C.N., D.C.B.C.N.

Abstract

A case study of a patient with hammer toes and lack of balance is presented.

Key Indexing Terms: Balance, talocalcaneonavicular joint, applied kinesiology.

Introduction

The contribution of afferent stimuli in coordinated gate largely relies on joint mechanoreceptors, muscle proprioception and Meissner' corpuscles within the skin and fascia of the foot.[1] The summation of this sensory stimuli provides for what has been termed the "magnet reaction" or positive support reaction.[2] Changes to this mechanism are evident in lower extremity dysfunction and are a hallmark of the anterior fifth lumbar.

Discussion

A fifty-five year old female presented for diagnosis and treatment of thirty-five year history of bilateral foot problems and disequilibrium. The patient described her feet as "unsightly" and "embarrassing" due to hammer toes. She continued to state that she had constant foot pain and since her toes no longer made contact with the ground she felt very unstable, especially with activities like yoga. Several podiatrist were consulted over the years for this problem; bilateral bunion removal from the great toes was performed nine years prior.

Postural assessment in the standing position showed a level pelvis, shoulder girdle and occiput with a posterior pitch en masse as to place the center of gravity posterior to the talocalcaneonavicular joint. Non contact of digits 2–5 was noted on both feet. Gait analysis illustrated a uniform heel strike with partial translation into mid-stance, toe off was greatly impaired and ataxia was evident. Movement from supination to pronation was premature.

Palpation of both feet produced marked tenderness over the metatarsal heads of digits 2–5 bilaterally and tenderness of the L5 spinous was exquisite producing a jump sign. Manual muscle testing of the lower extremities produced bilateral TFL, bilateral tibialis anterior and posterior, bilateral peroneus longus, bilateral flexor hallucis longus and bilateral flexor digitorum longus and brevis muscle weakness. All other muscles were graded +5 (0-5) in both patient induced and doctor induced testing.

Assessment of the spine and extremities found several cervical and mid-dorsal subluxations, with dropped metatarsals found bilaterally. Both feet were found to have lateral cuboids and profound restriction in anterior to posterior translation of the ankle mortice. Prone therapy localization to L5 was initially negative but showed with the center piece of the table lowered.[3]

Initial treatment included correction of cervical and dorsal subluxations, as well as correction of the lumbar spine. Specifically, the L5 anteriority was reduced in the supine position utilizing a quick traction of the patient cephalad with a contact to cradle the occiput and stabilize the chin. A noticeable release was felt by the patient and doctor. The ankle mortice and lateral cuboids were both manipulated and the feet were taped for stability. The patient was instructed to return in two days for follow up.

The patient was called the day after the adjustment to see how she was and if there were any questions. The patient remarked that her toes were much closer to the ground and her balance was better. Upon return to my office the following day the patient immediately stated that her toes were almost normal and that this was the first day in 10 years she felt comfortable wearing open toed shoes due to the appearance of her feet. The patient was treated several more times over the next 3 weeks with marked improvement in muscle strength and equilibrium doing yoga. The patient was instructed to continue her yoga and return for follow up in several weeks.

Conclusion

The above case is a classic example of the lack of understanding within the medical community on the relationship of spinal alignment and extremity function. As Dr. Duffy so eloquently stated "no symptom below the knee will be eliminated in the presence of an anterior 5th lumbar." In this case the patient was unfortunately mismanaged and unnecessarily operated on in hopes of correction. It is quite evident that every practitioner would greatly benefit from the knowledge and understanding of applied kinesiology!

References

1. Michaud, Thomas C., Foot Orthoses and Other Forms of Conservative Foot Care, Privately Published. (1997).

2. Walther, David S., Applied Kinesiology Synopsis, 2nd Edition, Systems D.C., Pueblo, Co. 1999.

3. Duffy, Daniel H., Managing the Hot Low Back Seminar, Lombard, IL., 1999.

Correction of Cranial Nerve Neuropathy Using Applied Kinesiology Chiropractic Care: A Case Study of the Treatment of Symptomatic Arnold-Chiari Malformation

Scott Cuthbert, D.C.

Abstract

A complex case of optic nerve neuritis producing visual loss in the right eye, resulting from cranial faults exacerbated by an Arnold Chiari Malformation Type I (ACM) of the brain in a twenty year old female, responds rapidly to applied kinesiology's cranial and spinal therapies.

Key Indexing Terms: Arnold Chiari Malformation (ACM), cranial fault, applied kinesiology.

Introduction

In the Arnold-Chiari syndrome there is a projection of the medulla and cerebellum that extends through the foramen magnum and into the cervical spinal canal. Presumably during fetal life, fixation of the lower spinal cord or its nerve roots may exert traction on the upper cervical cord and brainstem, causing the medulla and cerebellum to herniate through the foramen magnum. Hydrocephalus is frequently present in these cases. Other developmental defects may be associated, such as anomalies in the skull bones, spinal column, spinal cord, and meninges. Other causes of this herniation may be due to the excessive force of precipitate labor, a fall on the buttocks or feet, a forceful stretching of the head from the neck, or a blow on the vertex, among other things. The condylar parts may then be driven into the cranial base and relatively locked. This can produce a severe restriction in the craniosacral pattern of motion, especially at the occiput.[1]

Signs and symptoms of an Arnold-Chiari syndrome are usually evident in the first few months of life and are related to the associated hydrocephalus and other developmental neural defects. Prognosis is poor in these cases. Hydrocephalus is attributed to obstruction of the basal cistern. Occasionally there is a prolongation of the 4th ventricle into the spinal cord, creating CSF flow problems. Compression of the brainstem and stretching of cranial and cervical nerves produce other signs and symptoms. This appeared to be the causative factor in this case.

Medical treatment of infants includes surgical decompression of the posterior fossa and excision of the sac of the spinal region; in adults, surgical decompression of the posterior fossa is performed.[2]

Another finding in this case that is seen in other cases of ACM is what is called "see-saw nystagmus." This refers to a cyclic movement of the eyes; while one eye rises and turns in, the other falls and turns out. The vertical and torsional movements are then reversed, completing the cycle. Responsible intra-cranial lesions are generally in the inferior portion of the 3rd ventricle (para-sellar tumors) or midbrain (infarction). The interstitial nucleus of Cajal may play a role in the production of this type of nystagmus, or the disorder may be due to an unstable visuo-vestibular interaction control system. Lesions in the optic pathways may prevent retinal error signals, essential for vestibulo-ocular reflex adaptation, from reaching the inferior olivary nucleus, thereby making the system less stable.

Around 1930 it was discovered that a network of neurons in the brainstem connects the semicircular canals and the muscles of the eyes.[3] Around 1965 Szentagothai established the exquisite correspondence between each semicircular canal and each of the three pairs of eye muscles.[4] Breinin has shown there are receptors, probably muscle spindles, in the extraocular muscles that signal their static tension.[5] The vestibular reflex stabilizes retinal images during head movements. What is called the vestibulo-ocular reflex is the product of the network of neurons that join the vestibular receptors to the muscles of the eyes. The integration of sensory inputs between the visual and the vestibular mechanisms appeared to be a crucial factor in resolving the optic nerve pathway problem for the patient described in this case report.

ACM, with its associated intra-cranial tissue tensions and changes, may produce dysfunction in all or any of these areas.[6] In this case, cranial nerve, ocular muscle, and muscle and joint receptor dysfunctions *also apparently produced dysfunction in these areas*.

Case Report

A woman with a medically diagnosed case of "retrobulbar neuritis," or "optic nerve neuritis" of the right eye, came in for applied kinesiology evaluation and treatment. This twenty-year old had "gone blind" in her right eye three weeks previously. She was frightened about this, as was her mother. She could no longer see anything out of her right eye but the outlines of large objects and peculiar colors…nothing distinct could be seen. Everyone's face had suddenly gone blank when she awakened three weeks previously. Her mother had heard about applied kinesiology chiropractic and called to ask if I could help her daughter. I explained a few things about the relationship of the cranial nerves of the eye to the moving structures of the head, and the mother brought her daughter in the next day for examination.

Three months prior to her vision loss the young woman and her mother were involved in an automobile accident. Their car had slid backwards and down into a 15-foot ditch. No real damage to the car or the passengers was perceived at the time, but the young woman had her first migraine headache after the accident. She had had two disc injuries previously in the neck, and had chiropractic reduction of the disc injury under anesthesia. A lumbar disc was also treated in this way. Her energy had been low for a number of years; it was even lower after her accident. She had also been unable to lose weight since she was put on steroids in the 5th grade due to asthma; she was perhaps twenty pounds heavier than she thought she should be. She found that the best treatment over the years for her asthma had been injections of Vitamins B, C, and magnesium.

In relation to her vision loss, an MRI was performed a week after the onset of her symptoms. Results indicated a Type I Arnold-Chiari Malformation of the brain, with a 5 mm herniation of the cerebellar tonsils into the spinal canal. Her medical doctors had never suspected this problem. She brought the radiological report with her on her first examination. Five millimeters of the lower brain were displaced into the spinal canal, or had prolapsed down into and below the foramen magnum. Often, problems arise in these cases because of compression of the lower cranial nerves. Many misdiagnoses are given for this kind of problem in these patients (e.g., psychiatric disorders, multiple sclerosis, myasthenia gravis, all kinds of medical guesswork). Her physicians had scheduled her for multiple sclerosis evaluation before she came to us for treatment. Multiple sclerosis as a possible diagnosis was dismissed after treatment with us due to the complete resolution of her problem.

The patient also had a history of spinal scoliosis, and four sinus surgeries between the ages of ten and sixteen. She broke her nose at the age of ten, and suffered from sinusitis and sinus infections frequently in childhood; these were more severe after her broken nose.

Initial examination in our office revealed a dramatic example of the "ocular lock" phenomenon, with extreme saccadic motion in both eyes. Almost every angle of her gaze had drastic disorganization in the movement of the two eyeballs. This phenomenon is called "see-saw nystagmus,"[6] and is a common finding in ACM cases.

Motion of the eyes shows one eye rising and turning in, while the other falls and turns out during movement of the eyes through the cardinal fields of vision.

Manual muscle testing revealed very strong sternocleidomastoid, anterior scalene, and deep neck flexor muscles. However, strong flexion of the head onto the chest produced definite pain in the upper cervical area. The cervical stretching test was positive, as was cervical compression.[7–8] There was pain and weakness on testing the upper trapezius muscle bilaterally. Pincer palpation was positive to the rectus capitus posterior minor muscle, suggestive of a possible myofascial dural interaction.[9–11] The right external pterygoid had positive therapy localization (TL) and challenge. A category II went with this finding, as did an underlying category I. She demonstrated a classic finding of internal and external frontal bone subluxations on opposite sides (found by challenge), as well as a lateral sphenoid strain on the left. All of these faults were thought by the doctor to be changing the shape of the orbit and the tension on the tissues between the greater and lesser wings of the sphenoid, and therefore on the nerves that pass into the eye.

Freeman Wycke's one-leg standing test had also been very imbalanced on initial examination. The patient is asked to stand on one foot in front of the doctor, to find her balance if she can, and then to close her eyes. If she loses her balance, the test is positive. Upon asking the patient to therapy localize to the cervical spine, she may immediately improve her balance.[12–13] That occurred in this patient's examination.

The gluteus maximus muscles were bilaterally weak, and an Arthrostim(TM) instrument correction of her atlas (ASLP: Palmer listing system) strengthened both muscles. She also had an oculo-basic fault on the right that was corrected. The oculo-basic finding and correction in this case correlates with the relationship found between the sacrum and pelvis and the neck and head.[13, 17–18]

Although the spinal cord usually extends only to the L1 vertebral level, the dural covering continues to line the entire extent of the vertebral canal, creating a large spinal fluid filled subarachnoid space. The spinal dura, which anchors itself firmly at the 2nd sacral segment, continues down the sacral canal to the filum terminale to blend with the periosteum on the dorsum of the coccyx. There it secures the caudal end of the spinal cord and is called the central ligament of the spinal cord. The central ligament assists in maintaining the cord in position during movements of the body.[14] Brieg states that the primary source of adverse mechanical spinal cord tension is "set up directly by virtue of its anchorage at its two extremities, namely the brain and cauda equina."[15]

Recent anatomical studies have also demonstrated that the dura mater is attached to the vertebral canal in the lumbar region. The anterior attachments are short and strong while the posterior attachments are weaker and longer. The anterior and anterolateral connective tissue bands attach to the posterior longitudinal ligament. These bands are strongest at the L5-S1 level and less strong in the upper lumbar region. The dural nerve root sheaths are also attached to the posterior longitudinal ligament anteriorly and to the periosteum of the inferior pedicle laterally.[16]

The oculo-basic and category corrections should theoretically improve the dural tension upon the coccyx, sacrum, and lumbar spine while also integrating the visual righting reflexes with the pelvic righting reflexes. "Oculo" represents the visual righting reflexes, and "basic" is the Logan Basic technique for sacral and spinal treatment.[13, 17–18]

After the corrections made on her first visit I had the patient immediately do the Freeman Wycke test again; she was totally balanced. Finger-to-finger test had also been positive (right over left by one inch), and after these corrections she tested perfect.[7, 8]

It was fascinating to systematically peel this woman's ocular lock findings away. The young woman's mother watched closely every time as I re-tested her daughter, and could see the improvement in the synchronous movement of her daughter's eyes. After each cranial correction that influenced the eyeballs function, I re-tested and found less and less saccadic motion and ocular confusion in the eyes' movement. After my last cranial correction her eyes were gliding together properly like pinballs through melted butter.

At this point the patient resumed the supine position so I could re-check her corrections. As she began to lie back she exclaimed, "*O my gosh…I can see your face!*" I looked over to her mom, standing about 10 feet further away, and asked if she could see the same things better on her also. "Yes, O my gosh, *mom*!"

I took the excited young lady and her mother to the Snellen eye chart, and found she was now seeing 20-30 through her right eye.

On her second visit two days later, and before I re-examined her, she was tested on the Snellen eye chart as 20-20, and almost 20-15. There was still a little blurriness to fine print, but every day since her first correction two days before she was seeing better and better. She said that as she picked up the toothpaste tube the morning before she was able to read the writing on it and yelled out so everyone in her home could hear it, "I can see, I can see!"

An applied kinesiology re-examination indicated an improvement in her TMJ pattern (less deviation of the jaw, less weakening of the indicator muscle during challenge and TL); the upper cervical correction was holding; extreme flexion of the head on the neck produced no pain; the same frontal fault remained but did not produce dramatic weakening on challenge; there was now only slight evidence of the ocular lock phenomenon; and the category II remained stable.

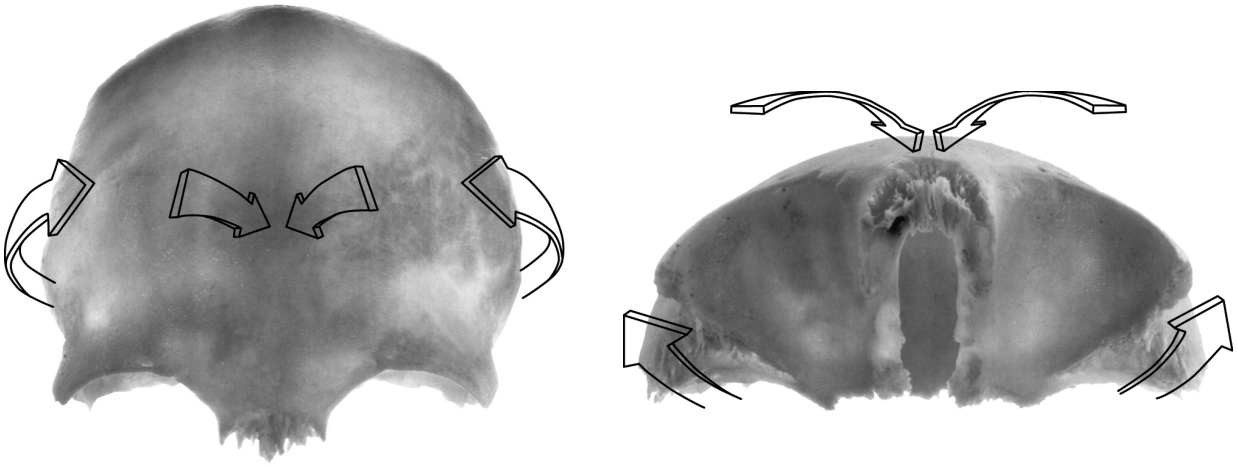
Results

The patient's vision continued to improve on follow-up visits. On her fourth visit, eight days after her initial correction, she checked at 20-13, with no more blurriness to fine print. She was also re-examined by her ophthalmologist. His report was sent to us, and concluded amusingly: "*Spontaneous improvement. No clinical or radiological evidence of M.S.*" He also wrote, "*Patient saw chiropractor who told her it was due to tightness of occipital spinal nerve or something like that. After manipulation she saw better. 7-12-02.*" If a subsequent MRI evaluation is performed, any change in the cerebellar herniation will be reported. The patient has remained asymptomatic 29 months after treatment. She also remarked that she had had pain on flexion of her chin to her chest for years, and that she never has that any more. Her asthma problems had also definitely improved since her initial treatments.

Discussion

With the numerous cranial faults found and corrected in this patient, in addition to the Arnold-Chiari malformation already present in her cranium, the physical and visual defects cited in this case may be explained. With an internal frontal cranial fault on one side and an external frontal cranial fault on the other, an obliquity of the reciprocal tension membranes (R.T.M.) will result.

Figure 1



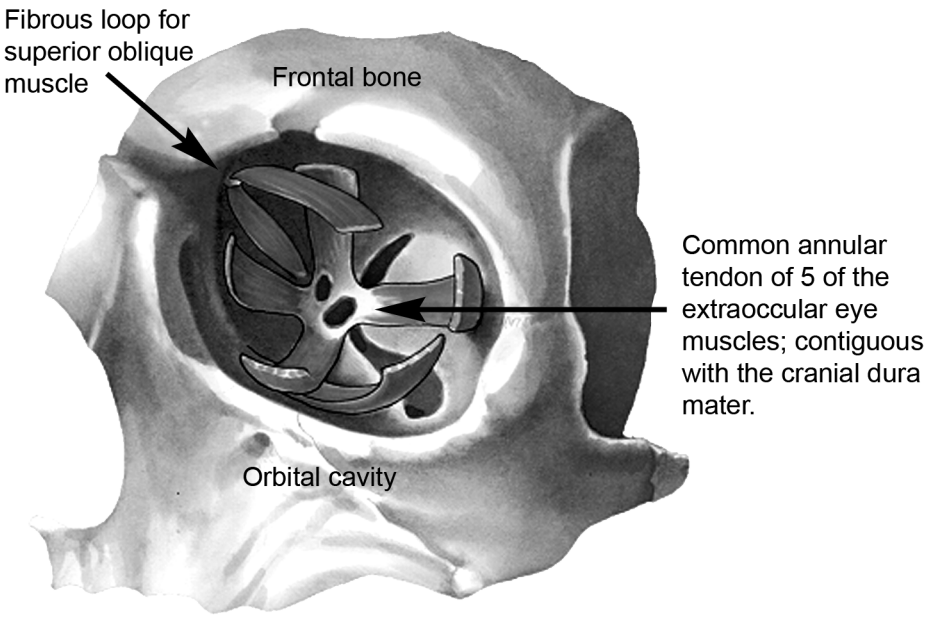
The frontal bone. Frontal cranial faults may affect the shape of the orbital cavity, the angulation of the orbital fissures, and the tension on the nerves and vessels passing into and out of the eyes. Under these conditions the cranial nerves may be subject to undesirable mechanical influences of various kinds including compression, stretch, angulation, torsion, edema, and many others. *

The anterior pole of the reciprocal tension membranes on the inside of the skull is altered. The inclination of, and tissue tensions between, the superior and inferior orbital fissures between the greater and lesser wings of the sphenoid were changed by these cranial faults. Frontal faults like these can result in imbalance in one or both superior oblique muscles; therefore ocular muscle deviation, or "ocular lock" would occur as a result of the change in the length between the origin and insertion of the eye muscles or a disturbance in its passage through the fibrous loop attached to the frontal bone.

In the literature, abnormal afferent input into the central nervous system has been linked to balance problems, as well as visual disturbances.[19–20] Traumatic injuries to the head and neck have been linked as risk factors for visual disorders in the literature.[21–23]

The anatomical relationship between the inner meningeal layer of the dura to the optic nerve is critical. The dural sleeve around the optic nerve (the dura, arachnoid, and pia mater all attach here) extends into the sclera of the eyeball and transmits tensions from the meninges to the nerves and muscles of the eyeball.[14] [Figure 2] The dural sleeve on the optic nerve contributes to the formation of the annular tendon, which is the origin for the extraocular muscles. Tension in the meninges can therefore be transmitted to the cranial nerves of the eye, the muscles of the eye, and to the eyeball itself. Ocular lock has been found most frequently to be secondary to cranial faults.[24–26, 28] Magoun, Walther, Blum, Pick, Upledger, and others have made detailed analyses demonstrating the possible effects of dural tension on the cranial nerves.[1, 24–28]

Figure 2



The sphenoid bone provides several important foramina for eye function. The optic canal through which cranial nerve II passes is vulnerable to disturbance when the sphenoid is shifted. The optic chiasm is created by the sphenoid bone, as well as the superior orbital fissure that lies between the greater and lesser wings of the sphenoid. Cranial nerves III, IV, and VI pass through this mobile opening, as well as the ophthalmic division of cranial nerve V, the frontal nerve, the lacrimal nerve, and the nasociliary nerves. *

Further disturbance to the sphenoid bone occurred due to the external pterygoid muscle dysfunction. As a result of the external pterygoid's attachment to the pterygoid plate of the sphenoid, abnormal tension here will change the position of the sphenoid bone and affect the optic foramen through which the optic nerve travels, as well as the superior orbital fissure through which the oculomotor, trochlear, ophthalmic division of the trigeminal, and abducent nerves travel.[14]

Added to this was the patient's lateral sphenoid strain and upper cervical fixation compounding the pressures on her R.T.M. The cranial physician would also recognize the probability that the four previous sinus surgeries she had between the ages of 10 and 16, as well as her broken nose, may have affected the movement, position, and tension around the foramina, fissures and nerve fibers of the optic canal. The "closed kinematic chain" of the cranium participates globally in any fault within the system.[24] With a basic understanding of the principles and anatomy involved here, one may eventually learn, in most cases, to make an accurate diagnosis of what has happened.

The cranial nerves, together with the cranial connective tissue operate as a continuum and are powerfully linked to vital organs (eyes, ears, cerebrum, cerebellum, brainstem) and pain-sensitive structures (e.g., cranial nerves and dura mater), and injury or impaired physiology or motion to these structures can make cranial nerve tissue dysfunctional.

Crisera has given a structural definition to ocular lock that fits this case perfectly.[27] The structural meaning of ocular lock is "the torsional strain on the bulbar portion of the eye and orbital cavity, secondary to inappropriate tension in one or more of the extraocular muscles…. If such a state is prolonged in time, structural changes will take place both in the cranium (Wolff's Law and the piezoelectric effect) and in the shape of the eyeball, and thereby influencing the light (photons) projected on to the retina (visual disturbances)."

Vision occurs in retinal space and is affected by the movements of the extraocular muscles of the eye. Proprioception of the eyes, neck, vestibular system and the limbs occurs in the space of the muscles, tendons, joints, and skin, all of which can be affected by abnormal biomechanics and aberrant sensory input or motor output. So sensory space is, in most cases, completely linked to motor space. The symptoms of visual disturbance can appear when the central nervous system receives conflicting messages from these differing frames of reference.

Dural tension and cranial nerve dysfunction should be suspected in any patient whose eyes are not moving in synchronicity with one another. As was the case with this patient, if synchronous movement can be restored to the eyes after correction of any faults affecting the R.T.M., dural membrane tension must be recognized as a causative factor in eye dysfunction.

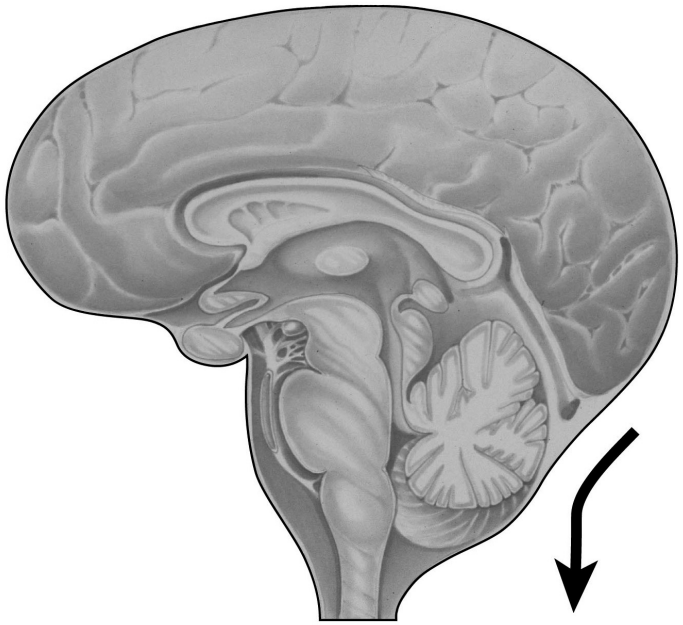
Conclusion

In this patient there were numerous cranial faults diagnosed through AK testing procedures and successfully treated. The ACM created a marked distortion in the cerebellum and tentorium, suspected to possibly affect the tension upon the cranial nerve nuclei and nerve roots to the eyes. Stress from cranial and spinal imbalance combined with ACM could be implicated in creating the physical and visual defects cited in this case. Returning the dura to a physiological range of tension by using specifically applied cranial corrections is a major goal of AK treatment.[24, 28] AK cranial evaluation and treatment seek to achieve zero defects both within and without the cranium. In 1991, Goodheart stated, "We, as a profession, are divided by the dura and its attachments. We, as a profession, can be united by an understanding of the dura and its attachments."[29]

With this specific case it was not determined whether the patient's condition was secondary to occipital influences on the visual center, related to actual cranial nerve entrapment, or myofascial dysfunction associated with the eye muscles. Further studies will need to be performed to evaluate similar patients with greater controls and concurrent neuroradiological studies. There is an anatomical relationship between the neck, skull, TMJ, intra-cranial dura mater and cranial nerves to the eye. This relationship suggests the possibility that chiropractic, and specifically AK, might warrant further study as a method of treatment and non-surgical conservative intervention for conditions related to ACM.

This has not been an attempt to cover the subject of symptomatic Arnold-Chiari malformations, but to point out an effective way of treating some of its consequences. It was also an attempt to give a practical method by which any chiropractic physician with suitable skills may discover for himself the fact that the cranial sutures do allow motion and to observe by experience the possibilities that lie ahead in the development of chiropractic techniques applied to the cranium. There are possibilities in the development of applied kinesiology's cranial therapy that should put our profession way out in front in the therapeutic world.

Figure 3



Downward pressure or cranio-dural stress upon the mid-brain and brainstem may produce tension on the cranial nerve nuclei and nerve roots in this area. In this case, treatment of cranial nerve and dural tension problems relieved the patient of the symptoms occurring in her right eye. *

In this case AK manual muscle testing was successful in helping to evaluate and monitor cranial, cranial nerve, and spinal dysfunction, which guided the treatment believed to have helped relieve this patient's symptoms and normalize her vision. This case was particularly profound in the patient's response to treatment for suspected "retrobulbar neuritis," or "optic nerve neuritis." It is always difficult to discern whether the patient's condition would have normalized without care; however, at the time of care there seemed a significant relationship between the symptomatology relief and treatment rendered. Future studies are clearly indicated with chiropractic treatment of "retrobulbar neuritis," or "optic nerve neuritis" possibly associated with ACM to determine if the results of this one study can be generalized or are isolated to this patient.

*** Images courtesy David S. Walther, DC Systems DC, Pueblo, CO, 81004**

References

1. Magoun, H. I. *Osteopathy in the Cranial Field*, Third Edition. (The Journal Printing Company, Kirksville, MO, 1976.).

2. Chusid, Joseph G. *Correlative Neuroanatomy & Functional Neurology, 19th Edition*. (Lange Medical Publications, Los Altos, CA, 1985).

3. R. Lorente de No, "Vestibulo-Ocular Reflex Arc," Archives of Neurology and Psychiatry, 1933; 30:245-291.

4. J. Szentagothai, "The Elementary Vestibulo-ocular Reflex Arc," Journal of Neurophysiology, 1950; 13:395-407.

5. Breinin,GM Electromyographic evidence for ocular muscle proprioception in man. Archives of Ophthalmology, 1957;57:176-180.

6. Brazis, Paul W., Masdeu, Joseph C., Biller, Jose. *Localization in Clinical Neurology, 2nd Edition*. (Little, Brown and Company, 1990).

7. Mazion, J. M. *Illustrated Manual of Neurological Reflexes/Signs/Tests for Office Procedure*. (Orlando: Daniels Publishing Co., 1980).

8. Evans, R.C. Illustrated Essentials in Orthopedic Physical Assessment. St. Louis, MO: Mosby; 1994.

9. Kenin S, Humphreys BK, Hubbard B, Cramer GD, "Attachments from the Spinal Dura to the Ligamentum Nuchae: Incidence, MRI Appearance, and Strength of Attachment." *Proceedings of the 2000 International Conference of Spinal Manipulation* Sep 2000: 202-4.

10. Hack, GD, Koritzer RT, Robinson WL, Hallgren RC, Greenman PE, "Anatomical relation between the rectus capitis posterior minor muscle and the dura mater" *Spine* Dec 1995; 20(23): 2484-2486.

11. Goodheart, G. J. *Applied Kinesiology 1997/98 Workshop Procedural Manual* (Privately published, 1998).

12. Ayres, A.J. *Sensory Integration and Praxis Tests*. Los Angeles: Western Psychological Services, 1989.

13. Goodheart, G. J. *Applied Kinesiology 1979 Workshop Procedure Manual* (Privately published, 1979).

14. Gray, Henry. *Anatomy of the Human Body*, 29th American edition, ed. Charles M. Goss (Philadelphia: Lea & Febinger, 1973).

15. Brieg, A. *Adverse Mechanical Tension in the Central Nervous System*. New York: John Wiley & Sons, 1978.

16. Spencer, S., et al. "Anatomy and Significance of Fixation of the Lumbosacral Nerve Roots in Sciatica," *Spine* 1983;8(6):672-679.

17. Walther, D.S., *Applied Kinesiology, Synopsis, 2nd Edition*. (Systems DC, Pueblo, CO, 2000).

18. Coggins, W.N. *Basic Technique: A System of Body Mechanics*. ELCO Publishing Company, 1983.

19. Heikkila HV, Wengren BI. Cervicocephalic kinesthetic sensibility, active range of motion, and oculomotor function in patients with whiplash injury. Arch Phys Med Rehabil 1998;79(9):1089-94.

20. Gimse R, Tjell C, Bjorgen IA, Saunte C. Disturbed eye movements after whiplash due to injuries to the posture control system. J Clin Exp Neurophychol 1996;18(2):178-86.

21. Parker, RS, Rosenblum, A. I.Q. loss and emotional dysfunctions after mild head injury incurred in a motor vehicle accident. J. Clin. Psychol. 1996 Jan; 52(1):32-43.

22. Kischka U, Ettlin, T Heim S. Cerebral symptoms following whiplash injury. Eur Neurol 1991; 31(3):136-40.

23. Bertucci, D, Aggugia M, Bolamperti L. Chronic post-traumatic headache associated with minor cranial trauma: a description of cephalgic patterns. Ital J Neurol Sci 1998 Feb;19(1):20-24.

24. Walther DS, *Applied Kinesiology Vol. II, Head, Neck, and Jaw Pain and Dysfunction—The Stomatognathic System*. Pueblo, CO, 81004: Systems D.C., 1983.

25. Blum, CL, "Cranial Therapeutic Approach to Cranial Nerve Entrapment Part I: Cranial Nerves III, IV, and VI" *ACA Journal of Chiropractic* Jul 1988;22(7): 63-7.

26. Upledger J.E., *Craniosacral Therapy II: Beyond the Dura* (Seattle, Washington, Eastland Press, 1987) pg.115-30.

27. Crisera, P. *Cranio Sacral Energetics, Vol. 1*. (Rome, Italy: Privately published, 1997).

28. Goodheart, G.J. *Applied Kinesiology Workshop Procedure Manuals*. (Detroit, MI: Privately published, 1982).

29. Goodheart, GJ. *Being a Family Doctor*, videotaped seminar, 1991.

The Temporal Bone Cranial Fault: A Resume of 'The Trouble Maker' with a Focus on the Proprioceptive and Vestibulo-Ocular Syndromes

Scott Cuthbert, D.C.

Abstract

For many years now I have been fascinated with the temporal bone. Several aspects of my appreciation and understanding of this bone will be discussed in this paper. Its complexity, its beauty, its diverse functioning, and its importance have become more apparent to me as the years have gone by.

Key Indexing Terms: Temporal bone, cranial fault, applied kinesiology.

Introduction

Soon after beginning my own practice in the clinic of applied kinesiology's Voltaire, our own movement's Encyclopedist *extraordinaire* (Dr. David Walther), I was told by the master that when he corrected inspiration and expiration assist cranial faults of the temporal bones, he would simultaneously correct many other cranial patterns found by AK examination. He said it also frequently eliminated KI-27 testing in a majority of patients. I have found this to be the case also after five years of consistent study and treatment.

Discussion

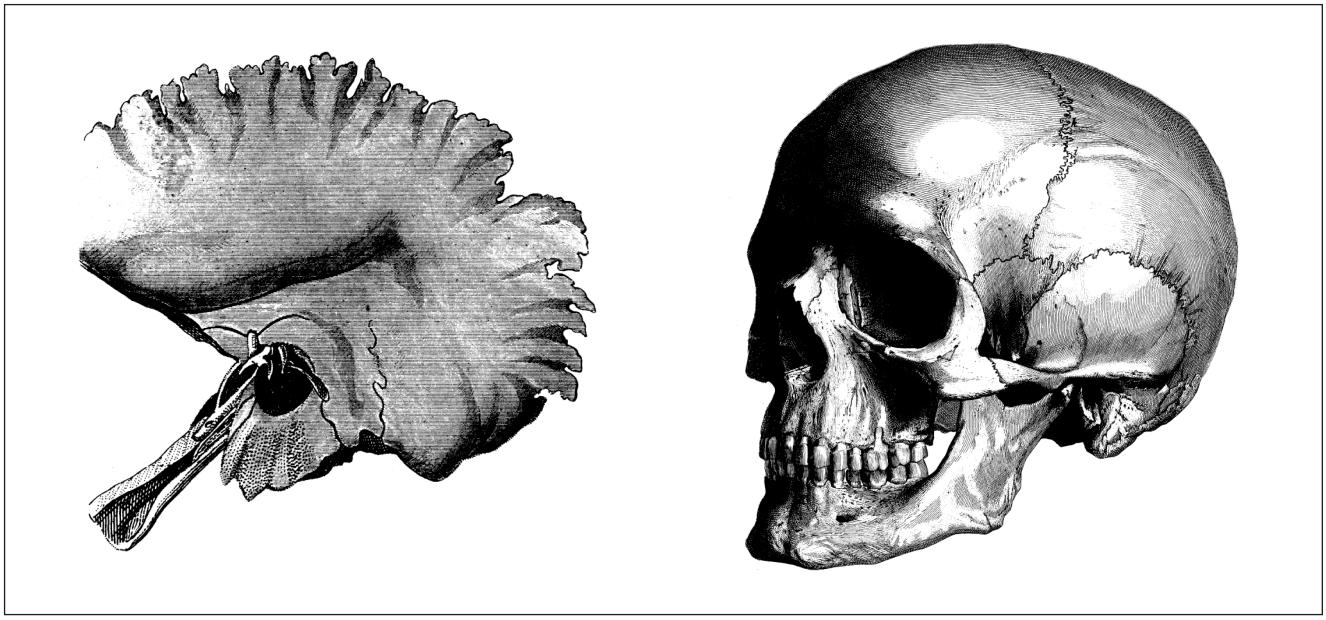
A lucid happiness becomes the portion of the physician who begins to discover his natural gifts in the work he does by the light of an incessantly active meditation and, thanks to it, one that frees him from the dross and the foreign deposits that hide his passion from him. Thus for me in my work with patients, I frequently find that the temporal bone plays some part in my patients' problems.

In reviewing about sixty-five recent case records I have found that temporal bone cranial faults have been present in the following widely differing patient conditions: dizziness, syncope, tension headache, migraine headache, cluster headache, tinnitus, TMJ problems of widely varying symptom type, earache, Bell's palsy, visual disturbances, conjunctivitis, lacrimal duct disturbance, nystagmus, muscular imbalance of the eyes or ocular lock, loss of visual acuity when measured on the Snellen eye chart, sinusitis, scalp pains, shoulder muscle pain and weakness, ischial tuberosity pain, sacro-sciatic notch tenderness, spinal muscle pain and weakness occurring literally anywhere in the patient when tested, hiatal hernia, esophageal spasm, heartburn, duodenal pain, digestive syndromes and lower bowel problems.

A single case does not prove much but under certain circumstances it may lead to some worthwhile discovery. In the cases that I treat in the clinic, these individual cases and the involvement of the temporal bone suggest how endemic and consistent this cranial fault is in the sick people I treat.

I like to remember, when reciting anecdotal reports of chiropractic's success in the treatment of sick patients, what Sir James Padget said: "Receiving thankfully all that physiology or chemistry or any other science can

give us, let us hold that, that alone is true, which is proven clinically and that which is clinically proved needs no other evidence."



The Structural Beauty of the Temporal Bone

We are poor indeed if, after many years of working with it, we do not learn how to see in the bones of the skull not only an admirably ordered landscape with its valleys and hills, its inner movements, its geological unity and its rhythm, but also a perfect piece of sculpture with its asymmetrical balance, its silent planes, its tapering lines, its expressive reliefs, its sinuous and pure profiles. The gulfs, promontories, and estuaries of the temporal bone seem embraced by the blood and "that highest known element in the human body," that fluid that washes away metabolic gravel and neurochemical sand, that fluid that is indefatigably persistent in striking, caressing, falling, or dripping through the surfaces of the majestic temporal bone.

If we do not apply an ardent curiosity to the study of the architecture and precise shapes of the temporal bone, to the muscles overlying the bone, to the sutures that fit it precisely into the skull, to the nerves that invest its matrix of forms, to the network of veins and arteries within the bone…will our work with the temporal bone ever quiver with that animation, at once distinct and mysterious, which causes the living surfaces and dynamics of the bone to vibrate as if under the continuous caress of the influx of the nerves and blood, lymph and CSF, and our own perception of innate intelligence manifesting itself?

In studying the temporal bone, see how the petrous portion looks like a greedy hand that reaches out to clutch its prey in order to feed on it. The petrous tip of the temporal bone is anchored into the posterolateral corner of the body of the sphenoid at the posterior clinoid process by the petroclinoid or apical ligament (usually, but not always present), like a ballerina-on-points. The petrous portion of the temporal is like an anteromedially pointed anvil on which the cranial base is suspended in toward the sphenoid bone at a 45- to 60-degree angle. The zygomatic arch is like a wing or a flame reaching out from the temporal. See the harmonious construction of all its parts, the ends of the bone slipping into its soft, padded, and lubricated sockets, ridges, and bevelings. See the way the bone's levers are moved by the pull of dura or turned by the play of muscles. See the close overlapping and the undercuts of the sutural surfaces, with the amphora of the squamous portion to contain the weight of the brain. There is a universal architecture built into the human skull that borrows its most forceful poetry from functional logic. If we study the harmonious geometry and architecture of any cranial bone long enough, the morphological formula of the skull will no doubt arise for us someday.

The architectural structure of the temporal bones competes with the sphenoid in complexity. They form the most impressive lateral structure of the cranium, and present highly variable and complex sutures. Because of its wedged shape, the temporal bone acts as a buttress for the head. It has a flying buttress formation that joins it with the zygoma. It has a rhino's-horn-shaped mastoid that is formed by the constant pulling of the sternocleidomastoid muscle as the infant raises their head. An infant who develops normally can hold his head up unassisted by six months, at which point the mastoids have ossified and been given their particular shape by the action of the SCM. The shape of the temporal's mastoid process feels like it was specifically designed to fit into the overlapping hand of the manipulative physician.

The temporals are functionally married to the sphenoid and mandible, and their connection to the tentorium is immediate and extensive. Temporal bone cranial faults always involve the tentorium and therefore influence other bones that attach to the tentorium – the sphenoid, occiput, and the other temporal bone. The pharyngeal muscles are suspended from the inferior aspect of the petrous portion. The core link means that the sacrum and ilia may also be affected, or be affecting the temporals. Muscles and joints in the lateral portions of the body, like the shoulders and hips, may echo the temporal bones' condition. M. L. Rees has written extensively about the separate portions and different treatment methods to be applied to the mastoid processes alone.[1]

Muscular and ligamentous attachments at the styloid and mastoid add potentially disturbing forces to the temporal bone. The muscles of mastication and the occlusion of the teeth both affect the temporal, especially the temporalis muscle. The temporomandibular joint, lateral pterygoid muscles, retrodiscal ligaments, muscles attaching the hyoid to the temporal bone, sphenotemporal ligaments, stylomandibular ligaments, and the most power neck muscles of all, the sternocleidomastoid, all affect the temporal bones.

The Temporal Bone Cranial Fault: The necessary integration of the vestibular mechanism with the visual righting mechanism

Watch a seagull or swan flying, or a cheetah or giraffe or ostrich running, or your own dog or cat walking along. They keep their heads held horizontally, such that the planes of the horizontal semicircular canals remain perpendicular to gravity. This constancy of position in relation to gravity makes the head a stabilized platform, which considerably simplifies the processing and fusion of vestibular, visual, and kinesthetic information as well as their coordination.

All mammals have three semicircular canals on each side of their heads. The canals are situated in three approximately perpendicular planes, though not the ones you might guess. To locate it in humans, just look at a person from the side and plot a line from the meatus of the ear to the external edge of the eye. The horizontal canal is in a plane that overhangs this line by 20 degrees. The two remaining planes of the canals are at 45 degrees with respect to the frontal and sagittal planes of the body.[2–4]

Figure 2

Photographs of a man running (drawn after DuCroquet). The visual righting, labyrinthine, and head-on-neck reflexes are responsible for evaluating and maintaining the body's orientation in space. The nervous system constantly attempts to keep the head held horizontally during movement, so that the plane of the horizontal semicircular canals remains perpendicular to gravity. The angular geometry of the semicircular canals helps coordinate the organization of the cerebral analysis of visual movement with the inputs from the eyes, joints and muscles, and other proprioceptors throughout the body. The line drawn indicates roughly the plane of the horizontal semicircular canal.

Why is this anatomical configuration important in our patients with symptoms? Because the three planes of the semicircular canals form a basic frame of reference to which our entire perception of movement through space is organized. ***The geometry of the canals*** dictates the organization of the cerebral analysis of visual movement and perhaps also many other movements. This perception of space may even prove to be the basis for Euclidean geometry.[5]

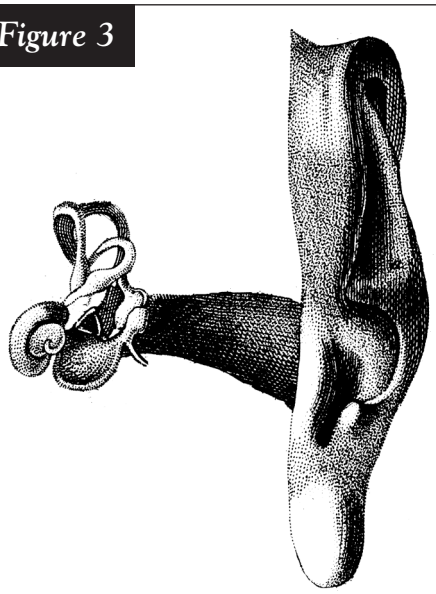
Now the labyrinthine and visual righting reflexes may be disturbed by joint or muscle problems in the neck, as well as by cranial faults that may result from either whiplash dynamics or a blow to the head during an automobile accident or other head and neck trauma.[6–7, 10–13]

The head-on-neck reflexes come from the equilibrium proprioceptors located in the upper cervical ligaments, and these also can be disturbed by subluxations or fixations of these vertebrae after trauma.[8–9]

During trauma mechanoreceptors throughout the body may be subjected to tensile stress and deformation, and trauma may increase dural tension throughout the craniosacral system.[14] Abnormal dural tension may impair the movement and elasticity of the CNS itself, causing symptoms to then arise from its own tissues.[15]

In the middle of the petrous portion of the temporal bone is the acoustical vestibular organ. This is an organ so delicate that it can detect a movement of the stereocilia on the hair cells the distance of the diameter of a hydrogen atom.[4]

Figure 3



The geometry of the semicircular canals is dependent upon the geometry of the petrous portion of the temporal bone.

The forces of trauma affect the membranous labyrinth within the temporal bones of the skull. Acceleration and deceleration forces following collision may activate in a violent and intense way the ciliated cells of the semicircular canals and the matrices of the otolithic organs.[16] The forces of acceleration and deceleration can "deprogram" the sensory cells of the membranous labyrinth. The endolymph no longer properly stimulates the sensory cells situated at the level of the crista ampullaris, semicircular canals, and membranes of the saccule and utricule. When the trauma is violent, some ciliated cells may be partially destroyed. More commonly, they are stunned to the point of no longer transforming body movements into accurate sensory signals. In some cases they become hyper-reactive and generate too many signals, such that the over-stimulated cerebellum can no longer provide reliable information to the body. ***Movement of the endolymph and the geometry of the stereocilia themselves are determined by the movement and positioning of the temporal bone in relation to the earth's gravitational field.***

Anti-gravitational muscle function, muscle tone, and posture are not properly maintained; consequently, the vestibular nuclei do not receive proper sensory input for decoding and distribution to the body, particularly in regard to cerebellar control. The cerebellum normally integrates labyrinthine, visual, and kinesthetic information that is used to provide motor output for correct balance.[16–17] In post-traumatic syndromes, some of the sensory inputs no longer yield proper responses.[18] Freeman-Wycke's one-leg standing, Hautant's, Romberg's and other cerebellar tests are frequently positive in these patients.

Because of incorrect activation and poor responses from the cerebellum after trauma, the patient has numerous symptoms found also in disturbed proprioceptive syndromes: loss of balance, vertigo, nausea, motion sickness, poor depth perception, headaches, vertebral pain, and poor visual accommodation.[6, 16, 19–25]

From this discussion it may be reasonable to assume that trauma may affect the functionality of the eyes, inner ears, muscles and joints. When any one of these sensory organs is dysfunctional, sensory conflict (dysponesis) may result. The importance of correcting the vestibulo-ocular, vestibulo-spinal, and opto-kinetic reflexes in the examination and treatment of patients with equilibrium disorders will be discussed next.

Eliminating disorganization between the equilibrium proprioceptors and the reflexes discussed in the next section is paramount to obtaining optimal results in patients who have persisting equilibrium syndromes after trauma. Failure to recognize this problem and correct it is often the reason that a post-traumatic equilibrium disorder or other vestibulo-ocular problem persists for years after an injury.

The Temporal Bone Cranial Fault's relevance to the vestibulo-ocular, vestibulo-spinal, and opto-kinetic reflexes

The visual righting, labyrinthine, and head-on-neck reflexes are responsible for evaluating and maintaining the body's orientation in space.[26] In 1948 Spector described the failure in the mechanisms of multi-sensory integration as the underlying cause of some types of vertigo and nausea, but he provided no therapeutic approach for correction.[27] ***If all three reflexes*** are not providing the same information about the body's orientation, this neurologic disorganization produces the misdirected effort known as dysponesia (sensory conflict).[28] This can happen when one reflex indicates the head is level and another that it is tilted. It also occurs when the body detects motion that the eyes cannot detect.

The combination of an upper cervical subluxation or fixation, a temporal bone cranial fault, and inhibition of muscles in the neck is frequently found in the clinic to produce this kind of problem.

It is well known in neurology that pathways descending from the vestibular sensors and flowing through the vestibular nuclei ensure postural reactions that keep the body upright, stable, and in balance. They also initiate compensatory reflexes during tilting, bouncing, braking, and other movements of the body. These are the so-called vestibulo-spinal reflexes. In principle, they are well identified and depend on neural networks that connect each semicircular canal with the postural muscles and the muscles of the neck.

Another major mechanism that allows us to follow the landscape while we are moving is a very old reflex action, the opto-kinetic reflex. The nerve pathway that processes these signals also influences the centers that control posture.

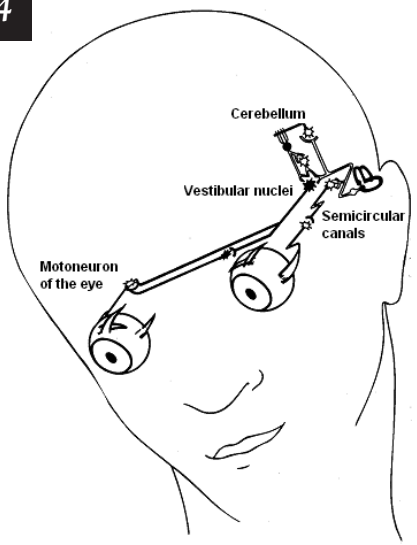
What is quite noteworthy about the opto-kinetic reflex to the hypothesis in this paper is that all along this pathway neurons respond to visual movements in preferred directions ***aligned along the planes of the semicircular canals of the vestibular system.***[29]

Around 1930 it was discovered that a network of neurons in the brainstem connects the semicircular canals and the muscles of the eyes.[30] Around 1965 Szentagothai established the exquisite correspondence between each semicircular canal and each of the three pairs of eye muscles.[31] Breinin has shown that there are receptors, probably muscle spindles, in the extraocular muscles that signal their static tension.[32] The integration of sensory inputs between the visual and the vestibular mechanisms appears to be a crucial factor in resolving many cases of proprioceptive disturbances in our patients.

The geometrical correspondence between the visual system and the semicircular canals extends to the motor system. ***The plane of action of the three pairs of extraocular eye muscles, which enable rotation of the***

eyeball, is approximately parallel to the planes of the semicircular canals.[33] The angular geometry of the semicircular canals is thus basic to the perception and control of movement.

Figure 4



The vestibulo-ocular reflex stabilizes retinal images during head movements. What is called the vestibulo-ocular reflex is the product of the synergism of neurons that join the vestibular receptors to the muscles of the eye. A common cause of sensory conflict in patients with proprioceptive syndromes is different signaling coming from the visual righting, labyrinthine, and head-on-neck reflexes. If these separate reflex mechanisms are not providing the same information about the body's orientation and movement through space, this sensory conflict produces the misdirected effort known as dysponesia.

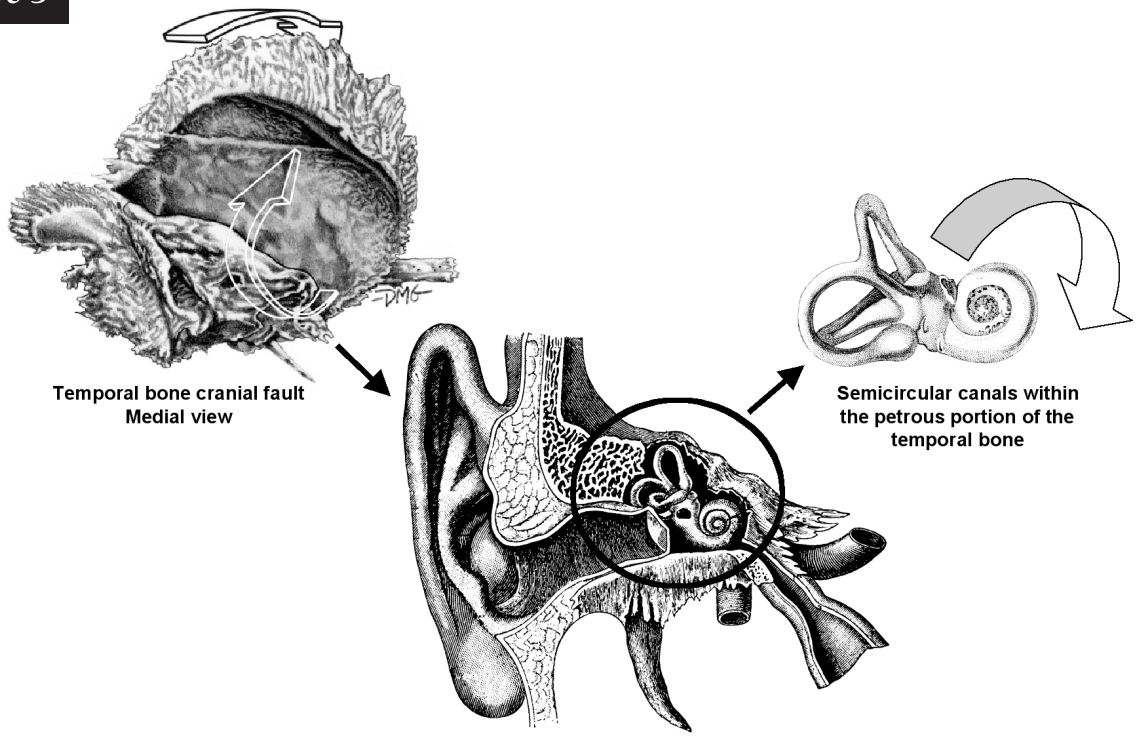
The active movement of the eyes detected by the receptors in the extraocular muscles influences the neurons of the visual cortex and leads to a preference in these cells for given lines of orientation. In this way, projection of the proprioceptors of the eye (and of the neck) on the neurons of the visual cortex contributes to their directionality. It suggests as well that ***the muscles of the eye constitute a frame of reference with the same capacity as the semicircular canals***.[4, 25, 34]

The importance of specifically testing the muscles of the eye becomes apparent in this context. It seems likely that the proprioceptors of the eye muscles contribute at least as much as other muscle receptors to the total integrative/afferent process and that the sensory input that acts upon those receptors is accordingly important. *Ocular lock testing* can evaluate just this factor in the patient's sensory ensemble.

Because the planes of action of the eye muscles are close to those of the semicircular canals, it is difficult in a particular patient to know which one is producing the problem. Specific testing of these separate but integrated mechanisms will have to be carried out on the patient.

With temporal bone cranial faults or malpositions, an obliquity of the reciprocal tension membranes (RTM) may result, and may change the angulation of the semicircular canals in relationship to the earth's gravitational field.[10–11, 35, 36–54] The RTM consists of the periosteal attachments of the meningeal dural membrane of the falx cerebri and cerebelli, tentorium cerebelli, and the diaphragma sella. The lateral attachments of the tentorium cerebelli on the internal aspect of the skull can be modified by temporal bone cranial faults. In the clinic the temporal bones are frequently found in counter-rotation, producing discordant integration once again from the vestibular sensory inputs coming from each side of the head.[35, 50–51] The tissue and fluid tensions inside the vestibular mechanism within the petrous portion of the temporal bone, as well as its geometrical inclination on either side of the head, can therefore be changed by cranial faults like these.

Figure 5



Temporal bone cranial faults may change the angulation of the semicircular canals and the movement of fluids through the vestibular mechanism. They may also change the shape of and tension within the jugular foramina, as well as the drainage of blood from the head through the jugular vein. They may also decrease the elasticity of the tentorium cerebelli and increase the tension on the cranial membranes throughout the skull. This distortion may be transmitted to all the other bones of the skull. The cranial nerves are ensheathed by dura mater and pia mater membranes that must remain supple; otherwise they may cause mechanical deformation and abnormal tension on the nerve bundles.[10–11, 36–54]

The measurable amplitude of cranial bone motion ranges between 40 microns to 1.5 millimeters, according to differing authorities.[44–50] When temporal faults are present in the patient, there are frequently visible and tactile signs of the faults. One mastoid process will be closer to the table than the other, and one mastoid process will be more prominent on one side and more depressed on the other.

Whenever a symptom in the patient suggests the temporal bone, a dependable diagnostic modality is available in applied kinesiology. The diagnostic method used to discover a temporal bone cranial fault in applied kinesiology is termed a "cranial challenge" or "therapy localization." The challenge can be described as follows. In Magoun's text, *Osteopathy in the Cranial Field*, 3rd Edition, in the chapter called "Principles of Treatment," there are five different methods described for "securing the point of balanced membranous tension which must be followed to secure the best results."

"A. EXAGGERATION. This is the ordinary procedure for the usual case and is employed when not contraindicated…To employ this method, increase the abnormal relationship at the (cranial) joint by moving the articulation slightly in the direction towards which it was lesioned…. To do this with the two members of an articulation augments the chance of securing a reduction because of the increased resilience of the membranes."

In the AK rebound challenge procedure, which employs Magoun's exaggeration procedure, the physician momentarily increases the fault pattern of a single bone or group of bones with the intention that this vector of force placed into the skull will cause a temporary increase in the tension of the reciprocal tension membranes of the cranio-sacral system. If this vector of force increases the RTM tension, it will produce a momentary lowering of the overall muscle tone of an indicator muscle.

The correction pressure is sustained through several respiratory cycles using the same vector as the challenge. This allows the R.T.M. to accumulate enough energy or tension to free itself and spring back, or rebound, into the correct relationship. Magoun calls this the exaggeration correction; this is the preferred method of correction in chiropractic, osteopathy, and "cranio-sacral" schools of teaching. This rebound correction is assisted by the patient's own inherent breathing, which further induces the membranes into correction.

With applied kinesiology challenge procedures we are guided into the proper vectors of force to be imparted to this architecturally impressive but neurologically delicate bone. However, we must be able to feel the movement occurring in the bone as we correct it, feeling both for the sutural response and for the synchronization of the dura. We are introducing a force designed to lubricate and then free the last threads of adherence or imbalance in the temporal's structural interrelationship with the other cranial bones and with the tentorium itself during an AK cranial correction.

The Temporal Fault and cranial nerve involvement

Physicians who perform neurological evaluations have numerous signs and symptoms to look for in patients with cranial nerve problems. Applied kinesiology has added a significant number of functional neurological tests that indicate cranial nerve involvement in our patients.

Cranial nerves 3, 4, 5, 6, 7, 8, 9, 10, and 11 all have relationships with the temporal bone. The nuclei of all the motor branches of the cranial nerves are located in the area between and below the occiput and the temporal bones, i.e., the brainstem. These are all true peripheral nerves in the sense that they synapse external to the central nervous system. In addition to the head, the cranial nerves also have significant functions in the neck, chest, and abdomen. Every time we change any relationship between the occiput and temporals, especially affecting the tentorium cerebelli, we affect the tissue tension around all of these nerves. This is particularly important if we think of the far-reaching influences of the vagus nerve and when we think of the frequency with which vestibular and auditory (cranial nerve VIII), neck (cranial nerve XI), and digestive problems (cranial nerve X) are encountered in clinical practice.

(The importance of the cerebellum, between and below the two temporals, can hardly be over-emphasized.)

The jugular foramen is the intervertebral foramina between the occiput and the temporal bone. The foramen contains (1) the jugular vein, (2) the vagus nerve, (3) the spinal accessory, and (4) the glossopharyngeal nerve. Dr. Goodheart describes it as though there are four fingers coming out of your jacket's sleeve.

The border of the temporal with the occiput, the occipito-mastoid suture, is an elongated crevice that remains open throughout life. The open architecture of the jugular and petrous portions makes the cranial base portion of the temporals, as well as the occiput and sphenoid, susceptible to subluxation. There can be foraminal encroachment here because of dural sleeve occlusion.

This may produce vagal signs and symptoms **(S/S)**, glossopharyngeal **(S/S)**, and spinal accessory **(S/S)**, and temperature differences in the face and neck. The diagnostic signs of cranial nerve entrapment at the jugular foramina, according to Dr. Goodheart, are as follows**:**[55]

Cranial Nerve IX (S/S):
- Uvula deviated to one side; swallowing problems.
- Upper pharynx muscles innervated by the glossopharyngeal nerve. Patients don't complain of it.
- Very few uvula hang down in the center. Uvula deviation will be found frequently if we look for it. Patients can't taste bitterness on the tongue…posterior 2/3 of tongue innervated by glossopharyngeal nerve.

Cranial Nerve X (S/S):
- Cardiac rate, rebound tenderness of the abdomen; digestion. Child not gaining weight properly. Spastic pyloric valve; vomiting. Common in children 2.5 years and up. Not so common in younger children.
- Check to see if one foot turns in better than the other.
- Check tone of abdomen between one side and another. Usually will be much tighter on one side than another.
- Both will improve after correction of universal cranial fault or jugular foramen decompression technique. Frequently, compression of ventricle 4 (CV-4) is also needed.
- It should be remembered that faults producing **(S/S)** attributable to cranial nerve X would likely be producing signs in cranial nerve IX because of their intimate association. The nuclei of both cranial nerves are in the floor of the 4th ventricle, and the ganglia are in the same jugular foramen.

Cranial Nerve XI (S/S):
- SCM, upper trapezius, or splenius capitus tension or weakness.
- AK testing of the SCM and upper trapezius and splenius capitus muscles offers cranial technicians one of the most immediately available and accurate windows on the cranial nerve system in the therapeutic world today. Cranial faults producing irritation to cranial nerve XI will frequently be affecting other cranial nerves when tested.
- Cranial Nerve XI and the entire cranial nerve system are so intimately related as to be inseparable due to dysfunctions at the jugular foramen.
- Whenever cranial nerve XI dysfunction is found, a complete evaluation of all the cranial nerves should be made. Because cranial nerves usually carry dural and pia mater sleeves with them, it is critical to evaluate all of them when dural tension is discovered in a patient. When this is done and treatment is given to all the faults found, patients may experience undreamed of improvements in their clinical pictures.

Jugular vein (S/S):
- Since 90–95% of the venous blood exits the brain at the jugular foramen, you will feel one side of the face to be of a different temperature than the other side. Measure the patient's facial temperature… usually one side will be warmer than the other. Thermistor, hand, or any other heat measuring devise can be used.

Conclusion

The evidence presented in this article allows the vertebral subluxation, temporal bone cranial fault, and equilibrium syndromes to be associated via sensory conflict and dysponesis. It is my hope that illustrating this relationship will lead to future investigation by a variety of researchers to further document the relationship between vertebral subluxations, cranial faults, abnormal muscle tone, equilibrium disturbances, sensory conflict, and other health problems.

The hypothesis derived from clinical and anatomic observations is that the nervous system operating within the disturbed geometric forms and mechanisms of patients with temporal bone cranial faults will not operate properly. Anatomical distortions of the geometry of the eyes or the vestibular mechanism within the temporal bones (producing a tilt in the angular geometry of the semicircular canals) may create discordant sensory input into the CNS compared to that coming into it from the spinal joints and muscles, thereby creating sensory conflict and producing poor stability and deficient motor activity. This lays the groundwork for pain and numerous proprioceptive syndromes. Thus the examination and treatment of the cranial mechanism, along with other chiropractic procedures that improve proprioceptive signaling throughout the body, assume a new significance and even greater importance than was formerly appreciated.

References

1. Rees, M.L. *The Art and Practice of Chiropractic*, International Systemic Health Organization, Inc. Third Edition, 1994.

2. J. Szentagothai, "The Elementary Vestibulo-ocular Reflex Arc," Journal of Neurophysiology, 1950; 13:395-407.

3. Eccles, J. C. Brain Mechanisms and Learning. Oxford: Blackwell Scientific Publications; 1961.

4. Parber, DE. The vestibular apparatus. Scientific American, 1980; 243:118-121.

5. Poincare, H. The Value of Science, trans. George Bruce Halsted. New York: Science Press;1907. p. 73.

6. Heikkila HV, Wengren BI. Cervicocephalic kinesthetic sensibility, active range of motion, and oculomotor function in patients with whiplash injury. Arch Phys Med Rehabil 1998; 79(9):1089-94.

7. Gimse R, Tjell C, Bjorgen IA, Saunte C. Disturbed eye movements after whiplash due to injuries to the posture control system. J Clin Exp Neurophychol 1996; 18(2):178-86.

8. Schieppati M, Nardone A, and Schmid M. Neck muscle fatigue affects postural control in man. Neuroscience 2003; 121(2):277-285.

9. Holm, S., Indahl, A., Solomonow, M. Sensorimotor control of the spine, Journal of Electromyography and Kinesiology, 12;3:219-234.

10. An annotated bibliography of over 250 papers relating to cranial manipulative therapy. Retzlaff, E.W., Mitchell, F.W. The cranium and its sutures. Berlin: Springer-Verlag; 1987. p. 68.

11. A list of over 400 papers related to the cranial concept, and over 30 books explaining this therapeutic modality. Friedman, H.D., Gilliar, W.G., Glassman, J.H. Osteopathic Manipulative Medicine Approaches to the Primary Respiratory Mechanism, San Francisco International Manual Medicine Society; 2000. p. 221-253.

12. Caffey, J. The whiplash shaken infant syndrome: manual shaking by the extremities with whiplash-induced intracranial and intraocular bleedings, linked with residual permanent brain damage and mental retardation. Pediatrics, 1974;54(4):396-403.

13. Magoun H, Entrapment neuropathy in the cranium. Journal of the American Osteopathic Association, Mar 1968; 67:779-87.

14. Butler, DS. Mobilisation of the Nervous System. London: Churchill-Livingstone; 1991.

15. Breig, A. Adverse mechanical tension in the central nervous system. Analysis of cause and effect. Relief by functional neurosurgery. New York: John Wiley and sons; 1978.

16. Berthoz, A. The Brain's Sense of Movement. Cambridge, MA: Harvard University Press; 2000.

17. Sherrington, Charles S. The Integrative Action of the Nervous System. New Haven: Yale University Press; 1906.

18. Lund, J.P., et al. The pain-adaptation model: a discussion of the relationship between chronic musculoskeletal pain and motor activity. Canadian Journal of Physiology and Pharmacology, 1991; 69:683-694.

19. Treleaven J, Jull G, and Sterling M. Dizziness and unsteadiness following whiplash injury: characteristic features and relationship with cervical joint position error. J Rehabil Med 2003; 35:3643.

20. Brandt T, Bronstein AM. Cervical Vertigo. J Neurol Neurosurg Psychiatry 2001; 71(1):8-12.

21. Humpfreys KM, Bolton J, Peterson C, Wood A. A cross-sectional study of the association between pain and disability in neck pain patients with dizziness of suspected cervical origin. J Whiplash & Related Disorders. 2002; 1(2):63-73.

22. Galm R, Rittmeister M, and Schmitt E. Vertigo in patients with cervical spine dysfunction. Eur Spine Journal. 1998; 7:55-58.

23. Heikkila HV, Wengren BI. Cervicocephalic kinesthetic sensibility, active range of motion, and oculomotor function in patients with whiplash injury. Arch Phys Med Rehabil 1998; 79(9):1089-94.

24. Gimse R, Tjell C, Bjorgen IA, Saunte C. Disturbed eye movements after whiplash due to injuries to the posture control system. J Clin Exp Neurophychol 1996; 18(2):178-86.

25. D. G. MacKay, The Organization of Perception and Action. New York: Springer; 1987. p. 7.

26. McCouch, G.P.; Deering, I.D.; Ling, T.H. "Location of receptors for tonic neck reflexes." J. Neurophysiol May 1951; 14.

27. Spector, B. "Neuroanatomic mechanisms underlying vertigo and nausea." Bull N Engl Med Center August 1948; 10(4).

28. Whatmore, G.B.; Kohli, D.R. The Physiopathology and Treatment of Functional Disorders. New York: Grune & Stratton, Inc.; 1974.

29. J. Simpson, "The Accessory Optic Systems," Annual Review of Neuroscience, 1984; 7:13-14.

30. R. Lorente de No, "Vestibulo-Ocular Reflex Arc," Archives of Neurology and Psychiatry, 1933; 30:245-291.

31. J. Szentagothai, "The Elementary Vestibulo-ocular Reflex Arc," Journal of Neurophysiology, 1950; 13:395-407.

32. Breinin,GM Electromyographic evidence for ocular muscle proprioception in man. Archives of Ophthalmology, 1957; 57:176-180.

33. C. De Eaele, W. Graf, A. Berthoz, and F. Clarac, "Vestibular Control of Skeletal Geometry," *Posture and Gait*. Elsevier, Amsterdam, 1988: 423-432.

34. J. I. Simpson and W. Graf, "The Selection of Reference Frames by Nature and Its Investigators," Adaptative Mechanisms in Gaze Control: 3-20.

35. Walther DS, Applied Kinesiology Vol. II, Head, Neck, and Jaw Pain and Dysfunction— The Stomatognathic System. Pueblo, CO: Systems D.C.; 1983.

36. DeJarnette MB, Cranial Technique, Nebraska City, Nebraska: self-published; 1979–1980.

37. Pederick FO, A Kaminski-type evaluation of cranial adjusting Chiropractic Technique. 1997;9(1):1-15.

38. Pederick FO, For Debate: Cranial Adjusting — An Overview. Chiropractic Journal of Australia, 1993; 23(3):106-12.

39. Blum, CL, The Effect of Movement, Stress and Mechanoelectric Activity Within the Cranial Matrix International Journal of Orthodontics, 1987; 25(1-2): 6-14.

40. Blum, CL, Biodynamics of the Cranium: A Survey. The Journal of Craniomandibular Practice, Mar/May 1985:3(2):164-71.

41. Peterson, K.; A Review of Cranial Mobility, Sacral Mobility, and Cerebrospinal Fluid. Journal of the Australian Chiropractic Association, Apr 1982; 12(3):7-14

42. Pederick FO, Developments in the Cranial Field. Chiropractic Journal of Australia Mar 2000; 30(1):13-23.

43. Magoun HL, Osteopathy in the Cranial Field, 3rd Edition, Meridian, Idaho: Sutherland Cranial Teaching Foundation; 1976. p.100.

44. Pick, M. A preliminary single case magnetic resonance imaging investigation into maxillary frontal-parietal manipulation and its short-term effect upon the intercranial structures of an adult human brain. Journal of Manipulative and Physiological Therapeutics, 1994; 17(3).

45. Baker, EG. Alteration in the width of maxillary arch and its relation to sutural movement of cranial bones. J Am Osteopath Assoc, 1971;70:559-564.

46. Heifitz, MD, Weiss M. Detection of skull expansion with increased cranial pressure. J Neurosurg, 1981; 55:811-812.

47. Heisey, SR, Adams, T. Role of cranial bone mobility in cranial compliance. Neurosurgery, 1993; 33(5):869-876.

48. Lewandoski, MA, Drasby E, Morgan M, Zanakis, MF. Kinematic system demonstrates cranial bone movement about the cranial sutures. J Am Osteopath Assoc, 1996; 96(9):551.

49. Retzlaff, EW, Michael DK, Roppel RM. Cranial bone mobility. J Am Osteopath Assoc, 1975; 74:138-146/869-873.

50. Magoun, HI. "The temporal bone: troublemaker in the head," The Journal of the American Osteopathic Association, Vol. 73 (June 1974).

51. Magoun, HI. "Entrapment neuropathy of the central nervous system, Part II: Cranial nerves I–IV, VI–VIII, XII," The Journal of the American Osteopathic Association, 67:779-787 (March 1968).

52. Frymann, V, A study of the rhythmic motions of the living cranium, Journal of the American Osteopathic Association, May 1971, p. 928–945.

53. Upledger JE, Craniosacral Therapy II: Beyond the Dura. Seattle, WA: Eastland Press; 1987. p.115–30.

54. Blum, CL, Cranial Therapeutic Approach to Cranial Nerve Entrapment Part I: Cranial Nerves III, IV, and VI. ACA Journal of Chiropractic, July 1988; 22(7):63-7.

55. Goodheart, GJ. Being a Family Doctor, Videotaped seminar and notes.

Subconscious Mind Repatterning Using Newly Invented, UNCUT Live Flower Essences

Brent W. Davis, D.C.

Abstract

Ever since the early years of applied kinesiology, flower essence therapy has been used with mixed results by AK practitioners to treat the mental side of the Triad of Health. Flower essences available in the marketplace all use the Bach method of essence preparation which captures healing frequencies from flowers placed in an extraction water bath after they have been cut from the mother plant. The cutting process disrupts the bio-coherence of healing frequencies, diminishing therapeutic possibilities.

In 1999 this author invented an entirely new form of flower essence which captures energy from UNCUT living flowers, resulting in healing frequencies orders or magnitude more powerful. This process was patented in 2001. Clinical use of the new flower essences is discussed in the context of AK practice.

Key Indexing Terms: Flower essence therapy, Triad of Health, applied kinesiology (AK).

Introduction

One of the ways to screen the need for treating the emotional side of the AK Triad of Health is by the use of "semantic screening." Semantic screening consists of having the patient state aloud certain test phrases to see if the statement changes the strength of an indicator muscle. For example, if a patient were to state, "I am filled with gratitude," and a previously strong indicator muscle weakened, it would signify that on the subconscious level the patient did **not** feel gratitude.

According to mind/body pioneer, John Diamond, M.D., certain herb-derived extracts[1] can change semantic screening responses. When that occurs, astonishingly the change correlates with actual transmutation of unhealthy subconscious memories or beliefs.

Materials and Methods:

From the time I patented the FlorAlive[2] UNCUT flower essence method in 2001 until the present, I have been heavily immersed in the discovery and clinical validation of essences from unusual flowers with unique healing characteristics. The way I find the flowers is through attunement with Nature and by being drawn to them intuitively. It is a metanormal process that defies scientific explanation. And yet, in every day clinical practice, using simple AK techniques, it is easy to validate the uses of the new essences. Many of the most powerful essences have the property of healing the subconscious scars from emotional or physical abuse.

From the mountains of the high Andes, to the woodland forests of Tennessee, I eventually devised a combination formula consisting of the following six flowers:[3] Maquilina, Wild Iris, May Apple, Tulip Poplar,

Blue-Eyed Grass and Pink Lady Slipper. This combination has the property of helping to remove several self-sabotaging beliefs. The test phrases to employ in semantic screening with this formula are:
- No matter what action I take, I cannot influence the outcome (Maquilina)
- I feel loneliness and separation from life (Wild Iris)
- I lose my power when others don't listen to me – or – I feel invalidated (May Apple)
- My self esteem is strong and balanced (Tulip Poplar)
- I am free from the influence of others' hurtful actions toward me (Blue-Eyed Grass)
- I have trust in my life – or – I trust in my future (Pink Ladyslipper).

The novel combination formula of these six flowers abolishes many if not all of the unhealthy responses to the above test phrases. These flowers also abolish abnormal responses to the basic orientation-to- wellness semantic screening phrases taught by Dr. John Diamond, which are:
- Health is good
- Life is good
- Love is good
- I want to be healthy.

Results

What I have observed in the context of integrative care is that most of the time one or two bottles of this formula permanently removes from the subconscious mind unhealthy responses to the test phrases. The implications of that finding are profound.

I am fascinated to know what the outcome of the use of this formula will be when tested in a rigorous double blind study by a world class scientist with expertise in the measurement of cognitive change. Results from that study may be available before the end of 2005.

Discussion

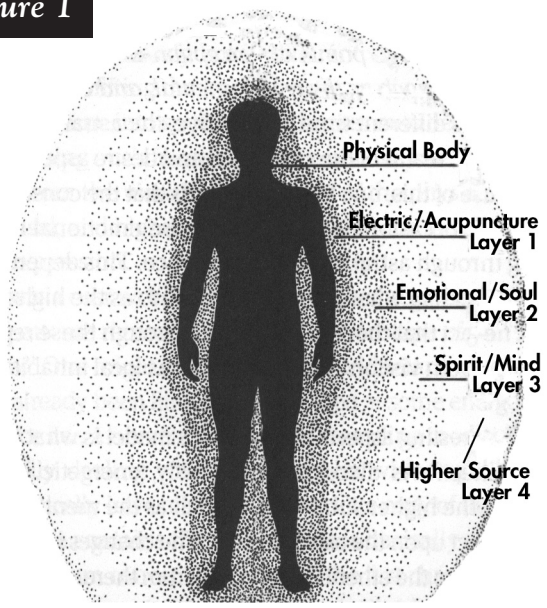
So-called "talk therapies" can be enormously helpful to increase coping abilities in the emotionally disabled, but in most cases do not change unhealthy patterning in the subconscious mind. During the last five years that I have been observing in patients rapid and profound beneficial changes in emotional outlook and subconscious patterning, I remain intrigued by the phenomenon of deep healing produced by the new essences. How is it possible?

The frequency of the uncut flower essences appears to "overwrite" negative subconscious patterning, diminishing or removing profound obstacles such as hopelessness, loneliness, poor self esteem, lack of trust, failure to allow receiving and prosperity, and more.

As remarkable as it may seem, all features of a flower including its color, shape and fragrance combine to form a "frequency" that acts as an antidote to unbalanced emotional states. Flowers appear to have been divinely placed on Earth for the great healing purpose of displacing negative subconscious memories and beliefs most people harbor, or for filling voids in us where we have emotional chasms. When a few drops of a highly energized FlorAlive essence are placed in water, the healing frequency is transferred to the water immediately. When the consumer drinks the water, the healing frequency is immediately transferred to him, and appears to transmute the part of the subconscious mind which is holding a negative memory or belief. To use a common analogy, it appears very similar to the process of reformatting a hard drive for better performance. The paradigm that discusses how this can happen is explained below.

According to a composite view derived from ancient medical systems and contemporary "vibrational medicine," Figure 1 depicts the several layers that comprise the full human being.

Figure 1



It is posited that the outermost layers essentially influence and determine the balance (state of health) of the inner layers. Each successive layer going outward from the physical body is believed to resonate at a higher frequency, possessing a more subtle character, than the layer inside it, yet exerting a greater power of influence over the state of the physical body. For example, if an individual is suffering from stubborn, unbalanced emotional states (layer 2), it can lead to strong imbalances in the electrical acupuncture meridians (layer 1) and reflect into the physical body causing disease symptoms. If this individual were treated with the appropriate, high frequency uncut flower essence for his condition (influencing layers 2, 3, and 4), he could receive a strong stimulus toward deep restoration of health.

Results from health professionals using electrodermal screening with Vegatest® and other instruments show that flower essences prepared according to Dr. Bach's method primarily influence the second layer, that of the emotions, while FlorAlive essences influence the layer of the emotions as well as the higher frequency layers 3, 4 and beyond. These findings are graphically depicted by GDV (gas discharge visualization) images that can be seen on the website, www.FlorAlive.com (Thanks to Dr. Robert Blaich for allowing me in 2004 to test a group of his patients with the gas discharge visualization instrument which yielded ignificant documentation of changes before and after administration of my flower essences.)

Conclusion

By employing newly invented uncut flower essences, it is possible to produce permanent changes in unhealthy subconscious memories and beliefs as measured by "semantic screening" and applied kinesiology muscle testing response.

Resources

In the late nineties, Dr. John Diamond reported to me in a private communication that for several years he had been testing herbal remedies and flower essences from around the world. He was searching for companies making products that would shift and displace unhealthy subconscious thoughts and memories as measured by "semantic screening" and clinical outcome. He commented that herbal products made by my company, PRL, Inc. had the unique property of regularly producing therapeutic subconscious shifts while other companies' products did not. He explained that the only other way he could obtain good results was when he made an herbal extract himself for his patients. He asked me how that could be so.

As a result of his call I became intrigued by the process of semantic screening, and traveled to Dr. Diamond's office to be treated by him and to observe him treating patients over 3 days. When I returned to practice I was inspired to begin using the semantic screening process on all my patients. Understanding this simple but profound procedure inspired me in the discovery of the new flower essences.

FlorAlive™ essences are distributed by PRL, Inc. Tel. 800 274 3727. The six flower essence described in this paper is called ReviveAll.™ On the web, www.FlorAlive.com.

This combination formula is called ReviveAll.™

Muscle Testing: Is It By Art Or By Inspiration? An Opinion Paper

Daniel H. Duffy, Sr., D.C., DIBAK

Abstract

The purpose of this essay is to discuss the difference between inspiration and art in the demonstration of muscle testing effects, especially as it relates to muscle testing in accordance with the protocols set forth by the International College of Applied Kinesiology.

Key Indexing Terms: Applied kinesiology, manual muscle testing.

Introduction

While watching a videotape presentation of a highly regarded motivational speaker, the critical difference between the inspirational vs. the art form of demonstration became apparent to this observer, especially as it relates to the sources of information passed on by those demonstrating muscle testing.

Discussion

The motivational speaker described is an awesome lecturer who appears to teach by inspiration. He presents his view of the world in an interesting and enlightening manner by discussing the absolute source of all that is and how one can connect to that source.

In regards to the transmission and teaching of the principles of applied kinesiology, the possibility of introducing error into any of the activities related thereto stems from the source of the information alleged to stem from the original teachings. This is true of any system of knowledge, all of which are subjected to immediate and continuous distortions by entrepreneurial types interested in quick profits and wishing to "astonish the mobs."

This essay suggests that there exists a higher probability of distortion of the original teaching when indulging in the use of the inspirational form of instruction or engaging in activity directed there from. When using the inspirational form of teaching one has to be very careful about the source of the inspiration. This writer considers the inspirational form of teaching to be classified as the teacher being possessed as is understood in the common idiom, thereby placing it in a lesser category of consideration.

The major purpose of this essay is to convey to the reader the difference between inspiration and art in the teaching of others and to demonstrate the pitfalls in the use of one vs. the other. There is nothing new or original in any of the following commentary. This is simply a restatement of what others before me have considered to be fundamental truths being reiterated herein via the current idiom.

The lecturer described above exemplifies the use of the inspirational type vs. the art form of instruction. Experts in applied kinesiology who have mastered the craft use the art form of muscle testing and look

askance at those making use of the inspirational form, most of which has been found to be ineffective and which often serves to bring discredit upon those using the art form.

To gain a fuller understanding of this subject matter, the reader is referred to the Dialogues of Plato, in particular, the Ion,[1] wherein this subject is given serious treatment via the dialectic approach of Socrates. Serious students of the great philosophical ideas will gain much insight by studying the Platonian dialogues prior to tackling the great philosophies. My experience suggests that the roots of most of the eternal questions studied and discussed by the great western philosophers can be found in the Platonian Dialogues which are actually the words of Socrates recollected by his student, Plato. Socrates, like Christ, never wrote anything down except in the sand.

A short discussion of the effect of the dialogues on myself is in order. One of the fundamental guiding forces in my life arises from a comment by Socrates in the Dialogues: "The unexamined life is not worth living." I couple this observation with another observation from a later philosopher, Kant, who was obviously a student of the Dialogues. My guiding phrase from Kant's Categorical Imperative[2] is "Act as if the maxim from which you act were to become through your will a universal law." Spoken in simple language this translates to give everything you do your best shot. Kant's Categorical Imperative suggests that you should perform everything you do as though it would be everyman's guide for all eternity in the performance of that specific act.

Teachers who use the inspirational form of teaching are channels through which flows information from a source. The source can be God, the Bible, the Koran, a great philosopher, or a fool. Such teachers are said to be inspired and when in such an inspired state, they deliver information, not from themselves, but from the source of their inspiration, of which a classical example is speaking in tongues. This can also be described as being possessed.

In the Platonian Dialogue entitled Ion, Socrates cleverly likens the force of inspiration to the force of magnetism in a magnet. Socrates described how, when a ring of plain metal is brought into contact with a magnet, the power of the magnet is transferred to the plain metal ring which becomes a passageway for the transference of the magnetism. The second ring of metal magically comes into possession of the same force contained in the original source and will transmit that force to other like materials such as metallic substances, metal scraps, shavings, etc. The power of the original magnet is transferred only through other like materials. The similarity of the substances involved in the transference of magnetism is a subtle key to further understanding of the phenomenon. Note that similarity of substance is one of those rarities in which an all and every definition fits. Socrates spent his life in search of all and every definitions for words such as virtue, knowledge, wisdom, etc.

Hence, we see that the inspirational form of teaching requires diligence in the selection of source since the transmitting medium simply passes on what stems from the source. Much of what is described as mob activity or herd instinct is of like nature. The inspirational state can be instilled in a single meeting with an overwhelming or strongly persuasive or titillating authority figure, particularly when such a figure presents something new and mysterious or highly interesting to the viewer.

The other form of teaching is the art form. Expertise in the use of the art form requires long, diligent effort and is not easily gained. For example, one cannot expect to be able to play a violin by reading a book entitled How To Play The Violin, or ride a bicycle after reading the How To Ride A Bike book. The amount of time and effort required is proportional to the difficulty and precision of the task to be learned. Washing dishes, sweeping floors, playing violins, and riding bicycles demand different investments in time and effort.

The discipline of applied kinesiology requires many hours over many years to master its art form. Applied kinesiology cannot be learned by reading a book or dabbling in its fringe or in its parts, although many

believe that to be so. One cannot hope to produce a Rembrandt quality painting without spending many years at the easel doing all sorts of artwork using many different mediums.

Achieving the expertise of the art form through great effort renders the achiever dependably independent of all worldly influences, while the integrity of the inspired form depends upon the integrity of the worldly or otherworldly source. It has been said that many worldly sources are false prophets. The phenomena called speaking in tongues, automatic writing, and fortune telling are examples of otherworldly sources of inspiration. The observer of such activity must judge on the merits of each.

The demonstration by the lecturer cited above (to be described below) involved the presence of certain energies. To facilitate appreciation for the writer's understanding of energies before discussing the actual event, the following is offered on the subject of energy. All matter is condensed energy ($E = MC^2$). The difference between matter and energy can be compared to the difference between steam and hot water. All energy is neutral, there is no such thing as positive and negative energy, these terms are inventions, not discoveries. All energy flows from an ultimate source which is non-material idea. Ideas come first, then things follow in substance. The esotericist, for example, states that thoughts are the precursors to things.[3] Ultimate source can be described as spiritual, mental, Mother Nature, the universe, the all, etc. It is the opinion of this writer that nothing is disconnected from ultimate source although its use is lost to most due to the lack of knowledge, will and understanding, or the connection with false teachings.

All matter is simply condensed energy. There is only one energy and it is neither good nor bad, except in how we perceive and use it or allow it to have an effect on us. The notion that "bad" energy exists is an incorrect idea that calls forth equally incorrect teachings. Such incorrect ideas are used in pseudo-scientific circles that describe such things as good and bad cholesterol. Good and evil are inventions, not discoveries. Inventions emanate from man; things that are discovered emanate from source (source can be considered God, the Ultimate, the universe, or whatever one accepts in that regard as mentioned above). Good and evil are often mixed up with pleasurable and unpleasurable from which stems much of the illogical thought processes that pressure us, affect our behavior, and cause us to form wrong thoughts which result in things or actual events. Evil effects are possible only when we forget our connection to source, form an ego that begins to believe itself to be the source, and then acts in an inappropriate manner, bringing upon ourselves unhealthy, unpleasurable, uninspiring states.

Acting out of the imagination-inspired ego exposes us to inspired forces greater than our own that flow from defective machines such as Hitler, Pol Pot, Stalin, or a doctor who misrepresents himself as an expert in certain fields such as medicine or muscle testing. A trusting person can easily be misled by such entrepreneurs. Indeed, many have been misled in applied kinesiology considering the amount of arm pull down tests being performed all over the world these days. The result of following the teachings of such misguided entrepreneurs is inspired activity, the source of which does not belong to the transmitter of the error. They merely channel someone else's energy like parrots or copy machines or like the magnets of Socrates. They are temporarily magnetized (energized) and simply allow the magnetism (energy expressed inspirationally) to flow through them like the magnetism flowing through an iron bar which becomes momentarily magnetized by a magnet. It is under such influence that they become unconscious "repeaters." For example, good and bad effects of substances have been demonstrated by placing the substances on the body during muscle testing and observing a weakening of the muscle tested. This exemplifies wrong teachings and is not part of the applied kinesiology protocol, although those attacking our discipline cite such examples of foolishness to undermine AK.

The subject lecturer mentioned above made a demonstration wherein he compared the difference in muscle testing results between the effects of a banana placed against the tested subject's chest against a CD of hard rock music. Supposedly, placing the CD against the subject's chest caused his arm to go weak while the banana did not. The lecturer then claimed that the bad energy in the CD rock music had weakened the subject's arm.

This type of testing (against substances placed upon the body or held in the hand) has been found to have a random effect caused by the spectroscopic color of the substance held against the body. To explain: while demonstrating this phenomenon in the Basic Applied Kinesiology 100-hour course, I often use folic acid placed on the stomach area. If this produces an observable response via a change in muscle strength, I then use a book with a yellow cover to demonstrate the same response, and jokingly tell the subject to take three yellow books a day and call me in a month. Colors affect the body in different areas in accordance with the colors of the rainbow. The spectroscopic color of folic acid is yellow, hence the similar effect that folic acid and a yellow book would have. Evidence exists suggesting the possibility of mentally transmitting or injecting color energy into patients via thought processes as does the notion that energy can be gathered and accumulated by certain breathing techniques and then projected from the body. I have successfully demonstrated these effects publicly. The transmission of color energy was demonstrated by this writer on a subject via biofeedback measurements of body temperature at a Goodheart seminar during the early 1970's just after Goodheart had discovered the phenomenon involved in the random effect observed from placing nutritive substances on the body. A biofeedback temperature probe with both temperature and sound indicators was placed on the subject's forehead. The temperature failed to change following several attempts by other chiropractors performing usual chiropractic adjustments, in particular, Basic Technique, due to the fact that the patient showed a low sacrum on one side. I suddenly enlightened to what Goodheart was trying to teach and volunteered to take a try at it. I took a basic contact on the patient's sacrotuberous ligament with my thumb and held it for a few moments. There was no change in the temperature or the tone of the sound emitted by the biofeedback instrument. I then began to mentally inject the color red into my thumb and the temperature almost instantly responded by increasing at the glabella with accompanying changes in the sound as it moved along the musical scale of tones. The temperature probe on the subject's forehead immediately began to register a change in the proper direction.

The arm pull down test is always used by novices in muscle testing demonstrations because the fulcrum effect makes it is easy to overcome the strength of even the most powerfully developed athletes. Archimedes claimed that, with a long enough lever, he could move the earth. It is my belief that the majority of people are not dishonest but are simply acting under wrong influences. They are victims of the inspirational form of involvement and like hollow reeds or magnets, simply pass through what they have learned from defective teachings.

It has been said that there is only one energy, neither good nor bad, but simply, energy. Good and evil are effects, not causes. In the causal chain, the examination of constant conjunction (see the writings of the radical empiricist, Hume)[4] gives us the notion of cause and effect. For example, every time we see a match put to paper we see the paper burn; therefore the burning match is the cause of the burning paper. This is called constant conjunction by Hume to demonstrate how we describe what we think we know about cause-effect. The energy burned by saving a drowning person is no different than the energy required to beat them to death. The energy stored in a CD of rock music is no different than the energy stored in a CD containing religious music or the energy stored in a banana. Any observable difference between what is considered good and evil energy is simply a function of how we choose to respond to it or to allow it to affect us. Such energy, although stemming from ultimate source, as does all energy, is completely under our own control for us to use as we so desire! Imagination can cut us off from using ultimate source while opening the door to actions stemming from inspiration.

Since the discovery of muscle testing as a diagnostic method, the alternative health field has spawned a flood of entrepreneurs peddling books and techniques grossly lacking in the art of muscle testing. The faulty techniques are then picked up by others who begin to repeat the faulty techniques by the inspirational form of transmission. Not having mastered the art form, the inspired reproducer, lacking the power to discriminate proper from improper muscle testing technique, are inspired to reproduce faulty muscle testing techniques, transmitting them to others of similar or lower levels of knowledge, understanding, and wisdom.

Many books have surfaced to profit (in all the myriad ways) from this latest discovery of George J. Goodheart, Jr., DC, of Detroit, Michigan, who, at the time of this writing, is in his 86th year and still productive in the generation of new methods of using muscle testing as a diagnostic indicator, possibly the greatest medical discovery of the 20th century. In spite of medical propaganda to the contrary, it is the opinion of this writer that precious few medical techniques of the latter 20th and early 21st century will stand the test of time and the doctor of the future will interest his/her patient in the proper care of their physical frame and the use of natural foods grown under natural conditions. The present indulgence in destructive drugs and surgical procedures will become a thing of the past as the public discovers the fallacy of the germ theory of disease and learns that almost every disease considered infectious and transmissible is in fact due to malnutrition. They will also discover that almost every disease described as "cause unknown," now treated symptomatically with destructive drugs, will also be revealed to be caused by malnutrition. The doctor skilled in the masterful art of applied kinesiology will be the doctor of choice in such a society except for those cases requiring crisis medical care.

Conclusion

When a muscle tester is energized by inspiration, if the source of the inspiration is flawed, the result is flawed. The inspired transmitter simply transfers whatever flows through them from the original magnet.

Muscle testing is an art form. It does not become useful as an inspirational form until long after one has paid the dues. Paying the dues means spending time in the trenches, doing the hard work, treating every patient, every day, in accordance with applied kinesiology protocols while keeping in mind the Categorical Imperative of Kant.

References

1. Ion, The Dialogues of Plato. Britannica Great Books. Vol. 7. W. Benton Publ: 1952. p. 142–148.

2. Fundamental Principles, The Critique of Pure Reason. Britannica Great Books. Vol. 42. W. Benton Publ. P. 253–87.

3. Heindel, M. The Rosicrucian Mysteries, An Elementary Exposition of Their Secret Teachings. 2nd Edition. Oceanside, CA. Rosicrucian Fellowship: 1916.

4. Hume, D. An Enquiry Concerning Human Understanding. Britannica Great Books Vol. 35. W. Benton Publ: 1952. p. 451–509.

Muscle Activation Technique

Simon J. King, B.App.Sc.(Chiro), DIBAK

Abstract

The author presents a clinical procedure based on the observation that an indicator muscle is inhibited when an inhibited muscle is put into contraction. The hypothesis is that spindle cell activity contributes to a cumulative summation of afferent input that is necessary for normotonic muscle response. Unfortunately, the lack of a gold-standard objective test for muscle inhibition has prevented the proper testing of this hypothesis. The clinical procedure is therefore presented for the interest of the membership.

Key Indexing Terms: Proprioception, muscle inhibition, muscle contraction procedure.

Introduction

Muscle spindles generate constant afferent input based on the amount of tension or stretch on the muscle fibres. This input creates reflex control of movement through the monosynaptic myotatic reflex as well as providing information to higher centres for volitional control and coordinated responses via interneuron connections to propriospinal pathways. Constant afferent input from the spindle cells generates resting muscle tone by activating a continuous flow of motor output from the alpha motor neurons.

Although chiropractors and physical therapists have traditionally concerned themselves with proprioception from joints and ligaments, ligaments are non-elastic and by definition will only become active at the end of the range of motion of a joint. Only muscle spindles give constant, proportional feedback to the nervous system about the state of tension and tone in the muscles.

Reflex activity via the myotatic reflex, flexor withdrawal reflex and crossed-extensor reflex contributes to the sum of proprioceptive input that adjusts and maintains muscle tone. Of these reflexes only muscle spindle cells spontaneously generate continuous output. Other sensors only contribute when stimulated.

The myotatic reflex generates monosynaptic activity to the muscle that originated the input, however the signalling becomes generalized when it passes to the crossed extensors and via the propriospinal fibres and ascending tracts.

I hypothesised that the myotatic reflex contributed to overall afferent input, including input to propriospinal tracts that had an influence on the summation required for facilitation at local and distant anterior motor neurons. Alterations of proprioception have been shown to be cause a decrease in muscle strength, both local[1] and distant[2] to the injury site. Poor muscle tone or muscle power has been shown to be a predictor of injury.[3]

Muscle inhibition (from any cause) might cause general deafferentation and this might be detected in muscles supplied via propriospinal pathways.

I observed that increasing the tension in an inhibited muscle by putting it into contraction would cause an indicator muscle to test weak by manual muscle testing. The decreased sensory input from the spindle cells of the weak muscle seemed to defacilitate the alpha motor neurons of otherwise normotonic muscles.

While I do not believe there is any substitute for testing muscles directly, there are some muscles that cannot be tested manually. The multifidus and rotatores muscles cannot be isolated and tested effectively using standard manual muscle testing and yet their strength and tone are probably essential for the protection and preservation of the joints of the spine.

Discussion

The MUSCLE ACTIVATION TECHNIQUE is presented for the interest of members. I have found it extremely useful in my daily practice and as a way of introducing new students of applied kinesiology to the art of muscle testing.

The Technique

Any muscle can be tested for inhibition using the muscle activation technique. If a muscle is weak, putting it into contraction will result in a strong indicator muscle failing. This might be thought of as a "muscle challenge." Also, if the muscle belly of the weak muscle is contacted while in contraction, an indicator muscle will stay weak for as long as the contact is maintained. Removing the pressure on the muscle belly returns the indicator muscle to normal.

In practice, the Muscle Activation Technique can be applied as follows:
The patient is placed prone and the strength of shoulder abduction is checked.
The doctor needs to begin with a strong muscle. Shoulder abduction is most convenient and most often used because contraction of shoulder abductors seems to be a necessary part of any startle response for most people (falling, loud noises, roofs falling in etc). If shoulder abduction is not strong with the patient prone, do not continue with this technique.

The technique is really very simple although its application can take a little practice. Systematically provide resistance to patient movements (involving muscle contraction) and test the strong shoulder abductors while the patient maintains contraction of each muscle group. Sometimes gravity provides enough resistance, other times the doctor will need to provide additional resistance by means of manual pressure, a strap, the table or an assistant.

Failure of the strong indicator is taken to mean that there is a failure of the muscle spindle afferent input from any of the muscle fibres that have been activated or put into contraction. Failure of the indicator usually means that the contracted muscle (or muscles) are inhibited. This can usually be verified with specific testing of the involved muscles.

If the strong indicator fails, alter the patient's proprioception until the weakness is negated and strength is maintained. In manual therapy terms, this usually means using sustained pressure (sustained challenge) at each vertebral level until you find the level and direction that negates the weakness (restores the strength). Other proprioceptive changes such as altering the patient's position may prove helpful to isolate the area of any spinal lesion.

The principles contained in this technique can be applied to a wide variety of situations.

There follows a basic routine for the checking various spinal areas. Each movement is presented in terms of instructions to the patient, except for cervical protrusion, which I define as cervical flexion with the chin forward.

Patient prone, test a strong shoulder abductor. Ask the patient to

Low back	Thoracics	Neck
Squeeze your right knee down	Opposite arm out	Cervical flexion
Squeeze your left knee down	Same arm out	Cervical protrusion
Lift your right leg up	Full internal rotation	Cervical extension
Lift your left leg up	Full external rotation	Right ear to table
Stretch your right leg long	Shoulder to hip	Left ear to table
Stretch your left leg long	Shoulder to ear	Right ear to feet
Make your right leg short	Handrest to shoulder	Left ear to feet
Make your left leg short	Handrest to floor	
Lift your right hip up	Handrest to feet	
Lift your left hip up	Handrest away from feet	
Lift your back up (lumbar flexion)		
Push your tummy down (lumbar extension)		

Keep testing until a weakness is found (if any). Asking the patient to maintain the position and contraction, carefully press each vertebra in turn, first generally from Inferior to superior, then from left to right and right to left (only if the first test negated the weakness). A vertebra should only be corrected if it negates the weakness in one direction, not both.

Adjust the vertebra in the direction that negated the weakness.

Re-check the contraction and the test to make sure the weakness is gone. If not abolished, you may have only taken care of one of the factors involved in creating the abnormal afferent input (presuming you were right about your diagnosis and performed the adjustment correctly).

Continue to check all of the available motions and muscle contraction combinations.

Remember that failures in proprioception may not be spinal in origin.

Conclusions

Such a general technique is not easily subjected to controlled evaluation. The technique fits with known neuroscience and seems to be clinically useful. The author encourages further research and development.

References

1. Herzog W; Suter E Muscle inhibition following knee injury and disease. Sportverletz Sportschaden 1997 Sep;11(3):74-8.

2. Bullock-Saxton JE Local sensation changes and altered hip muscle function following severe ankle sprain. Phys Ther 1994 Jan;74(1):17-28; discussion 28–31.

3. Orchard J et al. Preseason hamstring muscle weakness associated with hamstring muscle injury in Australian footballers. Am J Sports Med, 25(1):81–5 1997 Jan–Feb.

Hydor

Tyran Mincey, D.C.

Abstract

Hydor is Greek for water (Funk and Wagnall's). It is essential, and breathes breath into life. It allows us to exchange nutrients vital to function and remove those that would inhibit it. Most life on this planet could not survive with out it.

Key Indexing Terms: Water, muscle inhibition, applied kinesiology.

Introduction

With all the opinions about how much, how often, and what pH, this author was inspired to do a brief but simple blinded study that explores what effect gustatory challenge of different types of water would have on the patients central integrated state as evidenced by muscle responses or inhibitions.

Materials and Methods

10 unqualified randomly selected patients who had complaints that ranged from pain syndromes to visceral dysfunction were chosen and told, "we would like to see how your body reacts to water."* Using two 4-ounce bottles with a dropper containing water of two different pH's (one alkaline at 7.4 and the other acid at 6.5). Both bottles of water were filtered. One bottle of water was alkalized using a Mavellos™ water alkalizer (Jupiter). The device was preset at the pH of 8. These bottles were then labeled "A" and "B" by a third party leaving the doctor blind regarding the contents. Using the deltoid muscle as primary indicator muscle (long lever), a gustatory challenge was initiated; each patient initially had a "strong" (PIM) indicator muscle before testing with water. The patient was tested and told to hold the arm up after a drop of water was placed on the tongue. A muscle testing reaction was then studied. While there are many other factors that could have been studied, we just wanted to see what outcomes would be for this basic study.

Table of Outcomes

	Vial A – .01 micron filtered water (untouched) - pH 6.8	Vial B – .01 micron filtered water (alkalized antioxidant) - pH 7.4
1.	W	–
2.	W	–
3.	W	–
4.	W	–
5.	W	–
6.	W	–
7.	W	–
8.	W	–
9.	W	–
10.	W	–

Key: "W" = inhibited " – " = not reactive

*pH of the body is a very narrow range of between 7.35 and 7.4 and does not vary greatly.

Results

We have noted that a significant difference exists with all inhibitions coming from vial "A." Again, Vial A contained water that was filter with a .01 micron filter. In all subjects in this small study Vial "A" inhibited the deltoid muscle compared to Vial B, which elicited no muscle change.

Discussion

There have been papers published in the proceedings and elsewhere proclaiming the virtues of water (Motley). While fascinating based on our findings it seems prudent that we approach the recommendations of the use of water carefully and not jump on any band wagon of fantastic claims. Instead we should make decisions on an individual basis by testing a person and making an observation regarding what is best for that person's constitution. In a day and age where it seems that on a regular basis a new type of water is coming out (i.e. oxygenated, ozonated, with a "special" hydrogen bond, alkalized, reverse osmosed, magnetized, distilled and probably more) we have an ethical responsibility to help patients identify what type might react best in their body as one size does not fit all.

Conclusion

While our sample was small it does lead one to wonder what the outcomes may have been for distilled water or some the other types since this authors observation is that it is usually is slightly acidic. The outcomes also remind us of the need for more trials with different types of water and remind us that we may need to look at other factors that may impact our patient's need for different types of water. This Author suspects that some factors such as work place and geographic location will have an obvious impact on the amount of water a person should consume but the these factors may also impact the need for water of differing pH's. This needs to be developed further in other papers.

A more in depth study of water needs to be done, this should include more participants and several types of water. No conclusion as to health benefit can be made at this time except to say that it is obvious that not all patients will respond the same way to all types of water. Since the goal of the study was to identify if in fact the central integrated state would be impacted by water type, the author is satisfied that that there is evidence to support such a statement. In particular it seems the alkalized antioxidant water tested better than filtered water.

Acknowledgments

Special thank you to Mrs. Nicole Mincey for her work on this and Dr. John Lombardozzi for his encouragement.

Resources

Funk and Wagnall's New International Dictionary of the English language (World Publishers Inc, 2001).

Jupiter Marvello instruction manual Dong Yang Science Company LTD 2001.

Motley, Heath DC "Distilled Water, The only Water" ICAK proceeding Vol 1, 2001–2002.

The Four Dimensions of Being

Dale Schusterman, D.C., DIBAK

Abstract

The number four is common to many schools of thought when it comes to understanding the human being. It is the basis of nature, consciousness, and the human body. This paper will explore how to use this quad pattern in the body for the treatment and diagnosis of patterns that have deep roots and might be overlooked without this knowledge. One of the reasons behind switching is that we are an overlay of four separate energy systems. Using this method will quickly get past much of the neurological disorganization that we spend so much time trying to clear before we can effectively work on someone.

Key Indexing Terms: Quad pattern, switching, applied kinesiology.

Introduction

The number four is found in all cultures as a metaphor, or symbol, for understanding reality. Native cultures talk of the 'four corners of the earth,' 'the four directions (N, S, E, W), or the 'four winds.' Many of the healing rituals of indigenous peoples involved honoring, invoking, and balancing the four 'spirits.'

All living systems are based on the carbon atom, which creates four bonds to form tetrahedral shapes. The tetrahedron, the simplest of the Platonic solids, is the most compact of all shapes (four faces and four points) and is the building block of nature.

Discussion

Early medicine talks of the four humours (blood, yellow bile, phlegm, and black bile). A proper mix of these four humours, which relate to the four elements (air, fire, water, and earth), was required for optimum health. Disease was seen as an imbalance, excess or deficiency of the humours. They were said to give off vapors, which ascended to the brain. Here they would form the temperaments (sanguine, choleric, phlegmatic, and melancholic), which describe basic human personality characteristics.

It may sound like nonsense to us now when we hear of such things, but it would be wise not to dismiss outright the knowledge of the ancients. Certainly, they did not have the perspective of our scientific knowledge of the body and mind. But, it is also possible that scientific arrogance has overlooked some of the truths of previous paradigms, which are still valid.

Most wisdom traditions talk about the four dimensions of man/woman. The most common names for these dimensions are the physical, astral, causal, and spiritual planes of existence. We are said to have a garment in which we inhabit each of these four planes. They are called bodies or sheaths—physical body, astral body, causal body, and spiritual body. There are special names for each of these dimensions in the Hindu, Buddhist, Sufi, Jewish, and Taoist systems, but for consistency, we will use the English equivalents. What they all agree on is that there are four basic stages of existence set within a larger backdrop of reality (which is why some systems talk of 5 elements).

Dr. Alan Beardall based his system of Clinical Kinesiology on his observation that there are four 'biocomputers' in the body. He called them the Local, Spinal, Endocrine, and Primary computers. They are the mechanisms in the body that adapt and process all information that come into the system.

First, let us look at the form of the human body. It consists of four extremities—two arms and two legs. The four fingers and toes on each hand and foot are also an example of the four-pattern in the body. There is also a five-fold pattern, but the fifth 'wheel' is different from the other four. The thumb is in a different plane from the fingers, much as the head is different from the arms and legs. The four chambers of the heart is another area where we see the number four manifesting. Other examples could be given, but these are the most obvious areas that reflect the four-fold nature.

As kinesiologists, we can test the four dimensions in the body in a straightforward fashion. We can do this by testing muscles on the four extremities. Each arm or leg represents one of the four dimensions.

In my book, *Sign Language of the Soul: A Handbook for Healing*[1], I describe a complete system of energy healing, based on the Kabbalah (Jewish mysticism), which incorporates an in depth way to explore these four dimensions. The four dimensions or 'worlds' as they are known in the Kabbalah are part of the explanation of the Tree of Life. The Tree of Life (Figure 1) is a model used to describe the anatomy of human consciousness, but it also is an excellent representation of the physical body. Indeed, one of the central tenets of the Kabbalah, which is common to all wisdom traditions, is that the body and human consciousness are cut from the same mold. The symbol of the Tree of Life consists of ten major energy centers connected by twenty-two gates. These 32 pathways express in each of the four dimensions. The correlation in the human body is the four extremities. Each extremity consists of 32 bones, which is a direct correlation to the structure of consciousness.

Table 1.

Lower extremities	14 phalanges, 5 metatarsals, 7 tarsals, tibia, fibula, femur, ilium, ischium, pubis
Upper extremities	14 phalanges, 5 metacarpals, 8 carpals, radius, ulna, humerus, clavicle, scapula

Table 2 shows the four dimensions as they correlate with the four extremities. This information will form the basis for the simple testing technique that follows.

Table 2.

Left Arm	Spiritual Body
Right Arm	Mental Body
Right Leg	Emotional Body
Left Leg	Physical Body

There are many other such correlations between the body and the Tree of Life, but they are not pertinent to this discussion. However, one other important relationship needs discussing. There are four places in the body which act as doorways, in consciousness, to these four dimensions. When you focus upon one of these doorways, it brings the body/mind to the frequency of that dimension. The four doorways are the area several inches below the umbilicus (Tan Tien), the thymus/heart area, the third-eye, and the atlas (Table 3). Following is a brief summary of these doorways.

Tan Tien

The area several inches below the umbilicus and several inches into the body is know in Eastern philosophy as the Tan Tien, which means 'the elixir field.' In martial arts, and other similar systems, this area is known as the center of the being. All movement centers from this point and the Tan Tien is a power point that gives great bodily control and strength. This is the center of Qi or Chi in the body; therefore, it is here that one focuses to enhance his or her physical power.

Thymus/Heart
The area of the upper to mid sternum is the next doorway. This is the area of the thymus gland and also covers the heart. This area is the center of emotion, love, and the sense of self. When we talk about ourselves, we point to our thymus. All wisdom traditions talk of the importance of love and opening the heart. This is the doorway to the astral world and by focusing on the heart/thymus area it brings one into awareness of his or her feelings.

Third Eye
The third eye is important in many traditions as the seat of consciousness. Many people focus here during meditation. It is common to look upwards to visualize something or to see with the mind's eye. Looking upwards 20 degrees also shifts the brain rhythm towards the alpha state. This doorway brings one to his or her mental, or visual, state of awareness.

Atlas
The fourth doorway is located at the lower occipital bone and the atlas. This is an area of higher Self awareness. When one concentrates from this place, it is a meta position in which one sees him or herself from a place of objectivity, or from a witness perspective.

Table 3.

Left Upper Extremity	Atlas
Right Upper Extremity	Third Eye
Right Lower Extremity	Thymus/Heart
Left Lower Extremity	Tan Tien

Focusing or concentrating on these areas causes a subtle shift in consciousness, which we can use to our benefit as we muscle test the body. There are ways to activate these four doorways in the body to access deeper levels within the being/nervous system, but that is beyond the scope of this paper. What we can do with this knowledge, however, is to combine focusing on the doorway with muscle testing its corresponding extremity.

For example, test all of the major muscles in the left lower extremity and fix any imbalances that you find. The muscles that connect the torso to the left leg (psoas, quadratus lumborum, etc.) should also be considered. Then have your patient focus on his or her Tan Tien, while you retest the muscles of the left lower extremity. It can help your patient to focus if you to touch the Tan Tien, or place one of their fingers on the area. Often you will find one muscle that now tests inhibited. Fix this muscle according to your findings; however, the patient must remain focused on his or her Tan Tien while you apply the correction (NL, NV, cranial adjustment, spinal manipulation, etc.). There does not appear to be any connection between focusing on the Tan Tien and the muscles of the other three extremities.

Repeat the same procedure with the right lower extremity while the patient concentrates on his or her thymus/heart area. Often muscles that test quite strong in the clear, on the right lower extremity, will inhibit with this change of focus. Then test the right shoulder, arm, and hand muscles while the patient focuses on his or her third eye (1/2-1 inch above the center of the eyebrows). Finally, have the patient focus on his or her atlas area (touch the area to help them focus) while testing muscles of the left upper extremity.

You will often find inhibited muscles in each extremity when you test with the patient focusing upon the doorways. Remember, that the patient must maintain his or her focus upon the doorway during treatment of the inhibited muscle.

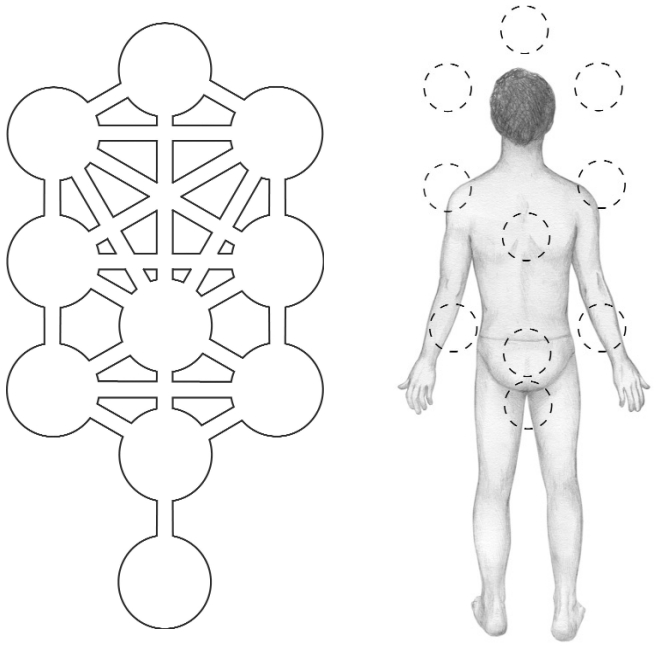
Conclusion

The next time you have a patient who is switched, try this method first and fix a few of the imbalances you find. Then go back and see if the patient is still switched. Instead of taking the time to unswitch the patient, just balance what you find through the four doorways/extremities. Use your time to treat instead of getting them ready to treat.

It is possible for the patient to think of a specific injury or emotional issue, or to hold a therapy localization while focusing upon each doorway. This will enable you to find new patterns that relate to the patient's problem in each of the four dimensions. Do not assume that all right arm problems are related to the mental body, and so on. That would be too simplistic an explanation. A right knee problem might show imbalances in all four extremities when you therapy localize the knee, while the patient focuses on the doorways, and you perform the muscle tests.

With this procedure you have engaged the consciousness, or attention of the patient, hence specific areas of the nervous system, as part of the diagnostics and the treatment. There are many parts to a person, physical, emotional, mental, and the inner sense of 'I.' With this simple procedure you can begin to incorporate these parts into your muscle testing and treating protocol.

Figure 1 **Tree of Life**



Summary of Procedure

1. Test the muscles in each extremity and fix them in your customary way.
2. Have your patient bring his or her attention to the doorway that corresponds to one of the extremities, while you retest the muscles.
3. Fix any muscles that inhibit when the patient focuses on the doorway. The patient must maintain focus on the doorway while you perform the correction.
4. When everything is clear, have your patient therapy localize a problem area and repeat steps 2 and 3.

Resources

Schusterman, Dale, (2003). *Sign Language of the Soul: A Handbook for Healing*, Cranston, RI: The Writers' Collective.

Division II

Critical Review

The Neuroregulatory Role of the Trigeminal Nerve in Dural Torque and the Reciprocal Tension Membrane System

Harlan Browning, D.C., C.C.N., D.C.B.C.N.

Abstract

Perpetuation of the semi-closed hydraulic system of the CNS is a function of the rhythmic coordination of the craniosacral respiratory mechanism and the integrative modulation of the trigeminal nerve. This paper will present neuroanatomical justification for the trigeminal nerve's role as a "weigh station" for dural tension and CSF flow, as well as the importance of local spinal and cranial fulcrums for regulating and maintaining the reciprocal tension membrane system.

Key Indexing Terms: Reciprocal tension, trigeminal nerve, applied kinesiology.

Introduction

Since its inception into Osteopathy, and with its propagation through applied kinesiology, treatment to what Sutherland termed the Primary Respiratory Mechanism has proved invaluable to the maintenance and vitality of health.[1] As kinesiologists, we are well aware of the dynamic function of the cranium in relation to the TMJ, spine and pelvic complex. But what is coordinating this mechanism and to what extent does this rhythmic motion rely on an intermediary monitor. Certain anatomical and biomechanical relationships give us a clue as to the driving force behind the stomatognathic system.[2] The following is a unique look at these factors.

Cranial Anatomy and Motion

The skull is a complex multi-articulated osseous structure composed of multiple bones and sutural plates. It can be divided into three anatomical elements: (a) the vault, consisting of portions of the frontal bone, two parietal bones, the occipital and the temporal squama; (b) the base, consisting of the body of the sphenoid, petrous and mastoid portions of the temporals and condylar portion of the occiput; and (c) the complex of facial bones.[3] Its sutural structure has a variable layout dictating a complexity of motion centering around the sphenobasilar synchondrosis.

From its rotation around the transverse axis, the motion of the basiocciput moves into degrees of flexion and extension. Its motion provides the driving cog in which the closed kinematic chain of the cranium rotates. From its flexed position we find the sphenoid and occiput rotate in opposite directions, the occiput rotates posteriorly while the sphenoid rotates anteriorly; these motions are reversed into extension. In turn the paired bones of the skull move into internal and external rotation as they follow flexion and extension at the sphenobasilar junction. This synchronization of flexion and extension of the unpaired midline bones, along with the internal and external rotation of the paired bones, allows for longitudinal and transverse distortion of the skull. So with motion into flexion we find an increase in longitudinal diameter and a decrease in vertical height and motion into extension brings about a longitudinal decrease and a ventricle increase.[4] This bidirectional mechanism provides the bellows like mechanism that drives the craniosacral respiratory pump.

Dural Anchors in the Cranium and Spinal System

The bi-layered membranous dura that envelopes the CNS is a mixture of a highly innervated and vascular outer endosteal layer, and an internal avascular meningeal layer.[5] Both layers have a close affinity throughout the cranium as the endosteal layer interdigitates within the periosteum of the vault. At junctures of dural folding, and throughout the dural venous system, we find the meningeal layer separate to provide a closed fluid conduit. The pachymenix or dura continues as osseous reflections and attachments are made within the cranium as well as the spinal column. Organization of the membrane system falls within four distinct anatomical divisions dividing the cranium into veiled partitions and demarcating the skull from the spinal column. The anatomical divisions are organized as follows:

Falx Cerebri

The sickle shaped dural reflection that makes up the falx cerebri has its anterior attachment firmly anchored from the vertical midline and ethmoid notch of the frontal bone, as well as the crista galli of the ethmoid bone. From its most forward position the falx passes posteriorly attaching to the undersurface of the frontal, parietal and occipital bones along the midline, as well as interdigitating via Sharpey's fibers within the sagittal suture. At its most posterior point the falx attaches to the internal occipital protuberance of the occiput before blending inferiorly with the tentorium cerebelli.

It is important to note that along its path several prominent enfoldings or dural reflections make up the venous sinus system of the CNS. Along the length of the sagittal suture we find the superior sagittal sinus which courses inferiorly to meet the strait sinus at the junction of the falx cerebri and the tentorium cerebelli. From here we find the free boarder of the falx cerebri forming the inferior sagittal sinus.

Tentorium Cerebelli

The roof of the cerebellum finds itself as a continuation of the inferior portion of the falx cerebri as it passes forward to make two significant attachments to the anterior and posterior clinoid processes of the sphenoid bone. From its anterior course we see the dura reflect laterally, attaching along the petrous ridge and mastoid portions of the temporal bone and ultimately securing itself to the inferior angle of the parietal bones. Finally, the tentorium cerebelli traverses the occipital bone to make its most posterior attachment.

Falx Cerebelli

Forming a reciprocal membrane, the falx cerebelli dives deep from its most superior attachment with the leaves of the tentorium cerebelli and the free edges of the straight sinus. It continues posteriorly to attach to the internal portion of the occiput before turning inferior and ending with a dense fibrous dural ring surrounding the foramen magnum. At this location we find the meningeal layer separate itself from the endosteal layer to make what we refer to as the spinal dura.[5] This separation forms the caudal continuation of the dural system starting as the atlanto-occipital membrane and coursing through the length of the spine to its final attachment at the sacrococcygeal complex.

Spinal Dura

From its cranial exit, the meningeal layer of the dura and its blending with the posterior atlanto-occipital membrane make a unique contact with the rectus capitis posterior minor muscle (RCPMI). As was recently found by the Maryland State Board of Anatomy, there seems to be a tendinous interfacing that passes through the atlantooccipital articulation anchoring the dura with the RCPMI. Further dissection found that tractioning at this point caused noticeable changes in local and intra cranial dural tension suggesting a proprioceptive role in dural activity.[6]

From this point of contact we find the dura makes two other areas of communication within the cervical spine. Dural slips or Hoffmans Ligaments are seen to make strong connections with the posterior longitudinal ligaments and vertebral bodies of C2 and C3.[7]

Continuing loosely through the dorsal spine, the dura again makes several anchors upon the vertebral bodies and IVD's of the lumbar spine. From a central and lateral ligament we find the anterior portion of the dura making a connection with the vertebral body and posterior longitudinal ligament including that portion encasing the IVD. Finally we have a 3rd set of connective bands attaching the dural sleeve with the inferior pedicle and IVF through a lateral root ligament.[7] Inferiorly, within the sacral spine we see the dura forming an anterior attachment at S2 and continuing caudally until it blends in with the periosteum of the coccyx.

Sutherland's Reciprocal Tension Membrane

The continuity of the dural sleeve is the basis behind what Sutherland coined the reciprocal tension membrane. Its propagation and regulation is the cumulative effect of osseous movement and fluid mechanics perpetuating a tension constant along the length of the dura. These relationships provide the driving force of the craniosacral respiratory mechanism.

The center of tension along the length of the dural train finds its axis of motion around "Sutherland's Fulcrum" or the straight sinus.[1] From its intra-cranial position, this fulcrum allows for the bidirectional motion of the dura, which is a response dependent on cranial bone movement. As described by Sutherland this "suspended automatically shifting fulcrum" pivots upon the anterior aspect of the straight sinus, adapting to tensile shear generated through cranial inspiration and expiration.

With sphenobasilar flexion there is an anterior and caudal shift of tension in the falx cerebri moving towards its most forward attachments within the ethmoid bone. Posteriorly we see the dura initially traction toward its occipital contacts before making an anterior inferior curve along the straight sinus. This reciprocation causes an overall shortening and deepening of the falx with movement into flexion and its reversal into extension.[2]

Along the length of the two leaves of the tentorium cerebelli we find a corresponding lengthening and flattening during inspiration. Through its anterior attachments along the sphenoid, its lateral anchors along the petrous portion of the temporal bone, and its posterior communication with Sutherland's Fulcrum, the tentorium moves anterior and inferior. This semicircular pathway imparts an overall cephalad traction on the falx cerebelli and ultimately the dural sleeve. Thus the craniosacral primary mechanism is facilitated by and initiated through the tension within the dural system. The end result being the pressure constant within the CNS.

CSF Flow & the "Pressurestat Model"

The production and maintenance of CSF is a byproduct of a closely monitored semi-closed hydraulic system. The dural sleeve serves as the ridged and non-porous closed container in which a constant exchange of flow enters and leaves the system. From its initial production via the choroid plexus and ependymal cells, to its reabsorption into the arachnoid granulations, CSF provides a fluid medium from which the pressurestat model revolves. According to Upledger, certain conditions must be met to fulfill this requirement. (1) Production must equal reabsorption due to the inelasticity of the dura; (2) The production pumps or choroid have to slow or stop to accommodate increased fluid dynamics; (3) The outflow mechanism via the arachnoid granulations must up-regulate to reduce pressure gradients. This regulation of fluid dynamics largely revolves around pressure sensitive areas within the cranial vault.[8]

Auto-regulation of CSF flow into the dural membrane is a largely unknown mechanism of multiple fail-safes. By virtue of its anatomical location, the trigeminal nerve has a unique responsibility in monitoring proprioceptive and nociceptive afferents of both supra and infra tentorial structures.

Microscopic examination has shown that the sagittal suture is well vested with mechanoreceptors monitoring shearing and compressive forces.[8] The trigeminal system by monitoring changes in sutural effacement, indirectly controls up- regulation and down-regulation of CSF production in a rhythmic manner. Efferents disseminate through nerve tracts running the length of the falx cerebri and ultimately terminate in the

Choroid Plexus. Upledger states that "the system seems to operate on a six-second cycle." Three seconds of production followed by three seconds of idle gives us the rhythmical change in fluid pressure.[8]

In contrast, the outflow of CSF is in part regulated by the transmission of dural tension along the straight sinus or "Sutherlands Fulcrum." This area is a juncture of the falx cerebri, the two leaves of the tentorium cerebelli and the falx cerebelli below. At its most anterior portion, the straight sinus has a large congregation of arachnoid granulations which seem to be quite unique in their role. Upledger feels that due to dural continuity, tension from anywhere within the system is transmitted to this area. He goes on to state "the arachnoid granulation bodies clustered there seem to have the ability to increase or decrease the general rate of reabsorption of CSF from within our semi-closed hydraulic system." Thus the outflow portion of the system is a product of the CSF reabsorption and deposition into the venous system.

Ultimately it seems that the trigeminal nerve is a central regulator of the inflow and outflow pumps within the CNS. Due to its sutural investment and dural networking, the trigeminal system has a unique responsibility in maintenance of CSF pressure and ultimately the craniosacral respiratory mechanism. This auto-regulation not only involves fluid mechanics but most importantly dural tension and the reciprocal tension membrane.

Discussion

The dynamic changes that take place within the dural kinematic chain is best illustrated by the far reaching neurological impact the trigeminal nerve has on afferent regulation within and outside the cranial vault. By integrating itself with the cranial outflow via brainstem nuclei, and through the continuation of the spinal trigeminal tract into the upper cervical spine, the trigeminal nerve positions itself as an critical integrative center. This anatomical distinction is the key component in the tension constant seen throughout the dural train.

The Trigeminal Nerve and Dural Integration

Maintenance of the reciprocal membrane is firmly based on a closely monitored proprioceptive system. From its three divisions, the trigeminal nerve contributes to the majority of afferent input from the head. Supratentorial structures including the dura of the anterior and middle cranial fossa, mucous membranes of the mastoid air cells, and the calvarium receive innervation from both V2 and V3, while a small recurrent branch of V1 supplies the tentorium cerebelli.[5] Posteriorly, we see the cranial fossa receive innervation from both the vagus nerve and the sinuvertebral nerve of the upper two cervical segments.

It is important to note that in addition to nociception, these areas also provide proprioceptive and mechanoreceptive input from sutural buttressing and dural tension. Additionally, when we consider the contribution of mechanoreceptors in the TMJ and spindle/GTO afferents from the muscles of mastication and cervical spine, we find that the stomatognathic system has many layers of integration. This is a vital component in the trigeminal nerve's intermediary role in the reciprocal tension membrane.

Due to its direct monitoring of supra tentorial structures and its association via the interneuronal pool within the spinal trigeminal tract of the upper three cervical segments, the trigeminal nerve has afferent accountability of the cranium as a whole.

This anatomical correlation seems to be the driving force behind the perpetuation and maintenance of the craniosacral rhythm and ultimately the "normal" production of dural torque.

Maintenance and Correction of Dural Tension: The Fulcrum System

We have often heard the expression that the TMJ is the pump handle of the cranium; and in fact it is one of several. By virtue of dural attachments to the cranium, vertebrae and the RCPMI, several handles or fulcrums are positioned to allow for localized correction and perpetuation of dural tension. In fact a close inspection of a cranial dissection illustrates that dural torque is inherent to the system due to its twisted design allowing for strategic and purposeful interdigitation within the skull. This model facilitates the perpetual multidirectional traction that is required to minutely equilibrate and adjust the membrane tension constant and ultimately CSF pressure, a direct product of trigeminal influence.

Through proprioceptive monitoring, the trigeminal system is poised to segmentally adjust cranial mechanics via the muscular pull of the mandibular sling, the suboccipital triangle and deep spinal stabilizers. By affecting local cranial tectonics and vertebral motion, the trigeminal system micro- manages motion of the cranium and ultimately the tension of the dural tube as a whole. This dynamic mechanism relies largely on a complex higher sensory and motor center as seen in the pre and post central gyri. It would seem logical that the large cortical area devoted to the TMJ and head would be involved in much more than mastication.

Systemically, if the CSF pressure is to be maintained at a relative constant, a higher level of control would be needed to coordinate the production, absorption and more importantly, the distribution of cerebrospinal fluid. Due to the narrow range of CSF pressure, it would make more sense to affect local flow by changing dural tension and position than it would to increase or decrease pressure within the system as a whole. Similar to air vents, the dural leaves serve as rudders to move or change CSF flow. And what better way to change dural positioning than by effecting specific local muscular contraction and relaxation of those muscles contacting the dura and cranium. A closer inspection of the cranial dura would find that its design is far more applicable for fluid mechanics than for compartmentalizing the nervous system. Further research into this mechanism is sure to confirm this hypothesis.

Conclusion

Neuroanatomically, the trigeminal nerve and its corresponding nuclei seem to be the cog between monitoring and effecting cranial motion. Through afferent integration of CSF pressure and dural tension , and efferent output to contractile structures, the fifth cranial nerve coordinates cranial mechanics and dural integrity. Either directly through the muscle of mastication or indirectly through interneurons of the upper cervical spine, the trigeminal nerve and nuclei are largely responsible for what we term the craniosacral rhythm.

References

1. Sills, Franklin, Craniosacral Biodynamics, Volume 1 - The Breath of Life, Biodynamics, and Fundamental Skills (Berkeley, CA., North Atlantic Books., 2001).

2. Walther, David S., Applied Kinesiology Synopsis, (Pueblo, CO, Systems DC, 1988).

3. Greenman, Philip E., Principles of Manual Medicine, 2nd Edition, (Baltimore, MD, Williams & Wilkins, 1996).

4. Duffy, Daniel H., The Basic Course, (Geneva, OH, AK Printing, 1998).

5. Moore, Keith L., Clinically Oriented Anatomy, 3rd Edition, (Baltimore, MD, Willimas & Wilkins,1992).

6. No author given, Anatomical Relation Between the Rectus Capitis Posterior Minor Muscle and the Spinal Dura Mater (editorial). http://www.chiro.org.

7. Cramer, Gregory D., Darby, Susan A., Basic and Clinical Anatomy of the Spine, Spinal Cord and ANS (St. Louis, MO, Mosby-Year Book, 1995).

8. Upledger, John E., Craniosacral Therapy 1 Study Guide, (The Upledger Institute, 2000).

The Sacrospinalis Stress Receptor

Timothy D. Francis, D.C., F.I.A.C.A., DIBAK, M.S., D.H.M.

Abstract

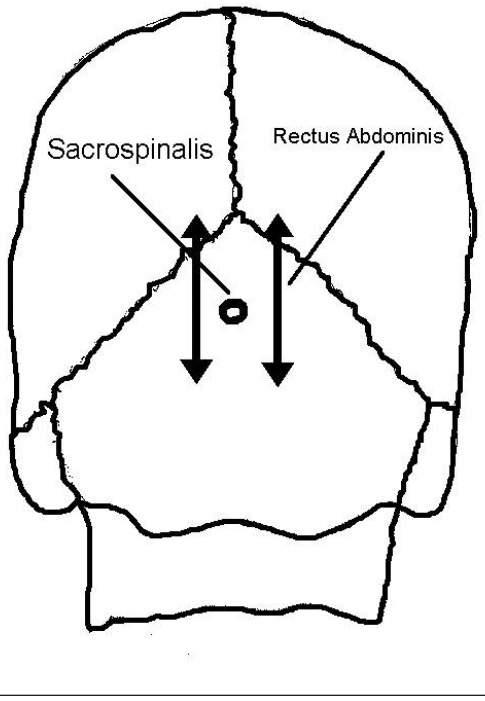
Cranial stress receptors were among the first reflexes to be used in applied kinesiology. Each of these reflexes correspond to a particular muscle/organ and are turn on/off switches. The stress receptor for the sacrospinalis is on the occipital bone.

Key Indexing Terms: Cranial stress receptor, applied kinesiology.

Introduction

Stress receptors have an influence on the corresponding muscle/organ. These may become active due to head trauma and/or emotional/biochemical trauma. The sacrospinalis stress receptor is on the occipital bone above the external occipital protuberance and below the lambda.

Figure 1



Discussion

Historically stress receptors have been thought to become active from head trauma. To reset the stress receptor, one takes a strong indicator muscle and challenges the stress receptor in a linear fashion on the scalp with digital pressure until the strong indicator muscle weakens, find a phase of respiration that negates the weakening effect and treat in the positive challenge direction on the phase of respiration that negated the challenge for four to five repetitions. It is this author's experience that emotional and/or chemical trauma will also initiate an active stress receptor. There is a stress receptor discovered located on the occipital bone below the lambda and above the external occipital protuberance between the stress receptors for the rectus abdominus. This particular stress receptor is in a circle which means the therapeutic directional force used can be in any direction. (Figure 1)

Conclusion

There exists a stress receptor for the sacrospinalis which exists on the occipital bone between the lambda and the EOP, between the stress receptors of the rectus abdominus in the shape of a circle. Stress receptors may be activated by physical, chemical, and/or emotional trauma.

Resources

Goodheart, George J. You'll Be Better, The Story of Applied Kinesiology. AK Printing. Geneva, Ohio.

Leaf, David, Applied Kinesiology Flow Chart Manual, Third Edition Private Published. (1995).

Walther, David., Applied Kinesiology Synopsis, Systems DC, Pueblo, Colorado. (1988).

The Extraordinary Meridians

Timothy D. Francis, D.C., F.I.A.C.A., DIBAK, M.S., D.H.M.

Abstract

Meridian therapy has been apart of applied kinesiology since 1970. Therapy localization was discovered in 1974 and applied to the pulse points to uncover meridian imbalances. Therapy localization to the pulse points combined with lateral flexion or rotation of the spine allows access to the extraordinary vessels utilized in the field of acupuncture.

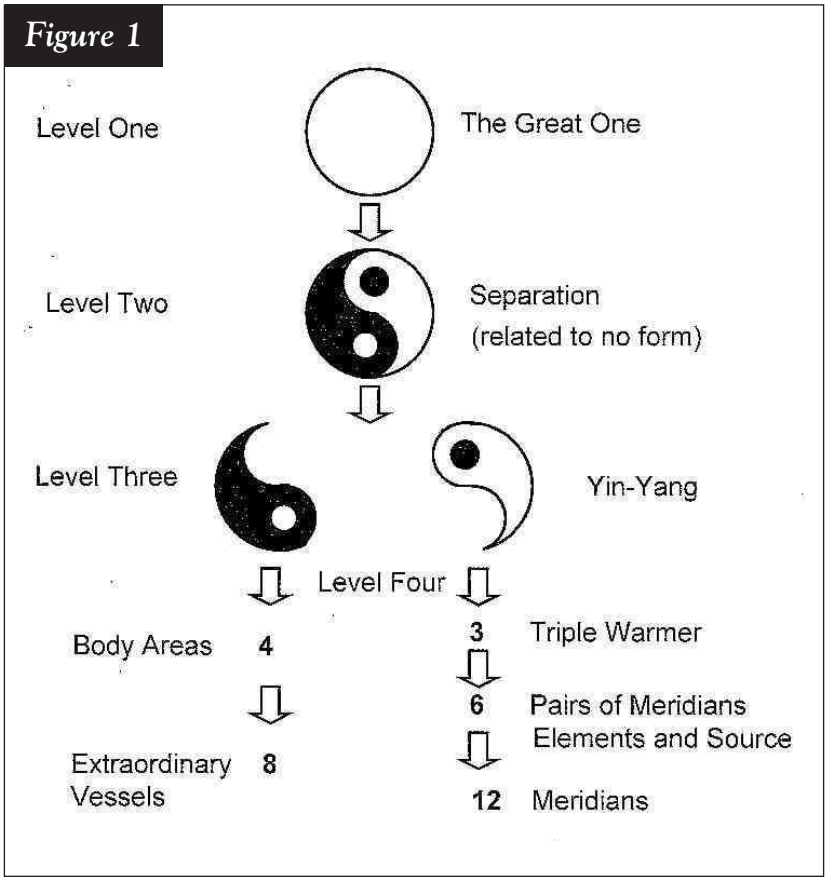
Key Indexing Terms: Acupuncture, meridian, applied kinesiology.

Introduction

The term extraordinary implies something wonderful, exceptional, rare, and unusual. It is considered in acupuncture that the extraordinary vessels are more primary than the twelve regular meridians. They are responsible for controlling, joining, storing, and regulating the Qi of the meridians.

Access to the eight extraordinary vessels is obtained by therapy localizing the pulse points with lateral flexion or rotation of the spine. The muscles related to the eight pulse points will display weakness via manual muscle testing only after they are stretched; that is a need for fascial flush. The structural corrections relate primarily to the spinal dural attachment areas and pelvic categories one and three; nutritional considerations are mineral in nature and emotionally the constitutional homeopathics are important as well as encoded memory technique.

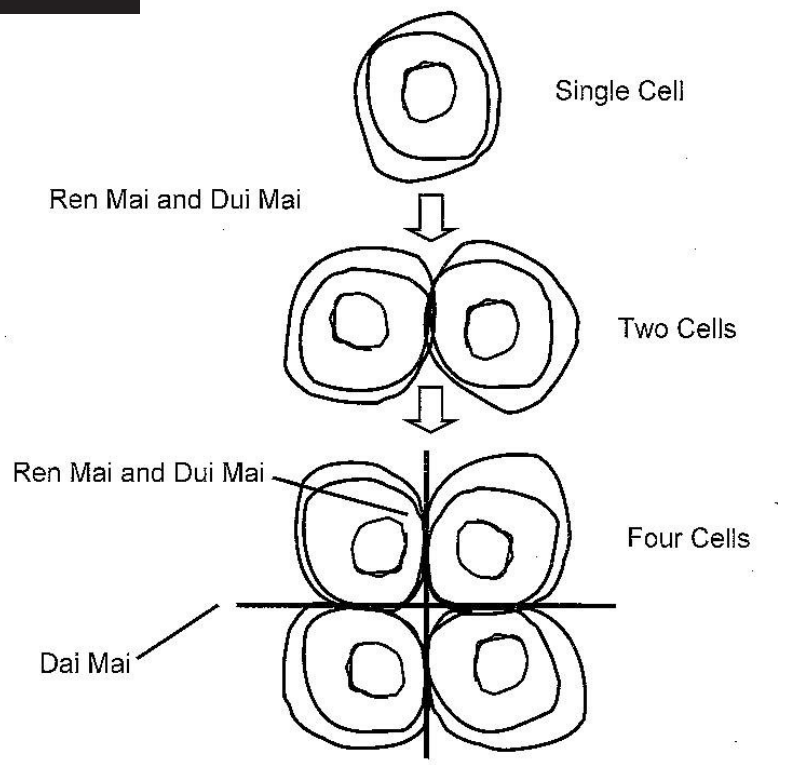
Figure 1



Discussion

Approximately one hundred thousand years ago china entered the clan commune period. It was during this period that acupuncture originated. The original acupuncture instruments were called bian and made of stone. New stone age (ten thousand to four thousand years ago) bian needles have been discovered in Mongolia and Shandong province. It appears acupuncture originated on the east coast and moxibustion in the north.

Figure 2



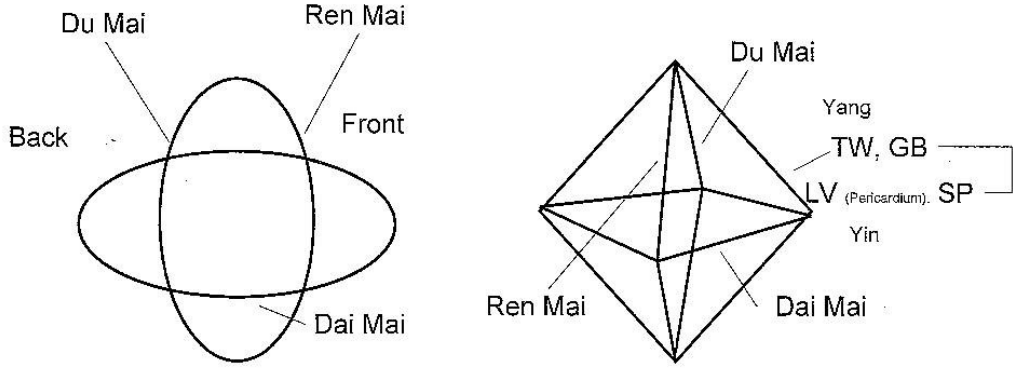
According to acupuncture theory, the cause of any disease is due to the imbalance of yin and yang. In the Miraculous Pivot it is stated "how to regulate yin and yang is most important."

The nascent of these ideas seems to have origin in the Han dynasty with the Su Wen and Ling Shu texts. During the Ming dynasty an important text called the Nan Jing appeared from which many of the systematizations follow. The Taiyi separates at the level of no form, (Figure 1) and forms the two; yin and yang. Yin and yang then separate giving rise to the eight extraordinary meridians on the one hand and the twelve meridians on the other. Therefore, all energies begin in the center. We cannot separate the energetic from the material structure in the body. This is represented by embryo development. (Figure 2)

The first division produces the ren mai and du mai meridians. The second division we have the dai mai. This is based on the work of Tohaku Ishi. Therefore, the ren mai ascends the front of the body, the du mai the back, and the dai mai encircles the middle dividing the body into anterior-posterior and superior-inferior portions. The classic yang refers to body portions as superior, posterior, and left; yin to inferior, anterior, and right. Therefore the boundary lines are yin-yang antagonisms.

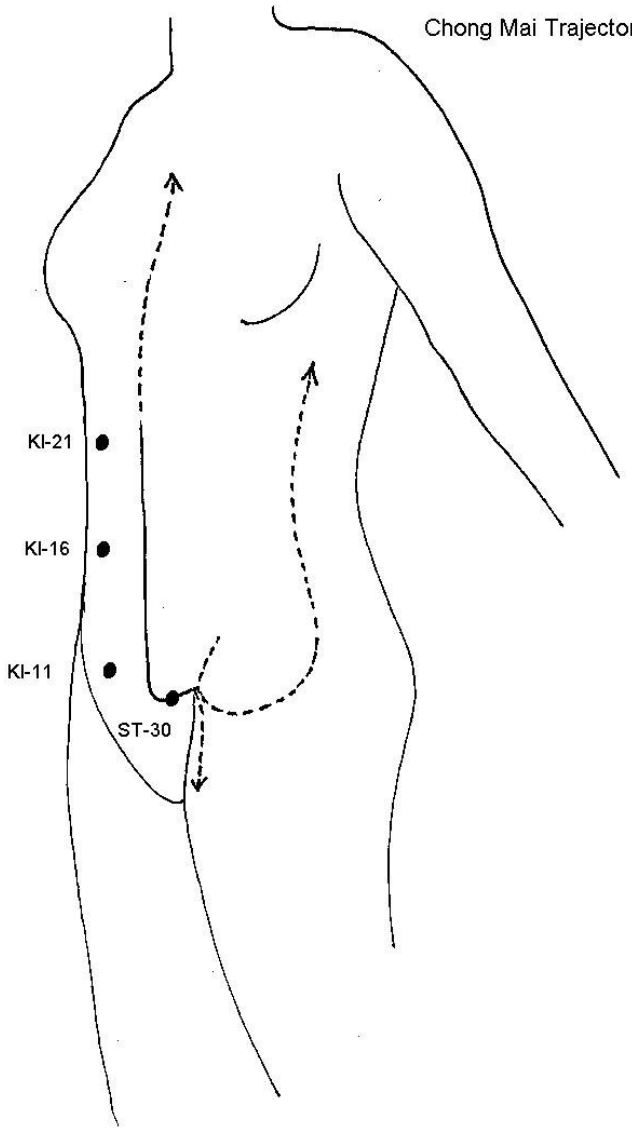
This forms topological relationships that express antagonistic-syntagonistic (yin-yang) expression of the body into octants. (Figure 3) These eight areas of the body are formed by the ren mai, du mai, dai mai, gall bladder, triple warmer, circulation-sex and spleen meridians. The gall bladder and triple warmer divide the anterior-posterior aspects of yang, the circulation-sex and spleen the yin. This relationship has eight octants corresponding to the eight extraordinary meridians. (Manaka) The point of convergence then is body balance and symmetry.

Figure 3



The du mai is the source of yang, ren mai is the source of yin, the chong mai is the source of the twelve regular meridians according to Li Shi Zhen. The ren mai, chong mai, and du mai relate to the endoderm, mesoderm, and ectoderm. (Figure 2) These extraordinary vessels are family to the formative energies of the body and are consequently at a deeper energetic level than the twelve regular meridians. Problems of the energetic center (ming men) are root problems, problems that arise in the meridians are symptomatic. The foundation of tradition in acupuncture is that great physicians treat root problems before symptomatic problems.
The root of all the body's energetic systems focus at ming men. This moving Qi between the kidneys is the source of yin-yang. Chong mai may be identified with this movement. This universal matrix of energy is sayoshi. Sayoshi is neither substance nor field, but is a tensor. A tensor is a generalized vector having function of position in an appropriate number of dimensions (interestingly another definition is a muscle that stretches a part). A tensor applied to a region is a tensor field. Therefore sayoshi is a quantity of state in space which has function. It is an operation with no substance that has a physiological effect. Sayoshi remains in space after an object has been removed from that space and can be carried by one object to another object. This may relate to Sheldrake's concept of morphic resonance. This propagation is called molecular waves. The change made by the distortion is the function. Acupuncture is intimately involved with sayoshi.

Figure 4



Physiological integration is essential to life. The extraordinary vessels function both energetically and informationally. The body is capable of responding both internally and externally; this is an open system. An open system is a system which exchanges information with its environment. Information refers to both energy and matter and yet it is neither. It is any pattern of events in time and space that is a measure of order in a system. Order is a degree of structure in a system. This signal system (known as the x-signal system) has embryological roots, locates and measures internal and external changes, changes physiology via transmission of signals and manifests topologically within holographic principles. Information therefore equals Qi! "Qi does work, is the result of work done, and is the medium of regulation."(Manaka) The extraordinary meridians function to regulate the body as fields within fields both in the sense of what does work and what regulates signals.

"Fields are non-material regions of influence." (Sheldrake) Gravitational fields are <u>not</u> in space and time, they <u>are</u> space – time. Fields are the medium of action at a distance. Electromagnetic fields are essential to

Figure 5

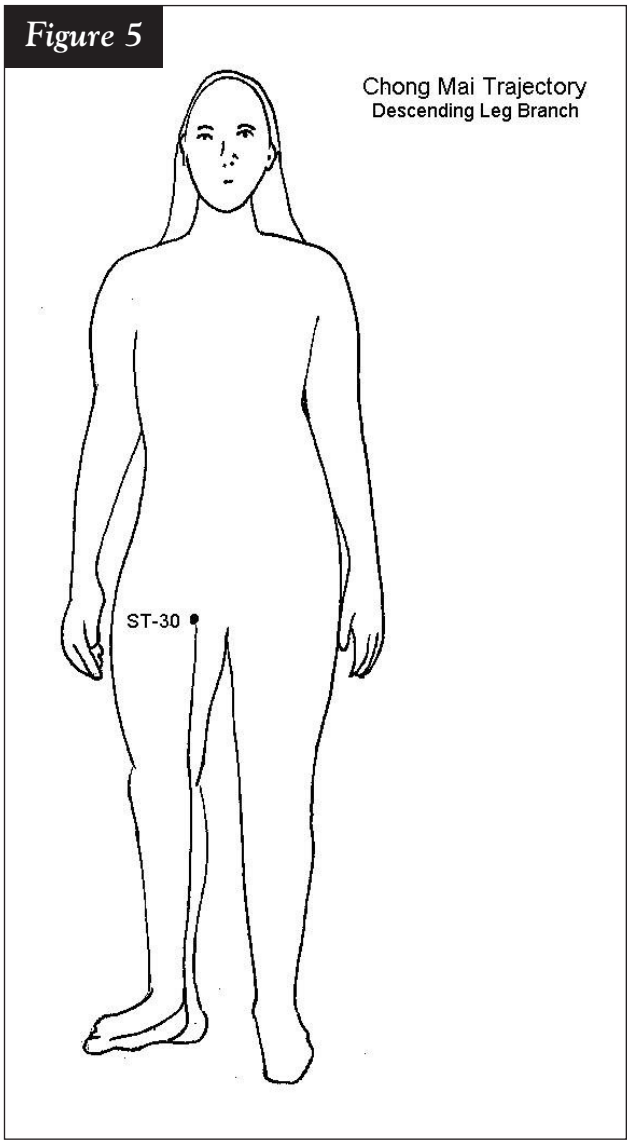
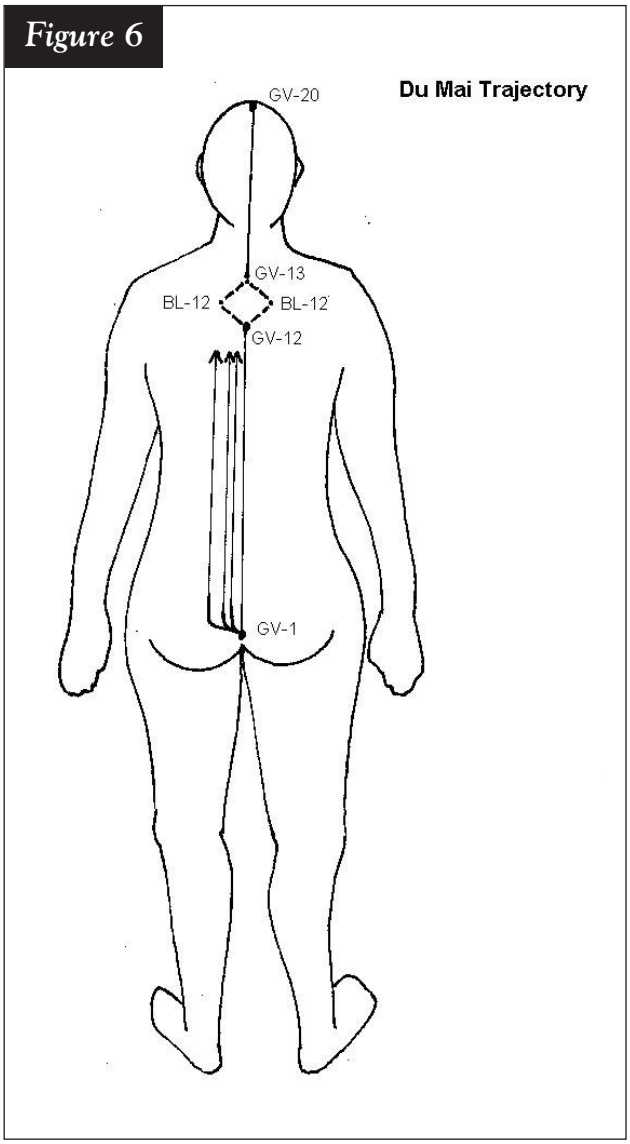


Figure 6



the function of our bodies. These fields affect each other even though they are not in contact. The fields around the heart, brain, muscles, and retina can be measured with magnetometers. The field is a description of the force that is exerted upon the object. The five elements is a description of a field where excess and deficiency change the distribution of energy within the field. (Matsumoto and Birch) Fields are more fundamental than matter. "Morphic resonance occurs between rhythmic structures of activity on the basis of similarity and through this resonance pass patterns of activity influence the fields of subsequent similar systems." (Sheldrake) This relates to isophasality (after Manaka) in which acupuncture points have something functional in common with all other points on the body that are in the same phase. This may be thought of as a homaccord in homeopathy, (the same remedy with different potencies; for example 6x, 12x, 24x, etc.) or an inversion of a musical chord or all the earth points in the chart of command points. This level of no-form is extremely energetic and informational. We can affect this energetic relationship by realizing the energies produced in the ming men are the main energetics of meridian Qi.

The eight extraordinary meridians are the chong mai, du mai, ren mai, dai mai, yin qiao, yang qiao, yin wei mai, and yang wei mai. (Figures 4 – 12) The chong mai is the ocean of the twelve meridians as it nourishes each of the yin and yang organs. It begins inside of the uterus (the moving Qi between the kidneys – approximately one inch below the umbilicus) and ascends internally in front of the spine, exits at ST-30 ascending

Figure 7

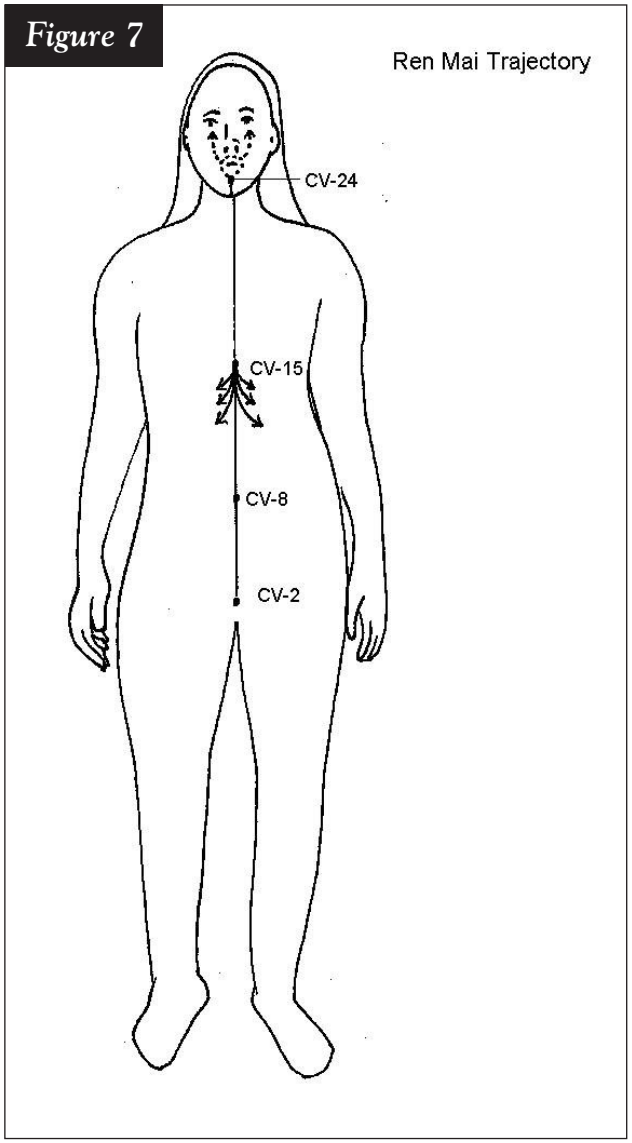
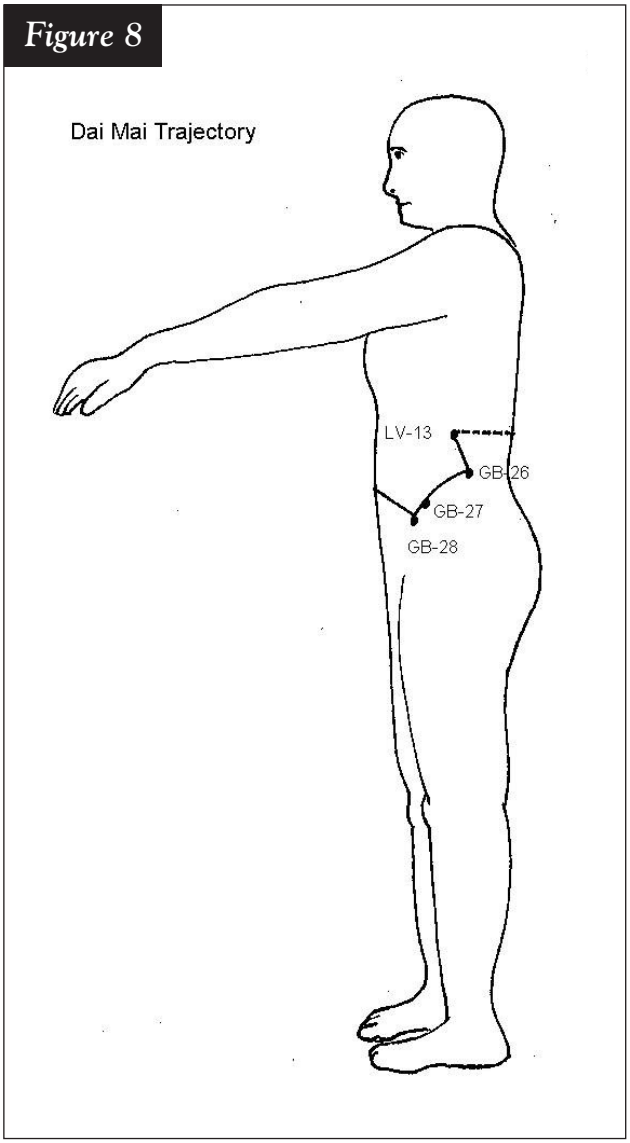


Figure 8



from KI-11 to KI-21 and finally to the mouth; descends to the feet passing through the three yin foot meridians. The chong mai is the general of the twelve meridians and according to Wang Shu He in his Maijing, Classic of the Pulse, "the chong and the du mai combined are the 'way' of the twelve meridians. If the chong and du mai do not function correctly, the twelve meridians do not return to the great meeting of the vessels. (LU-9)" (Figures 4 and 5)

The du mai (governing vessel) can control all vessels and may be considered the central ruler of the yang meridians. It too begins internally inside the uterus. The anterior branch exits at CV-1 following the ren mai passing to the genital organs, the posterior branch exits at GV-1 and ascends up either side of the spine sending branches to both scapula. (Figure 6)

The ren mai (conception vessel) is the ocean of the yin meridians. It starts in the uterus exiting at CV-1 ascending the anterior of the body to CV-2 through CV-24 surrounding the lips and ending at ST-1 (Figure 7).

The dai mai is like a belt that commands all the other meridians by loosening or tightening. It starts at LV-13 and passes through GB-26, GB-27, and GB-28. It is considered that the rectus abdominus is related to the dai mai vessel (Figure 8).

Figure 9

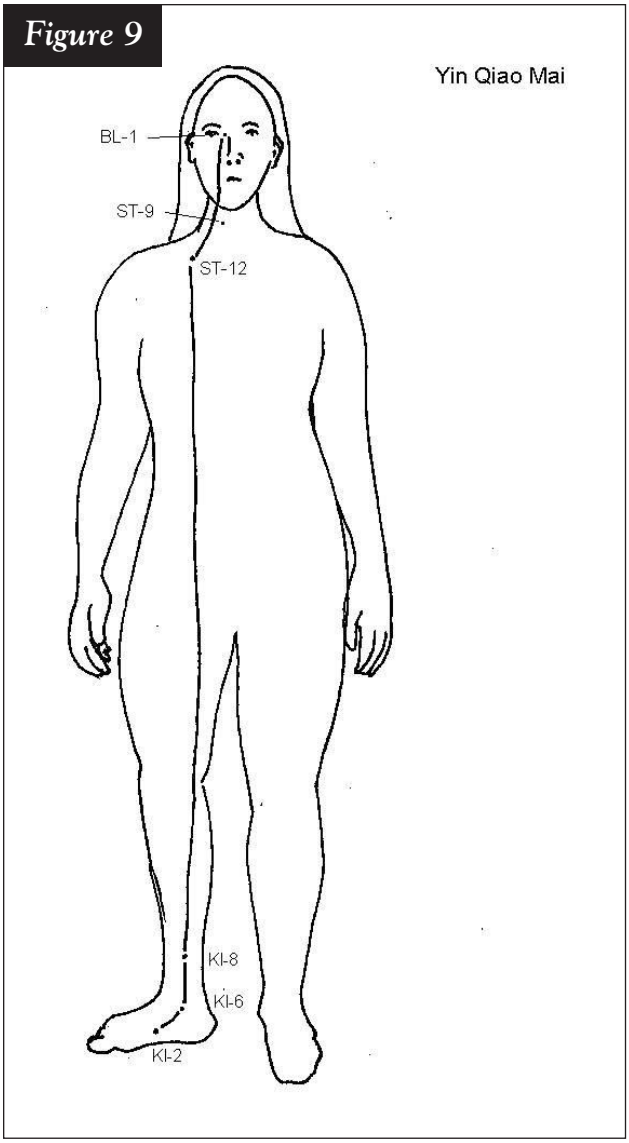
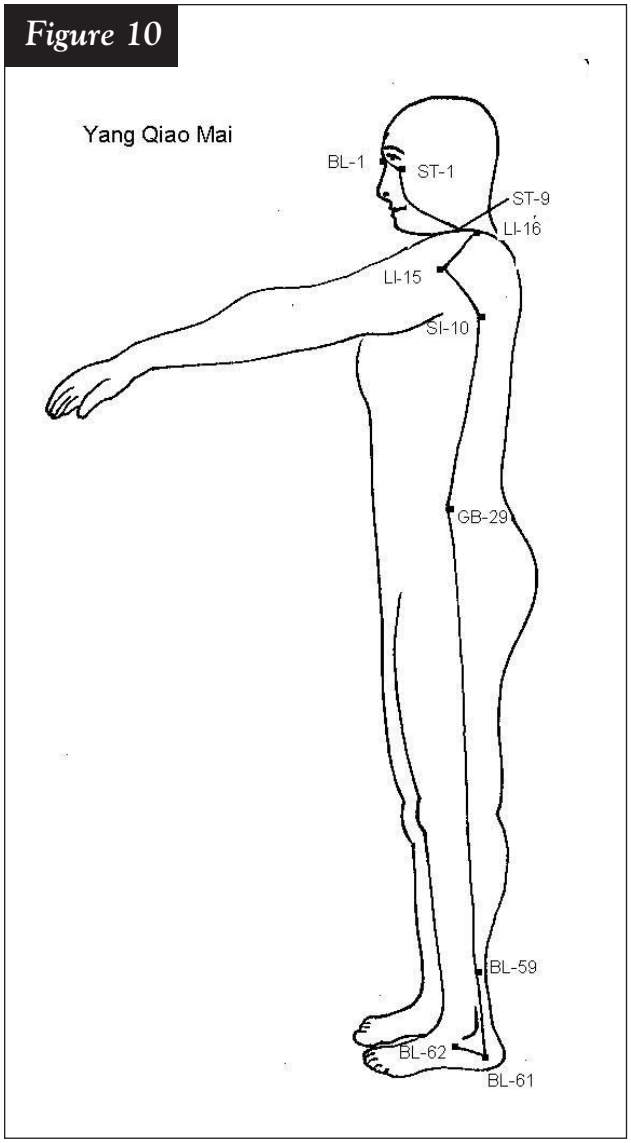


Figure 10



The yin and yang qiao mai meridians display the concept that "yang enters the yin, and the yin comes out to the yang." (Ma Shi) Remember that the nerve tracts from one side of the body enter the opposite side of the brain. The qiao vessels divide from the kidney meridian. The yin qiao mai then starts at KI-2 to KI-6, KI-8, up to ST-12 and ending at BL-1 (Figure 9) The yang qiao mai begins at BL-62 to BL-61, BL-59, GB-29, SI-10, LI-15, LI-16, ST-9, ST-4, ST-3, ST-1, and finally to BL-1. (Figure 10) Therefore remember that the bladder meridian divides in the brain to become the yin and yang qiao mai which cross and emerge at bladder 1.

The yang wei mai begins at the meeting of the yang vessels and the yin wei mai begins at the yin crossing according to the Nan Jing. Indicating the general function of dividing the left and right parts of the body. The yin wei mai starts at KI-9 to SP-13, SP-15, LV-14, and combines at CV-22 (from the left and right sides of body) travels together to CV-23 and ends on the forehead. (Figure 11) The yang way mai starts at BL-63 and travels up the outer part of the leg to GB-35, GB-29, LV-14, TW-13, TW-15, GB-21, SI-10, GB-20, GB-19, GB-18, GB-17, GB-16, GB-15, GB-14, to end at GB-13. (Figure 12)

These extraordinary meridians are related to the eight pulse points as follows: on the left wrist the distal to proximal points are the du mai, dai mai, yang qiao mai, and yang wei mai; and on the right wrist from distal to proximal are the ren mai, chong mai, yin qiao mai, and the yin wei mai. (Figure 13) The muscle correla-

Figure 11

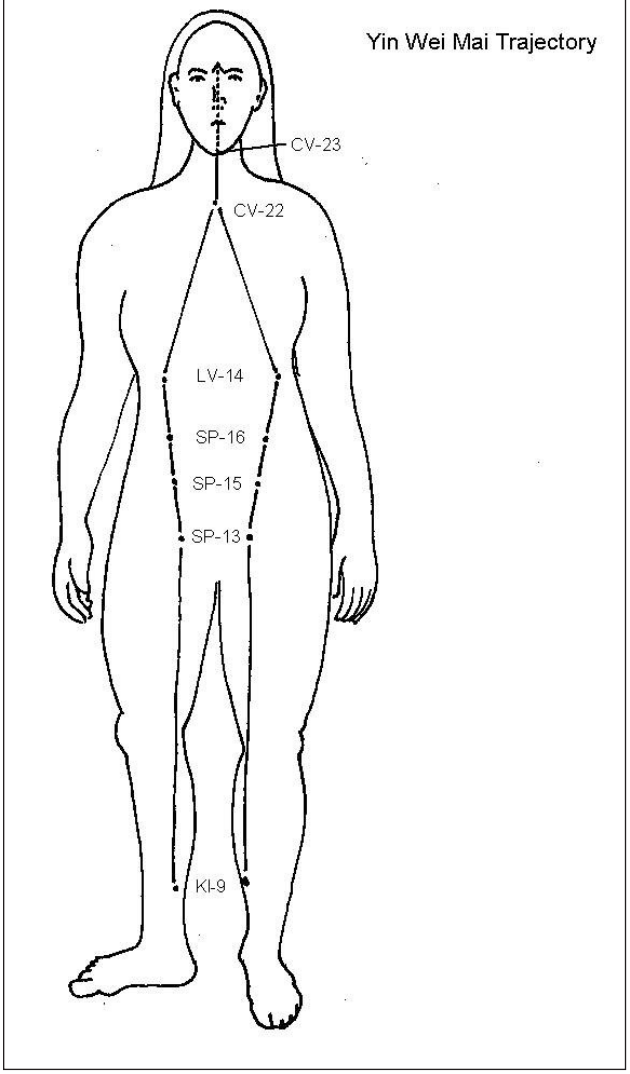
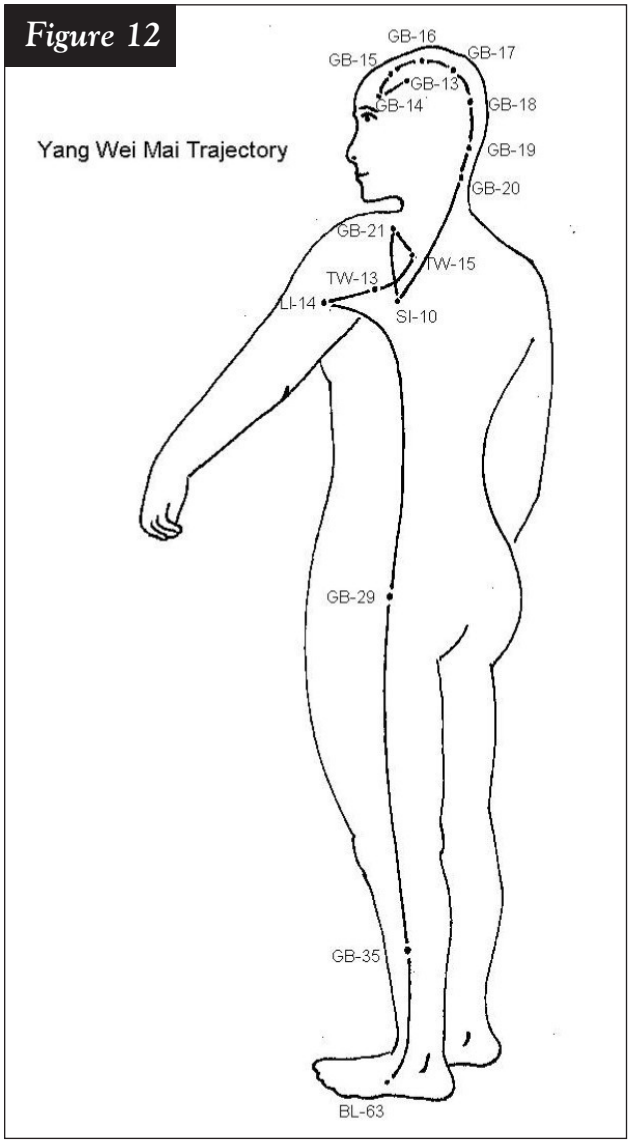


Figure 12

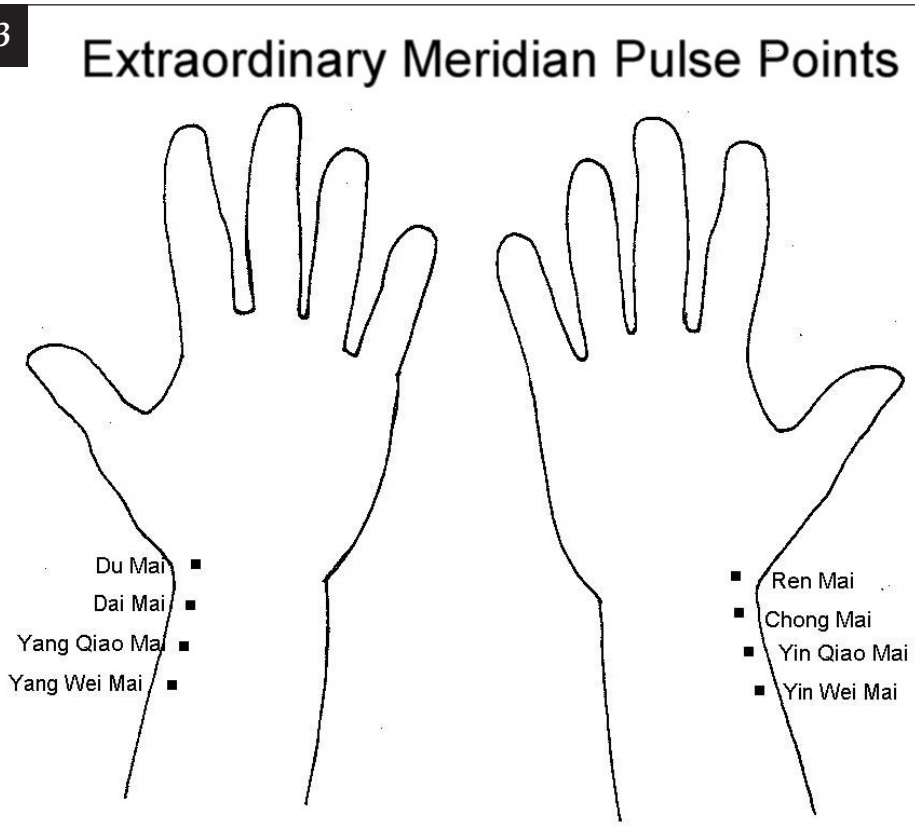


tions to the pulse points remains the same, however the muscles only display weakness after being stretched, and it is noted that all the element muscles will display this function. (For example, if the wood point therapy localizes with lateral flexion or rotation of the spine, then both the yin and yang meridian related muscles such as pectoralis major sternals, rhomboids, and popliteus muscles weaken on both sides of the body after a gentle stretch is applied.) The muscle stretch reaction will be negated by the patient therapy localizing the following corresponding points: du mai – GV-1, dai mai – LV-13, yang qiao mai – BL-1, yang wei mai – GB-14, ren mai – CV-24, chong mai – KI-27, yin qiao mai – ST-12, and the yin wei mai – CV-22.

The traditional methodology includes pulse point palpation, palpation along the meridian trajectories, usually using the thumbs beginning with a lighter pressure and progressing to a deeper pressure. Using lists of symptomatology, topological relationships, palpation of the meeting points, and abdominal palpation considered by some to be the most important component.

Classical treatments included treating the master point first and then its coupled point. (Figure 14) After the points were inserted, breathing exercises were prescribed according to the Daoist traditions. The side of the body the needles were inserted depended upon palpation, location of the symptom, and the side of the body effected. If the patient was not responding as expected, the He points (points located around the elbows and

Figure 13

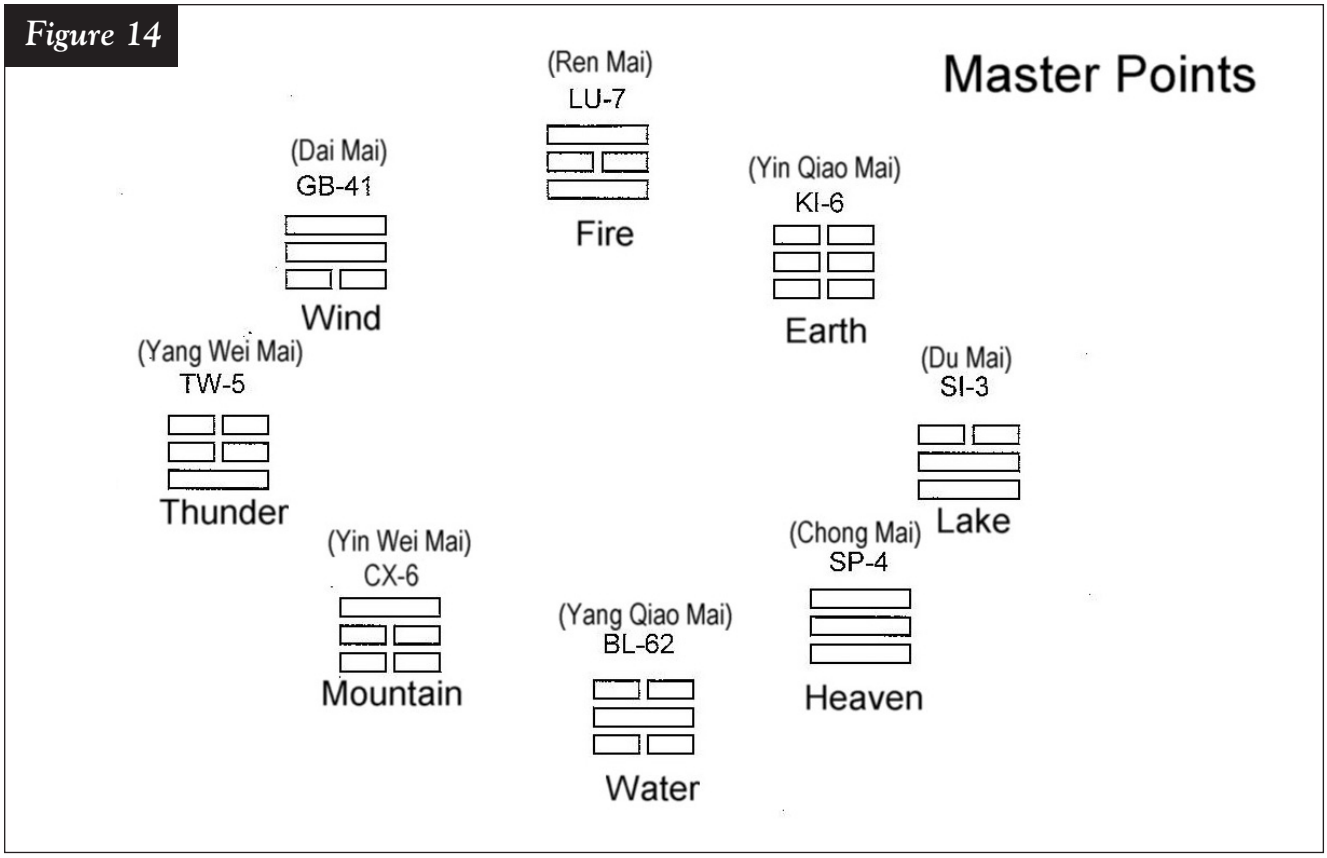


knees) were treated as well. Sometimes specific exercises were added to the treatment protocol after the needling was completed. Other practitioners also utilized herbal therapies in addition to needling of the points and sometimes in combination with other local points.

The master point correlations are as follows: chong mai – SP-4; du mai – SI-3; ren mai – LU-7; dai mai – GB-41; yin qiao mai – KI-6; yang qiao mai – BL-62; yin wei mai – CX-6; yang wei mai – TW-5. (Figure 14) These points are also sometimes called the respectable points and related to the I Ching's trigrams. Trigrams denote a heaven – person – earth relationship according to Daoist beliefs. This reflects the macrocosmic – microcosmic Chinese philosophy and the interaction of a triad of fields that is both energetic and informational as described earlier. (similar to a step – down transformer) These relationships are as follows: SP-4:Heaven; SI-3:Lake; LU-7:Fire; GB-41:Wind; KI-6:Earth; BL-62:Water; CX-6:Mountain; and TW-5:Thunder. (Figure 14) This may represent a kind of morse code for the body. Functionally the eight extraordinary vessels are four paired sets: chong mai (SP-4) with yin wei mai (CX-6); ren mai (LU-7) with yin qiao mai (KI-6); dai mai (GB-41) with yang wei mai (TW-5); and the du mai (SI-3) with yang qiao mai (BL-62). (According to Xu Feng) Matsumoto and Birch state that "we should not inordinately attend to the nature of the person's disease, but concern ourselves with the patient's bodily imbalances" and further "to diagnose the extraordinary meridians we should focus our attention to body balance and symmetry" and concluding "we can affect energetics and body balance by treating either meridians and organs or areas and octants."

Applied kinesiology utilizes therapy localization to the pulse points. A fourth pulse point was added by Dr. Goodheart distal to the classical distal pulse point which he relates to conception vessel/governing vessel. Usually a single pulse point will therapy localize, if multiple check the occiput for involvement. Then test the muscles related to the yin / yang meridian pair of the pulse point. Test the weak muscle found against the corresponding alarm point for strengthening. Depending on the response to the alarm point therapy localization will determine the treatment protocol. If the alarm point strengthens the weak associated muscle then check the corresponding nutrients, adjust the spine – rib heads at the associated point level as well as checking the

Figure 14



Lovett brother relationships. Stimulate the corresponding Tonification point on the ipsilateral side of weakness. If the alarm point does not strengthen the weak associated muscle, back up on the 24-hour clock (utilizing therapy localization) to see which meridian strengthens the weak muscle. The first meridian found to strengthen the weak muscle is considered to be in excess, test nutrients related to this meridian utilizing the weak muscle, adjust the spine – rib heads at the associated point level and check the Lovett brother relationship, stimulate the luo point on the first forward adjacent meridian (according to the 24-hour clock) on the ipsilateral side of the weak muscle. If there are no weak muscles related to the pulse point, go forward on the Sheng cycle according to the five elements. If the weak muscle is found on the first forward element, treat the element point (the pulse point that therapy localized) on the deficient meridian according to the chart of command points. (this happens to be its own tonification point) If the weak muscle is not found on the first forward element via the Sheng cycle, then go backwards on the Ko cycle, and again treat the element point on the deficient meridian according to the chart of command points. Nutrients given are related to the element point. This is a brief synopsis of applied kinesiology diagnostic – treatment protocol related to acupuncture.

To access the extraordinary vessels via applied kinesiology methodology we must therapy localize the pulse points in the clear first, that is, without lateral flexion or rotation of the spine and correct what is found as previously discussed. We next therapy localize the pulse points in a gait position, and if an imbalance is found (a pulse point therapy localizes) treat according to Anderson – Goodheart protocol. (musculotendinous meridians) Therapy localization to the pulse points in flexion or extension of the spine relates to umbilical reversal. (See the Fitness of Human Nature by this author) Finally, therapy localize the pulse points in lateral flexion or rotation of the spine.

If the pulse points therapy localize in lateral flexion, check the sacrum for a subluxation. There appears to be a correlation from distal to proximal pulse points and superior to inferior involvement on the sacrum (the most distal pulse points – ren mai and du mai relate to the S1 area, the chong mai and dai mai relate to S2,

yin qiao mai and yang qiao mai relate to S3, and yin wei mai and yang wei mai to S4). Find a phase of respiration that negates the challenge on the sacrum and adjust the sacrum in the challenged direction on the proper phase of respiration that negated the challenge while the patient visualizes the affiliated emotion or feeling. (Encoded Memory Technique) This procedure is greatly enhanced by having the patient insalivate the mineral that corresponds to the pulse point. The pulse point – mineral association has been discovered as follows: ren mai and du mai – cobalt, chong mai and dai mai – silver, yin qiao mai and yang qiao mai – zinc, yin wei mai and yang wei mai – selenium. Supplementation with these minerals is not necessary. The standard muscle – meridian nutrient correlation still applies. Next check the lovett brother relationship to the sacrum (occiput) for a subluxation and correct on the proper phase of respiration. Finally check for a Category 1 involvement of the pelvis.

If the pulse points therapy localize in rotation, check the upper cervicals for involvement. The following relationships have been found: ren mai and du mai – occiput, chong mai and dai mai – atlas, yin qiao mai and yang qiao mai – axis, yin wei mai and yang wei mai – C3. Adjust in the challenge direction on the phase of respiration that negated the challenge. Check the lovett brother association and correct as found. Check and correct a Category 3 involvement of the pelvis if found.

Retherapy localize to the pulse points as found in lateral flexion or rotation and they should now be negative. Supplement with the appropriate nutrients; herbs, homeopathics (the homeopathic remedies related are constitutional in nature), and/or nutrition.

Additional correlations have been found regarding the extraordinary meridians and the holographic spine. Ren mai relates to red and the musical note C, chong mai to orange and D, yin qiao mai to yellow and E, yin wei mai to green and F, yang wei mai to blue and G, yang qiao mai to indigo and A, dai mai to violet and B, du mai to black and C, making ren mai and du mai isophasal as discussed earlier.

The extraordinary pulse points will not therapy localize in the clear unless lateral flexion or rotation of the spine is utilized, however two pulse points therapy localized at the same time on the same side will display positive therapy localization which confirms the classical four pairing of the eight extraordinary meridians. For example, if involved the ren mai and yin qiao mai will therapy localize together, chong mai and yin wei mai, du mai and yang qiao mai, dai mai and yang wei mai. Of course this is the classical relationship of the four paired sets.

The master points will not therapy localize to negate the positive associated muscle stretch reaction. However the points mentioned earlier discovered by this author will. Again these points are CV-24 for ren mai, KI-27 for chong mai, ST-12 for yin qiao mai, CV-22 for yin wei mai, GV-1 for du mai, LV-13 for dai mai, BL-1 for yang qiao mai and GB-14 for yang wei mai. The associated classic alarm points related to the twelve regular meridians will only therapy localize with either lateral flexion or rotation of the spine according to how the pulse points displayed. In addition, both sides will therapy localize and both yin-yang relations.

Conclusion

"Extraordinary meridians are fields within fields that are capable of changing both the distribution and quality of energy used in the body and changing the signals that control the use of that energy." (Matsumoto and Birch) The energy transfer is of a qualitative and informational nature known as sayoshi. This is based on the knowledge that all energy begins in the center at ming men (the moving Qi between the kidneys), and it is not possible to distinguish the energetic structure from the material structure of the body.

These eight extraordinary meridians are related to the pulse points as yin on the right wrist and yang on the left wrist which only therapy localize while maintaining lateral flexion or rotation of the spine. The observed phenomena (muscle stretch reaction) will only display for approximately three minutes after therapy localization. The combination of structural correction (at the dural attachments of the spine) with emotional (encoded memory technique with associated mineral insalivation) and biochemical supplementation provide an effective holographic approach.

Resources

Becker, Robert O. & Selden, Gary. The Body Electric. Quill, New York (1985).

Becker, Robert O., Cross Currents. Tarcher/Perigee. New York (1990).

Capra, Fritjof. The Tao of Physics. Shambhala. Boston. (1991).

Daintita, John & Nelson, David. Dictionary of Mathematics. Penguin Books. London. (1989).

Francis, Timothy D., The Holographic Spine, Experimental Observations of the ICAK-USA, Volume 1 (2002-2003).

Ibid, The Fitness of Human Nature, Experimental Observations of the ICAK-USA, Volume 1 (2003–2004).

Gerber, Richard. Vibrational Medicine, Bear and Company., Santa Fe, New Mexico (1998).

Isaacs, Alan. A Concise Dictionary of Physics. Oxford. (1985).

Goodheart, George J., You'll Be Better, The Story of Applied Kinesiology, AK Printing, Geneva, Ohio.

Leaf, David, Applied Kinesiology Flowchart Manual, Privately published, Plymouth, MA, (1995).

Manaka, Yoshio; Itaya, Kazuko; & Birch Stephen. Chasing the Dragon's Tail. Paradigm. Brookline, MA. (1995).

Mann, Felix. Acupuncture: The Ancient Chinese Art of Healing and How It Works Scientifically. Vintage. (1962).

Manning, Clark A. & Vanrennen, Louis J. Bioenergetic Medicines East & West: Acupuncture and Homeopathy. North Atlantic Books. (1988).

Matsumoto, Kiiko & Birch, Stephen. Extraordinary Vessels. Paradigm, Brookline, MA. (1986).

McTaggart, Lynne. The Field. Harper Collins (2002).

Sheldrake, Rupert. A New Science of Life. Park Street Press. Rochester, Vermont. (1995).

Ibid, The Presence of the Past, Park Stress Press, Rochester, Vermont. (1995).

Walker, Brian Browne. The I Ching or Book of Changes. St. Martins Press. New York (1992).

Xinnong, Cheng. Chinese Acupuncture and Moxibustion. Foreign Languages Press. Beijing. (1987).

Youbang, Chen & Liangyse. Essentials of Contemporary Chinese Acupuncturist's Clinical Experiences. Foreign Languages Press. Beijing. (1989).

A Newly Discovered Muscle-Organ Relationship: The Pectoralis Minor and the Parotid Gland

Stephen C. Gangemi, D.C.

Abstract

The relationship between muscle dysfunction and organ or gland dysfunction has been a foundation of applied kinesiology since Goodheart began correlating the two in the early years of AK. There has been no previous consensus on a specific organ or gland relationship with the pectoralis minor muscle observed previously with consistency. The direct relationship of this muscle to the parotid glands will be proposed.

Key Indexing Terms: Parotid gland, immune system, applied kinesiology.

Introduction

The pectoralis minor muscle originates from the 3rd, 4th, and 5th ribs near the costal cartilage and inserts on the coracoid process of the scapula. Testing this muscle is usually done one of two ways. One test is to have the supine patient lift the shoulder of the tested side off the table while the doctor directs pressure to try to elongate the pectoralis minor fibers.[1] A second variation of the test explained by Beardall is to have the patient flex the arm 45 degrees across the body while keeping the elbow straight and the humerus at full external rotation while the doctor applies pressure on the forearm to try to abduct the arm.[2]

The parotid glands are the largest of the three main salivary glands. They overlie the mandibular ramus and are anterior and inferior to the external ear, one on each side. The function of the parotid glands is to produce serous fluid (saliva), secrete ptyalin (also know as amylase) for carbohydrate digestion, and also to stimulate the thymus gland to produce T cells for proper immune function. Goodheart also notes that the parotid works with the thymus to "tag" food for specialized use in the body[3] as well as deiodinate food in the mouth so that it is readily available for the thyroid after gastrointestinal absorption. The immune relationship of the parotid is clearly observed in diseases such as Sjogrens Syndrome and mumps. Saliva from the parotid glands typically contains 30-160 ug/ml of IgA immunoglobulin.[4]

Discussion

The visceral referred pain (VRP) areas are very important and useful in understanding muscle-organ relationships and how to properly treat a visceral imbalance. Activating a VRP area with some type of sensory stimulation, usually rubbing (mechanoreceptor stimulation) or pinching (nociceptor stimulation), will elicit a muscle response if the VRP is related to the organ with a problem. The VRP areas are extremely useful in guiding the physician whether to perform more sympathetic (pinching strengthens) or parasympathetic (rubbing strengthens) activity.[5]

The VRPs of the parotid glands are directly over the glands themselves. Rubbing over the parotid VRP will strengthen an inhibited ipsilateral pectoralis minor if there is a need for more parsympathetic activity. This also signifies the need for to rub Chapman's neurolymphatic reflex (NL). The NLs for the parotid glands, observed by Goodheart, are parasternal bilaterally at intercostal spaces 2, 3 and 4. If pinching over the parotid VRP strengthens the inhibited pectoralis minor then a need for more sympathetic activity is indicated. This is very common, as the parotid glands tend to act like sponges, interacting with toxins such as heavy metals, food allergies, and certain medications. This perhaps explains the association of pectoralis minor findings with poor lymphatic drainage.[6] The duct-associated lymph tissue (DALT) is an important part of the immune system of which the parotid is composed. The epithelial cells of the DALT in the parotids take up antigens and transport them to the adjacent immune cells.[7]

The most common stressors of the parotid glands include the following: heavy metals, food allergies and intolerances, chemical sensitivities, (notably aldehydes, hydrocarbons, and sulfites), partially hydrogenated (trans) fats, hormonal issues, and immune system issues such as viral or bacterial infections. This leads explanation to the common findings already known with the pectoralis minor, many of which have been observed by Schmitt: the need for molybdenum and selenium (chemical sensitivities)[8] – the need for essential fatty acids (trans fat intake) – the need for niacinamide (thymus and hormonal stress) – the need for vitamin A (thymus and thyroid gland nutrition) – and the need for antioxidants (heavy metal toxicity and food allergies).[9]

Conclusion

The pectoralis minor muscles have a direct relationship with the parotid glands. This is verified by correlation of the parotid glands VRPs, the NLs of the parotid glands, and the specific role of the parotid glands in regards to an individual's health and the findings associated with an inhibited pectoralis minor muscle when the parotid glands are compromised.

Due to the impact of proper parotid function on an individual's health, specifically the immune system and thyroid, the observation of an inhibited (or over facilitated) pectoralis muscle which previously had no organ relationship now enables the doctor to more thoroughly investigate and treat the patient leading to greater success.

References

1. Walther, David S., *Applied Kinesiology: Synopsis*, Systems DC, 1988, p. 313.

2. Beardall, Alan G., Clinical Kinesiology: Vol. IV. 2001. p. 51.

3. Walther, David S., Applied Kinesiology: Synopsis, Systems D, 1988, p. 137.

4. Kilian M. and D. Bratthall, Caries Immunology, Textbook of Clinical Cariology, Bratthall Publications, 1994, p. 307.

5. Schmitt, Walter H., Jr., "The Functional Neurology of Referred Pain", I.C.A.K. Collected Papers, Summer, 1989, p. 229.

6. Walther, David S., Applied Kinesiology: Synopsis, Systems DC, 1988, pp. 183-186.

7. Matsuda M, Ina K, Kitamura H, Fujikura Y, and T. Shimada. "Demonstration and Organization of Duct-Associated Lymphoid Tissue (DALT) of the Main Excretory Duct in the Monkey Parotid Gland." Archives of Histology and Cytology. Online. Internet. [28 January 2005]. Available www:http://www.ishc.net/summary/1997.12/summary.html.

8. Schmitt, Walter H., Jr, "Centering the Spine Functional Neurological and Biochemical Considerations", I.C.A.K. Collected Papers, Summer, 1987.

9. Schmitt, Walter H., Jr, private communication.

The Use of Low Level Laser Therapy in Treatment of Recurrent Temporal Bulge Cranial Fault with Attendant Digestive Complaints

James D.W. Hogg, D.C., DIBAK

Abstract

The Temporal Bulge cranial fault is a common finding in the practice of applied kinesiology.[1] It is usually accompanied by mild to severe digestive distress. While the standard cranial manipulation procedures used by applied kinesiologists are usually effective in eliminating this dysfunction, a minority of patients demonstrate chronic recidivism. This paper discusses the use of low level laser therapy (LLLT) to the suboccipital and retro mastoid areas as a method of improving longevity of the temporal bulge correction.

Key Indexing Terms: Laser, LLLT, temporal bulge, indigestion, hydrochloric acid, cranial adjusting.

Introduction

The temporal bulge cranial fault is a frequent encounter in clinical practice.[1] It is of special significance because of the, sometimes extreme, digestive disturbance it typically causes. In my practice I have observed gassiness and bloat after eating, greater tendency toward illeocecal valve problems, and difficulty digesting protein, calcium and iron.

The mechanism for the above reactions is thought to be inhibition to the parietal cells of the stomach via disturbed vagus nerve function. It is also common to see conditional inhibition of the muscles associated with digestive organs such as the abdominals, quadriceps, tensor fascia lata and pectoralis major clavicular division.

Although most patients respond well to the standard AK cranial adjustment for temporal bulge, some patients have chronic recurrence. The frustration inherent in such recurrence and our natural desire to help our patients has been the impetus for much advancement in technique in applied kinesiology and elsewhere. A previous paper by this author describes the importance of gall bladder meridian balance to enhance durability of temporal bulge correction.[2] Although addition of gall bladder meridian therapy has improved my results dramatically, I still was having more recurrence than I liked.

In the last two years I have become aware of the usefulness of low level laser therapy (LLLT). One of the most common applications has been the use of laser energy to improve nerve function.[3, 4, 5, 6] Although LLLT is most commonly applied to spinal nerves, it seems reasonable that cranial nerve function may also be enhanced with LLLT. Following is a discussion of my exploration of this concept as it applies to the vagus nerve and improved clinical outcomes with the temporal bulge cranial fault.

Discussion

For the sake of simplicity I will use the following conventions throughout this paper. When I am referring to a muscle that is conditionally inhibited on manual muscle testing I will refer to it as "weak". Likewise, when I am referring to a muscle that is conditionally facilitated on manual muscle testing I will refer to is a "strong".

The bilateral weakness of the clavicular division of the pectoralis major(PMC), tested simultaneously, is so common that I routinely include it in my initial exam along with the individual PMC tests. Since this bilateral weakness is considered in applied kinesiology (AK) to be associated with low hydrochloric acid and temporal bulge cranial fault,[1] it is well worth the few seconds it takes to perform. I find that a high percentage of my new patients, of all ages, exhibit this finding, with greater frequency in my older patients. This can result in serious metabolic problems for any patient but is especially serious for growing children and post-menopausal women because of the interference in protein and calcium absorption.

For most patients this problem is easily corrected via the temporal bulge cranial adjustment. A minority of patients seem to have trouble with recidivism. Various factors may contribute to this recidivism, including other cranial faults, temporomandibular joint dysfunction, neck and pectoral girdle muscular imbalances, foot and pelvic problems. As mentioned in the introduction, I have frequently found that gall bladder meridian imbalance as a contributing factor.

For several years I have been interested in the use of low level laser therapy (LLLT) for nerve regeneration and reactivation. I have been privileged to make the acquaintance of George Gonzalez, D.C. (another George!) who has developed many innovative applications of LLLT to chiropractic neurology. He was kind enough to suggest that vagus nerve function could be enhanced by applying LLLT near the base of the skull.

At the time I had just purchased a two diode 635nm, 5mw therapeutic laser with programmable pulse frequencies (Quantum 2 from A Major Difference). I also had a problem patient. She was the mother of a former AK student, and we were having quite a bit of difficulty with her digestion. She was on once a week visits for several months because of recurrent temporal bulge cranial faults with attendant extreme digestive distress.

In addition to cranial adjusting, and attention to the structural factors listed above, care involved frequent therapy for the gall bladder meridian. Although I prefer to treat low hydrochloric acid problems without resorting to supplementation of hydrochloric acid oraly, in this patient's case it was considered necessary.

Once the idea of applying LLLT to this difficult case occured to me I was excited to see what the results might be. I programmed the two laser diodes to 16 and 81 hz and found a spot at the base of the occiput which, when exposed to the laser at the above frequencies, negated the weakening effect of the patient's therapy localization to the area of the temporal bulge cranial fault. After treating the indicated location for 60 seconds, I turned off the laser and retested the patient. I was surprised and delighted to find that pectoralis major clavicular was no longer bilaterally weak. There was also now an absence of therapy localization and challenge for the temporal bulge cranial fault!

Upon retesting a week later, we were both happy to find that the temporal bulge cranial fault had not recurred. It has been several months since that time and, although she has had a few recurrences of her temporal bulge problems, she has shown dramatic improvement since the days it recurred every week!

I have found these results to be consistently repeated with other challenging patients. I have found that, If laser therapy is applied before manual adjustment, the findings for temporal bulge and bilateral pectoralis major clavicular weakness are often abolished. Recently I have discovered that, although the usual therapy localization and challenge for temporal bulge fault may be absent after laser therapy, sometimes challenging

twice, about one second apart (Goodheart's "cerebellar challenge") will yield a positive result (previously strong muscle becomes inhibited). In cases where there are positive findings either with regular challenge or the double challenge above, I finish up with the normal manual adjustment for a temporal bulge cranial fault.

Although I have gotten the best results with the equipment described above, I have gotten similar results with a simple 635nm 5mw laser pointer.

Protocol

I. Test each pectoralis major clavicular (PMC) muscle individually
   A. If weak, treat to strengthen

II. Test strong PMC muscles bilaterally, simultaneously
   A. If weak – indicates disturbed hydrocloric acid production

III. Determining temporal bulge fault
   A. Have patient touch (TL) temporo-parietal area on one side with both hands
   B. Test previously strong muscle (PSM)
      1. Weakening with TL indicates possible presence of temporal bulge fault
      2. Confirm with challenge
         a. On side of positive TL
         b. Contact patient's frontal bone with one hand and occiput with other hand
         c. Press contact points together with slight caudal torque on frontal contact, slight cephal torque on occipital contact
         d. Release contacts and test PSM
         e. Weakening – positive for temporal bulge fault
   C. If no weakening of a previously strong muscle (PSM) occurs with above procedure, repeat on opposite side of head

IV. Determining location for laser therapy
   A. Have patient TL to previously positive temporo-parietal area with one hand
   B. Test PSM – should be weak in response to TL
   C. Turn laser beam on
   D. If possible set pulse frequency to 16 and/or 81, if not just use unpulsed beam
   E. Apply laser beam to various locations along base of occiput
      1. Start from just posterior to mastoid process
      2. Test muscle that was weakened by TL (III, B above)
      3. Strengthening in response to laser beam – positive for laser treatment location
   F. Apply laser beam to positive area (above) for 60 – 120 seconds

V. Re-challenge and/or TL for temporal bulge cranial fault
   A. If positive (PSM goes weak) re-apply laser beam to see if it negates weakening
      1. If so, apply laser beam for another 60 – 120 seconds
      2. If not, continue with standard temporal bulge adjustment
   B. If negative, try double (cerebellar) challenge for temporal bulge fault
      1. If positive (PSM goes weak) proceed with standard temporal bulge adjustment
   C. Manual cranial adjustment may not be necessary after laser therapy

VI. Check for the parietal descent cranial fault and gall bladder meridian imbalance
   A. Correct as necessary to further enhance longevity of correction

Conclusion

The addition of low level laser therapy to my protocol for the temporal bulge cranial fault and associated digestive disturbances has greatly enhanced my clinical effectiveness and the longevity of correction. Current research on LLLT suggests many applications that may serve to enhance and expand the effectiveness of our standard applied kinesiology therapies. I have implemented LLLT in several of these procedures with initial promising results. I hope to report on these additional applications of LLLT in future papers.

References

1. Walther D, *Applied Kinesiology Synopsis, 2nd Edition*. Pueblo, colorado PP 390.

2. Hogg J., "Gall Bladder Meridian Imbalance in Recurrent Hydrochloric Acid Deficiency". Proceedings of the International College of Applied Kinesiology USA Summer, 1994.

3. Ehrlicher A, Betz T, Stuhrmann B, Koch D, Milner V, Raizen MG, Kas J. "Guiding Neuronal Growth with Light" Proc Natl Acad Sci U S A. 2002 Dec 10;99(25):16024-8. Epub 2002 Nov 27.

4. Gigo-Benato D, Geuna S, de Castro Rodrigues A, Tos P, Fornaro M, Boux E, Battiston B, Giacobini-Robecchi MG., "Low-power laser biostimulation enhances nerve repair after end-to-side neurorrhaphy: a double-blind randomized studyin`the rat median nerve model." Lasers Med Sci. 2004; 19(1):57-65. Epub 2004 Jul 30.

5. Anders JJ, Geuna S, Rochkind S. "Phototherapy promotes regeneration and functional recovery of injured peripheral nerve." Neurol Res. 2004 Mar; 26(2):233-9. Review.

6. Menovsky T, Beek JF. "Carbon dioxide laser-assisted nerve repair: effect of solder and suture material on nerve regeneration in rat sciatic nerve." Microsurgery. 2003;23(2):109–16.

Abnormal Muscle Testing Responses with Cerebellar Transneural Degeneration – A Case History

Datis Kharrazian, D.C., M.S., F.A.A.C.P., D.A.C.B.N., DIBAK, C.N.S., C.S.C.S., C.C.S.P.

Abstract

This case history of a 32-year-old female with multiple chronic disorders that was diagnosed and managed with cerebellar transneural degeneration.

Key Indexing Terms: Cerebellar transneural degeneration, applied kinesiology.

Introduction

The patient exhibited conditionally inhibited muscles with repeat testing that was not associated with aerobic/anerobic weakness or repeated muscle activation techniques. The patient would have a global weakness of muscles within two to three tests, with or without, a break of several seconds apart, regardless of challenge or testing in the clear. This pattern was not identified by previous applied kinesiologist management and therefore erroneous applied kinesiology diagnosis and treatments were offered to the patient.

Discussion

In applied kinesiology we tend to assume that the patient's motor system is intact, however if this is not the case then applied kinesiology muscle testing responses may not be correct. Standard muscle testing in applied kinesiology is based on a system in which the different sensory evoked stimuli with temporal and spatial summation is provided to the nervous system. The impacts of sensory evoked potentials are assessed using volitional muscle testing responses. The motor responses to the neuraxis reflect changes in the central integrative state of the anterior horn. If the evoked sensory stimuli modulates the central integrative state or anterior horn motor neuronal pools closer to sodium equilibrium potentials then we may see a conditionally facilitated muscle. If motor neuronal pools are brought closer to potassium equilibrium potentials, then we may observe a conditionally inhibited muscle. Once again the underlying premise for a muscle testing response is dependent on an integrative motor system that has the functional capacity to respond predictably through canalized and non-canalized pathways.

Canalized pathways of the anterior horn, which is considered the final common pathway, include rubrospinal, vestibulospinal, corticospinal, reticulospinal, tectospinal pathways. Ipsilateral modulation of the anterior horn also includes neocortex excitation of the pontomedullary reticulospinal tract which results in excitation of the anterior horn cells via inhibition of inhibitory interneurons termed dysinhibition. This dysinhibition pattern will allow for increased sensitivity and decreased gain of ipsilateral muscle spindles which increases the probability of large diameter afferent's proprioceptive excitation of the ipsilateral cerebellum and increased summative states of spinal cord reflexes. The pontomedullary rectiulospinal tract is also excites anterior muscles above T6 and posterior muscles below T6. The net result of the integration of these

pathways with neocortical and basal ganglionic systems results in the clinical motor responses we term as "strong or weak" or conditionally facilitated or inhibited muscles.

This case history illustrates how a pattern of a non-integrative motor system caused by a cerebellum that expressed patterns of transneural degeneration lead to non-dependable sensory evoked responses that are used commonly in the practice of applied kinesiology.

History

A thirty-two -year-old female presented with a history of depression, fatigue, nausea, anxiety attacks, vertigo, compromised digestion, abnormal menstrual cycles, infertility, multiple chemical and food sensitivities and inability to lose weight despite exercise and low calorie diet. She stated that she was perfectly healthy until age twenty-three. She had no health problems and was engaged in numerous physical activities. At age twenty-three she started graduate school in an M.B.A. program and noticed her health started to decline. She first noted episodes of fatigue and was starting to gain weight. She had a physical examination and routine blood test that found her to be healthy. Her problems were blamed on stress of graduate school. By the end of her first year she had gained twenty pounds and stated that friends were not able to recognize her anymore. She was no longer working out or physically active. She always felt like she had an injury and chronic left sided hamstring tightness and mid-back pain. She visited a chiropractor and was placed on a treatment protocol for three months. After the second month she started to develop dizziness, and nausea. She stopped her chiropractic care and but the dizziness and nausea symptoms did not improve. By age twenty-four she graduated from school and was married. Her symptoms kept progressing and she started suffering from anxiety attacks and abnormal menstrual cycles. She was also developing episodes of depression and was not able to work. In the next few years her symptoms gradually got worse. She visited different physicians during this time period and was diagnosed with irritable bowel syndrome, Meniere's, fibromyalgia and chronic fatigue syndrome. At age twenty-nine she visited a holistic minded M.D. and was diagnosed with hypothyroidism. She was placed on armour and felt improved energy and mood immediately. After the second month on armour she was back to her original symptoms. Attempts made to increase her dosage and to switch to other thyroid hormones did not help. Eventually she stopped taking thyroid hormones because it was not changing how she felt. She even mentioned she thought it was making her sicker. Between the age of twenty-eight and thirty-two she visited many alternative healthcare providers and tried numerous nutritional, detoxification and homeopathic programs. At age thirty she went on a detoxification fast with limited foods for two weeks and after finishing the program she could no longer tolerate any nutritional supplements or even homeopathic remedies. She was unable to digest most of them and when she was able to digest them, she felt immediate nausea, fatigue and occasional headaches after consumption. Her last visit before she presented to our office was to a chiropractor applied kinesiologist. Initially she felt better with treatments, but after her third visit she started to notice an increase in nausea and headaches. She was referred to our office for a second opinion.

Laboratory Testing History

The patient had numerous lab tests conducted before presenting to our office. An adrenal salivary test performed last year indicated altered adrenal circadian rhythm. The findings of suppressed pooled DHEA, elevations of night time cortisol and with depression of morning cortisol were consistant with adrenal maladaption stage II.

A comprehensive digestive stool analysis was conducted approxiamtely eighteen months ago that revealed infections with ameba histolytica and cryptosporodium. The total secretory IgA was severely suppressed.

A female hormone panel conducted over 36 days was completed last year. Lab results indicated a severely elevated testosterone and slight elevations of estradiol and progesterone. The luteal phase is less than 12 days. Altered progesterone and estradiol surges were noted during both the luteal and follicular phase.

A urinary amino acid test was performed two years ago. The isoleucine, leucine, methionine, phenylalanine, taurine, tryptophan cysteine, proline and threonine were within the lower limits of the laboratory range. Dietary peptide related markers anserine and 1-methylhistadine were elevated. Intermediary metabolites of phosphoserine and cystathionine were within the lower limits of the laboratory reference range.

A blood chemistry test which included a complete CBC with differential a chem-24, a lipid panel, a T7 thyroid panel and a lipid panel were performed the following day after her initial examination. Test results of the thyroid indicated an elevated TSH and a decreased T3 Uptake. The lab was called and instructed to add a TPO auto-antibody which returned elevated. The CBC indicated a pattern consistent with megablastic anemia. The lab was called and instructed to also add an intrinsic factor auto-antibody test which returned positive. Total protein levels and globulin levels were consistent with a pattern reflecting hypochlorhydria. The LDH was below 140 which has been associated with reactive hypoglycemia. The rest of the blood chemistry was equivical.

Diagnostic Imaging History

X-rays taken of her cervical, thoracic, lumbar spine and full-spine over the past three years demonstrated mild cervical hypolordosis with no patterns consistent with disease of the joints of bones. MRI studies of her brain conducted three years ago were equivical. Full-body non-contrast CT performed 4 months ago indicated moderate atherosclerosis. EKG testing completed on numerous occasions over the years were all normal. Abdominal and pelvic ultrasound tests completed in the past three years were all normal.

Standard Physical Examination

A physical examination which consisted of a complete neurological exam, EENT,and an applied kinesiology exam was performed. Blood pressure was tested simultaneously and bilaterally. The supine blood pressure on the right was 139/74 mm/Hg and 140/73 on the left. When placed into a seated position and tested again the blood pressure on the right was118/84 mm/Hg and 74/56 mm/Hg on the left. Deep tendon testing demonstrated hyperreflexia of the right biceps and right Achilles tendon. Pulse oximeter testing performed simultaneously and bilaterally was 100/77 on both the right and left upper extremity. Pulse oximeter testing performed simultaneously and bilaterally on the right lower extremity was 100/82 and 98/83 on the left lower extremity. Decreased pulse amplitude was noted with the left radial and tibial arteries when compared to the right. The abdominal reflex was absent on the right. Dysdiadochokinesia with rapid pronation/supination arms extended and elbows flexed was noted on the left. Finger-to-nose testing demonstrated past-pointing on the right and dysmetria on the left. Planter reflexes were intact with decreased response on the left when compared to the right. Coroneal reflex was absent on the right. Direct light stimulation demonstrated a alternating hippus between right and left with repeat testing. Tearing of the left eye was noted with direct light stimulation with accompanying bletherospasms. Vergence testing demonstrated an exophoria on the right with convergence initially and then a switch to left exophoria. Visual examination of the head and face demonstrated a slight facial paresis of the left lower face with weakness of the levator palabrea muscle. A right hypertropia was observed and identified to be related to a weakness of the left superior oblique muscle during cardinal fields of gaze testing. Slow saccadic glissades were observed with slow pursuit down to the right. Diminished internal rotation of the eye was noted on the right pupil passive head lateral bending to the left when compared to the left. Increased sensation was noted with pinwheel examination of the VI, V2, V3, cervical and lumbar dermatomes on the left. Increased vibrition sense was noted with the left lower and upper extremity. Weber's test laterized to the right. Ophthalmoscopic examination with a panoptic revealed an artery to vein ratio of 1:1. Optikinetic testing to the left reveled slow pursuits to the left and optikinetic testing of the right indicated overshooting with fast phase saccades. A right palate paresis was noted. A non-spastic pyramidal paresis demonstrated with 4/5 responses of the distal wrist extensor and toe extensor was noted. Tongue deviation indicated deviation to the right of 10%. Hyposmia was noted on the right. Abdominal auscultation demonstrated sluggish bowel sound on the left. Cardiac auscultation indicated tachcardia. Examination of the skin demonstrated xerodermia.

Applied Kinesiology Examination

Applied kinesiology testing as presented in the Synopsis by Walter and by papers published in the proceedings of the Annuals of the International College of Applied Kinesiology were conducted. Muscle testing as described by Kendall and Kendall were performed. Intial muscle testing of one muscle followed by another exhibited a conditionally inhibited response graded 4/5 of bilateral triceps, bilateral pectoralis majors, bilateral tensor fascia lata, left hamstrings, right teres minor and right quadraceps.

The patient was tested for a variety of chemical factors used in applied kinesiology challenge techniques. Ammonia sniff test was positive and negated by arginine. CO2 challenge test was positive and negated by B-vitamins and alpha-ketoglutaric acid. Aspirin challenge was positive and negated with omega-3 essential fatty acids. Homocysteine challenge test was positive and negated by broad-spectrum methyl-donar product (Methl-SP Apex Energetics), Glutathione challenge was positive and negated by factors of altered methlylation and citric acid cycle function as previously discussed. The patient was tested with various 6x homepathics and had facilitation responses to insulin, 4-hydroxyestradiol, testosterone, GABA and serotonin.

During the examination and challenge testing of the patient, the author noted that the patient was testing with inconsistent muscle testing responses when double checking patterns. Upon further observation the patient appeared to inhibit with all three types of muscle testing (type I, II and III) upon the second or third muscle test despite what the challenge presented. Pattens of original muscle inhibition patterns were different than initial testing when re-examined. Challenge testing with chemical factors proved to not be accurate, despite the substance tested, a muscle inhibition was exhibited upon repeated testing. The patient was assessed with aerobic/anaerobic challenge techniques as described by Maffatone as well as with Repeated Muscle Activation testing and not found to be a factor. Even repeat testing with the same substances caused inconsistent muscle responses.

Discussion About Physical Examination Findings

The physical examination findings are consistent with a pattern that reflects transneural degeneration of the left cerebellum with concomitant fatigue and wind-up of the cerebellar-dentato-rubro-thamlamo-cotrico-olivilo-cerebellar feedback loop in relation to the right neocortex. This pattern also appears to cause concomitants of a left hemisphericity due to increased frequency of firing of the left cerebellum and it postsynaptic counterpart, the right neocortex. The decreased frequency of firing of the left hemisphericity presented with decreased presynaptic activation of the left pontomedullary reticuospinal activation and the concomitants associated with this pattern.

Hemisphericity is a term used to indicated decreased function of the neocortex. Transneural degeneration (TND) is a term used to describe neuronal pools that have lost their electrochemical gradient negativity from decreased activation of early cellular protein gene transcription caused by decreased firing or presynaptic neuronal pools. A neuron needs three things to survive which include oxygen, glucose and stimulation. Stimulation of neurons increases the production of intracellular proteins which allow for production of transmitter substances, mitochondria and the ability to maintain the negative electrochemical gradient potentials. When there is decreased protein production of the cell membrane potential gradients are brought closer towards sodium equilibrium potentials. The net result clinically is pools of neurons that are easily excited with presynaptic stimulation but fatigue easily.

The neurological system is modulated in such a way in which one side of the neuraxis increases the function of the other side. The neocortex monsynatpically excites the pontomedullary reticulospinal tract which inhibits inhibition of motor neuronal pools and therefore leads to excitation of these neurons which include the gamma motor apparatus. Excitation of the gamma motor neurons increase the sensitivity of the ipsilateral muscle spindles which then increase the amount of proprioceptive bombardment of the ipsilateral cerebellum. The cerebellum then exicites the contralateral neocortex via the mesencephalon. Now the other neocortex

monsynaptically stimulates the ponotmeduallary reticulospinal tract which increases the sensitivity of the ipsilateral muscle spindles, which increase the excitation of the ipsilateral cerebellum, which then excites back the opposite cortex via the mesencephalon again. Therefore, one side of the nervous system promotes the survivability of the other side. In this patient we have a pattern of left cerebellar TND. The left cerebellum is brought closer to sodium equilibrium potentials and therefore we see patterns that are clinically expressed as both excitation of the right cortex with suppression of the left neocortex due to its postsynaptic relationship with eventual patterns of fatigue and decreased activity of the right neocortex and left cerebellum.

The cerebellum receives afferent input from contralateral neocortical projections to the central tegmental tract that excite the inferior olivary nucleus in the medulla and via the pontine nuclei in the pons, from the vestibular nuclei in the pontomeduallary junction, and from spinal cord projections of the spinocerebellar tracts and the cuneocerebellar tracts. The cerebellum projects into the vestibular nuclei of the pontomedullar junction bilaterally, to the contralateral thalamus and neocortex via the mesencephalon.

Information that arrives to the cerebellum projects into the deep cerebellar nuclei and to the granuloyctes. The granulocytes excite the cerebellar purkinje cells, basket cells and stellate cells. The basket and stellate cells inhibit the purkinje cells and the purkinje cells then inhibit its own deep cerebellar nuclei. These series of co-inhibitions have been termed "surround inhibition" and refer to the cerebellum's role in inhibiting its own output. If the cerebellum is not healthy, such as in our case of transneural degeneration, it loses the ability to inhibit its own output and may increase its projections to the pontomedullary centers and contralateral mesencephalon and neocortex. Increased output to the pontmedullary center may excite the solitary nucleus and increase vagal activation which may cause nausea. An expression of cerebellar excitation of the solitary nucleus from failure of cerebellar surround inhibition was observed when the patient's blood pressure went from 140/73 in a supine position to 74/56 in a seated position. As the patient moved into a seating position she increased receptor amplitude potentials of the vestibulo-cerebellum which projected into the solitary nucleus and created a drop in blood pressure. Note that this type of orthostatic hypotension presentation was only evident on the left side and therefore less likely to be related to metabolic (adrenal) manifestations which would most likely have bilateral expressions.

The physical examination also presented with alternating exophoria (lateral deviation of the eye) with repeated convergence testing. The contralateral cerebellum functionally pushes the eyes outward. If one cerebellum has decreased firing potential in comparison to the other side one may see an exophoria on the side of the decreased firing cerebellum. In this patient we saw switching of the exophoria with convergence which may be attributed to alternating fatigue of the cerebellum from TND. We also noted alternating hippus (fatigue of pupillary constriction) which may also correlate with TND of the cerebellum from dentato-rubro pathways.

The patient noted hyperalgesia on the entire left side of the body and face. This cannot be attributed to spinal nerve pathology, but more likely to be related to increased activity of the right somatic sensory cortex.

If the left cerebellum exhibits a TND pattern we may see expressions of increased summation of the right cortex followed by decreased summation due to the cerebellar neurons being closer to sodium equilibrium potential but with decreased intracellular protein to maintain activation. As the neocortex is brought up to threshold it functionally may inhibit the contralateral neocortex. In this patient we observed expressions of both left and right hemisphericity. The clinical expressions of hemispericity are noted by postsynaptic neocortex projections. Ten percent of neocortex projections is via the corticospinal pathways which predominantly send projections to the contralateral anterior horn. Ninety percent of neocortical projections are to the ipsilateral pontomedullay reticulospinal tract which include the cranial nerve nuclei of the pons (CN V, VI, VII) and medulla (CN VIII, IX, X, XI, XII). The neocortex also projects to the ipsilateral basal ganglionic system. The basal ganglia inhibits the ipsilateral thalamus and excites the mesencephalon ipsilaterally and contralaterally.

The pontomedullary reticulospinal tract also inhibits the intereomedial lateral (IML) cell column responsible for sympathetic autonomic output, it inhibits inhibition of the anterior horn and it inhibits the anterior muscles above T6 and the posterior muscles below T6.

In this case we observed right neocortex hemisphericity patterns of hyposmia on the right which may be attributed to decreased frequency of firing of the right temporal lobe, the right v entral posterior lateral nucleus of the thalamus or due to increased or decreased frequency of firing potentials of the right superior salivatory nucleus in the pons that is responsible for nasal mucous production. We observed tongue deviation to the right which may be attributed to decreased frequency of firing of the right hypoglossal nuclei. An absent corneal and abdominal reflex on the right may be attributed to multimodal somatosensory pathways that are most likely modulated by postsynaptic expressions of the right neocortex. The patient was observed to have past pointing on the right and dysmetria on the left which may be attributed to loss of integration of cerebellar-cortical pathways. A right hypertrophia (elevated pupil) was observed and found to be related to a left superior oblique weakness when the hypertrophia was assessed with cardinal fields of gaze and head tilts. The left superior oblique is innervated by the trochlear nucleus on the contralateral mesencephalon which maintains its central integrative state from ipsi and contralateral basal ganglia projections, contralateral cerebellar projections and from projections into the superior and inferior colliculus from visual and auditory pathways. The patient exhibited a non-spastic pyramidal paresis (inhibition of distal extensors) which may be attributed to decreased activation of the pontomedullary reticulospinal tract which is presynaptic to the right neocortex.

Tachycardia was noted in this patient. From a neurological perspective it may be attributed to a right hemisphericity. The right neocortex has descending reticulospinal projections into the S-A node of the heart. The tachycardia may also be attributed to anemia present with this patient's lab findings. When anemia is present there may be compensatory tachycardia to increase oxygen delivery to cells which are deprived due to decreased hemoglobin capacity to carry oxygen.

The patient also presented with numerous clinical indications of failure to inhibit the interomediate lateral cell (IML) column that is responsible for autonomic sympathetic output including decrease oxygen saturation readings on the left by pulse oximeter, increased artery to vein ratio of the left eye, and decreased pulse amplitude on the left. These expression may be a consequence of the failure of the left pontomeduallary reticuospinal tract to inhibit the left IML or from long standing mesencephalic integration patterns.

Discussion of Biochemical Factors

This patient suffers from numerous biochemical vicious cycles. The adrenal axis is up-regulated which promotes insulin resistance, suppression of pituitary-ovarian feedback, suppressed gastrointestinal secretory IgA, and a TH-2 lymphocyte subset shift which may increase vulnerability to autoimmune patterns. The patient is already demonstrating positive thyroid perioxidase auto-antibodies as well as antibodies for intrinsic factor. Anemia also increases the stress to the adrenal axis because it decreases mitochondira oxidative phosphorylation potentials due to insufficient oxygen delivery. When oxidative phosphyrolation pathways are uncoupled it places ATP output production on glycolysis which places stress on the adrenals.

The patient also demonstrated patterns consistent with polycystic ovary syndrome (PCOS) due to elevations in testosterone. It appears that insulin up-regulates 17-20 lyase of the theca cells of the ovaries and increases androgen and estrogen output. These shifts also increase the production of sex-hormone binding globulin which increases the percentage of free hormones in circulation and alter LH surge outputs. The depressed T3 uptake is most likely related to endogenous elevations of estrogen promoted by PCOS patterns.

Management of Patient

Since the patient was not able to tolerate any nutritional substances due to unhealthy digestive function, the patient was placed liposomal delivery of gluthathione and superoxide dismutase (Oxicell from Apex Energetics) and liposomal delivery of phosphatidylserine (Adrenacalm from Apex Energetics). Liposomal

delivery allows nutrients to bypass the gastrointestinal tract and go directly into the blood stream. Both glutathione and phosphatidylserine are powerful brain nutrients and antioxidatns. During the initial physical examination the patient was given caloric stimulation to modulate the frequency of firing of the left cerebellum by warm air stimulation of the right cerebellum and cold air stimulation of the left cerebellum. This immediately normalized planter stroking reflexes, corneal reflexes, abdominal reflexes, pinwheel sensation,and optikinetic activity.

The patient was instructed to conduct caloric stimulation to herself during the day with warm and cold air as described with a syringe and a hair dryer. On follow-up examination in two weeks the patient reported normalization of nausea and anxiety. The patient was then given left cerebellar exercises which includes spinning counter clockwise while her eyes were fixated on her thumb, visualization of physical activity exercises and instructed to play catch with her left hand by throwing a ball against the wall. She was also instructed to play with a jigsaw puzzle to increase activation of the right neocortex. The patient made general improvements in her overall health in function in subsequent weeks. After the end of the first month the patient no longer had conditionally inhibited muscles with repeated muscle testing procedures and was able to be tested and treated with standard applied kinesiology protocols.

Conclusion

This case demonstrated how imbalances in the neuraxis may lead to unpredictable muscle testing responses. One of the assumptions that we have in AK is that the patient's nervous system is always intact. This may not be the case with many patients that we encounter in our practices.

I remember when first starting practice I was trying to evaluate a chronically ill patient that had inconsistent muscle testing responses. I was frustrated and felt that the patient was not participating adequately. Now I realize that she may not have had a neurological system that could withstand numerous manual testing procedures. This case history was presented to increase awareness of abnormal muscle responses that we may face as applied kinesiologists when a patient may be neurologically compromised to the point where manual muscle testing procedures may exceed metabolic threshold.

Resources

Power, K. Applied kinesiology, neurology and our patient population. ICAK-U.S.A News Update, Vol. 15, No. 1, May 1998.

Schmitt, W., Yanuck, S. Expanding the neurological examination using functional assessment part I: methodological considerations. International Journal of Neuroscience. 1999; 97:61-76.

Schmitt, W,. Yanuck, S. Expanding the neurological examination using functional assessment part II: neurologic basis of applied kinesiology. International Journal of Neuroscience. 1999;97:77-108.

Caruso W., Lesiman, G. The clinical utility of force/displacement analysis of muscle testing in applied kinesiology. International Journal of Neuroscience. 2001; 106:147-157.

Leisman, G., Shambaugh, P., Ferentz, A. Somatosensory evoked potential changes during muscle testing. International Journal of Neuroscience. 1989; 45:143-151.

Chiropractic Applied Kinesiology Integration with TMJ Dental Care – Two Cases

David Leaf, D.C., DIBAK

Abstract

This paper describes two cases where the need of TMJ splinting techniques were needed to stabilize the patients corrections.

Key Indexing Terms: Applied kinesiology, chiropractic, muscle weakness, stroke, vision, speech.

Introduction

One involves symptoms brought on by improper dental care, the other the recovery from a stroke.

Materials and Methods

The first case is of a 16 year old female who began orthodontic work at age thirteen. As part of the care, 6 teeth were removed and then braces were fitted.

On her initial visit, the girl reported the following symptoms:

1. Weakness on the left side of her body causing her to walk with a staggering gait and an inability to run
2. Severe headaches that limited her attendance in school to an average of two days a week.

These symptoms slowly began after the dental work described above was initiated.

Testing showed very poor muscle strength on the left with normal muscle strength on the right side of the body. When she was in a supine position, she was unable to lift her left leg over 3 inches off of the table. Using a two-pound weight, she was able to lift it only 4 inches with her left arm.

Examination of her cranium showed classical tenderness over the temporalis, the pterygoid pocket, masseter on the left and extreme tenderness in the sub occipital muscles on the left. Her left eye was retracted in the socket.

Cranial corrections showed an immediate increase in strength on the left side of the body and a 50% decrease in the headache that she was experiencing. However, within two minutes the strength was lost.

Placing a tongue depressor between the teeth on the left following the cranial corrections allowed the strength increase to stabilize but also further dramatically increased the strength in the muscles. In fact when her gait was observed, she had a normal gait pattern. She was also able to lift her leg through a complete ROM with a five-pound weight attached to her ankle. This was also the case with the arm.

Results

She was referred to a qualified dentist specializing in the equilibration of the TMJ. She was fitted with a splint. The result of the combined care has been a complete return to normal muscle strength and coordination and almost a complete reduction in headaches. She has not missed a day of school for the headaches since the combination of cranial correction supported by the correct dental splint.

Materials and Methods

The second case involves a 57-year-old man who suffered a stroke that left him with poor coordination, overall loss of strength, speech pattern changes and an inability to coordinate the eyes so that he could focus and read.

The patient entered my office 8 weeks after the stroke and he was half way through his rehabilitation.

The patient presented himself with an uncoordinated gait, losing balance to both sides as his weight transferred across his foot. He was able to only minimally hold a conversation without inappropriate words being interjected into his sentence structure. He was unable to read except for three to four words at a 48-point type on the computer screen. Initial testing showed global weakness on both sides of his body.

Initially, he responded to both right and left-brain activity increasing his strength on the appropriate side. He also strengthened to a homolateral gait pattern, moving the extremities on both sides simultaneously. He also responded to cranial mobilization of the sphenoid and the occiput bilaterally. These three activities were performed in the office and the patient's wife was told to continue these at home with the patient.

Results

On his return, he was found to now strengthen to the normal cross pattern of walking, using the arm and leg on the opposite side. Cranial examination and correction for frontal problems was added to the returning sphenoid imbalance. Palpation of the pterygoid pockets showed bilateral tenderness. This was reduced with correction to the pelvis and upper cervical areas. On this visit, he was now able to read two to three lines at a 32-point font and at the completion of the visit at a 24-point font. He was able to speak for approximately two minutes without his speech pattern breaking down.

On the next visit, he had progressed to speaking for 5 minutes without his speech pattern breaking down. His cranial pattern had returned. After correction, cotton rolls, as used by dentists, were placed between the teeth and he was asked to walk. This resulted in an immediate improvement in his ability to read print down to a 14-point font. He was referred to have a splint fabricated to stabilize his TMJ and aid in the stabilization of his cranial corrections.

He progressed steadily except for one visit when his splint was out of equilibration. On this visit his vision and speech pattern had severely declined. The patient stated that he felt that his splint was incorrect. He waas referred back to the dentist for adjustment of the splint which immediately corrected the cranial imbalance and the vision and speech patterns improved.

Discussion/Conclusion

These two cases show the need of coordinated dental and chiropractic care in the treatment of complex and severe cases.

A Fifty-five year old female presented for diagnosis and treatment of thirty-five year history of bilateral foot problems and disequilibrium

Resources

Leaf, D.W., D.C., "What is TMJ Imbalance?" 2003 ICAK-U.S.A. Fall Regional Meeting, October 4–5, Boston, MA.

Mehta, Noshir, D.D.S., "Concepts of 3-Dimensional Dental Analysis," 2003 ICAK-U.S.A. Fall Regional Meeting, October 4–5, Boston, MA.

Mehta, Noshir, D.D.S., Driscoll, Stephen, D.D.S., "Temporalis, Pteygoids, Sphenoid, Splints," 2003 ICAK-U.S.A. Fall Regional Meeting, October 4–5, Boston, MA.

Effects of Proper Walking on Spinal Fixations

David Leaf, D.C., DIBAK

Abstract

Walking can have both positive and negative effects on physiological and biomechanical parameters. This paper discusses how proper waking can increase spinal flexibility and increase rib expansion while improper walking can reverse these patterns and initiate negative effects.

Key Indexing Terms: Applied kinesiology, spinal fixations, flexibility, walking, rib expansion.

Introduction

Thirty patients with multiple spinal fixations and decreased rib expansion were chosen for this study. The patients ranged from 18 to 75 years of age.

Materials and Methods

They were tested for the following parameters:

1. Spinal flexion as measured as how far they could bend over and reach. A mark was placed on the leg at the lowest level that they could reach.
2. Passive arm abduction was measured.
3. Lateral bending of the head and neck were measured
4. Rib expansion at the xiphoid process was measured
5. Motion palpation of the spine was performed
6. Muscle testing for applied kinesiological correlations with spinal fixations was performed testing for bilateral weakness of the deltoids, Popliteus, teres major, lower trapezius, psoas, gluteus maximus and the neck extensors were performed

The following results were found.

The average was 4 areas of spinal fixation, a rib expansion of 2.5 inches, arm abduction at 135 degrees, spinal flexion at 4.25 inches below the inferior pole of the patella.

Treatment consisted of testing and correction of the following:

1. Anterior talus
2. Dropped navicular
3. Lateral cuboid
4. Posterior calcaneus
5. Correction of tibialis posterior and peroneus longus
6. Correction of gastrocnemius
7. Proprioceptive neuromuscular facilitation (PNF) to the ankle and foot muscles.
8. Instructions on proper waking including heel strike, toe off and length of stride

The patients were then asked to walk for 100 steps on a treadmill and the above testing procedure was repeated.

Results

The results of the testing showed:

All but 5 had all spinal fixations corrected. The area that remained was the upper cervical fixation. Rib expansion increased an average of 1.3 inches. Spinal flexion increased an average of 3.75 inches.

Those patients that did not show a marked increase in rib expansion were shown to have a diaphragm imbalance associated with rib fixation/subluxation patterns.

The patient's were then told to walk 30 steps with their prior gait pattern. In all but two cases, the original findings were found to have been reestablished.

Discussion

In an other short study, patients with no spinal fixations were asked to walk with one shoe off for 300 steps on the treadmill. Following this the patients were retested for signs of spinal fixations. All of these showed at least three areas of fixation and as many as five.

Conclusion

Normal walking creates patterns of muscle action that reinforces normal facilitation and inhibition patterns that helps to normalize spinal mechanics. In the absence of diaphragm and rib subluxations, these same walking patterns increases rib expansion

Resources

Walther, David., D.C., Applied Kinesiology Synopsis, Systems DC, Pueblo, Colorado. (1988).

Leaf, David, D.C., Applied Kinesiology Flow Chart Manual, Third Edition Private Published. (1995).

Usage of Different Tapes in Supporting Injured Structures

David Leaf, D.C., DIBAK

Abstract

Kinesio® tape is a new type of tape that is designed to stay on the patient for a number of days without allergic reaction. It's use in skin proprioception imbalances aids in the treatment of both acute and chronic problems. Elastikon is an elastic firmer supporting tape that is useful in correcting ligament injuries.

Key Indexing Terms: Applied kinesiology, skin proprioception, taping.

Introduction

Normal athletic type taping is used to support joints during activities or to limit motion. For example, after spraining an ankle, the athlete will have a very firm tape support using layers and layers of inflexible tape. This type of taping is also used in an attempt to prevent injury. It is not uncommon for athletes, especially in football and soccer, to have their ankles taped daily, even for a light practice.

Materials and Methods

In a sentence, the purpose of regular taping is to support the muscles and joints limiting excessive motion. However, this causes a restriction in the flow of fluids in the area and interferes with the normal proprioception from the skin in the area. In addition, excessively tight taping will stop the normal expansion of muscles that lie under the tape causing inhibition.

These problems have limited the use of taping in the general population and in many types of injuries. Additionally, one of the problems with taping is the allergic response that many have had. It is difficult to have a patient have sensitive skin taped for support for more than a few hours without having a large percentage of them have allergic responses. Due to this, the use of taping has not been a part of the treatment protocols for correcting skin proprioceptive imbalances.

Skin has proprioceptive input into the normal functioning of the muscles that lie under it. For example, the skin over the lateral side of the foot will be stretched in almost all ankle sprains. The peroneus tertius will be found weak and usually responds to a directional stretch in a superior direction. If the ankle is swollen, it is difficult to treat this skin imbalance. Using Kinesio® tape, this is easily done.

Kinesio® tape is a highly elastic tape that can be applied for up to four days to continually normalize the proprioceptive imbalance caused by the skin trauma. After determining the direction that the skin should be held in, the tape is applied and stretched so that the skin will be held in its normal position. This can be done by using the elastic property of the tape. Apply it at one end, stretch it and apply it to the skin so when it contracts it pulls the skin in the desired direction to pull and hold the skin. An alternative method is to stretch the joint and the skin, apply the tape, and when the joint returns to a normal position the tape will be supporting the skin. The problem with this method is in making sure that the skin is being held in the corrective position.

Results

Alas, in the first 25 patients that this procedure was used, two had minor skin eruptions due to the tape.

Discussion

For supporting ligaments, especially at the ankle and the wrist, Elasikon offers support with elasticity. This tape, from Johnson & Johnson, is cloth and elastic. In ankle sprains, it is common to find the distal tibia and the fibula to have separated slightly. This allows the talus to become unstable. After correcting the structural problems, palpation of the ligaments of the ankle will usually show marked tenderness. Approximation of the tibia and the fibula may markedly reduce the tenderness over the ligament. Elastikon offers a good support for this type of injury. Double backing it to keep the adhesive off of the skin and wrapping the ankle gives support that is somewhat flexible. You have the option of stretching the tape or leaving it on unstretched. If the tape is stretched, care must be taken not to over tight the tape. Leaving it unstretched lets the tape expand with the underlying structures.

Conclusion

Rule #1. If taping to correct skin imbalances, make sure the underlying muscle tests strong with the tape applied.

Rule #2. If taping surrounds a joint, test to ensure that the support is not too tight causing inhibition of muscles that lie under the support. Then have the patient walk or move the joint under stress and retest for any possible abnormal inhibition.

Resources

Kinesio® Tex Tape, Kinesio® USA, LLC, Bldg. C, Suite 6, 3939 San Pedro Dr. NE, Albequerque, NM, 87110.

Leaf, D.W., D.C., Milan A.C. Centro Sportivo Milanello, Personal observations, 1998 to present.

Leaf, D.W., D.C., "Important Tools in AK Dealing with Athletic Injuries," 1999 ICAK-U.S.A. AK Sports Symposium, February 26-28, Los Angeles, CA.

Effectiveness of Applied Kinesiology Procedures on Foot Size

David Leaf, D.C., DIBAK

Abstract

A combination of common applied kinesiology procedures, when done in coordination with each other, result in sometimes dramatic decrease in overall foot size outlines indicating an increase in intrinsic foot (or pedal) support. The paper describes the treatment protocol used and the results in over 100 cases.

Key Indexing Terms: Applied kinesiology, muscle testing, orthotics, foot.

Introduction

It has become common for footprints to be used as a marketing tool for foot orthotic companies. There are foot support stores in malls currently using this tool to attract and sell their product. In an attempt to show patients and students in applied kinesiology classes the effectiveness of appropriate care, this same simplified procedure was used.

Materials and Methods

The following is an outline of the procedure.

Step I.

1. Place a clean paper on the floor and have the patient place their foot lightly on the paper and trace around the foot holding the pen at a 90 degree angle to the outside of the foot.
2. Have the patient stand and retrace the foot with another color.

Normally, there will be only a slight (1/4 inch at the most) expansion of the foot from a non-weight bearing to weight bearing posture. In over 100 cases, the largest increase was slightly over 1-inch length and 1 and 3/4 inches laterally. An increase of more than 1/4 inch indicates loss of intrinsic foot support.

The following protocol was then used to correct intrinsic foot support.

Step II.

1. Test the peroneus tertius, peroneus longus and brevis, tibialis posterior, tibialis anterior, extensor hallucis brevis and flexor hallucis brevis and the gastrocnemius.
2. Challenge the skin especially for peroneus tertius and Tibialis posterior weaknesses.
3. Challenge for related foot subluxation patterns – tibialis posterior – navicular – peroneus tertius – cuboid – peroneus longus/brevis – proximal fibular head – extensor hallucis brevis – anterior talus – flexor hallucis brevis – posterior calcaneus.
4. Correct the skin imbalance and tape using Kinesio® tape.
5. Correct the foot subluxations found.

6. Test for trigger points in the muscles above and correct any strain counterstrain or fascial imbalances.
7. Test for involvement of the gastrocnemius using the new procedure described by Goodheart. This is easily done in the supine position with the patient contracting the opposite triceps while the gastrocnemius is tested for weakening. Correct this imbalance.
8. Perform a proprioceptive neuromuscular facilitation (PNF) procedure for the ankle/foot muscles using an "X" pattern. Have the patient always begin with the toe action followed by either dorsiflexion or plantarflexion.
9. Palpate the plantar muscles for trigger points and perform either strain counterstrain or fascial technique if found.
10. Correct any subluxation found from L – 4 to the sacrum.
11. Ask the patient to walk approximately 30 steps.

Step III.
Place a new clean paper and repeat the tracing process above and compare to the original.

Results

In over 180 cases, done in my office and by students in applied kineisiology classes, less than 15% had equal weight bearing and non-weight bearing tracings. This is defined as 1/4 inch or less increase in either length or width.

Conclusion

In all of the other cases, when the above protocol was used, the second tracing was markedly less that the original tracing. This has proven to be an excellent tool for showing the effectiveness of our care to the patient. The patient can leave with the tracing and show their friends and family. This procedure should be done prior to fitting for orthotics as it can change the size of the foot and the need for support in the different arches of the foot.

Resources

Kinesio® Tex Tape, Kinesio® USA, LLC, Bldg. C, Suite 6, 3939 San Pedro Dr. NE, Albequerque, NM, 87110.

Leaf, D.W., D.C., Milan A.C. Centro Sportivo Milanello, Personal observations, 1998 to present.

Leaf, D.W., D.C., "Important Tools in AK Dealing with Athletic Injuries," 1999 ICAK-U.S.A. AK Sports Symposium, February 26–28, Los Angeles, CA.

Clinical Response to a Neurologically Based Comprehensive Clinical Protocol Developed by Dr. Walter H. Schmitt

Kerry M. McCord, D.C., DIBAK

Abstract

OBJECTIVE: Demonstrate the utilization of a neurologically based comprehensive clinical protocol utilizing applied kinesiology (AK) techniques in the evaluation and treatment of four patients whose clinical presentations were dissimilar.

CLINICAL FEATURES: The patients chosen were of various ages and genders, and presented with a diversity of symptoms. They included three pain syndromes and a child with attention deficit disorder.

INTERVENTION & OUTCOME: The application of the comprehensive clinical protocol led to a successful resolution of presenting symptomatology regardless of presenting complaint.

Key Indexing Terms: Applied kinesiology, manual muscle testing, pain, attention deficit disorder, food allergies.

Introduction

Utilizing AK techniques in the context of a neurologically based comprehensive clinical protocol allows the physician, regardless of patient symptoms, to investigate physiology gone awry in an ordered and efficient manner, leading, sequentially, to the application of appropriate therapies, and subsequently, to the desired outcome.

Materials and Methods

A neurologically based comprehensive clinical protocol (included at papers end), developed by Walter H. Schmitt, D.C., DIBAK, D.A.B.C.N.,[1] was used in the evaluation and treatment of four patients, whose case histories appear below. The clinical protocol proposed by Schmitt utilizes the manual muscle testing response (inhibited, facilitated, over-facilitated) as a reflection of the status of the anterior horn motor neuron pool (AMN) for the muscle being tested. Sensory receptor based diagnostic challenges are then used to direct appropriate therapy. AK assessment procedures are prioritized for effectiveness and efficiency and ordered to facilitate the resolution of muscle inhibition secondary to systemic neurological phenomena prior to addressing local problems. Results of initial consultation, examination and treatment, as well as visit by visit follow up, are presented in a format that is intended to clarify the clinical presentation and outline treatment in a way that can be easily followed by referring to the Quintessential Applications: A(K) Clinical Protocol attached.[2] Phone calls were made the week prior to submission of this paper for publication in an effort to assess each patient's current condition and perceived, long-term response. The four cases presented below include three patients in pain (one acute, one chronic, one mixture of acute and chronic) and a child with attention deficit disorder. The positive and rapid responses to vastly differing clinical presentations, employing the same clinical protocol (with specific therapeutic differences dictated by individual patient response to testing), suggests applicability in the care of all patients in any AK practice.

Discussion

CASE #1

HISTORY: Master A. and his parents, presented in our office complaining that Master A. has trouble mentally focusing (previously diagnosed as attention deficit disorder - A.D.D.), suffers from constipation, and experiences stomach pain at bedtime. As a matter of history, Master A.'s parents state that, on his father's side, there is a family history of what is termed "My O. Mind" (scatterbrained). According to Master A.'s parents, his focusing disorder is so severe that a 30-minute homework assignment turns into a 2 to 3 hour ordeal. Master A. complains that his stomach hurts at bedtime and his parents report that he has infrequent bowel movements (possibly two to three times per week) and lots of gas. His parents also report that he is allergic to citrus, which causes sores on his lips. Reportedly, his problems are getting worse, and his home life and schoolwork have been adversely affected by the symptoms he now suffers. From his parent's point of view, it is more than marginally disconcerting that Master A. has to be "prodded" constantly to do schoolwork and perform routine daily tasks. He and his parents state that he has no physical restrictions, does not use any medication, and has just recently begun supplementing with vitamin B-12 and DHA.

CHIEF COMPLAINTS: Constipation, gas, stomach upset at bedtime; occasional, mild, pins and needles in both hands; A.D.D. or difficulty focusing ("easily distracted") and food sensitivities.

PHYSICAL EXAMINATION: Visual examination revealed a well nourished, soon to be nine-year-old boy, with no significant visible postural deficits, who was alert and oriented times three.

ASSESSMENT and TREATMENT: Postural analysis and Temporal Sphenoid (TS) Line analysis were used to help identify specific muscle weakness(es). Manual muscle testing weakness of the Pectoralis Major, Sternal Division (left), the Tensor Fascia Lata (right) and the Latissimus Dorsi (left) was initially observed. Autogenic facilitation (AF) did not strengthen weak muscles indicating active nociceptive pathways overriding normal muscle spindle cell control mechanisms. Injury recall technique (IRT) was used on areas of past injury restoring normal muscle spindle response to AF. Aspirin/Acetaminaphen/Ibuprophen Mix (A/A/I) challenge was negative. Antihistamine Mix challenge was positive, strengthening weak indicator muscles and suggesting the presence of elevated histamine. Histamine metabolizing agents (folic acid, B-6, etc.) were tested for strengthening weak indicator muscles and revealed a favorable response to folic acid. Visceral challenge technique (VCT) was used to identify hypersensitivity to wheat. IRT with therapy localization (TL) to Chapman's reflexes for the liver, small and large intestine, with wheat in the mouth, was performed. Systemic structural factor assessment revealed immune driven, cranial related neurological disorganization (switching) that was treated by stimulation of the thymus Chapman's reflex. Citric Acid Cycle (CAC) factors and heart-focused (HF) activity were not assessed. Systemic endocrine evaluation revealed a hyperadrenia pattern that was treated by IRT with TL to the adrenal Chapman's reflexes with homeopathic histamine (offender) in the mouth. GI tract assessment revealed no weak muscle patterns (in the clear) associated with digestive organ function, and no apparent ileocecal valve (ICV) difficulty upon evaluation with standard AK challenge procedures. Rubbing and pinching visceral referred pain (VRP) areas for digestive organs revealed no significant findings. However, challenging the ICV with sugar in the mouth revealed the presence of an open ICV pattern. A discussion regarding dietary ingestion of sugars and starchy carbohydrates followed and led to a recommendation that Master A. decrease the intake of same. Treatment was delivered by rubbing the small intestine Chapman's reflexes with sugar in the mouth. Ileal Brake challenge was negative. Challenge of the gastrocolic reflex was positive and milk products were identified as an offender. Stomach Chapman's reflex was rubbed with milk in the mouth. Emotional stress was not assessed. All initially and subsequently observed muscle weakness patterns had spontaneously resolved. Flexor reflex afferent (FRA) activity was used to challenge for spinal manipulative need. An L-5 subluxation was identified and corrected in harmony with coupled mechanics. Gait assessment revealed a disruption of the normal gait inhibitory patterns immediately following pinching of the pancreas VRP. The pancreas Chapman's reflex was rubbed and gait disruption resolved. Gait inhibitory patterns were challenged again by pinching the pancreas VRP while sugar was held in the mouth.

This challenge was positive and the pancreas Chapman's reflex was again treated while Master A. held sugar in his mouth. Treatment continued until all gait inhibitory pattern disruption was resolved. Dietary changes were suggested including the elimination of wheat, milk products, sugar and starchy carbohydrates. Anti-gliadin SIgA salivary assessment was ordered to rule out gluten sensitivity. It was suggested that Master A. return for follow-up in one month at which time reassessment of his then current condition and dietary patterns would be made.

Master A. did not return for follow-up as recommended, however, his mother called our office one to two weeks post initial treatment and reported that the focusing disorder (A.D.D.) had significantly improved, that his teacher had observed a remarkable change in his performance at school, that his stomach aches had disappeared, and that bowel function was better.

PHONE FOLLOW-UP: (3.5 months after last office visit) Master A.'s mother reports that all presenting symptoms are greatly improved. With the awareness that she gained from the single visit in our office, she felt comfortable following the dietary recommendations made and has continued on her own to improve the dietary choices of Master A. ("and the entire family"). She reports that Master A.'s grades have risen significantly, as has his ability to focus in the classroom. Digestion and bowel issues previously reported have completely resolved, with the exception of responses to the occasional exposure to wheat and sugar ("the holidays were a challenge"). She expressed her deep appreciation and remarked, "He is a new little boy".

CASE #2

HISTORY: Mr. B. presented in our office complaining of severe and debilitating left shoulder pain. Mr. B. states that ten days prior to his visit with us, he awakened with a mild ache in the left shoulder. That day, as he reached into the back of his truck to pick up his toolbox, he experienced sharp pain in the shoulder that "almost brought him to his knees" and since that time has suffered continued pain as well as inflammation and severely restricted motion. He was seen by a medical physician, prescribed a muscle relaxant (Skelaxin) and an anti-inflammatory (Vioxx), and after ten days has seen little to no improvement. He had continued working until two days prior to his presentation in our office and finds that resting the shoulder eases the persistent pain and discomfort, but does nothing for the restricted range of motion, nor aggravation associated with same. As a matter of history, he reports that his knees hurt and swell regularly, that he had Osgood-Schlatter's as a teenager and injured his right quadriceps playing softball some years past. He also reports breaking his right hand and left foot. When asked regarding current medications and past surgeries, he denies taking medication with the exception of that recently prescribed, but admits to having had surgery on the fractured right hand ("pinned") and a tonsillectomy.

CHIEF COMPLAINTS: Frequent, severe, sharp pain in the left shoulder joint, with limitation of motion; occasional, mild, neck stiffness, with pain across the left shoulder; intermittent, moderate, pins and needles in the left forearm and hand; frequent, mild, swelling in both knees, especially right.

PHYSICAL EXAMINATION: Mr. B., a 39-year-old male, was alert and oriented times three. His seated blood pressure was 130/80. His pulse was 72 and respirations 14 per minute. Postural analysis revealed an elevated left scapula and an elevated right occiput.

NEUROLOGICAL EXAMINATION: Deep tendon reflexes, including biceps, triceps and brachioradialis, were symmetric and bilaterally equal (2+). Dynamometer grip strength measured 105/110/115 pounds of pressure on the dominant right hand and 95/95/95 pounds of pressure on the left. Dermatomal evaluation for sensory acuity in the upper extremity revealed hyperesthesia overlying the C5 and C6 dermatomes, on the left.

ORTHOPEDIC EXAMINATION: Orthopedic examination of the cervical spine revealed pain on: Maximum foraminal compression. Orthopedic examination of the left shoulder revealed pain on: Shoulder abduction, supraspinatus tendon, Apley's scratch and shoulder apprehension. Left shoulder range of motion, as measured by inclinometer: Forward elevation 100 degrees, backward elevation 30 degrees, internal rotation 90 degrees, external rotation 80 degrees, adduction 20 degrees, abduction 95 degrees. Pain and guarding was noted on all ranges of motion, but especially abduction. Palpation with mild digital pressure revealed no significant paravertebral tenderness in the paraspinal musculature palpated. Spinous percussion elicited no significant pain over the spinous processes percussed.

ASSESSMENT and TREATMENT: Postural analysis and TS Line analysis were used to help identify specific muscle weakness(es). Manual muscle testing weakness of the Psoas (left) and Pectoralis Major, Sternal Division (left) was initially observed. AF was performed and did not strengthen weak muscles. IRT was used on areas of past injury restoring normal muscle spindle response to AF. Nociceptor stimulation blocking (NSB) and set point (SP) techniques were performed to relieve pain in the shoulder. During the application of NSB and SP, pain on movement decreased and range of motion increased until 90% of the pain was eliminated and range of motion was, for all practical purposes, fully restored. Systemic structural factor assessment revealed an immune driven, cranial related neurological disorganization (switching) that was treated by stimulation of the spleen Chapman's reflexes, effectively restoring shunt stabilizer muscle activity (middle & lower trapezius) in the left shoulder. CAC factors, HF activity, systemic endocrine factors, the GI tract and emotional stress were not assessed. Evaluation of local (shoulder) muscle function revealed a weak Posterior Deltoid (left) that was treated with origin-insertion (OI) technique followed by IRT (micromanipulation of the mortis joint). Gait assessment revealed an interruption of the normally expected pattern when challenging gait with head tilt to induce tonic labyrinthine reflex activity (TLR) indicating an under functioning endocrine gland. Therapy localization to adrenal Chapman's reflex restored the expected gait response and stimulation of adrenal Chapman's reflexes was performed. Following Chapman's reflex stimulation, gait patterns now exhibited the normally expected response. Follow-up was recommended in one week.

NOTE: As Mr. B. approached the front desk to pay his bill, he remarked that he had come on the recommendation of family, but having had a past history in nursing and a less than favorable opinion of chiropractic in general, his expectations were that he would "waste some money, satisfy his family, and go get some real help elsewhere."

2nd VISIT: Mr. B. reported that his shoulder had remained significantly improved (95%), that he had returned to work and was only experiencing a "slight catch" (on certain movements). Since his prior visit, having contemplated his past history of injury, he now recalled that he had been shot and stabbed in the right thigh, fell off of a roof and landed on his "butt," was struck by lightening and had a nail go through his right foot. Postural analysis and TS Line analysis were used to help identify specific muscle weakness(es). Manual muscle testing weakness of the Psoas (right), and the Middle and Lower Trapezius (left) was initially observed. IRT was performed on areas of past injury. SP was used to relieve residual pain (catch) in the shoulder. Muscle weakness previously identified had spontaneously resolved. A Category 1 pelvic fault was corrected. Gait assessment was negative. Mr. B. was dismissed to return as needed.

PHONE FOLLOW-UP: (4 months after last office visit) Mr. B. reports that his primary complaint remains 100% resolved. He states that he has had no additional symptoms since his last visit, with the exception of mild shoulder pain due to overexertion, occurring soon after his last office visit. He reports that the transient pain resolved within one hour and that he has had no problems since. He further reports that he is maintaining full range of motion and expressed his appreciation for the excellent outcome.

CASE #3

HISTORY: Ms. C. presented in our office for injuries sustained in a motor vehicle accident that occurred in November 2002. Ms. C. states that she was the driver of a motor vehicle stopped in traffic when another vehicle struck hers in the rear. She states that her vehicle was struck with enough force to be pushed into the vehicle immediately ahead and reports that her head and face hit the steering column and headrest as the seatbelt that she was wearing bruised her chest. Immediately following the accident, she was taken to the hospital by ambulance, seen in the emergency room, examined, x-rayed, given a cervical collar and medication, but not admitted. She has consulted her primary care physician, a neurologist and a chiropractic physician. She reports that her pain has diminished over time, but she still suffers with middle back, lower back, right buttock, hip and thigh pain, as well as ringing in the ears and daily headaches. She also states that she has had significant digestive problems since the accident in question. She states that her problems are staying the same; lying down makes them better; sitting aggravates same. She rates her level of stress as severe; states that she sits more than 50% of her time at work; engages in no regular physical activity/exercise and reports that her home life, work life, recreation, rest and sleep have all been adversely affected by the pain she now suffers. She states that she used to walk regularly and go to the gym, but now she hurts after exercising, and therefore, she is relatively inactive. When asked regarding current medications and past surgery, she denies taking medication but reports a gastric bypass in 1999 (lost 110 pounds), a hysterectomy in 2002, two C-sections, Morton's neuroma surgery (twice), as well as elective cosmetic surgery (after gastric bypass) including a facelift, tummy-tuck, breast lift and implant removal.

CHIEF COMPLAINTS: Constant, moderate, right buttock and hip pain, with radiation into the mid lateral thigh, on the right; frequent, moderate, swelling in the ankles, with accompanying foot pain; constant, mild, daily, sinus area headaches, ringing in the ears, blurred vision, loss of balance, memory loss and a blood shot eye (secondary to face lift); intermittent, mild, left -sided, mid back tension; frequent, moderate, constipation, indigestion, and gas. (All preceding symptoms, except as otherwise noted, are a direct result of, or have become worse since, the accident in question.) She also reports anxiety, apprehension, disturbed sleep, fatigue, depression, inability to concentrate, canker sores, recurrent infections, insomnia and recent weight gain.

PHYSICAL EXAMINATION: Ms. C., a 53-year-old Caucasian female, was alert and oriented times three. Her seated blood pressure was 138/86. Her pulse was 72 and respirations 18 per minute. Postural analysis revealed an elevated right ilium and an elevated left scapula.

NEUROLOGICAL EXAMINATION: Deep tendon reflexes, including biceps, triceps, brachioradialis, Achilles and patellar, were symmetric and bilaterally equal (2+). Heel and toe walk were able to be performed, but not without incident, as heel walk caused pain in the right foot. Dynamometer grip strength measured 45/50/55 pounds of pressure on the dominant right hand, 45/50/55 pounds of pressure on the left. Dermatomal evaluation for sensory acuity in the upper and lower extremities revealed no significant alteration in sensation.

ORTHOPEDIC EXAMINATION: Orthopedic examination of the cervical spine revealed pain on: Foraminal compression, maximum foraminal compression and O'Donahue's maneuver. Orthopedic examination of the lumbar spine revealed pain on: Leg lowering, forced leg lowering and Nachlas. Cervical range of motion, as measured by inclinometer: Flexion 50 degrees, extension 75 degrees, right and left lateral bending 35 degrees, right rotation 70 degrees, left rotation 75 degrees. Lumbar range of motion, as measured by inclinometer: Flexion 60 degrees, extension 15 degrees, right lateral bending 20 degrees, left lateral bending 15 degrees, right rotation 15 degrees, left rotation 20 degrees. Palpation with mild digital pressure revealed paravertebral tenderness from the occiput through C7, bilaterally, from T1 through T7, on the right, and from L2 though L4, on the right. Spinous percussion elicited pain over the following spinal segments: C5 through T2.

ASSESSMENT and TREATMENT: Postural analysis and TS Line analysis were used to help identify weak muscles. Manual muscle testing weakness of the Gluteus Medius (bilateral), the Tensor Fascia Lata (bilateral) and the Pectoralis Major, Clavicular Division (right) was initially observed. AF did not strengthen weak muscles. IRT was used on areas of past injury restoring normal muscle spindle response to AF. Systemic nutritional factors were not addressed. Systemic structural factor assessment revealed immune driven, cranial related neurological disorganization (switching) that was treated by stimulation of spleen and thymus Chapman's reflexes. Systemic endocrine evaluation revealed evidence of hypoadrenia that was treated by rubbing adrenal Chapman's reflexes. Due to constraints of time, further treatment was suspended and Ms. C. was asked to return for follow-up in one week.

2nd VISIT: Ms. C. reported that since her initial visit her mid-back has significantly improved, however, the low back, hip and leg pain persisted. She also admits to taking antibiotics for a urinary tract infection (past 3 days). Postural analysis and TS Line analysis were used to help identify specific muscle weakness(es). Manual muscle testing weakness of the Gluteus Medius (left), the Pectoralis Major, Clavicular Division (right) and the Pectoralis Major, Sternal Division (right) was initially observed. AF did not strengthen weak muscles. IRT was used on areas of past injury, not initially addressed, restoring normal muscle spindle response to AF. SP was used on the left leg to relieve pain. Systemic nutritional factors were not addressed. Systemic structural factor assessment again revealed immune driven, cranial related neurological disorganization (switching) that was treated by stimulation of spleen and thymus Chapman's reflexes. CAC and HF activity were not addressed. Systemic endocrine evaluation revealed evidence of hypoadrenia that was treated by rubbing adrenal Chapman's reflexes. Adrenal challenge technique revealed a hyperadrenia pattern that was treated by IRT to adrenal Chapman's reflexes with caffeine in the mouth. Manual muscle testing weaknesses initially observed had, by this juncture, spontaneously resolved. Liver VRP area challenge revealed a need for IRT to liver Chapman's reflexes with caffeine and cortisol in the mouth, suggesting a disturbance of phase 1 and phase 2 liver detoxification pathways. GI tract assessment revealed a sugar induced, open ileocecal valve (ICV) pattern. Stimulation of the small intestine Chapman's reflexes while the patient held sugar in the mouth resolved the sugar induced, open ICV challenge. Pain in the right lower rib cage and over an abdominal scar (not previously reported) but observed while treating the sugar induced, open ICV, was addressed with SP technique. Emotional stress assessment was negative and local problems were now addressed. A pelvic Category 1 fault was identified and corrected (out of sequence) prior to assessing the iliolumbar ligament. The iliolumbar ligament was subsequently assessed and treated with origin insertion (OI) IRT. (Had the iliolumbar ligament been treated first, the Category 1 may have spontaneously resolved and not needed mechanical correction.) FRA spinal subluxation evaluation was performed and an anterior T11 was adjusted (no other spinal subluxations were identified). Gait assessment revealed a disruption of the normal and expected gait patterns after pinching the pancreas VRP. Pancreas Chapman's reflex was rubbed and gait disruption resolved. This observation and the sugar induced, open ICV pattern previously observed led to a discussion regarding dietary habits resulting in a recommendation that Ms. C. decrease starchy carbohydrates and sugar intake and increase dietary protein and complex carbohydrates. Location quality memory technique (LQM) was used to resolve mild residual pain in the left buttocks. It was recommended that Ms. C. return for follow-up in one week.

3rd VISIT: Ms. C. reported that the pain she had experienced in the leg for the past two years was now "gone!" The pain in the buttocks was intermittent (but not daily) and more aggravated during 2 days of gut irritation following inappropriate dietary choices. Postural analysis and TS Line analysis were used to help identify specific muscle weakness(es). Manual muscle testing weakness of the Gluteus Medius (right), the Pectoralis Major, Sternal Division (bilateral) and the Tensor Fascia Lata (right) was initially observed. AF strengthened weak muscles. SP was used to relieve residual buttock pain and pain in the area of the ischial tuberosity. Following the application of SP technique, all previously identified weak muscles were now strong. Weakness of the sartorius (right), the gracilis (right) and the sternocleidomastoideus (left) was now observed. Systemic nutritional factors were assessed revealing a strengthening response to A/A/I Mix suggesting the need for improved fat metabolism and Prostaglandin (PG) production. She responded favorably to Black

Currant Seed Oil and pyridoxal-5-phosphate (P-5-P). An adverse muscle testing response to the tasting of trans fats and animal fats led to a recommendation that both be eliminated or reduced, respectively. IRT to liver and small intestine reflexes with trans fats (vegetable shortening) and animal fats (lard) in the mouth resolved the weakness associated with tasting same. Systemic structural factors did not show a need for immune stimulation but rather a need for mechanical correction of an external frontal bone fault. CAC and HF activity assessment was negative. Systemic endocrine assessment revealed a pattern consistent with hypoadrenia that was treated by rubbing adrenal Chapman's reflexes. Liver VRP area challenge revealed a need for increased sympathetic activity. IRT to liver Chapman's reflexes with cortisol in the mouth was performed and resolved the positive challenge. The tasting of cholesterol resulted in Pectoralis Major, Sternal Division weakness (bilaterally) and was treated with stimulation of liver Chapman's reflex with cholesterol in the mouth. GI tract assessment revealed a weak tensor fascia lata that responded to rubbing the stomach VRP. Rubbing of the stomach Chapman's reflex resolved the weak tensor fascia lata (right). A gastrocolic reflex challenge was positive and revealed milk as an offender. Rubbing the stomach Chapman's reflex with the offender (milk) in the mouth negated the positive challenge. FRA activity, gait assessment and the need for further chronic pain relief were negative. It was recommended that Ms. C. return for follow-up in two weeks.

4th VISIT: Ms. C. stated that her pain was now "98% relieved." She reported that she was still experiencing intermittent, mild problems sitting, but also reported that she was working 70 plus hours weekly and under a significant amount of stress. She states that her digestion is somewhat improved. Postural analysis and TS Line analysis were used to help identify specific muscle weakness(es). Manual muscle testing weakness of the Pectoralis Major, Sternal Division (left), the Pectoralis Major, Clavicular Division (left) and the Tensor Fascia Lata (right) was initially observed. AF did not strengthen weak muscles. IRT to the left shoulder restored normal muscle spindle response to AF. SP to the area of the ischial tuberosity was performed. Systemic structural factor assessment again revealed what appeared to be immune driven, cranial related neurological disorganization (switching), however, a favorable response to the insalivation of hypothalamus tissue by immune and non-immune related muscles suggested that the immune difficulty was secondary to the hypothalamus adapting to some other organ imbalance. A need for sympathetic challenge of organ VRPs was identified and gut related VRPs (small and large intestine) were treated with IRT to related Chapman's reflexes with an identified offender (milk) in the mouth. Endocrine assessment revealed a pattern consistent with hypoadrenia that was treated by rubbing adrenal Chapman's reflexes. No other endocrine assessments were positive (in the clear), however, darkening the room revealed endocrine related muscle weakness. Correction of a sphenoid compression cranial fault resolved the dark induced weakness. GI tract assessment revealed a weak Tensor Fascia Lata (right) that responded to the insalivation of probiotics and rubbing of the large intestine Chapman's reflex. Local problems then assessed revealed a need for OI to the Oblique Abdominus and the sacrotuberous ligaments, bilaterally. Assessment of abdominal muscle function led to further evaluation of the cranium revealing a sagittal suture jam, the correction of which favorably affected abdominal muscle function. FRA activity, gait assessment and the need for further relief of chronic pain were negative. Ms. C. was then asked to close her eyes and touch her finger to her nose. This resulted in global muscle inhibition when performed with the left hand. The global muscle inhibition was interpreted as evidence of cerebellar transneural degeneration (TND) and addressed with appropriate in office neurological rehabilitation techniques (repetitive left finger to nose activity while patient breathed O_2 at 2 liters per minute for approximately 3 minutes). Ms. C. was asked to perform cerebellar exercises (finger to nose, from 3 different positions, with her eyes closed) 2 times daily for 2 to 3 weeks, and reduce/eliminate milk products. Supplementation with probiotics was recommended.

Ms. C. returned two weeks later reporting that all chronic symptomatology had been resolved, that she was pain free, and wished to be enrolled in a supervised personal training program so that she might re-engage in regular physical activity.

PHONE FOLLOW-UP: (2.5 months after last office visit) Ms. C. states that she considers herself "a success story" and hopes that "others can be assisted" with the procedures that resolved her 24 months of pain and discomfort. She rates the resolution of her primary complaints as 9.5 on a scale of 0 to 10 (10 being complete resolution of all complaints). She states that after sitting at her desk for long periods of time (she still works 70+ hours per week), or upon overexertion, she has "twinges" of pain, but they have become less and less frequent. She is walking regularly without discomfort, and states that she is "delighted to no longer be a chronic mess."

CASE #4

HISTORY: Mr. D. presented in our office complaining of severe right groin, buttock and hip pain. He stated that groin pain had been severe for four days, but especially the past 24 hours, and reported taking medication for pain. He stated that, due to the intensity of the pain in the groin, he had suffered with "chills and sweats" all night and he had not slept. As a matter of history, and integrally related to his presenting complaint, he reports having suffered with the affects of Polio since 1954 and had sprained his groin two months past, shortly after having had an orthotic made to provide support for his right lower extremity. He reports that three weeks prior to presentation in our office, the pain in the groin had become significantly more aggravated. He also stated that he had begun sleepwalking 3 nights ago and fell forward into the TV set bruising his ribs. Mr. D. admits that he has suffered with chronic pain most of his adult life and that, for the past 20 years, it has prevented gainful employment.

CHIEF COMPLAINTS: Constant, mild to severe, neck stiffness, tightness, tension, on both sides, with muscle spasms and weakness, and pain across both shoulders; constant, mild to severe, mid back pain, spasms and tension; constant, mild to severe, low back, sacroiliac, buttock and hip pain; intermittent ache radiating down the legs to the knees, with aching in both feet, aggravated by activity (These are symptoms he has reportedly suffered for 25 to 30 years.) He also reports anxiety, irritability, disturbed sleep, fatigue, inability to concentrate and severe groin pain (the primary reason for seeking care at this time).

PHYSICAL EXAMINATION: Mr. D., a 53-year-old Caucasian male was alert and oriented times three. His seated blood pressure was 148/80. His pulse was 84 and respirations 20 per minute. Postural analysis revealed an elevated left ilium and an elevated left scapula. He has an anatomic short leg (on the right) reported at a loss of three-quarters of an inch. The right leg is atrophied, consistent with the history of polio.

NEUROLOGICAL EXAMINATION: Deep tendon reflexes, including biceps, triceps and brachioradialis, were symmetric and bilaterally equal (2+). Achilles and patellar reflexes on the left were brisk (2+), and, on the right, Achilles reflex was normal (2+) and patellar reflex absent. Heel and toe walk were able to be performed without incident. Cerebellar function tests were essentially normal. Dynamometer grip strength measured 80/75/90 pounds of pressure on the dominant left hand and 65/60/60 pounds of pressure on the right. Dermatomal evaluation for sensory acuity revealed hyperesthesia overlying multiple dermatomes in the cervical, thoracic and lumbar regions on the posterior trunk.

ORTHOPEDIC EXAMINATION: Orthopedic examination of the cervical spine revealed pain on: Shoulder depression. Orthopedic examination of the lumbar spine revealed pain on: Leg lowering, Gaenslen and Patrick-Fabere. Cervical range of motion, as measured by inclinometer: Flexion 50 degrees, extension 75 degrees, right and left lateral bending 35 degrees, right and left rotation 70 degrees. Lumbar range of motion, as measured by inclinometer: Flexion 60 degrees, extension 20 degrees, right and left lateral bending 20 degrees, right and left rotation 20 degrees. Palpation with mild digital pressure revealed paravertebral tenderness from the occiput through T11, bilaterally. Spinous percussion elicited no significant pain over the spinous processes percussed.

ASSESSMENT and TREATMENT: Postural analysis and TS Line analysis were used to help identify specific muscle weakness(es). Manual muscle testing weakness of the Gluteus Medius (bilateral), the Adductors (right) and the Pectoralis Major, Sternal Division (left) was initially observed. AF did not strengthen weak muscles. IRT was used on areas of past injury restoring normal muscle spindle response to AF. SP was used to relieve pain in the groin and buttocks. Due to constraints of time and patient fatigue, systemic assessments were postponed. Weaknesses initially observed had resolved following IRT and SP, with the exception of the right adductor, which was treated with OI IRT. Emotional recall techniques were also employed in an attempt to relieve the emotional distress associated with the past days of severe pain. Following treatment with emotional recall techniques, Mr. D. remarked that the "chills and sweats" had stopped and that he felt like he may now be able to sleep. Mr. D. was sent home to rest and asked to return the following day.

2nd VISIT: Mr. D. reported that he had slept soundly for the first time in days and that the pain he had suffered was significantly reduced. Postural analysis and TS Line analysis were used to help identify specific muscle weakness(es). Manual muscle testing weakness of the Gluteus Medius (right), Tensor Fascia Lata (right) and Pectoralis Major, Clavicular Division (left) was observed. AF strengthened weak muscles. Systemic nutritional factors assessed revealed a strengthening response to A/A/I Mix. A discussion regarding potential dietary inhibitors of Delta-6-Desaturase revealed daily consumption of significant quantities of alcohol and resulted in a recommendation to avoid same. Evaluation for nutrients to support conversion of Omega 6 and Omega 3 fatty acids into PG 1 and PG 3 families, respectively, revealed a favorable muscle testing response to Sesame Seed Oil and vitamin B-6. Oral challenge with trans fats weakened strong muscles suggesting recent or consistent dietary exposure. Trans fat induced weakness was negated by TL to small intestine Chapman's reflexes and treated by IRT with TL to Chapman's reflexes with trans fat (vegetable shortening) in the mouth. Dietary restriction of trans fats was recommended. Systemic structural factor assessment revealed immune driven, cranial related neurological disorganization (switching) that was treated by stimulation of spleen and thymus Chapman's reflexes. CAC and HF activity were not addressed. Systemic endocrine effects now assessed revealed disturbed liver detoxification pathways exhibiting a muscle strengthening response to glucuronic acid suggestive of tendencies toward hyperinsulinism. GI tract and emotional stress patterns were not apparent and local problems were then assessed. Muscle weaknesses initially observed had spontaneously resolved. However, the need for OI IRT was identified and treatment applied to address manual muscle testing weakness of the Gluteus Maximus (right) and Medial Hamstrings (right). Other local challenges performed uncovered no further need for therapeutic intervention. Gait assessment revealed interruption of the normally expected gait inhibitory pattern upon pinching of the pancreas VRP. Pancreas Chapman's reflex stimulation resolved the disruption until challenged again with sugar in the mouth. Treatment of the pancreas Chapman's reflex with sugar in the mouth restored normal and expected gait inhibitory patterns. Tenderness in the groin, though reduced, was persistent, and treated with SP and LQM (Location, Quality, Memory Technique). Mr. D. was instructed to reduce starchy carbohydrates and supplement with B-6 and Sesame Seed Oil. It was recommended that Mr. D. return for follow-up in one week.

3rd VISIT: Mr. D. reported "significant overall improvement" with only mild buttock and groin pain, but states that he his right leg was "giving way" and that he "almost fell twice in the past 24 hours". Postural analysis and TS Line analysis were used to help identify specific muscle weakness(es). Manual muscle testing weakness of the Gluteus Medius (right), Adductors (right), Oblique Abdominus (right), Rectus Femoris (right) and Pectoralis Major, Sternal Division (right) was observed. AF strengthened weak muscles. Systemic nutritional factors were not assessed. Systemic structural factor assessment again revealed immune driven, cranial related neurological disorganization (switching) that was treated by stimulation of spleen and thymus Chapman's reflexes. Systemic endocrine evaluation revealed disruption of the normally expected muscle testing response to rotation of the head (inducing TLR) indicating endocrine hypofunction. TL to thyroid Chapman's reflexes restored the normally expected manual muscle testing response, and thyroid Chapman's reflexes were rubbed restoring normally expected manual muscle testing response to turning of the head. GI tract and emotional stress patterns were not apparent and local problems were then assessed. Gluteus Medius and Adductor weakness initially observed had spontaneously resolved. OI IRT treatment of the Oblique

Abdominus, Rectus Femoris, Piriformis and Gluteus Maximus on the right was painful and an apparent stress induced re-manifestation of TLR disruption was observed on assessment of gait with head tilt, indicating endocrine hypofunction. TL to adrenal Chapman's reflexes restored expected patterns and treatment by rubbing of the adrenal Chapman's reflexes resolved disruption. Due to persistent muscle dysfunction in the right pelvis and groin, an MRI was recommended to rule out partial or complete muscle tear. Mr. D. was asked to return for follow-up in one week.

4th VISIT: MRI and x-rays performed elsewhere were presented for review and were negative for muscle tear. Atrophy of upper thigh musculature, secondary to the history of polio, was observed. Mr. D. reported that he had fallen over the weekend, bruising his right arm and hip. Postural analysis and TS Line analysis were used to help identify specific muscle weakness(es). Manual muscle testing weakness of the Gluteus Medius (left) and the Pectoralis Major, Clavicular Division (left) was observed. AF did not strengthen weak muscles. IRT to the areas bruised during his recent fall and the lumbosacral region restored muscle spindle response to AF. SP was used to relieve pain in the right arm and neck (base of the skull). Retesting of systemic nutritional factors revealed that the A/A/I Mix still strengthened weak muscles and discussion confirming the implementation of lifestyle intervention strategies previously recommended ensued. Systemic structural factor assessment revealed a persistent immune driven, cranial related neurological disorganization (switching) that was treated by stimulation of the thymus Chapman's reflexes. Assessment of systemic endocrine effects revealed persistent manifestations of patterns of hyperinsulinism that led to an expanded discussion of current dietary habits, alcohol as it relates to blood sugar, the ingestion of milk products (stressor/offender of pancreatic function), the impact of same on the perpetuation of blood sugar handling stress and the prolongation of his progress toward full symptom resolution. Emotional stress related issues addressed with emotional recall techniques were followed by home care instructions in the use of emotional neurovascular points and tapping acupuncture head points (Stomach 1). Muscle weakness initially observed was resolved, other local problems were not found, and gait assessment was essentially normal. Weekly visits were recommended as Mr. D. informed me that he was to leave town in approximately 6 weeks, not to return for some months.

5th VISIT: Mr. D. reported that though the groin was still somewhat tender and the ribs a bit aggravated from the fall, he felt "great" overall. (During this visit, while testing the right Gluteus Maximus, his rib (anterior right) "popped" and he experienced excruciating pain. The acute pain was relieved by IRT and SP techniques.) Postural analysis and TS Line analysis were used to help identify specific muscle weakness(es). Manual muscle testing weakness of the Tensor Fascia Lata (right) and the Gluteus Medius (right) was initially observed. AF did not strengthen weak muscles. IRT was used on areas of past injury restoring normal muscle spindle response to AF. SP was performed to relieve pain over the right lower rib cage. Systemic structural factor assessment revealed no immune driven, cranial related neurological disorganization but did reveal a temporomandibular joint (TMJ) disorder secondary to involvement of a tooth on that same side. The TMJ was treated with IRT to the involved tooth. CAC, HF activity, systemic endocrine effects and GI tract were not assessed. Emotional recall techniques were performed and local problems assessed. A pelvic Category 1 fault was identified and corrected (out of sequence) prior to assessing the iliolumbar ligament. The iliolumbar ligament was subsequently assessed and treated with OI IRT. (Had the iliolumbar ligament been treated first, the Category 1 may have spontaneously resolved and not needed mechanical correction.) This is the point that the Gluteus Maximus (right) was tested and the rib "popped." Relief was obtained by the application of IRT and SP techniques. FRA spinal (and foot) subluxation evaluation was performed and anterior subluxations of T8 and T10 were adjusted. Gait assessment revealed interruption of the normally expected gait inhibitory patterns and a right sacroiliac subluxation was identified and corrected with pelvic blocking. Gait assessment with head tilt (inducing TLR) disrupted normally expected muscle testing patterns, indicating endocrine hypofunction. TL to adrenal Chapman's reflexes restored expected patterns and treatment by rubbing of the adrenal Chapman's reflexes resolved disruption. Mr. D. was asked to return in one week.

6th VISIT: Mr. D. stated that the only real bother was the right rib cage (pain radiated from front to back). He said it was "like a spear straight thru". He reported that his groin, though admittedly "not perfect" was

much better and that he experienced little to no pain in his neck, upper, middle or lower back (pain that had been his constant companion for 20 years). Postural analysis and TS Line analysis were used to help identify specific muscle weakness(es). Manual muscle testing weakness of the Gluteus Medius (right) was initially observed. AF did not strengthen weak muscles. IRT was performed over the ribs and Oblique Abdominus (right) restoring normal muscle spindle response to AF. SP was performed to relieve persistent pain over the right lower rib cage. Systemic structural factor assessment revealed an immune driven, cranial related neurological disorganization (switching) that was treated by stimulation of the thymus Chapman's reflexes. CAC and HF activity were not assessed. Systemic endocrine assessment revealed a pattern consistent with hypoadrenia that was treated by rubbing adrenal Chapman's reflexes. Local problems were then assessed and revealed the need for OI IRT on right-sided core and rib stabilizing musculature including the Latissimus Dorsi, the Anterior Serratus, the Lower Trapezius and the Oblique Abdominus (especially posterior). Gait assessment was negative. Mr. D. was asked to return in one week.

7th VISIT: Mr. D. reported a "trace" of discomfort in both the rib and the groin but otherwise was feeling "great". He said, "(his) brain (was) racing" as he was "excited about the future". He also reported that he had been drinking a lot of beer for the past week or two. Postural analysis and TS Line analysis were used to help identify specific muscle weakness(es). Manual muscle testing weakness of the Gluteus Medius (left), Sartorius (left) and the Pectoralis Major, Sternal Division (left) was initially observed. AF strengthened weak muscles. (Systemic nutritional factors were not assessed. However, intense consultation with Mr. D. and his wife regarding alcohol use and its effect on him and his family was engaged. Avenues of assistance were identified and recommendations made.) Systemic structural factor assessment revealed an immune driven, cranial related neurological disorganization (switching) that was treated by stimulation of the spleen and thymus Chapman's reflexes. CAC and HF activity were not assessed. Systemic endocrine evaluation revealed disruption of the normally expected muscle testing response to rotation of the head (inducing TLR) indicating endocrine hypofunction. TL to adrenal Chapman's reflexes restored expected patterns and treatment by rubbing of the adrenal Chapman's reflexes resolved disruption. GI tract assessment revealed a sugar induced, open ICV, treated with stimulation of the small intestine Chapman's reflexes with sugar in the mouth. Emotional recall techniques were performed. Manual muscle testing weaknesses initially observed had spontaneously resolved. Gait assessment was negative. Mr. D. was asked to return in one week.

8th VISIT: Mr. D. reported that he felt "great" and had "no pain for the (entire) week". He did report that he was "fatigued some around the shoulders (upper back)". During the 5th through the 8th visits, the habitual use of alcohol, its effect on him, his wife and family, community resources to help with his ongoing addiction (A.A.), and recommendations regarding the use of HF techniques to relieve chronic emotional pain (associated with years of disability) were openly discussed. On this visit, the emotional pain endured by his spouse (dealing with alcoholism) surfaced and was confronted. Postural analysis and TS Line analysis were used to help identify specific muscle weakness(es). Manual muscle testing weakness of the Gluteus Medius (right) and Oblique Abdominus (right) was initially observed. AF strengthened weak muscles. Systemic structural factor assessment revealed persistence of an immune driven, cranial related neurological disorganization (switching) that was treated by stimulation of the thymus Chapman's reflexes. A disturbance of the right TMJ was identified and treated by IRT tooth. HF activity was performed, though manual muscle testing indications were not present. Systemic endocrine evaluation revealed evidence of adrenal hypofunction that was treated by rubbing adrenal Chapman's reflexes. Manual muscle testing weaknesses initially observed had spontaneously resolved. Gait assessment was negative. Mr. D. was asked to return in one week.

9th VISIT: By this juncture, just less than two months following his initial visit, Mr. D. wrote, "I feel better than a normal person!" Upon further specific questioning, he did report slight awareness of the rib and groin as well as mild upper body fatigue. He further reported that he was sleeping only 3 to 4 hours per night. Following the same clinical protocol, identifying weak muscles and testing for appropriate response to AF, no need for IRT or pain relief techniques was observed. Systemic structural factor assessment revealed a need for thymus Chapman's reflex stimulation. FRA activity revealed a need to adjust the right foot and T-11

(anterior). Gait assessment with head tilt (inducing TLR) disrupted normally expected muscle testing patterns, indicating endocrine hypofunction. TL to adrenal Chapman's reflexes restored expected patterns and treatment by rubbing of the adrenal Chapman's reflexes resolved disruption.

Having dealt with chronic pain most of his adult life, Mr. D. now felt that he was largely relieved of same. Though he looked forward to the future, engaging in establishing his own business, he was acutely aware that he must continue to courageously struggle with his ongoing dependence on alcohol.

NOTE: Just prior to leaving town, Mr. D. was again sleepwalking and fell into the TV set (the corner of which struck him in the right rib cage). He had no other reported complaints as he presented for treatment prior to his departure. Treatment was rendered to relieve pain utilizing IRT and SP techniques. Systemic structural factors and endocrine effects were addressed, and spinal adjustments made.

PHONE FOLLOW-UP: (one week short of 3 months after last office visit) Mr. D. was unable to be contacted during the week prior to publication.

Conclusion

The use of a basic science driven comprehensive clinical protocol, expressed as a neurological hierarchy, organizes clinical thought, allowing the physician to navigate the terrain of the patient's physiology and be led through a forest of symptoms toward a favorable, and often remarkable, outcome. Those utilizing AK techniques and/or the concepts of chiropractic neurology achieve favorable outcomes frequently enough to steel their resolve in the practice of their art and science. Having previously utilized virtually all of the procedures presented in this clinical protocol, albeit haphazardly (i.e. applied in no particular order), and having since disciplined myself to organize my thinking and clinical application around the principals inherent within it, the results achieved are predictably more efficient (i.e. less time) and effective (i.e. more favorable outcomes).

The understanding that injury recall patterns must be addressed first in order to restore normal muscle spindle cell control mechanisms; that systemic nutritional, structural, endocrine, GI and emotional stress factors impact descending neural pathways undermining favorable and lasting response to local treatment procedures; and, that gait assessment helps confirm that no further therapy is needed prior to releasing the patient to daily activity; provides the functionally oriented physician a clear cut path to the eagerly anticipated, remarkable outcome, that each patient so richly deserves.

The case histories presented herein were selected for their diversity (gender, age, clinical presentation). The phone call follow-ups were made (by someone other than the author) after the body of the paper was already written. Both were done to demonstrate that the physiologically organized application of the clinical protocol presented herein applies regardless of clinical presentation. It has become indubitably clear that initial procedures common to all practices (history and examination), followed by well-ordered diagnostic challenges and treatment, based on the basic sciences of neurology and biochemistry, organize neurological expression in such a way that a favorable outcome is substantially encouraged.

References

1. Schmitt, W.H., *The Neurological Rationale for a Comprehensive Clinical Protocol Utilizing Applied Kinesiology Techniques*. Submitted for Publication in the Proceedings of the Annual Meeting of the I.C.A.K., U.S.A., Volume 1, 2005 – 2006.

2. McCord, K.M., and Schmitt, W.H., *Quintessential Applications: A(K) Clinical Protocol*. St. Petersburg, Florida: Privately Published, 2005.

QUINTESSENTIAL APPLICATIONS: AK CLINICAL PROTOCOL

BEGINNING PROCEDURES

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

SYSTEMIC NUTRITIONAL FACTORS

5. Test Aspirin, Acetaminophen, Ibuprofen Mix - If Strengthens:
    a. Check Essential Fatty Acids (BCSO, FSO, EPA, etc.)
    b. Check EFA Cofactors (B-6, Mg, Zn, Niacin)
6. Test Antihistamine Mix – If Strengthens:
    a. Challenge Orally for Offender(s) (Allergen)
    b. IRT Chapman's Reflexes with Offender(s)
7. Sniff Tests – Aldehydes, Bleach, Ammonia
8. Test Nutrients Based on Patient History:
    a. Vitamin E (Low Back Muscles); Vitamin C (Shoulder Muscles)
    b. Iron, Folic Acid, Vitamin B-12
    c. Cholesterol Lowering Nutrients (If Indicated)
    d. Chondroitin Sulfate – If Strengthens:
        i. Check Sulfur (Cysteine) & Associated Nutrients
        ii. Check Blood Sugar Handling (Insulin, Magnesium)

SYSTEMIC STRUCTURAL FACTORS

9. Is TL to K-27 Positive?
    a. Straight TL – Cranial – Pre-Test Imaging (Go to 11) – Immune or Mechanical?
    b. Crossed TL – TMJ – IRT to TMJ (Go to 10) – Treat Immune Circuit
    c. Dorsal Crossed TL – Use Tooth Techniques
10. Does TL to TMJ Strengthen Weak Muscle? (and/or) Is TMJ Present with IRT?
    a. Right TMJ – Thymus (or Lower Sternum)
    b. Left TMJ – Spleen (or Lower Sternum)
    c. Check Nasosphenoid Cranial Fault
    d. Check Temporoparietal Jam
    e. Check Sphenoid Compression Fault
    f. Correct TMJ / TMJ Muscles – Correct with IRT and/or Mechanically

11. Does Pre-Test Imaging Strengthen? If Yes – Check Cranial Bones
    a. If IRT Positive:
        i. Right Side – Check Thymus (or Lower Sternum)
        ii. Left Side – Check Spleen (or Lower Sternum)
    b. If No IRT – Make Mechanical Correction

SYSTEMIC NUTRITIONAL FACTOR

12. Does Rebreathing in a Paper Bag Strengthen?
    a. If Yes: Check Citric Acid Cycle & Electron Transport Chain Nutritional Factors

HEART-FOCUSED ACTIVITY

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

SYSTEMIC ENDOCRINE EFFECTS

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

GASTROINTESTINAL TRACT

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

EMOTIONAL STRESS

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

LOCAL PROBLEMS

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

GAIT ASSESSMENT

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

CHRONIC PAIN

32. If Chronic or Persistent Pain: Use LQM Technique

Preliminary Study on the Effects of Sucralose on Metabolic Pathways

Scott E. Muzinski, D.C., N.M.D. • Kimberly R. Muzinski, D.C.

Abstract

Clinical trials using applied kinesiology (AK) muscle testing of Splenda® (sucralose) by clinicians have been difficult to accurately conduct because of the wide range of weakness in muscles that are created with oral testing of Splenda®. A non-blinded test was performed by two practitioners on three test volunteers to determine if a tool for analysis and treatment could be determined to hypothesize a chemical pathway which is most effected. An oral dose of Sucralose was administered to each subject and found to create generalized muscle weakness in all test subjects. A subsequent dose of SP Cleanse™, Ultra Clear®, UltraClear PLUS®, and Silymarin were added orally to each subject while the Sucralose was still present and the subjects were re-tested. The results demonstrated that all subjects responded to broad-spectrum detoxification support best.

Key Indexing Terms: Sucralose, detoxification, applied kinesiology.

Introduction

Splenda® (Sucralose) is one of many artificial sweeteners available in today's market. It is a non-caloric sweetener about 600 times sweeter than sucrose. It is available in many diet soft drinks, diet foods such as meal replacement bars, and single serving packets.[1]

Sucralose is manufactured by chlorinating sucrose. Three chlorine (Cl) atoms replace three hydroxyl groups yielding 1,6-dichloro-1,6 dideoxy-Beta-D-fructofuranosyl-4-chloro-4-deoxy-alpha-D-galactopyranoside.[2] The new chemical, marketed as Splenda®, is marketed as a biostatic source of sweetening that is not bioavailable for the body to use as a caloric source. However, research has shown that while up to 80% of Splenda® is passed through to the rectum and excreted unchanged, approximately 11%-27% is absorbed.[3] Of this, 20%-30% is metabolized and the remainder is excreted by the kidneys. The metabolized portion is broken down into 1,6-dichlorofructose.[4] The pathway for this chemical is unknown in the human body.

The manufacturer claims that the Cl- is processed by the body the same as NaCl (table salt) and produces a negligible effect.[5] However, speculation has been made that the absorbed portion is handled in much the same way as carbontetrachloride xenobiotic (otherwise known as certain type(s) of pesticide) in hepatic metabolism.[6]

Because of the broad range of effects on muscle testing by Splenda® and the speculative similarities to that of xenobiotics, an examination of hepatic detoxification pathways is warranted. Additionally, detoxification pathways for the kidneys and digestive tract should be examined as well as sugar handling pathways, because Splenda® could have an effect on all.

Materials and Methods

Three volunteers where chosen for this preliminary study. A group of muscles where tested in the clear using standard AK technique. Each test subject was also tested for switching and cranial faults. None were detected. Psoas, Pectoralis Major Sternal division (PMS), Quadriceps, Hamstring, and Latissimus Dorsi muscles,[7] both right and left, were all tested and found to be strong.[8] These muscles were selected because each has a relationship to an organ-system function according to standard AK practice. Psoas to the kidney, PMS to the liver, Quadriceps to the small intestine, Hamstrings to the large intestine, and Latissimus Dorsi to the pancreas.[9]

Each test subject was placed supine and given approximately .25 gm of Splenda® orally and all muscles were re-tested. In the re-test all muscles were weak according to AK terminology. Then approximately 1/3-1/2 of a capsule of SP Cleanse™ was added orally to each test subject while the Splenda® was still in place. All muscles were re-tested and results recorded. SP Cleanse™ is a product that is designed to support multi-organ system detoxification pathways with over 20 whole foods and botanicals and phytonutrients. Some of these are shown to be effective in liver, kidney and intestinal detoxification as per Standard Process catalog and product information.[10]

Next, each test subject's palate was cleared with a wash of filtered water. K27 and all muscles were re-tested to confirm that all had returned to a normal strong state. Then Splenda® was re-introduced with the same results. Subsequently, approximately 1/4 tsp. of UltraClear® was introduced orally to each test subject, and all muscles were re-tested. UltraClear® is a product from Metagenics that provides a broad-spectrum multi-organ detoxification support including phase1 hepatic detoxification.[11]

The same procedure was repeated for a third time adding UltraClear PLUS® to the subject orally. UltraClear PLUS® from Metagenics is a broad based support that is advertised as a support for both Phase1 and Phase 2 hepatic detoxification pathways.[12]

As a final sequence to this test the patient's palate was again cleansed and Splenda® was again introduced. The muscles were re-tested with the same results. One tablet of Silymarin™ from MediHerb was added to each subject. Silymarin is known to help in hepatic function by improving Phase 1 detoxification.

Results

Each time Splenda® was introduced, all muscles tested weak. The addition of SP Cleanse™ and UltraClear® strengthened all muscles. All muscles remained weak with the addition of UltraClear PLUS®. Only one muscle, the PMS, strengthened with Silymarin.

Discussion

Because Splenda® effects so many of the body's detoxification pathways, each system that could be identified as being involved had a representative muscle test incorporated into this study. The hepatic detoxification pathways of Cytochrome p450 are essential in detoxification of chemicals in hepatic function.[13] This particular pathway is responsible for processing many things, including xenobiotics like carbontetrachloride.[14] If Splenda®, a carbontrichloride, breaks down into compounds that are bioequivalent to xenobiotics, hepatic function would be effected and could be measured by testing the muscles that are specific to that organ relationship, as was demonstrated by a strong PMS upon re-testing.

However, because Splenda® is only partially processed by the liver, the portions that are moved from the bloodstream and filtered by the kidneys could affect kidney function. Muscles such as the Psoas would be affected and this was demonstrated. Because about 80% of the Splenda® is passed directly to the large intestine and rectum, muscles like the Quadriceps and Hamstrings would be affected.

Because Splenda® is non-caloric, the sugar handling organs like the pancreas should not engage when it is added but Latissimus Dorsi muscles were weak after adding Splenda®. This shows that there is probably some relationship to the pancreas. There has been some research showing that Splenda® does not affect insulin levels but does raise HemaglobinA1c.[15] There must be a relationship that would explain the failed test of the Latissimus Dorsi muscle which remains undefined.

Because Splenda® affects almost all major muscle-organ systems, many or all muscles that could be tested could show up weak. By using products like SP Cleanse™ or UltraClear®, which are broad-spectrum multi-system detoxifiers, a positive change could be effected. It is an unusual result that UltraClear PLUS® did not help any test and follow up studies should be conducted to determine the reason for this.

Because Silymarin aides hepatic function by improving Phase 1 detoxification, it follows that the hepatic function would be affected by this support as was demonstrated by the positive PMS muscle test. Silymarin is also supportive of healthy intestinal function.[16] However, in this experiment it had no effect upon Hamstring or Quadricep function, probably because the intestinal interaction of Silymarin is not enough to support the effect Splenda® has upon the intestines.

The test with Silymarin demonstrates that individual support for specific organ systems can help that part of the detoxification pathway but will leave the others in this complex pathway unaffected. Further testing with nutritional and herbals targeting specific organ systems should be conducted to confirm this, such as using Argenix™ from Standard Process, to detoxify the kidney pathways.[17]

Lab tests such as Functional Liver Detoxification Capacity Testing (from Great Smokies Diagnostic Laboratory)[18] could help establish future study criteria. This could also provide an opportunity to support scientific exploration of lab analysis in conjunction with muscle testing, possibly establishing a correlation between detoxification pathways, affects of Splenda®, and muscle-organ relationships, if one exists. This testing would show the potential detoxification ability for individual organs and pathways, such as hepatic or kidney, and could be correlated with test subjects' reactions during muscle testing. If, for example, some subjects were found not to have a weak Psoas test during provocative testing of Splenda®, labs might correlate a greater potential for detoxification of that pathway or organ-system in that subject. This could provide more effective analytical results and clinical guidance for individualizing treatment.

Conclusion

The link between Splenda® and detoxification pathways is difficult and complex to establish at first glance. It had proven to be difficult to perform muscle testing and record data at first because of the complex relationship of all of the involved muscle-organ relationship pathways involved.

While many who muscle test and practice AK have known that Splenda® would weaken strong muscles, it proved difficult to find a link to individual pathways to strengthen the total body imbalance created.

Research proved to be the most effective tool in this study. By establishing the involvement of Splenda® with many organ detoxification pathways it became clear that a multi-faceted approach to the chemistry involved would prove most beneficial.

By establishing the relationship between the organ-muscle relationships, treatment protocols can be established. For those patients who need artificial sweeteners in life, such as diabetics, support can be offered in the form of a general daily detoxification support. As stated above, this could be supported by lab testing in the clinic if needed.

An additional project could be proposed to determine if pre-loading a test subject with daily doses of a broad-range detoxification support could counter any effects of Splenda®.

References

1. "Sucralose—A New Artificial Sweetener," The Medical Letter, 1030, (July 3, 1998).
2. Gain B., "FDA Approves J&J Sweetener," Chemical Week, Vol. 160, Issue 14, (June 10, 1998).
3. Food and Drug Administration "Final Rule" for Sucralose, 21 CFR Part 172, Docket No. 87F-0086.
4. Gain B., "FDA Approves J&J Sweetener," Chemical Week, Vol. 160, Issue 14, (June 10, 1998).
5. "Q&A: Is newly FDA approved sweetener Sucralose good for you?" Executive Health's Good Health Report, Vol. 35, Issue 2, (Nov/1998).
6. Hunter BT., "Sucralose," Consumers' Research Magazine, Vol. 73, Issue 10, (Oct/1990).
7. Walther, David S., Applied Kinesiology, Synopsis, (Pueblo, CO., Systems DC, 1988).
8. Walther, David S., Ibid.
9. Walther, David S., Ibid.
10. Standard Process, Inc., SP Cleanse™, Product Information No. 2670.
11. Metagenics Product Catalogue, UltraClear®, pp. 52.
12. Metagenics Product Catalogue, UltraClear PLUS®, pp.53.
13. Pizzorno Jr., Joseph E., Murray, Michael T., Textbook of Natural Medicine, Volume 1, (New York, Churchill Livingstone), 1999.
14. Pizzorno Jr., Joseph E., et al, Ibid.
15. "Q&A: Is newly FDA approved sweetener Sucralose good for you?" Executive Health's Good Health Report, Vol. 35, Issue 2, (Nov/1998).
16. MediHerb, Inc., Silymarin™, Product Information No. M1420.
17. Standard Process, Inc., SP Cleanse™, Product Information No. 1140.
18. Ball, E., Runkel, P., Holmes S., Great Smokies Diagnostic Laboratory, Functional Assessment Resource Manual, (Asheville, NC, 1999).

The Effects of Sucralose on Muscle/ Organ Relationships with Nutritional Testing

Scott E. Muzinski, D.C., N.M.D. • Kimberly R. Muzinski, D.C.

Abstract

A non-blinded test was performed by two practitioners on three test volunteers to further analyze the effects of Sucralose on muscle/organ relationships and treatment with specific nutritional substances. An oral dose of Sucralose was administered to each subject and found to create generalized muscle weakness in all test subjects as demonstrated in the authors' original paper. Psoas, Hamstring, PMS, Latissimus Dorsi, and Quadriceps were tested and found to be weak. Arginex®, Silymarin, Gastro-fiber®, Okra Pepsin® and Pancreatrophin PMG® were added orally to each subject while the Sucralose was still present and the subjects were re-tested. The results demonstrated that individual muscle organ pathways respond to specific nutritional support for specific pathways.

Key Indexing Terms: Sucralose, nutritional testing, applied kinesiology.

Introduction

As was demonstrated in the authors' first paper on this subject, Splenda® has an effect on several muscle/organ relationships.[1] Nutritional substances were chosen to determine if specific nutrition could support individual muscle/organ relationships without crossover into other muscle/organ pathways in relation to the effects of Splenda®. This was done to further define the nutritional chemistry involved in Splenda® reactions in the body.

Materials and Methods

Three volunteers where chosen for this study. A group of muscles where tested in the clear using standard AK technique. Each test subject was also tested for switching and cranial faults. None were detected. Psoas, Pectoralis Major Sternal division (PMS), Quadriceps, Hamstring, and Latissimus Dorsi muscles, both right and left, were all tested and found to be strong. These muscles were selected because each has a relationship to an organ-system function according to standard AK practice. PMS to the liver, Quadriceps to the small intestine, Hamstrings to the large intestine, Latissimus Dorsi to the pancreas, and Psoas to the kidney.[2]

Each test subject was placed supine and given approximately .25 gm of Splenda® orally and all muscles were re-tested. In the re-test all muscles were weak according to AK terminology.[3] Then one tablet of Arginex® was added orally to each test subject while the Splenda® was still in place. All muscles were re-tested and results recorded. Arginex® is a product that is designed to support kidney function and kidney detoxification as per Standard Process® catalog and product information.[4]

Next, each test subject's palate was cleared with a wash of filtered water. K27 and all muscles were re-tested to confirm that all had returned to a normal strong state. Then Splenda® was re-introduced with the same results. Subsequently, one tablet of Silymarin was introduced orally to each test subject, and all muscles were re-tested. Silymarin is a product from MediHerb® that contains Milk Thistle® extract, which is known to assist liver detoxification.[5]

The same procedure was repeated for a third time adding Gastro-fiber® to the subject orally. Gastro-fiber® from Standard Process® is advertised as a support for intestinal elimination.[6]

The same procedure was repeated for a forth time adding Okra Pepsin® to the subject orally. Okra Pepsin® from Standard Process® is advertised as a support for intestinal elimination and absorption.[7]

As a final sequence to this test the patient's palate was again cleansed and Splenda® was again introduced. The muscles were re-tested with the same results. One tablet of Pancreatrophin PMG® from Standard Process® was added to each subject. Pancreatrophin PMG® is advertised as a support for pancreatic function.[8]

Results

Each time Splenda® was introduced, all muscles tested weak. With the addition of Arginex® the Psoas tested strong bilaterally, there was no change with the other muscles. With the addition of Silymarin the PMS tested strong bilaterally while the others remained weak. When Gastro-fiber® was introduced the Hamstrings tested strong bilaterally while the other muscles in the test group remained weak. Okra Pepsin® strengthened the Quadriceps only and Pancreatrophin PMG® strengthened the Latissimus Dorsi only.

Discussion

Splenda® affects many of the body's detoxification pathways and muscle/organ relationships. Therefore each system that could be identified as being involved had one representative muscle test incorporated into this study.

The hepatic detoxification pathways are responsible for processing many things. Hepatic function could be affected and measured by testing the muscles that are specific to that organ relationship, as was demonstrated by a strong PMS upon re-testing with Silymarin.

Splenda® is only partially processed by the liver, and the portions that are moved from the bloodstream and filtered by the kidneys could affect kidney function. Muscles such as the Psoas would be affected and this was demonstrated. With the addition of Arginex® the Psoas tested strong providing support for kindey function.

About 80% of the Splenda® is passed directly to the large intestine and rectum, muscles like the Quadriceps and Hamstrings would be affected. Adding Okra Pepsin® demonstrated strengthening of the Quadriceps, the small intestine associated muscles. And the addition of Gastro-fiber® strengthened the Hamstrings.

Splenda® is non-caloric, the sugar handling organs like the pancreas should not engage when it is ingested but Latissimus Dorsi muscles were weak. The addition of Pancreatrophin PMG® strengthened the Latissimus Dorsi.

Silymarin aids hepatic function by improving Phase 1 detoxification, it follows that the hepatic function would be effected by this support as was demonstrated by the positive PMS muscle test. Silymarin is also supportive of healthy intestinal function. However, in this experiment it had no effect upon Hamstring or Quadricep function, probably because the intestinal interaction is not enough to support the effect Splenda® has upon the intestines.

Conclusion

Establishing the relationship between the organ/muscle relationships, as was found in the first paper, allows for further exploration of the effects of Splenda® upon these pathways. The ability of specific nutritional supplimentation to have an effect on these muscle/organ pathways further demonstrates the point that Splenda® has a negative effect upon the functional muscle strength of individual muscle/organ relationships. More exploration of nutritional items could further define the chemical pathways that are influenced by Splenda® and the support that will correct these effects.

References

1. Muzinski, S., Muzinski, K., "Preliminary Study on the Affects of Sucralose on Metabolic Pathways," (June 2005).

2. Walther, David S., Applied Kinesiology, Synopsis, (Pueblo, CO., Systems DC, 1988).

3. Walther, David S., Applied Kinesiology, Synopsis, (Pueblo, CO., Systems DC, 1988).

4. Standard Process, Inc., Arginex®, Product Information No.1140.

5. MediHerb, Inc., Silymarin, Product Information No. M1420.

6. Standard Process, Inc., Gastro-fiber®, Product Information No.4615.

7. Standard Process, Inc., Okra Pepsin®, Product Information No.6080.

8. Standard Process, Inc., Pancreatrophin PMG®, Product Information No.6650.

The Results of Nutritional Preloading Against Sucralose Muscle Testing

Scott E. Muzinski, D.C., N.M.D. • Kimberly R. Muzinski, D.C.

Abstract

A volunteer was preloaded with UltraClear® for one week to determine if preloading would negate the effects of Sucralose on observed muscle weaknesses as recorded in two previous papers.[1, 2] The results showed that preloading had no noticeable effect upon observed muscle weaknesses when Sucralose was introduced after one week of preloading.

Key Indexing Terms: Nutritional preloading, Sucralose, muscle testing, applied kinesiology.

Introduction

As was demonstrated in the authors' first and second papers on this subject, Splenda® has an effect on several muscle/organ relationships.[1, 2] Further, those effects can be negated by adding nutritional supplementation specific for the individual involved muscle/organ relationship. A broad-spectrum detoxification support can have an effect on all of the effected pathways involved. This was demonstrated in the author's first paper by using UltraClear® from Metagenics®.[3] Based on these results a clinical trial was conducted to determine if preloading a volunteer with a broad spectrum support, specifically UltraClear®, could prevent the noted muscle/organ weaknesses upon standardized AK muscle testing after a week of preloading.

Materials and Methods

One volunteer was chosen for this study. A group of muscles were tested in the clear using standard AK technique. The test subject was also tested for switching and cranial faults. None were detected. Psoas, Pectorals Major Sternal division (PMS), Quadriceps, Hamstring, and Latissimus Dorsi muscles, both right and left, were all tested and found to be strong. These muscles were selected because each has a relationship to an organ-system function according to standard AK practice. Psoas to the kidney, PMS to the liver, Quadriceps to the small intestine, Hamstrings to the large intestine, and Latissimus Dorsi to the pancreas.[4]

The test subject was placed supine and given approximately .25 gm of Splenda® orally and all muscles were re-tested. In the re-test all muscles were weak according to AK terminology.[5] This demonstrated that Splenda® has an effect upon the volunteer consistent with the results from the previous studies.

The test subject then had approximately .25gm of UltraClear® added orally along with the already present Splenda® and the muscles were re-tested. All muscles were found to be strong at this time.

Next, the test subject's palate was cleared with a wash of filtered water. K27 and all muscles were re-tested to confirm that all had returned to a normal strong state. The test subject was then given .25gm of UltraClear® orally and tested to determine if they had a nutritional need for the product when Splenda® was not present. No need for the product was found in this volunteer when Splenda® was not present according to AK oral nutritional testing, using the muscles outlined above.

The test subject was then given instructions as to the proper daily use of UltraClear®. This was to be done with one scoop in water TID for 5 days. The test subject was also to avoid any food or food product that contained Splenda® or any known form of Sucralose until the re-testing, to be conducted at the end of the 5th day approximately 1 hour after the 3rd scoop of UltraClear® had been taken.

The test subject returned for re-testing on the 5th day about 1 hour after taking the 3rd scoop of UltraClear®. Their pallet was cleansed with filtered water to be sure that any remaining UltraClear® was cleared from their oral cavity.

The test subject was placed supine and tested for switching and cranial faults. None were detected. Psoas, Pectorals Major Sternal division (PMS), Quadriceps, Hamstring, and Latissimus Dorsi muscles, both right and left, were all tested and found to be strong. The test subject was then given approximately .25 gm of Splenda® orally and all muscles were re-tested following the procedure outlined above.

Results

When Splenda® was added to the nutritionally pre-loaded test subject, Psoas, Pectorals Major Sternal division (PMS), Quadriceps, Hamstring, and Latissimus Dorsi muscles, both right and left, were all tested and found to be weak according to AK terminology.

Discussion

Splenda® affects many of the body's detoxification pathways and muscle/organ relationships. Therefore each system that could be identified as being involved had one representative muscle test incorporated into this study, as has been the protocol for the previous papers on the subject. The hepatic detoxification pathways are responsible for processing many things. Hepatic function is affected and measured by testing the muscles that are specific to that organ relationship, as was demonstrated by PMS upon testing. Splenda® is only partially processed by the liver, and the portions that are moved from the bloodstream and filtered by the kidneys have an effect on kidney function. Muscles such as the Psoas are affected, as has been demonstrated.

About 80% of the Splenda® is passed directly to the large intestine and rectum, muscles like the Quadriceps and Hamstrings are also affected. Splenda®, a sugar substitute, has some effect on the sugar handling organs like the pancreas and this was demonstrated by the Latissimus Dorsi muscle test.

The results of this trial demonstrated that the effect of Sucralose was not preemptively counteracted by nutritional support. It would appear that the body uses UltraClear® as a food substance and that it is used up or excreted at the time of ingestion. Some nutrients may be indivually stored such as fat soluble vitamins, but the net effect is lost in about the same time as normal digestion, absorption, circulation and excretion, i.e., the normal digestive cycle. If this is in fact the case, then taking the substance will generally not work as a preventative, because it will only be effective while it is actively circulating in the blood stream. It certainly demonstrates that pre-loading does not leave extra support available for the various detoxification pathways except while still actively in the blood stream. The possibility of boosting patient's detoxification pathways so they can continue with the intake of substances such as Splenda® without the negative effects apparently does not work in oral nutrient testing. It might be possible to take a substance such as UltraClear® at the same time as Splenda® or just before or after it (during the same digestive cycle) and have the effects countered systemically, but that would not be measurable easily by AK muscle testing and that was not the scope of this paper.

Conclusion

Establishing the relationship between the organ/muscle relationships, as was found in the first paper, allows for further exploration of the effects of Splenda® upon these pathways. The ability of specific nutritional supplimentation to have an effect on muscle/organ pathways further demonstrates the point that Splenda® has a negative effect upon the functional muscle strength of individual muscle/organ relationships. Pre-loading a patient with nutrients was demonstrated to be ineffective in preventing the effects of Splenda®. It might be possible to take a substance such as UltraClear® at the same time as Splenda® or just before or just after it (during the same digestive cycle) and have the effects countered, but that was not demonstrated in this paper. If there is a way to prevent the effects of Splenda® in advance it was not found in pre-loading.

References

1. Muzinski, S., Muzinski, K., "Preliminary Study on the Effects of Sucralose on Metabolic Pathways," (June 2005).

2. Muzinski, S., Muzinski, K., "The Effects of Sucralose on Muscle/Organ Relationships With Nutritional Testing," (Dec. 2004).

3. Metagenics Product Catalogue, UltraClear®, pp. 52.

4. Walther, David S., Applied Kinesiology, Synopsis, (Pueblo, CO., Systems DC, 1988).

5. Walther, David S., Applied Kinesiology, Synopsis, (Pueblo, CO., Systems DC, 1988).

The Brainstem and Manual Muscle Testing

James Otis, D.C., D.A.C.N.B.

Abstract

This article offers a brief review muscle physiology, spinal cord function, and the modulating effects of norepinephrine (NE) and serotonin (5HT) on muscle function, all with an emphasis on factors that effect muscle test outcome. It describes five manual muscle test procedures that are used to evaluate brainstem function, the neuro-physiological relevance of each procedure, and expected muscle test outcomes in response to physiological brainstem stimulation. It proposes that in the context of a full neurological exam, specific muscle test procedures can be used as a sensitive, easily administered diagnostic tool for the evaluation of brainstem function.

Key Indexing Terms: Reticular formation, reticular activating system, norepinephrine, serotonin, manual muscle testing.

Introduction

Each muscle participates in a number of functions. Shoulder muscles participate in posture and locomotion, reaching, throwing, and lifting. Each function requires the activation of specific combinations of motor neurons in the spinal cord and each function is controlled from different areas of the brain. Muscle tests can be designed to evaluate specific motor functions and by extension the areas of the brain that control those functions. This article describes five manual muscle test procedures that are used to evaluate brainstem function, the neuro-physiological relevance of each procedure, and expected muscle test outcomes in response to physiological brainstem stimulation.

Section 1: Motor Control

Overview

The spinal cord and brain coordinate all of our movement, voluntary and automatic. The cerebral cortex initiates and sets the direction for voluntary movements. Cortical motor areas connect with a variety of neurons in the brainstem that in turn project to the spinal cord. Cortical motor areas also project to the spinal cord where they connect with interneurons, which in turn project to the alpha motor neurons (AMNs) that activate muscle fibers. Cortical motor areas have very few direct connections to the AMNs that activate muscle cells, and the direct connections that do occur are mainly with AMNs that innervate hand muscles.

The cerebral cortex delegates the detailed work of coordinating muscle movement to groups of cells in the brainstem and spinal cord. Commands from the cerebral cortex and other areas of the brain activate specific areas of the brainstem, which in turn activate pattern-generating neurons in the spinal cord. The brainstem coordinates automatic and repetitive movements, and muscle tone.[1] Mammals that have had their forebrains removed can be made to walk, trot and gallop by stimulating different locomotor regions in the brainstem. The movements are well coordinated and the animal is able to maintain largely adequate balance.[2]

Muscle Physiology

Movement and posture is accomplished by muscle activity. Each muscle contains hundreds of thousands to millions of independent, contractile units called muscle fibers. There are three types of muscle fibers, slow-twitch fibers (S), fast-twitch fatigable fibers (FF), and fast-twitch fatigue-resistant fibers (FR). Type-S fibers are smaller, and type-FF are larger. Fibers of the same type are grouped together as motor units. Each motor unit is controlled by a single motor neuron in the spinal cord or brainstem.

Motor units within each muscle have a wide range of properties. Type-FF motor units can generate 100 times the force of type-S motor units and they can contract five times as fast. On the other hand type-S motor units have ten times the endurance of type-FF motor units.[3]

Each muscle contains a mixture of fiber types. Proximal, postural muscles have a higher percentage of slow-twitch fibers, and distal hand muscles have a higher percentage of fast-twitch fibers. Routine posture and locomotion is accomplished almost entirely by slow-twitch fibers. Fast, explosive forceful movements of proximal muscles engage a higher percentage of fast-twitch fibers as do movements of the eyes and fingers.

Alpha motor neurons (AMNs)

Every part of the nervous system involved in the control of muscle function must do so by acting directly or indirectly on AMNs. AMNs innervate the extrafusal muscle fibers that do the work of the motor system.

AMNs vary in size, physiological properties, and function.[3, 4, 5, 6, 7] Small AMNs and their associated type S motor units are most active in tonic postural activities. Large AMNs and the associated type FF motor units are most active in fast forceful motions. A higher percentage of small AMNs innervate postural muscles, and a higher percentage of large AMNs innervate finger muscles.

Small AMNs are more excitable; they have a lower threshold of activation. They have a lower frequency of firing, thinner axons and slower conduction speeds. They innervate as few as 100 small, slow-twitch muscle fibers.

Large AMNs are less excitable; they have a higher threshold of activation. They have a higher frequency of firing, large diameter axons and faster conduction speeds. They innervate as many as 1000 large, fast-twitch muscle fibers.

Large and small AMNs have different properties, different functions, and they are activated from different parts of the nervous system. Many parts of the nervous system preferentially activate one group or the other, often facilitating one and inhibiting the other.[3, 4, 5, 6, 7]

- Rubrospinal and corticospinal input facilitates small AMNs and inhibits large AMNs.
- Group 1-a afferent input projects primarily to small AMNs.
- Group 2 afferent input projects primarily to large AMNs (as well as gamma motor neurons GMNs as discussed later)
- Group1-b afferent input inhibits small AMNs and facilitates large AMNs.
- Recurrent inhibition from Renshaw cells and reciprocal 1A inhibition is distributed uniformly between small and large AMNs.
- Input from the lateral vestibular nuclei preferentially stimulates large AMNs.

Plateau Potentials (PP)

Until the 1960's it was thought that the membranes of motor neurons were passive and the amount of synaptic input to the cell correlated directly with the output from the cell. In the 1970's researchers discovered that motor neurons could exhibit periods of sustained firing that were relatively independent of the amount of

synaptic input. It was discovered that this sustained firing is due to persistent inward currents that were large enough to create a sustained shift in the membrane potential, called a plateau potential (PP)[6, 7, 8]

In the 1980's it was discovered that these phenomena are dependent upon projections of NE and 5HT from the brainstem. 5HT and NE facilitate AMNs by creating persistent inward currents in the cell membrane that create a long lasting (up to a minute) increase in the membrane potential called a plateau potential. This increases the cell's excitability so that the same amount of synaptic input creates up to six times greater output from the cell.[6]

PPs are most likely to occur in small AMNs and least likely to occur in large AMNs. PPs last for up to a minute for small AMNs, but only 1–2 seconds for large AMNs[3, 5, 6] This is discussed more in a later section of this article about pre-loaded muscle tests.

Gamma Motor Neurons (GMN) and Muscle Spindle Cells (MSC)

GMNs innervate the intrafusal muscle fibers in muscle spindle cells that keep stretch receptors at a relatively constant length as the muscle changes length. MSCs contain stretch receptors that signal how fast the muscle is changing position (phasic signal) and how much it has changed position (tonic signal). MSCs are embedded in muscles. The ends of the MSC are firmly attached to the connective tissue matrix of the muscle and the length of MSCs changes in tandem with the length of the muscle. The sensory stretch receptors are suspended between the ends of the muscle spindle cell by intrafusal muscle fibers. As the muscle changes length, intrafusal fibers contract and relax in order to keep the stretch receptors at a relatively constant length. If the stretch receptors are overly slack or overly tensed they will send inappropriate signals to the central nervous system. GMNs regulate the strength of the stretch reflex by effecting the sensitivity of stretch receptors, which in turn determine the strength of the stretch signal that travels back to the spinal cord.[7, 9, 10]

GMNs are activated independently of AMNs. GMNs receive input about muscle length from group-2 afferents, propriospinal networks and a variety of suprasegmental sources.[11, 12]

5HT and NE provide strong (and opposite) modulation to the amount of group-2 feedback to GMNs.[13, 14] This is discussed more in a later section of this article about post-movement muscle tests.

Brainstem Reticular Formation and Muscle Tone

The brainstem consists of the medulla, pons, and mesencephalon. It controls many important functions in the body including respiration, cardiovascular function, gastrointestinal function, equilibrium, posture and automatic, stereotyped movements of the body.

The reticular formation and the vestibular nuclei located in the brainstem play an especially large role in the control of posture. Reticulospinal pathways project primarily to small AMNs, and vestibulospinal pathways have a larger effect on large AMNs. This article discusses reticulospinal control of posture. A future article will discuss vestibulospinal influence on posture and muscle function.

The reticular nuclei are divided into two major groups; the pontine reticular nuclei that extend throughout the pons and into the mesencephalon, and the medullary reticular nuclei that extend the length of the medulla. These two sets of nuclei function largely antagonistically to each other. The pontine reticular formation excites extensor antigravity muscles and the medullary reticular formation inhibits them. They transmit signals to the spinal cord through medial and lateral reticulospinal tracts respectively.[15]

The pontine reticular nuclei receive especially strong input from vestibular nuclei. The medullary reticular nuclei receive especially strong input from rubrospinal, corticospinal and other higher brain centers. When the brain stem is severed between the pons and mesencephalon (leaving the pontine and medullary reticular systems as well as the vestibular system intact) the animal develops decerebrate, or extensor rigidity. This

occurs because the medullary reticular formation fails to inhibit extensor muscles when it is deprived of its normal excitatory input from the cerebral cortex, red nucleus, and basal ganglia. Rigidity occurs primarily in antigravity muscles of the neck, trunk and the extensor muscles of the legs.[15]

In simple terms the pons facilitates extension and the medulla facilitates flexion, and these effects are strongest in muscles of the neck, back and legs. When a person is standing, tonic activation of the pons facilitates extensor antigravity muscles. When the person begins to walk, signals from higher brain centers activate specific parts of the medulla to inhibit extensor muscles and allow the swing phase of gait.[1]

Reticular Activating System and Muscle Tone

The reticular-activating system (RAS) acts through reticulospinal projections to modulate posture, muscle tone, and locomotion. It is comprised of three primary cell groups in the mesopontine tegmentum; the pedunculopontine nucleus that secretes acetylcholine (ACH), the locus coeruleus that secretes NE, and the raphe nuclei that secrete 5HT. These three groups of neurons each project to widespread areas of the nervous system where they have modulatory effects.[16, 17, 18]

The three groups of neurons which make up the RAS interact with each other, facilitating or inhibiting each other as they project to their respective targets to control the sleep-wake cycle and arousal, modulate the fight or flight response, and regulate posture and locomotion. It is not surprising that the RAS is linked to the motor system in order to optimize attack or escape. During deep sleep and REM sleep the RAS causes a loss of muscle tone so that we don't act out our dreams. During waking the RAS modulates muscle tone and locomotion via the reticulospinal tracts.[16, 17, 18, 19]

This article will focus on NE and 5HT projections from the RAS, and their effect on muscle test outcomes.

NE and Muscle Tone

NE modulates the function of many diverse parts of the nervous system. Neurons containing NE have the widest divergence and project to more parts of the nervous system than any other neurons in the brain.[20] All of the NE in the central nervous system is produced in the brainstem, most of it in the locus coeruleus (LC).

The LC neurons lie in a cluster in the periventricular gray area in the dorsolateral corner of the fourth ventricle in the rostral pons. It is named for the bluish color that is cast from its melanin content onto the floor of the fourth ventricle. The LC is located immediately medial to the medial vestibular nucleus and to the caudal end of the mesencephalic trigeminal nucleus.

The pattern of distribution and the types of contact that projections from the LC make with other neurons is similar to the pattern of distribution and the types of contacts that are made by peripheral sympathetic neurons. The proximity and close association of the LC with the mesencephalic trigeminal nucleus suggests that it, like the mesencephalic trigeminal nucleus, developed from neural crest cells and migrated into the central nervous system to form the equivalent of a widely divergent peripheral sympathetic ganglion.[20]

The close association of the LC and mesencephalic trigeminal nucleus is the basis of a physiological brainstem challenge discussed in a later section of this article.

The LC projects ipsilaterally to effect multiple areas in the spinal cord where NE has the following actions.

- Facilitates small AMNs[3, 5, 6]
- Inhibits renshaw cells (disinhibits AMNs)[21, 22]
- Dampens stretch reflexes[23]
- Strongly depresses synaptic actions of group-2 muscle afferents (which monitor muscle position) on GMNs and on intermediate zone neurons that provide input to AMNs and GMNs.[13]

The LC exerts prominent facilitory influence on posture by directly exciting AMNs for extensor (and flexor) muscles and by releasing those AMNs from recurrent renshaw cell inhibition.[21, 22]

NE has its greatest effects on proximal extensor, abductor, and external rotator muscles, especially in the legs. (See the discussion in Brainstem Reticular Formation and Muscle tone above.)

5HT and Muscle Tone

Cells that produce 5HT are all located in raphe nuclei in the midline of the brainstem from the medulla to the mesencephalon. They project extensively through the nervous system. Raphe nuclei in the pons and mesencephalon project rostrally, and those in the medulla project to the spinal cord. Jacobs and his research group (24) implanted micrometers in order to study the function of 5HT in behaving cats. He reports that the primary role of the medullary serotonergic system appears to be increasing motor tone and facilitating repetitive motor activity. 5HT inhibits nociception and increases sympathetic nervous system activity in order to support its primary function of increased motor tone. 5HT levels decrease during sleep, and fall to zero during periods of REM sleep, when muscle tone is profoundly reduced.[24, 25]

5HT has its greatest effects on proximal flexor, adductor, and internal rotator muscles, especially in the legs. (See the discussion in Brainstem Reticular Formation and Muscle tone above.) Medullary raphe nuclei project bilaterally to the spinal cord where 5HT has the following effects.

- Facilitates small low threshold AMNs[3, 5, 6]
- Facilitates gamma motor neurons, GMNs, and thereby enhances stretch reflexes.
- Strongly facilitates synaptic actions of group 2 afferents on GMNs and on intermediate zone neurons that provide input to AMNs and GMNs.[12, 13]

Serotonergic neurons in the medulla are sensitive to carbon dioxide and/or pH. Increased carbon dioxide (3%, which is fairly sensitive) causes increased serotonergic activity and increased breathing. Decreased levels of carbon dioxide cause decreased serotonergic activity in medullary neurons that project to the spinal cord. There does not appear to be phasic activity of serotonergic neurons related to the respiratory cycle. [24]

Summary: Contrasting effects of 5HT and NE on Muscle Function

- 5HT and NE each increase muscle tone of both flexor and extensor muscles. As a pontine nucleus the LC and its adrenergic projections (NE) have a greater effect on extensors than on flexors. Serotonergic projections from the raphe nuclei in the medulla have a greater effect on flexors than extensors.
- 5HT enhances feedback from group-2 muscle afferents to GMNs and to the intermediate zone neurons that provide input to GMNs. NE depresses group-2 muscle afferent feedback to those same targets.
- 5HT and NE have opposite effects on GMNs and the stretch reflex. 5HT weakly facilitates the muscle stretch reflex and NE strongly dampens the stretch reflex.

Spinal cord functions that effect muscle tests

Stretch reflex

Abrupt non-volitional lengthening of a muscle elicits a stretch reflex that causes the stretched muscle to contract and resist lengthening. The reflex is a postural mechanism to prevent unintended perturbations of a desired posture. It is accomplished by the activation of dynamic bag stretch receptors in MSCs. The receptors send signals to the cord through 1A afferents that have direct monosynaptic input to small AMNs that maintain posture. (Group 2 afferents also contribute to the stretch reflex in a lesser extent.)

In response to the stretch signal from 1A afferents, AMNs in the spinal cord increase their rate of firing to activate the muscle and resist lengthening. This process is not under voluntary control. An inadequate

stretch reflex causes muscle tests that involve an abrupt increase of force to fail.

The effectiveness of the stretch reflex depends upon
1. The sensitivity of the dynamic bag stretch receptors which is in turn dependent upon activation from dynamic GMNs in the ventral horn of the spinal cord.
2. The ability of the AMNs to respond to excitatory input.

NE and 5HT modulate both factors determining the efficacy of the stretch reflex.
1. NE facilitates AMNs and inhibits GMNs. It has a strong net damping effect on the stretch reflex.[23] (NE agonist medications are helpful in reducing exaggerated stretch reflexes and spasticity after spinal cord injuries.)
2. 5HT provides weak facilitation for stretch reflexes.

Group-2 afferent input to the cord

As a muscle changes length the nervous system is challenged to maintain adequate feedback from the stretch receptors that are embedded in the muscle. Muscle spindle cells are embedded in the muscle, and stretch receptors are embedded in the muscle spindle. MSCs change length when the muscle changes length. Muscle fibers within the MSC contract and relax to maintain a relatively constant length in the receptor portion of the spindle as the muscle and the muscle spindle gets shorter or longer. This is coordinated by GMNs in the spinal cord.[7, 9, 10]

GMNs activate intrafusal muscle fibers (inside the muscle spindle cells) to maintain optimal length of the receptor as the muscle changes length. GMNs are activated (independently of AMNs that innervate the extrafusal muscle fibers) by descending commands from the brainstem and cerebral cortex, from spinal propriospinal networks, and by feedback from group-2 muscle afferents.

Group-2 afferents convey signals from receptors (static bag and chain fibers in muscle spindle cells) that register muscle length. Group-2 afferents project to numerous targets in the spinal cord including neurons in the dorsal horn, intermediate zone and GMNs in the ventral horn. GMNs use information from group-2 afferents to calculate appropriate drive to intrafusal fibers and thereby maintain optimal spindle sensitivity.

5HT and NE modulate the input from group-2 afferents to spinal cord targets. 5HT facilitates the transmission of information from group-2 afferents to GMNs and NE depresses that transmission.[13, 14]

Modulation from NE and 5HT allows the brainstem to reconfigure spinal circuits and spinal reflexes on a second by second basis to meet the needs of different movements, and appropriate modulation is necessary in order to maintain spindle sensitivity during and after movement. The post-movement muscle tests described below evaluate the nervous system's ability to maintain spindle sensitivity and an appropriate stretch reflex during and after movement.

Interactions of Homologous columns in the spinal cord

The AMNs and GMNs that innervate a muscle reside in a continuous column of cells that span three or four spinal segments in the ventral horn of the spinal cord. There is a somatotopic organization of motor neurons (MNs) such that MNs innervating proximal muscles are in the medial portion of the ventral horn and MNs innervating distal muscles are in the lateral portion of the ventral horn. MNs to flexor muscles are more dorsal and MNs to extensors are more ventral.

Neurons that go to muscles with similar functions are grouped in close proximity in homologous columns. MNs in homologous columns interact in predictable ways. Excitation and inhibition spread from one group to another. When a muscle is activated, antagonist muscles are inhibited through reciprocal inhibition, and muscles with synergistic actions are facilitated.[10]

For example, when a flexor muscle is activated,
- Other flexors in that limb are activated to some degree.
- The antagonist extensor is inhibited, and
- Other extensors in that limb are inhibited to some degree as well.

Recurrent Inhibition

Recurrent inhibition is a negative feedback system that is used in many areas of the nervous system. Renshaw cells in the ventral horn of the spinal cord function as part of a recurrent inhibition loop that inhibits AMNs. AMNs send collateral axons that excite Renshaw cells that in turn inhibit the AMNs.

Renshaw cells are inhibitory cells in the ventral horn with a variety of targets and a variety of segmental and suprasegmental input sources. They are part of the recurrent AMN inhibitory loop described above. They serve as a regulator that can vary the gain of AMN recurrent inhibition.

NE depresses the activity of Renshaw cells, thus reducing the degree of recurrent inhibition to AMNs. NE facilitates the action of AMNs directly and through disinhibition (inhibiting the Renshaw cells that inhibit the AMNs)[21, 22]

Section 2: Manual Muscle testing to Evaluate Integration of Small AMNs

Introduction

AMNs have a range of sizes, physiological properties and functions. Small AMNs innervate slow-twitch muscle fibers that predominate in proximal muscles and perform most of the work of posture and locomotion. Large AMNs innervate fast-twitch muscle fibers that predominate in distal muscles (especially hand) that perform fine, delicate, fractionated movement. Fast-twitch muscles also supply fast explosive power for proximal muscles.

Small and large AMNs perform different functions and they are activated from different areas of the nervous system. Muscle tests can be modified so that they challenge one group of AMNs more than the other. This article will focus on tests that challenge small AMNs. (A future article will discuss tests that challenge large AMNs)

5HT and NE are projected to the spinal cord from different parts of the brainstem and in the spinal cord they have opposite effects on GMNs and on group-2 afferent input to GMNs. GMNs regulate stretch reflexes during and immediately following movement. Muscle tests can be designed so that they challenge the ability of GMNs and the brainstem systems that regulate them to maintain appropriate stretch reflexes during and immediately following movement.

This article describes manual muscle tests that are used to evaluate the integration of small AMNs. Within the context of a complete neurological examination, manual muscle testing provides a sensitive, easily administered method of evaluating brainstem function. Each test involves examiner application of a fast final force that elicits a stretch reflex. The muscle is evaluated to determine whether it can in can resist lengthening.

Muscle test variables

In addition to the final eccentrically applied test pressure, tests for small AMNs have the following variable factors.

1. **Contraction of muscles distal to the test muscle**. The test force can be applied while the patient maintains simultaneous isometric contraction of muscles distal to the test muscle.

2. **Amount and duration of pre-load force**: The test force can be applied after the muscle is already contracting strongly to resist a pre-loading force, after it is contracting lightly to resist a pre-loading force, or when the muscle has not been subjected to a pre-loading force.

3. **History of the antagonist muscle**. The test force can be applied after the antagonist muscle is contracted or stretched

4. **Pre-test movement.** The test force can be applied immediately after the muscle is lengthened, immediately after it is shortened, or while the muscle is at a constant length.

Muscle test procedure: Isometric contraction of muscles distal to the test muscle

Isometric contraction of muscles distal to the test muscle: Mechanics

The patient maintains isometric contraction of muscles distal to the test muscle while the test is performed.

Isometric contraction of muscles distal to the test muscle: Physiology

Isometric contraction activates Golgi tendon organs that send 1b afferent input to the spinal cord. 1b input inhibits small AMNs and facilitates large AMNs[4, 26, 27, 28] Due to the interactions of homologous columns of neurons in the spinal cord**,** excitation or inhibition of one group of muscles spreads to other muscles in that limb which have a similar function. For example inhibition of small AMNs that innervate distal flexor muscles causes inhibition of the small AMNs that innervate proximal flexor muscles and vice versa.

Isometric contraction of muscles distal to the test muscle: Significance

Isometric contractions of distal muscles can be used to enhance the sensitivity of proximal muscle tests. For example holding a fist (isometric contraction of finger flexors) will cause a higher percentage of pre-load tests (described below) for shoulder flexor muscles to fail, and holding the fingers and wrist in extension (isometric contractions) causes a higher percentage of rebound tests (described below) for shoulder abductors to fail.

Muscle test procedure: Pre-loaded muscle test

Pre-loaded muscle test: Mechanics

- Have the patient resist light isometric pressure for two seconds prior to applying the test pressure.
- Pre-load weakness is most often observed in proximal rather than distal, leg more than arm, flexor rather than extensor, and adductor, internal rotator muscles rather than abductor external rotator muscles (muscles that are most strongly effected by medullary, serotonergic projections)
- **The sensitivity of pre-load tests is enhanced when homologous muscles distal to the test muscle are isometrically contracted for the duration of the test.** For example, a pre-load test of arm flexors is enhanced when the fingers are held in flexion ("make a fist"), and a pre-load test of hamstring muscles is enhanced if the test is performed while the patient maintains foot dorsiflexion.

Pre-loaded muscle test: Physiology

The final test force causes a stretch that reflexively causes the muscle to resist lengthening. The stretch reflex occurs through activation of small AMNs. If the stretch signal is dampened, or if the small AMNs fail to respond adequately, the stretch response is inadequate and the muscle test fails.

Light pre-load pressure creates an isometric contraction that inhibits the small AMNs that respond to the stretch signal. Heavy pre-load pressure activates large AMNs that are then available to resist the test pressure. (Large AMNs are not activated by the stretch reflex) For this reason, light pre-load pressure often causes a test that fails, while heavy pre-load pressure does not.

PPs of small AMNs persist for up to a minute. PPs of large AMNs decay after 1-2 seconds. The two-second pre-load activation period is longer than the PPs produced for large AMNs, and the length of time reduces the force generated by large AMNs and fast-twitch fibers. (3,5,6)

5HT and NE have the following effects on pre-load muscle tests.

- Low 5HT causes decreased integration of small AMNs. It has little effect on the stretch reflex. It tends to cause pre-load muscle weakness.
- Low NE causes decreased integration of small AMNs and it also causes an increased stretch reflex. Because of the increased stretch reflex it does not tend to cause pre-load muscle weakness.

Pre-loaded muscle test: Significance

Pre-load weakness is frequently indicative of decreased serotonergic drive from the ipsilateral medulla.

Muscle test procedure: Rebound muscle test

Rebound muscle test: Mechanics

- Tap the arm or leg in a direction that stretches the antagonist of the muscle that is to be tested. Do this immediately prior to applying the test pressure.
- Rebound tests are best seen with extensor, abductor and external rotator muscles (that receive relatively large pontine/NE drive)
- Rebound tests are enhanced if the wrist or ankle is held in extension. (Plantar flex the foot while performing a rebound test with the piriformis muscle and hold the wrist and fingers in extension when performing a rebound test with the middle deltoid)

Rebound muscle test: Physiology

Tapping elicits a stretch response. If the stretch response is exaggerated the muscle that is stretched inhibits the test muscle (through reciprocal inhibition) to an extent that the test muscle fails to resist test pressure.

Rebound muscle test: Significance

Rebound test weakness is frequently due to an increased stretch reflex secondary to decreased NE.

Muscle test procedure: Antagonist-activation muscle test

Antagonist-activation muscle test: Mechanics

- Apply light isometric pressure to cause contraction of the antagonist muscle for two seconds immediately prior to applying the test force.
- The test is enhanced if distal muscles homologous to the antagonist are held in isometric contraction for the duration of the test. (Hold a fist while performing an antagonist activation test for the middle deltoid muscle)

Antagonist-activation muscle test: Physiology

Under normal conditions, light isometric contraction inhibits small AMNs. This author believes that in the case of excessive 5HT facilitation of AMNs, type 1b afferent feedback fails to inhibit the AMNs. Instead the antagonist AMN are facilitated and cause reciprocal inhibition of the test muscle.

Antagonist-activation muscle test: Significance

This author believes that weakness following antagonist activation frequently indicates exaggerated reciprocal inhibition due to increased 5HT drive to the AMNs. (The weakness pattern is frequently temporarily abolished by physiological challenges that decrease CO2 as described below)

Muscle test procedure: Post-movement muscle test

Post-movement muscle test: Mechanics

- **Post-shortening test**: Have the patient move the muscle to be tested through (at least) 30 degrees of its range of motion in a direction which shortens the muscle. Have them stop at a predetermined position. Immediately apply the test pressure.

- **Post-lengthening test**: Have the patient move the muscle to be tested through (at least) 30 degrees of its range of motion in a direction which lengthens the muscle. Have them stop at a predetermined position. Immediately apply the test pressure.

Post-movement muscle test: Physiology

Post-shortening and post-lengthening tests evaluate the ability of GMNs to maintain spindle sensitivity during (and immediately after) movements. **Two primary factors influence the GMNs ability to maintain post-movement receptor sensitivity**.

1. The efficiency of synaptic input from group-2 afferents to the GMN, as discussed earlier.
2. A variety of descending commands from supersegmental sources to the GMN (subject of a future paper)

The efficiency of group-2 afferent input is strongly modulated by the ratio of NE and 5HT in the spinal cord. The higher the 5HT / NE ratio, the more group-2 feedback to GMNs. (13)

- **A low 5HT/NE ratio** (low 5HT and /or high NE) causes decreased feedback from group-2 afferents to GMNs. GMNs think that the muscle has moved less than it has. GMNs fail to maintain adequate drive to intrafusal fibers and the receptor portion of MSCs is offloaded. It goes slack and fails to generate an adequate stretch signal**. The muscle fails to resist test pressure after it is shortened.**
- **A high 5HT/NE ratio** (high 5HT and /or low NE) causes increased feedback from group-2 afferents to GMNs. GMNs think that the muscle has moved more than it has. GMNs generate excessive drive to intrafusal fibers and the receptor portion of MSCs is loaded excessively. This generates excessive tone in the muscle that in turn weakens the antagonist muscle through reciprocal inhibition. **A muscle fails to resist test pressure after it is lengthened** (after its' antagonist is shortened)

Post-movement muscle test: Significance

- **Post shortening weakness might indicate**:

1. Inadequate drive to GMNs from suprasegmental command signals.
2. Decreased feedback from group-2 afferents to GMNs due to a decreased ratio of 5HT/NE.

- **Post-lengthening weakness might indicate**

1. Excessive drive to GMNs from suprasegmental command signals.
2. Excessive feedback from group-2 afferents to GMNs due to an increased ratio of 5HT/NE.

Muscle test procedure: Physiological brainstem challenges

Carbon dioxide (CO2) and trigeminal challenges can be used to evoke predictable changes in muscle function. Challenges are helpful in determining whether positive muscle test results are due to physiological problems in the brainstem or to lesions elsewhere in the nervous system.

Muscle test procedure: CO2-evoked changes in muscle function

Increased CO2: mechanics of the challenge

Have the patient wear a mask or breathe into a paper bag for 5 or 6 respirations immediately prior to performing the tests.

Increased CO2: physiology of the challenge

- Breathing into a mask causes increased CO2 in the serum that triggers an increase of 5HT.
- This increases the 5HT / NE ratio and muscles fail to resist test pressure after they are lengthened (or no longer weaken after they are shortened)
- Increased 5HT causes antagonist activated weakness (or abolishes pre-load muscle test weakness)

Decreased CO2: mechanics of the challenge

Have the patient take 5 or 6 respirations deep rapid respirations immediately prior to performing the tests.

Decreased CO2: physiology of the challenge

- Hyperventilation causes decreased CO2 in the serum that triggers a decrease of 5HT.
- This decreases the 5HT / NE ratio and muscles to fail resist test pressure after they are shortened (or no longer weaken after they are lengthened)
- Decreased 5HT causes pre-load muscle test weakness (or abolishes antagonist activated muscle test weakness).

Significance of the challenges:

If these CO2 challenges abolish a positive muscle test finding, it is likely that the weakness is due to a reversible, physiological medullary raphe nuclei lesion on that side. If CO2 challenges fail to abolish a positive muscle test outcome, there is increased likelihood that the causative lesion is elsewhere in the nervous system.

Muscle test procedure: Trigeminal-evoked changes in muscle function

Bite challenge: mechanism

Have the patient lightly bite a tongue depressor between the molar teeth on one side of the mouth, and test extensor/external rotator muscles on the same side of the body.

Bite challenge: physiology

- This author believes that because of their proximity and (presumed) shared lateral crest origins (Jones, 1991), activation of the mesencephalic trigeminal nucleus causes activation of the locus coeruleus, which results in increased noradrenergic projections to spinal cord neurons, especially those innervating extensors.

- Increased NE causes decreased stretch reflex for extensor muscles and they often fail to resist examiner initiated test pressure.
- Increased NE causes decreased stretch reflexes and abolishes rebound muscle test weakness.
- Increased NE causes a decreased 5HT/NE ratio, and muscles weaken after they or shortened (or no longer weaken after they are lengthened)

Bite challenge: significance

If the trigeminal bite challenge does abolish a rebound or post-lengthening muscle test weakness, there is increased likelihood that the weakness is due to a reversible metabolic lesion of the pons on that side. If it fails to abolish the weakness, there is increased likelihood that the weakness is due to a lesion elsewhere in the nervous system.

Mouth-open trigeminal challenge

Holding the mouth open often causes muscle test outcomes indicative of decreased activation of the locus coeruleus and decreased NE projection to the spinal cord. This is not as consistent as the biting challenge

Discussion

A muscle test evaluates whether a muscle can perform a certain function or group of functions; resist a perturbation, move and then resist a perturbation, generate a large force, etc. Each muscle function requires the activation of specific combinations of large and small AMNs, and large and small GMNs[7] and each function is most effectively directed from a particular area of the brain.

The brainstem coordinates posture and locomotion. Higher brain centers initiate and direct posture and locomotion, but delegate the details to the brainstem and spinal cord. Higher centers of the brain are more directly involved with the generation of fast powerful forces and with the control of hand and finger movement. Corticospinal and rubrospinal pathways carry signals from higher centers in the brain to the large AMNs that activate fast-twitch muscle fibers that are necessary for those actions.

Muscle tests evaluate the performance of particular muscle functions, and by extension the areas of the brain that control those functions. Muscle testing is a sensitive, easily administered diagnostic tool for the evaluation of brainstem (and other supraspinal) function.

Conclusion

This article has focussed on the evaluation of motor function controlled or modulated by the pontine and medullary reticular formation, the LC and the medullary raphe nuclei. It has hopefully extended the theoretical understanding of how to use muscle testing to evaluate brainstem function.

The tests described in this paper can be (and in the population of patients seen by this author, usually are) positive (weak) due to physiological, reversible brainstem dysfunction. They can also be positive due to dysfunction in other areas of the nervous system including the ipsilateral cerebellum, contralateral cerebral cortex or basal ganglia, and the spinal cord or peripheral nerve.

If CO2 and trigeminal challenges reverse positive findings, it is likely that those findings are due to physiological brainstem dysfunction. Physiological brainstem dysfunction can in turn be due problems in other areas of the nervous system. The brainstem does not function well if it receives aberrant input from any area of the

brain. For this reason, positive muscle test findings must be considered in the context of a complete neurological examination.

If CO2 and trigeminal challenges do not reverse positive muscle test findings, it is more likely that the lesions are due to lesions other than physiological brainstem lesions.

Abbreviations

- Abbreviations used in this article
- 5HT serotonin
- AMN alpha motor neuron
- GMN gamma motor neuron
- LC locus coeruleus
- MN motor neuron
- MSC muscle spindle cell
- NE norepinephrine
- PP plateau potential
- RAS reticular activating system

Acknowledgements: The author thanks Virginia Roe, D.C., DABCO, DACNB for editorial review.

References

1. GRILLNER S AND WALLEN P, Innate versus learned movements- a false dichotomy? *Progress in Brain Research, vol. 143, Brain Mechanisms for the Integration of Posture and Movement* vol. 143, edited by Mori S, Stuart D, and Wiesendanger M, Elsevier, 2004, p 3–12.

2. GRILLNER S AND WALLEN P, Innate versus learned movements- a false dichotomy? *Progress in Brain Research, vol. 143, Brain Mechanisms for the Integration of Posture and Movement* vol. 143, edited by Mori S, Stuart D, and Wiesendanger M, Elsevier, 2004, p 3–12.

3. BINDER, HECKMAN, AND POWERS, The physiological control of motor neuron activity, in *Handbook of Physiology, Section 12: Exercise, Regulation and Integration of Multiple Systems*, ed. Rowell and Shepherd, Oxford University Press, New York, Oxford, 1996, p. 3–53.

4. BINDER, HECKMAN AND POWERS, Relative strengths and distributions of different sources of synaptic input to the motor neuronal pool; implications for motor unit recruitment. In: *Sensorimotor Control of Movement and Pos*ture, Edited by Simon Gandevia, Kluwer Academic, New York, 2002, p. 207–212.

5. HECKMAN AND LEE, Synaptic integration in bistable motoneurons, *Progress in Brain Research, Vol. 123, Peripheral and Spinal Mechanisms in the Neural Control of Movement*, ed. M. Binder., Elsevier Science, Amsterdam 1999, p. 49–56.

6. HECKMAN AND LEE, Advances in measuring active dendritic currents in spinal interneurons in vivo, in *Motor Neurobiology Of The Spinal Cord*, ed. Timothy Cope, CRC Press, New York, 2001.

7. OTIS, JAMES. The use of manual muscle testing to assess functional integration of high-threshold versus low-threshold alpha motor neurons. *Proceedings of the Annual Meeting*, ICAK, 2004.

8. HULTBORN H, BROWNSTONE R, TOTH T, AND GOSSARD J, Key mechanisms for setting the input-output gain across the motorneuron pool. *Progress in Brain Research*, *Brain Mechanisms for the Integration of Posture and Movement* vol. 143, edited by Mori S, Stuart D, and Wiesendanger M, Elsevier, 2004, p. 77–96.

9. GUYTON A, AND HALL J, Contraction of Skeletal Muscle, *Textbook of Medical Physiology*, WB Saunders, 2000, p 67–79.

10. PEARSON, K. AND GORDON, J. Spinal Reflexes. *Principles of Neural Science, 4th Edition*, edited by Kandel, Schwartz, and Jessel, McGraw Hill, 2000, p. 713–736.

11. TAYLOR, ELLAWAY, AND DURBABA, Why are there three types of intrafusal fibers? *Progress in Brain Research, Vol. 123*, *Peripheral and Spinal Mechanisms in the Neural Control of Movement*, ed. M. Binder., Elsevier Science, Amsterdam 1999, p. 121–131.

12. TAYLOR A., Give proprioceptors a chance, *Sensorimotor Control of Movement and Posture*, Edited by Simon Gandevia, Kluwer Academic, New York, 2002.

13. JANKOWSKA AND GLADDEN, A Positive Feedback Circuit Involving Muscle Spindle Secondaries And Gamma Motor Neurons In The Cat. , *Progress in Brain Research, Vol. 123*, *Peripheral and Spinal Mechanisms in the Neural Control of Movement*, ed. M. Binder., Elsevier Science, Amsterdam 1999, p. 149–156.

14. JANKOWSKA, HAMMAR, CHOJNICKA AND HEDEN Effects of monoamines on interneurons in four spinal reflex pathways from group 1 and/or group 2 muscle afferents, *European Journal of Neuroscience*, Vol. 12, p. 701–714, 2000.

15. GUYTON, ARTHUR C, Cortical and brainstem control of motor function, *Basic Neuroscience, Anatomy and Physiology*, WB Saunders Company, Philadelphia, 1991, p 214–217.

16. GARCIA-RILL E, Disorders of the reticular activating system, *Medical Hypotheses* 49, p. 379–387, 1997.

17. GARCIA-RILL E, HOMMA Y, AND SKINNER RD, Arousal mechanisms related to posture and locomotion: 1. Descending modulation, *Progress in Brain Research*, *vol. 143, Brain Mechanisms for the Integration of Posture and Movement* vol. 143, edited by Mori S, Stuart D, and Wiesendanger M, Elsevier, 2004, p 283–290.

18. GARCIA-RILL E, HOMMA Y, AND SKINNER RD, Arousal mechanisms related to posture and locomotion: 2. Ascending modulation, *Progress in Brain Research*, *vol. 143, Brain Mechanisms for the Integration of Posture and Movement* vol. 143, edited by Mori S, Stuart D, and Wiesendanger M, Elsevier, 2004, p 291–298.

19. TAKAKUSAKI K, OOHINATA-SUGIMOTO J, SAITOH K, AND HABAGUCHI T, 2004, Role of basal ganglia-brainstem systems in the control of postural muscle tone and locomotion *Progress in Brain Research*, *vol. 143, Brain Mechanisms for the Integration of Posture and Movement* edited by Mori S, Stuart D, and Wiesendanger M, Elsevier, 2004, p. 231–238.

20. JONES, BE, Noradrenergic locus coeruleus neurons: their distant connections and their relationship to neighboring (including cholinergic and GABAergic) neurons on the central gray and reticular formation. *Progress in Brain Research, vol. 88, Neurobiology of the Locus Coeruleus*, edited by Barnes CD and Pompeiano O, Elsevier Science, Amsterdam 1991, p 15–30.

21. POMPEIANO, O. Relationship of noradrenergic locus coeruleus neurons to vestibulospinal reflexes, *Progress in Brain Research, volume 80, Afferent Control of Posture and Locomotion*, edited by Allum J and Hulliger M, Elsevier Science Publishers, Amsterdam, 1989, p. 321–328.

22. POMPEIANO O, Noradrenergic locus coeruleus influences on posture and vestibulospinal reflexes, *Alpha And Gamma Motor Systems*, Ed A Taylor, 1995, p 429–434.

23. MEUNIER, S., KATZ, R., AND SIMONETTA-MOREAU, M., Central nervous system lesions and segmental activity, *Sensorimotor Control of Movement and Pos*ture, Edited by Simon Gandevia, Kluwer Academic, New York, 2002, p. 309–313.

24. JACOBS, MARTIN-CORA, FORMAL, Activity of medullary serotonergic neurons in freely moving animals. *Brain Research Reviews, 40*, p. 45–52, 2002.

25. JACOBS B AND FORNAL C, Serotonin and motor activity, *Current Opinion in Neurobiology*, 1997, 7: p 820–825, 1997.

26. BINDER, M.D. Comparison of effective synaptic currents generated in spinal motorneurons by activating different input systems. In*: Biomechanics and Neural Control of Posture and Movement*. J. M. Winters and P.E. Crago, (eds.) Spinger-Verlag, New York, 2000, p. 74–81.

27. POWERS AND BINDER, Input-Output Functions of Mammalian Motor neurons, *Reviews of Physiology, Biochemistry, and Pharmacology,* 143, Springer, 1999.

28. POWERS RK, AND BINDER M.D., Input-Output Functions of Mammalian Motorneurons, in *Reviews of Physiology, Biochemistry, and Pharmacology, 143*, Springer, pp 137–263, 2001.

Enterogastric Reflex; Powerful Duodenal Factors That Inhibit Stomach

Jose Palomar Lever, M.D., O.S., DIBAK

Abstract

There are many disorders caused by the malfunction of the stomach. We know many syndromes and diseases that are consequences of under or over production of Hydrochloric acid, pepsin, gastric enzymes or less motility of the stomach.

Key Indexing Terms: Arthritis and bursitis of alkalosis source, ileocecal valve syndrome, calcium, protein and fats mal absorption, bloating, colitis, bacterial acid barrier, applied kinesiology.

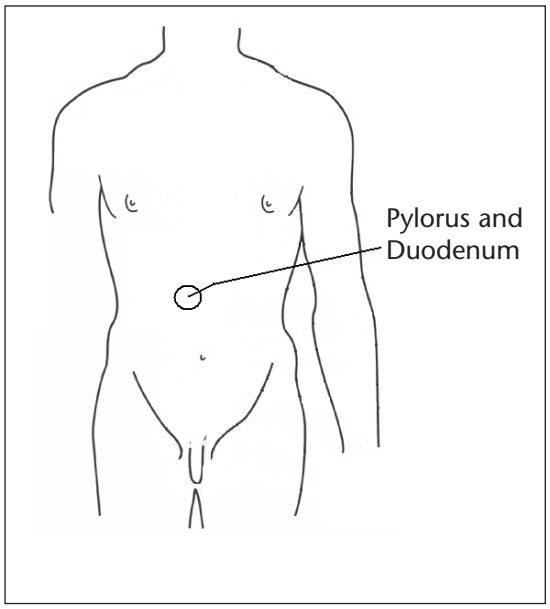
Introduction

We can treat those patients with Hydrochloric acid pills, but after discontinue the HCL we found the patient re-starts to show symptoms again.

The most common causes of the under working stomach are: Temporal cranial fault, Zinc deficit, Vitamin B6 deficit, Guanidine production, Antacid treatment, etc.

The treatment of these causes may not result in correcting the patient's problem; in many cases, it may show frequent recurrences or require supplementation for long periods of time.

We found helpful to search and treat problems by means of fixing the enterogastric reflex.
We found the Pylorus and Duodenum anterior referred pain area between the Stomach and Small intestine referred pain areas, and one inch from midline (right side).



We found the Pylorus and Duodenum posterior referred pain area over 10th Thoracic vertebrae.

Inhibitory Effect of Enterogastric Nervous Reflexes from the Duodenum.
When food enters the duodenum, multiple nervous reflexes are initiated from the duodenal wall and pass back to the stomach to slow or even stop stomach emptying as the volume of chyme in the duodenum becomes excessive.

These reflexes are mediated by three routes:

1. Directly from the duodenum to the stomach through the enteric nervous system in the gut wall,
2. Through extrinsic nerves that go to the prevertebral sympathetic ganglia and then back through inhibitory sympathetic nerve fibers to the stomach, and
3. Probably to a slight extent through the vagus nerves all the way to the brain stem, where they inhibit the normal excitatory signals transmitted to the stomach through the vagi.

All these parallel reflexes have two effects on stomach emptying: first, they strongly inhibit the pyloric pump propulsive contraction, and second, they probably increase slightly to moderately the tone of the pyloric sphincter.

The types of factors that are continually monitored in the duodenum and that can excite the enterogastric reflexes include the following:

1. The degree of distention of the duodenum.
2. The presence of any degree of irritation of the duodenal mucosa.
3. The degree of acidity of the duodenal chyme.
4. The degree of osmolarity of the chyme.
5. The presence of certain breakdown products in the chyme, especially breakdown products of proteins and perhaps to a lesser extent of fats.

The enterogastric reflexes are especially sensitive to the presence of irritants and acids in the duodenal chyme, and they often become strongly activated within as little as 30 seconds. For instance, whenever the pH of the chyme in the duodenum falls below about 3.5 to 4, the reflexes frequently block further release of acidic stomach contents into the duodenum until the duodenal chyme can be neutralized by pancreatic and other secretions.

Breakdown products of protein digestion also elicit enterogastric reflexes; by slowing the rate of stomach emptying, sufficient time is ensured for adequate protein digestion in the duodenum and other upper portions of the small intestine.

Finally, either hypotonic or hypertonic fluids (especially hypertonic) elicit the reflexes. Thus, too rapid flow of non-isotonic fluids into the small intestine is prevented, thereby also preventing rapid changes in electrolyte concentrations in the whole-body extracellular fluid during absorption of the intestinal contents.

Hormonal Feedback from the Duodenum Inhibits Gastric Emptying-Role of Fats and the Hormone Cholecystokinin. Not only do nervous reflexes from the duodenum to the stomach inhibit stomach emptying, but hormones released from the upper intestine do so as well. The stimulus for producing the hormones is mainly fats entering the duodenum, although other types of foods can increase the hormones to a lesser degree.

On entering the duodenum, the fats extract several different hormones from the duodenal and jejunal epithelium, either by binding with “receptors” in the epithelial cells or in some other way. In turn, the hormones are carried by way of the blood to the stomach, where they inhibit the activity of the pyloric pump and at the same time slightly increase the strength of contraction of the pyloric sphincter. These effects are important because fats are much slower to be digested than most other foods.

Precisely which hormones cause the hormonal feedback inhibition of the stomach is not fully clear. The most potent appears to be cholecystokinin (CCK), which is released from the mucosa of the jejunum in response to fatty substances in the chyme. This hormone acts as a competitive inhibitor to block the increased stomach motility caused by gastrin.

Other possible inhibitors of stomach emptying are the hormones secretin and gastric inhibitory peptide (GIP). Secretin is released mainly from the duodenal mucosa in response to gastric acid released from the stomach through the pylorus. This hormone has a general but only weak effect of decreasing gastrointestinal motility. GIP is released from the upper small intestine in response mainly to fat in the chyme but to a lesser extent to carbohydrates as well. (Although GIP does inhibit gastric motility under some conditions, its effect at physiologic concentrations is probably mainly to stimulate secretion of insulin by the pancreas.

In summary, several hormones are known that could serve as mechanisms for inhibiting gastric emptying when excess quantities of chyme, especially acidic or fatty chyme, enter the duodenum from the stomach. CCK is probably the most important.

Summary of the Control of Stomach Emptying

Emptying of the stomach is controlled only to a moderate degree by stomach factors such as the degree of filling in the stomach and the excitatory effect of gastrin on stomach peristalsis. Probably the most important control of stomach emptying resides in inhibitory feedback signals from the duodenum, including both enterogastric nervous feedback reflexes and hormonal feedback. These two feedback inhibitory mechanisms work together to slow the rate of emptying when:

- **Physical**: Too much chyme is already in the small intestine or
- **Chemical**: The chyme is excessively acidic, contains too much unprocessed protein or fat, is hypotonic or hypertonic, or is irritating.

In this way, the rate of stomach emptying is limited to that amount of chyme that the small intestine can process.

The Gastrointestinal System

The gastrointestinal system has its own intrinsic set of nerves known as the intramural plexus or the intestinal enteric nervous system, located in the walls of the gut. However, both parasympathetic and sympathetic stimulation can affect gastrointestinal activity, mainly by increasing or decreasing specific actions in the intramural plexus. Parasympathetic stimulation, in general, increases the overall degree of activity of the gastrointestinal tract by promoting peristalsis and relaxing the sphincters, thus allowing rapid propulsion of contents along the tract. This propulsive effect is associated with simultaneous increases in rates of secretion by many of the gastrointestinal glands, described earlier.

Normal function of the gastrointestinal tract is not very dependent on sympathetic stimulation. However, strong sympathetic stimulation inhibits peristalsis and increases the tone of the sphincters. The net result is greatly slowed propulsion of food through the tract and sometimes decreased secretion as well-even to the extent of causing severe constipation.

Procedure

Findings

I found that in normal patients, if we put direct pressure through the abdominal wall to stretch the Duodenum, and test the Pectoralis major muscles (Clavicular), they become weak, if tested together.

But if we do the same test, but previously tap the meridian point Stomach 1 (St-1), the test will be negated, probably regulating the CCK production (St-1 is related to Cholecystokinin (CCK)).

Method

I studied 90 random patients, 40% of which were males and 60% were females, seeking consultation for various reasons, and *who either showed problems related to hypo or hyper-function of the stomach*.

1. Initially I checked for and corrected switch if found; structural imbalances, emotional conditions, injury recall technique (IRT) problems and eliminated any medical treatments even for unrelated diseases. If drug treatments were not possible to avoid, then the patient was rejected from the research.

2. We handled each condition with the standard AK procedures, and treated stomach condition with hydrochloric acid and the nutrition the patient required showed by muscle testing.

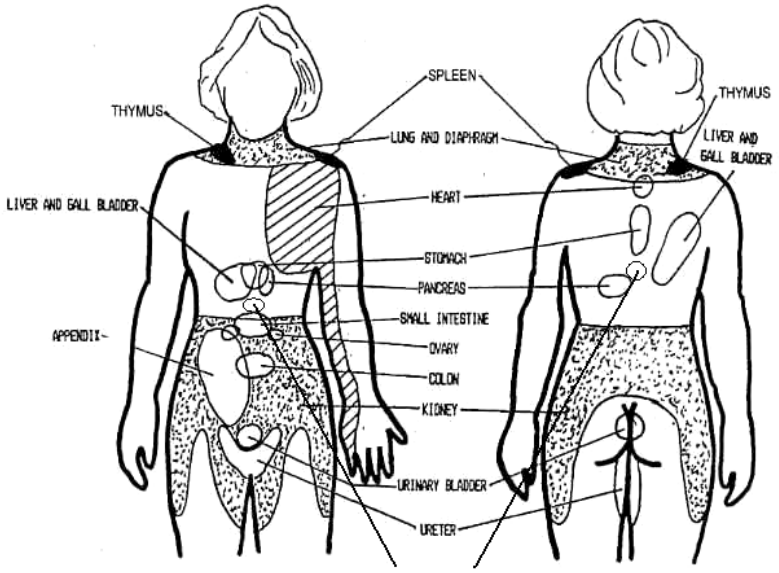
   We fixed (if found) Temporal Cranial Faults, Zinc deficit, Vitamin B6 deficit, Guanidine production and discontinued all kind of Antacid treatment, etc.

3. If Pinching the Duodenum Referred Pain area was negative, and the PMC kept strong, but putting direct pressure through the abdominal wall to stretch the Duodenum, and the Pectoralis major muscles (Clavicular) becomes weak if tested together, we consider it a normal enterogastric reflex (EGR).

   But if we found a patient that his PMC becomes weak after pinching the Duodenum RP (Together or individually tested) or has a weak PMC in the clear that becomes strong after rubbing the Duodenum RP, we consider an overactive EGR.

   If we found a patient that his PMC doesn't becomes weak after putting direct pressure through the abdominal wall to stretch the Duodenum, but becomes normal after rubbing the Duodenum RP (then you can make PMC weak after autogenic inhibition), we considered it an under-active EGR. PMC could be hypertonic in the clear from other sources!

   Yet, if the related problem (for example: ICV challenge is positive, but after rubbing the Duodenum RP becomes negative if overactive or pinching if under-active), we can establish the link between the EGR and the problem.



Duodenum and Pylorus RP

TREATMENT TO INCREASE PS in over-active EGR
(Stomach is under-functioning).
When rubbing the DRP (Duodenum Referred Pain) strengthens the associated muscle, use neurolymphatic (Chapman's) reflex (NL) activity. Rubbing the NL will strengthen the weak muscle. However, you can use the DRP to determine how long to perform NL activity. When enough NL activity has been performed, pinching the VRP will not cause a recurrence of the weak muscle. If pinching the DRP causes a recurrence of the muscle weakness, more NL activity must be performed.

TREATMENT TO INCREASE SYM in under-active EGR
(Stomach is over-functioning)

When pinching the DRP strengthens the associated weak muscle, one or more of the following techniques will be effective:

1. Set Point Technique must be using St1 (St1 is related to cholecystokinin (CCK))
2. IRT to the NL.
3. Visceral Challenge Technique (VCT).

VCT is performed when oral challenging with an offending substance (allergen, bad fat, sugar, drug, neurotransmitter, hormone, etc.) causes positive TL to the NL area. Correction is IRT to the NL with the offender in the mouth.

After treatment, check for a normal test (putting direct pressure through the abdominal wall to stretch the Duodenum, then the Pectoralis Major Clavicular muscles becomes weak.

Results

From the 90 patients studied, 82 patients had their EGR altered.

Four patients were found with under active reflex and complaining of some degree of duodenal peptic disease.

Seventy-eight patients have an over-active EGR. All of them complaining of different problems but related to the under functioning stomach. Most of them were ICV syndrome, bursitis or arthritis due to alkalosis.

All 82 patients with EGR altered were fixed by the treatment described and their symptoms improved.

Patients with under active EGR: All improved at least 90 % or more.

Patients with over active EGR:
48 Patients improved 90 % or more of their symptoms.
11 Patients improved 70 % or more of their symptoms.
16 Patients improved 50 % or more of their symptoms.
3 Patients improved 20 % or less of their symptoms.

Discussion

There are many sources of inhibition of the stomach and the digestive system, many of them are well known, but in difficult patients we need to search for less common causes.

Aside from the enterogastric reflexes, which have been discussed in this paper, several other important nervous reflexes also can affect the overall degree of bowel activity. They are the peritoneointestinal reflex, renointestinal reflex, vesicointestinal reflex, and somatointestinal reflex, duodenocolic, gastrocolic, gastroileal and defecation reflexes.

The *peritoneointestinal reflex* results from irritation of the peritoneum; it strongly inhibits the excitatory enteric nerves and thereby can cause intestinal paralysis, especially in patients with peritonitis. *The renointestinal* and *vesicointestinal reflexes* inhibit intestinal activity as a result of kidney and bladder irritation, respectively. Finally, the *somatointestinal reflex* causes intestinal inhibition when the skin over the abdomen is irritatingly stimulated.

Conclusion

Now we have another tool to treat stomach related problems, but we need to make more research about the neurology and chemical influence over the gland function.

Resources

Schmitt, Walter H., Jr. Injury recalls technique. Chiropractic journal of NC Vol. VI, 1990. pp. 25–30.

Schmitt, Walter H., Jr. Treatment to increase PS and SYM. The Uplink Issue 10, Spring, 1998.

Walther, David S., Applied kinesiology Synopsis, Pueblo, Colorado, Systems D.C. 2000, 2nd Ed.

Arthur C., M.D. Guyton, John E. Hall. Textbook of Medical Physiology, 2000.

Henry L. Bockus, J. Edward Berk. Bockus Gastroenterology, W B Saunders Co; 5th edition, 1995.

The Connection Between Homeocysteine, the Psoas Minor Muscle, and Low Back Pain

Thomas Rogowskey, D.C., DIBAK

Abstract

There appears to be a connection between excess levels of homocysteine and bilateral weakness of the psoas minor muscle as tested in the manner described by Dr. Alan Beardall. This weakness can cause instability in the lumbar spine and result in recurrent anterior lumbar vertebra subluxations. In turn, a relative stenosis of the spinal canal can result, causing dysfunction and pain in areas receiving innervation inferior to the anterior vertebrae. L3 seems to be the vertebrae most affected, often resulting in a spasmed closed ileocecal valve (ICV). The negative consequences of a closed ICV have been observed in applied kinesiology for some time, and can result in a very toxic condition, often depleting the available sulphate stores affecting joint stability and detoxification pathways.

Key Indexing Terms: Homocysteine, low back pain, applied kinesiology, psoas minor muscle, anterior lumbar vertebra, neuromuscular manifestations.

Introduction

The purpose of this paper is to demonstrate that excess homocysteine levels exist in a great number of patients and are the source of many of their presenting problems. There will be discussion of the research done on the long-term pathology resulting from high homocysteine. The musculoskeletal imbalances occurring as a result of this biochemical imbalance and the ramifications of these imbalances will be explored. Further discussion will be devoted to the correction of musculoskeletal conditions resulting from excess homocysteine. General remedies will be discussed in the context of using applied kinesiology to determine the specific needs of the patient.

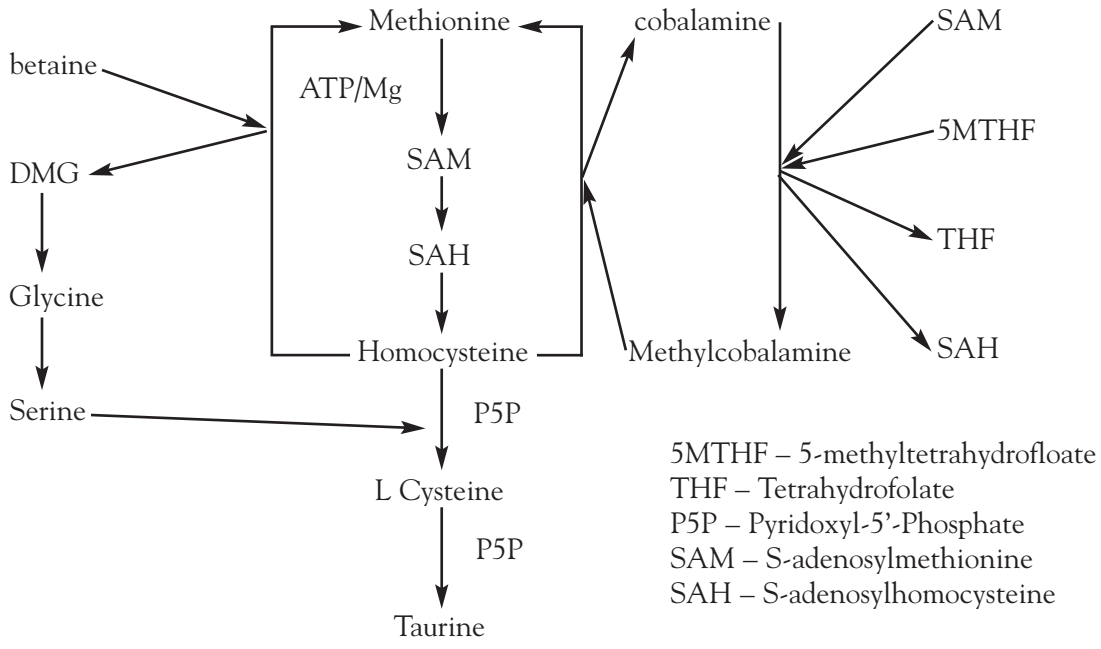
Discussion

High homocysteine has been shown to be associated with many chronic cardiovascular diseases. The normal serum levels for the female adult range from 4.0 to 10.4 micromol/L and for the male from 4.0 to 12.0 micromol/L. It appears that high homocysteine causes a weakening of the cardiovascular system, possibly due to oxidative stress to the blood vessel wall. Over time this condition increase the chances of strokes, heart disease, Parkinson's disease, Alzheimer's disease, complications of pregnancy, neural tube defects, neuropsychiatric disorders, strokes,[1] joint disrepair and compromised liver detoxification.

Homocysteine is an intermediate compound formed when S-adenosylhomocysteine is converted by the enzyme adenosylhomocysteinase (Figure 1). Homocysteine is broken down to cystathione by the enzyme cystathione-B-synthase.[2] In about 30 percent of the population, there is a deficiency in this enzyme, which prevents full conversion of the homocysteine. This is the result of a polymorphism that controls the production of the enzyme cystathione-B-synthase. This enzyme is dependent upon B6/Pyridoxal-5'-Phosphate (P5P) and serine for the conversion of homocysteine to L cysteine. Homocysteine is also remethylated to form methionine. This remethylation is dependent upon adequate amounts of the methylcobalamine form of B12, folic acid in

the form of 5- methyltetrahydrofolate (MTHF), or another methyl donor, betaine. The Methylenetetrahydrofolate reductase (MTHFR) C677T polymorphism can cause an excess of homocysteine by preventing a partially metabolized form of folic acid from being converted to MTHF. It is estimated that 10 to 15 percent of the population has this deficiency.[3]

Figure 1 **Homocysteine Metabolism**



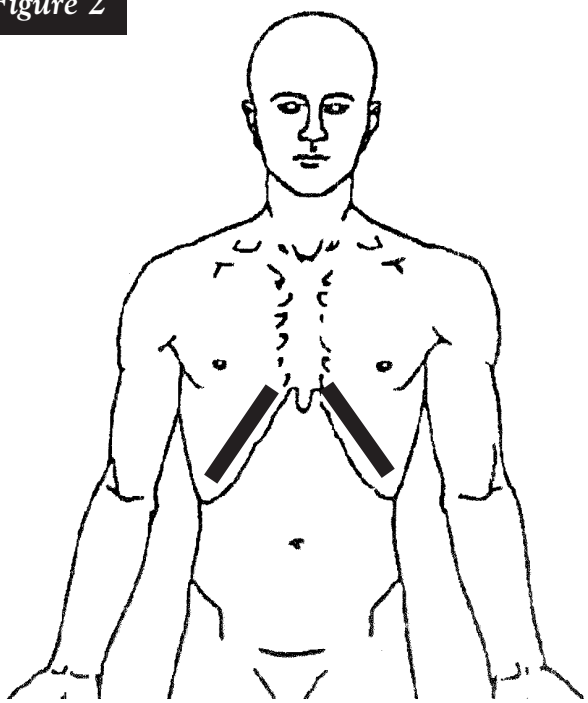
Numerous studies have indicated homocysteine elevation does not increase the risk of fatal heart attack, but it appears as a co-variable risk factor along with other agents for increased risk of arteriosclerosis.[3, 9] Elevated homocysteine is a modest independent predictor of ischemic heart disease and stroke in the healthy population. [8, 11] Low folate levels largely accounted for a trend toward greater cognitive decline with elevated homocysteine levels.[4] Lower levels of P5P were associated with greater risk for a venous thrombosis. High levels of P5P reduced the risk of arterial and venous thrombosis in patients with homocystinurea due to cystethionine-B-synthetase deficiency.[5, 8] Homocysteine concentrations were slightly increased in normotensive pregnancies that later develop preeclampsia and were considerably increased once preeclampsia was established. However, because of a lack of consistency in data, dose-response relationship, and biologic plausibility, the observed association cannot be considered causal from the current literature.[6]

There is now substantial evidence of a common decrease in serum/red blood cell folate, serum vitamin B12 and an increase in plasma homocysteine in depression. Furthermore, the MTHFR C677T polymorphism that impairs homocysteine metabolism is over represented among depressive patients, which strengthens the association.[7] Elevated serum homocysteine is associated with the presence of retinal vein occlusion, independent of other risk factors.[10]

Clinical Observations

I have found anterior lumbar subluxations to be an important factor causing low back pain, bilateral lower extremity symptoms, bilateral upper extremity symptoms, as well as gastrointestinal disturbances such as bloating and constipation. Attempts at stabilizing the anterior lumbar were not always successful using standard approaches such as facilitating conditionally inhibited muscles involved with anterior lumbar spine instability. I started testing the psoas minor muscle as diagrammed and described by Alan Beardall.[12] It was

Figure 2



conditionally inhibited in many of the unstable anterior lumbar conditions I was treating. I searched for a body function that would involve this bilateral psoas minor muscle weakness and found that a reaction to a source of homocysteine was common to all cases. A strong muscle would become conditionally inhibited when the patient was exposed to a source of homocysteine. The psoas minor muscles would be facilitated when the proper nutrient for alleviating excess homocysteine was administered. In addition to the bilateral psoas minor muscle weakness, there was often a subscapular muscle weakness when tested with the upper arm flexed to 90 degrees.

The Chapman's reflex or applied kinesiology's neurolymphatic (NL) reflexes I found related to excess homocysteine were the four intercostals spaces beginning just level with the Xiphoid process. This would be the fifth, sixth, seventh, and eighth intercostal spaces at the costal cartilage border bilaterally. (Figure 2) The NL for the subscapularis was present and was treated as well. Posterior reflexes appeared to be paraspinally starting at the spinous of T9 and going inferiorly several spinal segments. Treatment consisted of digital pressure until the reflex changed or no longer therapy localized. Often the correction of the psoas minor muscle reflex points stabilized the anterior lumbar region and often decreased the anterior subluxation so no further correction was necessary.

Recently I checked twenty existing and new patients and found that the psoas minor muscle was bilaterally conditionally inhibited in six of them. Those that were conditionally inhibited also exhibited an inhibition of a previously facilitated muscle upon exposure to homocysteine and would strengthen to one of the nutrients associated with this methylation defect. Though this ratio is close to the 30 percent of the population that is affected by high homocysteine, several of the existing patients that were tested were already taking B12, MTHF, P5P or betaine for a previously diagnosed homocysteine problem. Thus I would say that this method of screening yields more than the predicted 30 percent of the population, perhaps due to finding functional rather than pathological levels of excess homocysteine.

Classically, there are no acute signs of homocysteine excess. The literature addresses conditions that result from this condition, but by the time these conditions become evident, it is often too late for adequate recovery. Genotypes will help identify those at risk of the two polymorphisms involved with this methylation defect. Using this muscle test one can screen for possible methylation problems and the proper therapies to be administered for prevention of the conditions associated with excess homocysteine. The lack of homocysteine conversion will result in lower levels of L cysteine and consequently the sulfate ions for proper detoxification and joint repair. I have found that excess homocysteine can cause inflammation in the body. C reactive protein (CRP) is an independent measurement of inflammation, particularly in the cardiovascular patient. One patient had just found out her CRP was high, and I found it associated with high homocysteine. The high homocysteine reflexes were treated and the appropriate nutrients administered. Within a week, her CRP was back to normal. I have treated several cases where homeopathic 6x CRP conditionally inhibited a strong indicator muscle and excess homocysteine was found to be the culprit.

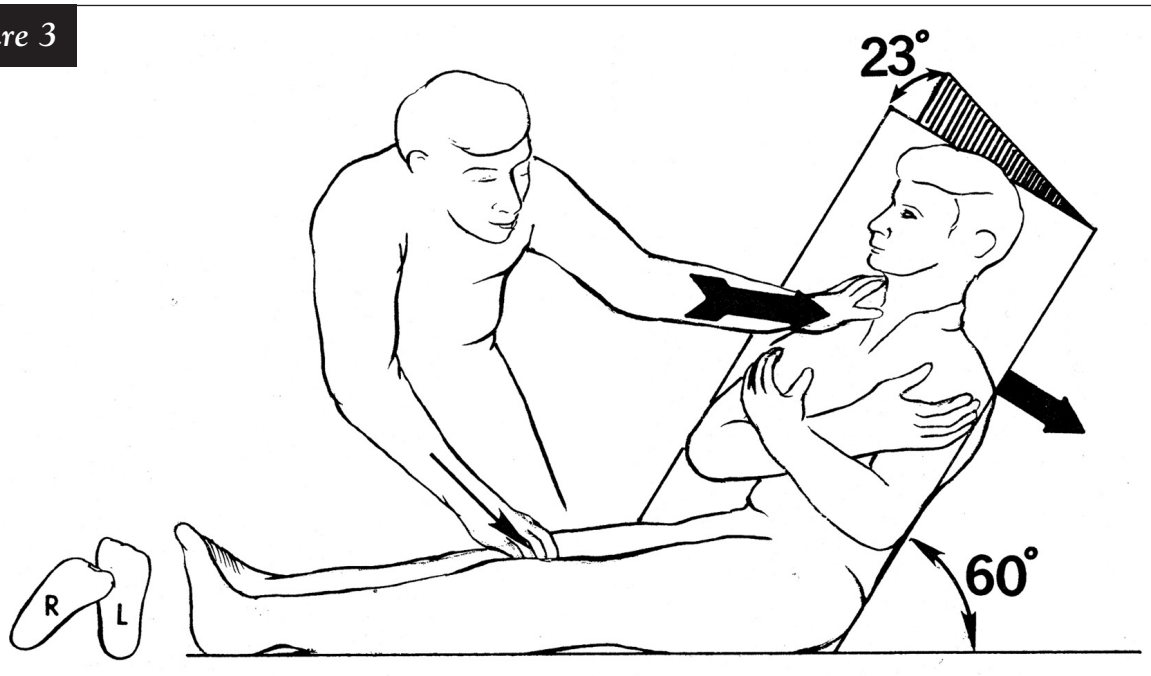
Nutrients associated with excess homocysteine can be seen in Figure (1). Facilitating conversion of homocysteine to L cysteine requires P5P and serine. Remethylating homocysteine requires B12 or betaine. To convert cobalamine to methylcobalamine one needs MTHF, methionine, and/or SAM (magnesium/ATP). MTHF requires a source of folate provided there is no MTHFR C677T polymorphism; otherwise, a source of MTHF is necessary. Administering L arginine creates a hypotensive effect as well as a reduction in homocysteine with oral administration.[13] L cysteine may be deficient and needed for joint repair and detoxification.

Treatment

Some of the symptoms that would lead a practitioner to look for excess homocysteine include: difficult centrally located low back problems; bilateral knee problems; bilateral foot problems; any bilateral neurological symptoms inferior to L3, any bilateral upper extremity symptom; any digestive difficulty that would be associated with a closed illeocecal valve; and any cardiac symptoms. Check for bilateral muscle weakness in the lower extremity in association with the symptoms presented. For example, with bilateral knee problems, check the rectus femoris muscle. It is often conditionally inhibited with an anterior L3. When the bilateral conditioned muscles relating to symptoms are found, apply and hold pressure anterior to posterior in the centerline approximately over the individual lumbar bodies and retest the involved muscles. (Patient may assist here applying the appropriate pressure.) The muscles will be facilitated when the associated anterior lumbar subluxation is held anterior to posterior.

Once it is determined that an anterior lumbar subluxation exists, check the psoas minor muscle bilaterally, testing one side at a time. See Figure 3 for the physical set up. The psoas minor test, as shown by Dr. Alan Beardall, is tested with the patient seated; the torso is flexed 60 degrees with a straight spine. The torso with arms crossed is then rotated away from the muscle being tested by 23 degrees, and the ipsilateral femur is internally rotated. Pressure is directed through the sagittal plane with a midclavicular contact while bracing the ipsilateral thigh. A conditionally inhibited muscle will result in a break or further extension of the torso from 60 degrees.

Figure 3



Used by permission from Human bio-Dynamics, Inc., Portland OR Phone 800-888-5931

If the psoas minor muscle is bilaterally conditionally inhibited, test a previously strong muscle with exposure to a source of homocysteine. If the indicator muscle does not conditionally inhibit with homocysteine, expose the patient to a source of methionine followed by the homocysteine. Using one of the conditionally inhibited muscles, test the various nutrients that correct for the excess homocysteine. These include: methylcobalamine, 5-methyltetrahydrofolate, folic acid, pyridoxyl-5'-phosphate, serine, betaine, and/or arginine. The patient may also need a source of L cysteine since there is a problem converting the homocysteine to L cysteine.

While still exposed to homocysteine, therapy localize the 5–8 intercostal spaces and treat where appropriate. Test and treat the subscapularis muscle reflexes if they are active. Treat the posterior reflexes starting approximately at the level of T9 spinous. Therapy localize to get the exact number and location of the paraspinal reflexes. It is better to treat these points during exposure to the homocysteine.

The apparent paradox of upper extremity symptoms can be explained by the occurrence of a "hidden cervical disc (HCD)" that is reciprocating the anterior lumbar subluxation. Use the standard applied kinesiology protocol for detecting the HCD. Check strong wrist extensor muscles while caudal pressure is applied through the cervical region. If the wrist extensors become conditionally inhibited, apply pressure to the abdomen from the anterior starting at the level of L3 (approximately at the umbilicus), and recheck the wrist extensors with the caudal pressure. If this abolishes the inhibition, suspect an anterior L3 subluxation. Check for the psoas minor bilateral inhibition and excess homocysteine. If L3 does not correct the wrist extensor inhibition, check the lumbar vertebrae inferior and superior to L3 in the same manner as when you challenged L3 involvement. If there is no anterior lumbar involvement, correct the HCD in the usual manner.

Conclusion

Excess homocysteine has been shown to be a risk factor for cardiovascular disease. Most likely it is also a factor in strokes and other vascular diseases, dementia, perinatal, eyesight, and neuropsychiatric disorders. This paper elucidates the musculoskeletal and functional biochemical problems that exist as a result of excess homocysteine in addition to these traditionally associated diseases and disorders. Correlating these findings with lab results is an area remaining to be investigated. The patient should methionine load before any lab test for homocysteine to better uncover any borderline cases. Supplementation with nutrients that effectively treat excess homocysteine and methylation disorders will impact the homocysteine levels as well as the musculoskeletal and functional biochemical problems. Methylcobalamine, 5-methyltetrahydrofolate, folic acid, pyridoxyl-5'-phosphate, L serine, betaine, L cysteine, and/or L arginine help optimize methyl and sulphur metabolism and will significantly help prevent and treat a wide array of conditions. The practitioner who treats musculoskeletal and functional biochemical problems now has a new tool for analysis and treatment.

References

1. Miller A, Kelly G. Homocysteine Metabolism: Nutritional Modulation and Impact on Health and Disease. *Alternative Medicine Review* 1997; vol.2, No.4: 234–54.

2. Astill-Smith C. Metabolic Seminar, Neural and Humeral Modulation. Chicago IL April 8–9,2000.

3. Bland, J. The Institute for Functional Medicine. Nutigenomic Modulation of Inflammatory Disorders: 2004 seminar series.

4. Hung J, Beilby JP, Knuiman MW, Divitni M. Folate and vitamin B12 and the risk of fatal cardiovascular disease: cohort study from Busselton, Western Australia. *BMJ*. 2003; 326:131-37.

5. Echinger S. Homocysteine, vitamin B6 and the risk of recurrent venous thromboembolism. *Pathophysiol Haemost Thromb*, 2003/2004, 33: 342–44.

6. Mignini L, Latthe P, Villar J, Kirby M, Carroli G, Khan K. Mapping the Theories of Preeclampsia: The Role of Homocysteine. *Obstetrics & Gynecology* 2005:105:411–25.

7. Coppen A, Bolander-Gouaille C. Treatment of depression: time to consider folic acid and vitamin B12. *Journal of Psychopharmacology*; (2005),Vol.19, No. 1: 59–65.

8. Den Heijer M, Lewington S, Clarke R. Homocysteine, Methylenetetrahydrofolate reductase and risk of venous thrombosis: a meta-analysis of published epidemiological studies. *Journal of Thrombosis and Haemostasis*.2005 Vol: 3 No: 2: 292–99.

9. Gupta M, Sharma P, Gitanjali G, Kaur K, Bedi G, Vij A. Homocysteine: an independent or an interactive risk factor for coronary artery disease. *Clinica Chimica Acta* February 2005, vol.352, Issues 1–2: 121–25.

10. Chua B, Kifley A, Wong T, Mitchell P. Homocysteine and retinal vein occlusion: A Population-based study. *American Journal of Ophthalmology*. Jan 2005 Issue 1: 181–82.

11. Casas J, Bautista L, Smeeth L, Sharma P, Hingorani A. Homocysteine and stroke: evidence on a causal link from mendelian randomization. *Lancet* 2005; 365: 224–32.

12. Beardall, Alan G., Clinical Kinesiology, Volume 1: Muscles of the Low Back and Abdomen. 1980. (PO Box 1752, Lake Oswego, OR 97034).

13. West S, Likos-Krick A, Brown P, Mariotti F. Oral L-Arginine Improves Hemodynamic Responses to Stress and Reduces Plasma Homocysteine in Hypercholesterolemic Men *The American Society for Nutritional Sciences J Nutr* 2005, 135: 212–17.

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

Walter H. Schmitt, Jr., D.C., DIBAK, D.A.B.C.N.

Abstract

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

Key Indexing Terms: Applied kinesiology, manual muscle testing.

Introduction

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

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

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

One of AK's most important and attractive features is that it is an open system. That is, AK allows for evaluation of the impact on a patient's nervous system of virtually any modality or therapy. This protocol is no exception to the openness of the AK approach. The hierarchy presented here is comprehensive, but the

therapies listed are not all-inclusive. For example, evaluation and treatment of food allergies is presented at a certain point in the protocol as identification of the existence of a food allergy response is imperative for optimal progression through patient assessment and treatment. Yet any additional allergy diagnostic activity may be added and, although one particular allergy treatment procedure is included, any effective allergy treatment procedures may be used. The key is when to employ various diagnostic and therapeutic steps. This protocol is open to the clinician's choice of techniques as it is based on fundamentally sound principles of the basic sciences of neurology and biochemistry.

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

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

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

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

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

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

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

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

AK procedures may be combined with the awareness derived from observing of autonomic windows on neurological function to address these issues, but as mentioned, that discussion is for another paper. However, in either approach, AK (the facilitation – inhibition approach) or chiropractic neurology (the neuron

metabolic state approach), muscle testing can be seen as a somatic window on neurological function and is a valuable tool in assessing the patient and directing therapy to the most optimal outcome. In fact, all of our therapies, and each and every therapy of any discipline can be seen as having their clinical influence by affecting one or more of the above states of neural activity.

Discussion

BEGINNING PROCEDURES

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

Summary: Identify a weak / inhibited muscle first. Then fix injuries to restore normal function of the ipsilateral cerebellum, the contralateral cortex, and the descending muscle spindle cell control mechanisms that are necessary for muscular and postural control.

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

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

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

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

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

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

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

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

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

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

When one cerebral hemisphere fires, there are three important patterns of muscle activity: 1) Ten percent of cerebral neurons fire via the corticospinal tract, corticoreticulospinal tract, and corticorubrospinal tract to muscles on the contralateral side of the body for meaningful, purposeful movement. Ninety percent of the cerebral neurons fire into the ipsilateral brainstem creating two additional patterns: 2) a general increase in muscle tone on that side of the body, and 3) an ipsilateral increase in muscle tone of posterior (extensor)

muscles above T-6 and an increase in muscle tone of anterior (flexor) muscles below T-6. This third phenomenon creates a gait-like distribution of muscle facilitation and inhibition. These latter two patterns create muscle tone on the opposite side of the body from the meaningful movement generated by the corticospinal tract fibers which decussate.

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

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

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

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

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

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

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

The one exception that allows nutrient testing prior to performing IRT or NSB is to do nutrient testing to see if any nutrient negates an IRT or NSB challenge. This can be a valuable tool in identifying which nutrient(s) will be most effective at reducing the pain and inflammation related to the injury and to speed the healing of the injured area. Other systemic nutritional testing should be postponed until later in the protocol as indicated.

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

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

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

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

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

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

SYSTEMIC NUTRITIONAL FACTORS

5. Test Aspirin, Acetaminophen, Ibuprofen Mix - If Strengthens:
   a. Check Essential Fatty Acids (BCSO, FSO, EPA, etc.)
   b. Check EFA Cofactors (B-6, Mg, Zn, Niacin)
6. Test Antihistamine Mix – If Strengthens:
   a. Challenge Orally for Offender(s) (Allergen)
   b. IRT Chapman's Reflexes with Offender(s)
7. Sniff Tests – Aldehydes, Bleach, Ammonia
8. Test Nutrients Based on Patient History:
   a. Vitamin E (Low Back Muscles), Vitamin C (Shoulder Muscles)
   b. Iron, Folic Acid, Vitamin B-12
   c. Cholesterol Lowering Nutrients (If Indicated)
   d. Chondroitin Sulfate – If Strengthens:
      i. Check Sulfur (Cysteine) & Associated Nutrients
      ii. Check Blood Sugar Handling (Insulin, Magnesium)

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

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

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

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

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

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

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

Prostanoids, which are derived from essential fatty acids (EFA) include prostaglandins (PGs), leukotrienes (LTs), and thromboxanes (TXs). Prostanoids influence inflammation, immune function, platelet aggregation, cholesterol synthesis, and modulate the responses of intracellular chemistry to membrane receptor driven functions. This includes neurotransmitter (NT) activity. In addition, EFA imbalances are reflected in a variety of muscle imbalances that must be normalized for proper joint support and afferentation that is necessary for healing.

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

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

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

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

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

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

Histamine is also one of the substances that depolarizes NOC resulting in an increase of potentially pain-inducing sensory activity. Therefore, we must neutralize the effects of histamine in order to normalize

afferentation from NOC in the same manner that we must control the prostanoids for their effects on both intracellular modulating and peripheral inflammation.

Food allergies and chemical sensitivities stress the immune system (thymus and spleen) which is why we address them prior to immune function. Detoxification of excess histamine stresses the spleen and liver, uses methyl groups related to sulfur amino acid metabolism that is essential in joint repair (as discussed below), and can deplete reserves of vitamin C. Degradation of histamine uses up the nutrients B-6 and folic acid. Both are essential for neurotransmitter (NT) synthesis. B-6 is necessary for ammonia metabolism, in particular as a transaminase enzyme (transports ammonia groups) activator. Ammonia groups are necessary for synthesis and degradation of amino acids as well as urea cycle function. Folic acid is important for many vital functions, not the least of which is cell replication. Its deficiency can also cause right brain – left brain imbalances and switching related to the hyoid. All of the B-6 and folic acid related functions can be compromised in the presence of histamine mediated allergic reactions.

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

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

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

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

7. Sniff Tests – Bleach, Ammonia

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

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

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

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

Free radical pathology is often driven by allergies and poor EFA metabolism, two issues that have already been addressed. So the bleach sniff test may be already negative by this juncture. If not, assessment of free radical chemistry and the antioxidants required to negate it are necessary at this point. This may include vitamins E and C, but we include these two important nutrients in a separate step immediately below since their need may be present even in the absence of a positive bleach sniff test.

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

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

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

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

8. Test Nutrients Based on Patient History:
   a. Vitamin E (Low Back Muscles); Vitamin C (Shoulder Muscles)

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

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

Vitamin E is important for so many low back and pelvic muscles that to leave it out of the patient's program is to perpetuate musculoskeletal problems. The same is true for vitamin C and shoulder problems. So early

on in the care of these symptoms, we test for these important antioxidants. Supplementation will help the associated muscle(s) maintain correction as well as provide the obvious biochemical effects.

8. Test Nutrients Based on Patient History:
   b. Iron, Folic Acid, Vitamin B-12

Oxygen supply to the tissues is of course, essential. But we first want to make sure that we have reduced any sources of excess oxidation (EFA, allergies/hypersensitivities, antioxidant depletion.) So we place the assessment of the oxygen carrying capacity of the blood here. But we also place the oxygen supplying nutrients ahead of CAC and ETC assessment since adequate oxygen is necessary for these oxidative phosphorylation pathways.

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

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

8. Test Nutrients Based on Patient History:
   c. Cholesterol Lowering Nutrients (If Indicated)

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

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

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

8. Test Nutrients Based on Patient History:
   d. Chondroitin Sulfate – If Strengthens:
      i. Check Sulfur (Cysteine) & Associated Nutrients
      ii. Check Blood Sugar Handling (Insulin, Magnesium)

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

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

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

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

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

SYSTEMIC STRUCTURAL FACTORS

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

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

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

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

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

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

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

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

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

When the immune system is dysfunctional, there will be an impact on the hypothalamus (HPT.) This is the area that psychoneuroimmunology and neuroimmunomodulation research addresses. When the immune system is dysfunctional and the HPT responds, HPT connections to the mesencephalic reticular formation (RF) cause the manifestation of the immune system problem to be seen in several ways. One factor observed is related to changes in TMJ muscle function and consequent patterns of body activity that are referred to as centering the spine patterns.[15] HPT adaptations to endocrine dysfunction may be manifested in the same manner (i.e., TMJ disturbance and/or muscle imbalance.) Another observation will be seeing changes in autonomic function. These include neurological signs such as alterations in pupillary reflexes and their fatigability that is mediated by cranial nerve III in the mesencephalon. They also include digestive problems such as ileocecal valve syndromes which can be monitored via muscle testing.

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

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

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

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

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

There also appears to be a sidedness to immune system problems. Thymus dysfunction is manifested in right-sided cranial and/or TMJ problems. If TL to a right-sided cranial or TMJ with the neck in extension is positive, there will be a weak infraspinatus on the right, possibly bilateral. Spleen problems are manifested in left-sided cranial and/or TMJ problems. If TL with the neck in extension to a left-sided cranial or TMJ is positive, there will be a weak lower trapezius on the left, possibly bilateral. Chemical hypersensitivities (and parotid problems) are manifested by either right-sided or left-sided cranial/TMJ faults and are accompanied by

TL to the lower sternum and a bilateral pectoralis minor weakness. It appears that there are no specific patterns of muscle inhibition associated with any of the three immune patterns other than the infraspinatus, lower trapezius, and pectoralis minor involvements. That is, any muscle weaknesses may be secondary to immune involvement. So important is this that no local problem (other than the systemic effects of injuries) should be addressed without first checking for cranial or TMJ patterns secondary to an immune problem.

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

In the case of the pupillary light responses, treating the spleen will have an affect on the left pupil reflex and treating the thymus will impact the right pupil response. Treating the lower sternum area could affect either or both pupil reflexes. The net effect of treating immune system Chapman's reflexes seems clearly to be one of inhibiting the mesencephalon: thymus inhibits the right mesencephalon, spleen inhibits the left mesencephalon, and treating lower sternum / parotid problems may inhibit either right or left mesencephalon.

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

9. Is TL to K-27 Positive?
   c. Dorsal Crossed TL – Use Tooth Techniques

10. Does TL to TMJ Strengthen Weak Muscle? (and/or) Is TMJ Present with IRT?
    c. Check Nasosphenoid Cranial Fault
    d. Check Temporoparietal Jam
    e. Check Sphenoid Compression Fault
    f. Correct TMJ / TMJ Muscles – Correct with IRT and/or Mechanically

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

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

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

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

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

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

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

This has brought us to switching and the relationship to cranial faults, pre-test imaging and the immune system. Normalizing immune function stimulates hypothalamus pathways to inhibit the mesencephalon and favorably influence pattern generator cells that operate via descending reticulospinal (RS) pathways. Cerebellar responses to injuries that have been corrected and the pathways for efferent copy, feedback and feed forward mechanisms have been addressed. The impact from mechanical cranial activity, so prominently affecting cerebellar firing, probably into the same pontomedullary descending pathways normalized by treating immune system function via hypothalamic-mesencephalic-pontomedullary descending RS pathways, is redressed. The effects of cranial correction to normalize the cerebellum via the dentatorubral tract directly into the opposite mesencephalon help to normalize potential mesencephalon imbalances from a direction other than the hypothalamus inputs.

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

SYSTEMIC NUTRITIONAL FACTOR

12. Does Rebreathing in a Paper Bag Strengthen?
    a. If Yes: Check Citric Acid Cycle & Electron Transport Chain Nutritional Factors

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

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

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

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

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

It is important to note that CO_2 combines with ammonium ion to generate the urea cycle, the major source of ammonia waste for the body. Inability to rid the body of ammonia results in hyperammonemia, resulting in changes in NT synthesis as ammonia groups are added to certain amino acid based NTs converting them into different substances altogether. For example, glutamate will become GABA, but in the presence of excess ammonia, it will be converted to glutamine and GABA will become deficient. Similarly, aspartate can be converted to asparagine and its NT function negated.

Speaking strictly about energy production rather than the other ramifications of CAC problems, we see that this is also the proper placement of CAC and ETC assessment. As stated previously, the need for CoQ_{10} could be performed earlier. But the CAC and the ETC are a continuum in the oxidative phosphorylation process for the production of ATP. ATP provides energy for every cellular energy-using function: muscles,

organs, and neurons. Once we have eliminated sources of CAC blockade (inflammation and/or allergy causing cytokines and nitric oxide) we can look to the possible need for B vitamins and manganese (and sometimes iron) necessary for efficient CAC activity.

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

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

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

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

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

HEART-FOCUSED ACTIVITY

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

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

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

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

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

HF activity, once performed, should be taught to the patient and used daily at home. HF activity is placed before other endocrine and GI tract techniques due to its powerful and positive effects on these systems. Likewise, it is placed prior to other emotional stress related techniques. HF activity helps to correct and/or maintain endocrine (especially stress-related) and GI tract symptoms. Once the patient has been placed on daily HF home activity, check the endocrine system and GI tract prior to performing HF activity in the office. HF activity may have positive impact on these systems, but they still may require further treatment. Performing HF activity in the office may obscure your endocrine and GI tract findings – similar to jump starting a car with a dead battery, the next time you start the car, the battery is dead again - unless you have addressed the underlying problem. Therefore, after initially giving the patient HF activity as homework, make sure that the patient is following your home care instructions, but do not do it in the office again, at least not until you have assessed and addressed any endocrine or GI tract dysfunctions.

SYSTEMIC ENDOCRINE EFFECTS

14. Does TLR Strengthen as expected?
    a. No: Identify and Treat Appropriate Endocrine Chapman's Reflex
    b. Yes: Check for Endocrine Related Muscle Weakness – Treat Appropriately
15. Does Rubbing Adrenal Chapman's Reflexes cause Pituitary Chapman's Reflex to TL?
    a. Yes: IRT to Adrenal Chapman's Reflexes with Offender.
16. Does Adrenal Challenge (Pinching) Induce Adrenal Muscle Weakness? If Yes:
    a. TL to Adrenal Chapman's Reflexes – If Strengthens: Rub Reflexes
    b. TL to Pituitary Chapman's Reflex – If Strengthens: Go to 15a
17. Does Ligament Stretch Cause Muscle Weakness?
    a. Yes: Rub Adrenal Chapman's Reflexes

18. Test Endocrine Related Muscles – Identify and Treat Primary Chapman's Reflex
    a. Test PMS (Liver) and TFL (Colon) – Treat Primary Chapman's Reflex
19. Test PMS – Rub and Pinch Liver VRP area – If Positive:
    a. Test Liver Detoxification Nutrients
    b. Challenge Liver Chapman's Reflex with Offenders – If Positive:
        i. IRT Liver Chapman's Reflex with Offenders
    c. Challenge PMS with Cholesterol – If Weakens: Go to 8c
        i. Rub Liver Chapman's Reflex with Cholesterol in Mouth.
20. Pinch Pancreas VRP & Test Biceps Brachii (or Other Upper Limb Flexor) – If Weakens:
    a. Test Chromium, Vanadium, Zinc, Pancreas Tissue, Sesame Seed Oil
    b. Challenge Pancreas Chapman's Reflex with Offender – Offenders include:
        i. Milk, Cortisol, Bad Fats, NE, Other Allergens

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

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

As mentioned previously, Goodheart observed that muscles would respond differently to right brain activity (e.g., humming a tune) and to left brain activity (e.g., counting.) [12] He taught that right brain activity would often respond to therapies that increased steroid activity. Similarly, he noted that muscles that responded to left brain activity (e.g., counting) would often respond the therapies that increased thyroid activity. Right-left brain imbalances of this nature can contribute to emotional stress (and cognitive) disturbances and the correction of endocrine imbalances at this point will set the stage for effective assessment of emotional recall issues later in the protocol.

14. Does TLR Strengthen as expected?
    a. No: Identify and Treat Appropriate Endocrine Chapman's Reflex
    b. Yes: Check for Endocrine Related Muscle Weakness – Treat Appropriately

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

Hypofunction of an endocrine organ (adrenal, thyroid, reproductive) will be seen to cause an inappropriate muscular response to changes in head position relative to gravity. The first step in endocrine assessment is testing to see if the TLR are operating properly by testing inhibited muscles with the head in a position for

TLR to facilitate those muscles. These reflexes operate primarily via the inferior vestibular nuclei, which receive direct input from the cerebellum. Failure of the TLR tells us that there is a under functioning endocrine organ, as long as other pathways to the vestibular nuclei are not interfering with its descending output.

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

15. Does Rubbing Adrenal Chapman's Reflexes cause Pituitary Chapman's Reflex to TL?
    a. Yes: IRT to Adrenal Chapman's Reflexes with Offender.
16. Does Adrenal Challenge (Pinching) Induce Adrenal Muscle Weakness? If Yes:
    a. TL to Adrenal Chapman's Reflexes – If Strengthens: Rub Reflexes
    b. TL to Pituitary Chapman's Reflex – If Strengthens: Go to 15a
17. Does Ligament Stretch Cause Muscle Weakness?
    a. Yes: Rub Adrenal Chapman's Reflexes

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

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

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

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

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

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

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

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

Many systemic muscular patterns will resolve instantly following one specific endocrine correction related to centering the spine. Of course, adaptations to injuries will also cause centering the spine problems, but these postural and visual (such as EID) responses to trauma will have long since been corrected by IRT, NSB, and/or SP techniques.

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

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

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

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

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

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

20. Pinch Pancreas VRP and Test Biceps Brachii (or Other Upper Limb Flexor)–If Weakens:
    a. Test Chromium, Vanadium, Zinc, Pancreas Tissue, Sesame Seed Oil
    b. Challenge Pancreas Chapman's Reflex with Offender – Offenders include:
        i. Milk, Cortisol, Bad Fats, NE, Other Allergens

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

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

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

Insulin has a significant effect on the autonomic nervous system. Insulin increases sympathetic outflow (which is one of the reasons hyperinsulinism patients often present with hypertension, many times misdiagnosed as "idiopathic hypertension" when it is really secondary to the sympathomimetic effects of the excess

insulin.) The neurological SYM response to increased insulin includes the spinal flexion pattern. Spinal flexion is usually accompanied by a spinal torque (gait) pattern as part of normal spinal coupled motion. Repetitive gait in the hyperinsulinism patient frequently leads to a spinal gait torque pattern that results in an iliolumbar ligament IRT pattern. Correcting the hyperinsulinism pattern normalizes the SYM spinal flexion and its concomitant spinal torque rotation. Certainly, if present, this should be addressed prior to checking the iliolumbar ligament as well as prior to administering any spinal and pelvic adjusting procedures. More will be said of this pattern in the discussion regarding gait.

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

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

GASTROINTESTINAL TRACT

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

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

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

Injuries must be corrected prior to investigating GI tract (or any autonomic) function because one of the three spinal cord effects of nociception is sympathetic activation at the intermediolateral columns (IML.) Nociception also activates the spinothalamohypothalmic pathway and its systemic autonomic (SYM) outflow. Muscle reactions to injuries that are maintained in an IRT pattern will cause cerebellar adaptation. The cerebellum fires directly into autonomic centers (parasympathetic) in the vital centers in the pontomedullary reticular formation (PMRF) including the nucleus of the tractus solitarius, dorsal motor nucleus of the vagus,

and glossopharyngeal nerves and also affects the mesencephalon which has systemic autonomic consequences. Therefore, any autonomic evaluation in the presence of an IRT injury can be misleading because the real autonomic status may be misinterpreted due to the multiple effects arising from local or systemic autonomic reaction to the injury.

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

We will also impact the GI tract when we correct endocrine problems. Liver – bowel interactions will have been investigated and corrected as part of our comprehensive treatment of the endocrine system. Further, there are well described (although not as well defined) relationships between female hormonal fluctuations and bowel function (i.e., the common diarrhea or constipation that parallels the menstrual cycle in many women.)

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

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

21. Challenge for Hiatal Hernia / GERD

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

22. Challenge Ileocecal Valve – Open or Closed
23. If Digestive Problem – Rub and Pinch Visceral Referred Pain area(s)
    a. If Rubbing Strengthens: Rub Chapman's Reflex for that Organ
    b. If Pinching Strengthens: Use VCT – IRT Chapman's Reflex with Offender

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

Neurologically speaking, aberrant visceral sensory activity will impact the ipsilateral cerebellum (via the GI muscle imbalances) and the contralateral cortex and can contribute to right – left brain imbalances and the emotional recall weakening effects. Cerebellar adaptations to long standing GI tract related somatic muscle imbalances can also cause changes in axial (old, midline cerebellum) sensory feedback from the cerebellum into the limbic system (old, midline cortex) and aggravate or enhance emotional perceptions. For this reason, the emotional recall techniques are placed after GI tract correction in the clinical protocol. This allows for correction of numerous secondary emotional recall problems during GI tract treatment.

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

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

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

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

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

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

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

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

When there is a psychological reversal, there is also a physiological reversal with a switching type effect on muscle testing findings. It is logical (and correct) that any type of switching should be corrected earlier in the course of treatment than at this point. The protocol addresses this by screening for allergies and bad fats very early on when screening with anti-histamines and aspirin, ibuprofen, acetaminophen mix, respectively. Most of the time, there will be small intestine involvement in both allergies and bad fat ingestion and correction will eliminate both psychological and physiological reversals at this point early in the treatment. There are other cases of small intestine involvement that do not cause these reversals and these will be corrected during other GI tract treatment in this section.

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

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

EMOTIONAL STRESS

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

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

It is acknowledged that emotional stress related techniques play an important role in treating patients. Therefore, the placement of the assessment of emotional stress related issues at this late point in the protocol

may be perplexing, even disconcerting to those who give high regard to the importance of the emotional aspects of health. If this is your position, consider that we have already corrected the following factors that can influence emotional stress and the ability to cope with it, biochemically and neurologically:

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

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

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

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

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

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

LOCAL PROBLEMS

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

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

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

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

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

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

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

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

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

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

26. Check Fascial Sheath Shortening

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

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

27. Check Iliolumbar Ligament

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

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

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

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

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

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

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

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

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

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

Identifying subluxation correction hierarchy by the flexor reflex afferent (FRA) subluxation nociceptive challenge is usually a multi-step procedure. NOC afferents create the FRA (flexor withdrawal response.) The nervous system can only respond to one NOC input at a time. In fact, any FRA inhibits all other FRA afferents. Hence the "worst" FRA overrides the "lesser" FRAs and causes its specific pattern of muscle withdrawal preferentially. Certain other non-NOC sensory pathways also create an FRA response. These include receptors in joints and secondary muscle spindle cell receptors (from the nuclear chain) which would fire in the presence of a subluxation.

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

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

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

GAIT ASSESSMENT

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

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

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

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

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

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

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

CHRONIC PAIN

32. If Chronic or Persistent Pain: Use LQM Technique

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

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

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

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

Conclusion

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

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

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

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

References

1. Schmitt, W.H., & Yanuck, S.F. Expanding the neurological examination using functional neurological assessment part II: neurologic basis of applied kinesiology. Intern J Neuroscience, 1999, 97, 77–108.

2. McCord, KM, and Schmitt, WH, Quintessential Applications: A(K) Clinical Protocol. St. Petersburg, Florida: Privately Published, 2005.

3. McCord, Kerry M. Clinical response to a neurologically based comprehensive protocol developed by Dr. Walter H. Schmitt. Submitted for inclusion in Proceedings of the ICAK-U.S.A. Volume 1, 2005–2006.

4. Belli, Richard. Deep Tendon Reflexes. ICAK-USA News Update, 1994.

5. Belli, Richard. Autogenic Inhibition. Proceedings of the ICAK-U.S.A., volume I, 1995–1996. pp. 15–18.

6. Schmitt, Walter H., "A Critical New Concept" (Transneural Degeneration) *ICAK News Update*, (1999) 15:4. p. 5–7.

7. Schmitt, Walter H., Jr. Transneural degeneration and the links between the nervous system and the body chemistry. Proceedings of the ICAK-U.S.A. Volume 1, 1999–2000. p. 149–156.)

8. Schmitt, Walter H., Jr. A neurological rational for injury recall technique. Proceedings of the ICAK-U.S.A. Volume 1, 1999-2000. p. 137–139.

9. Gerald R. Polino, personal communication, 1993

10. Burt, Alvin M.; Textbook of neuroanatomy. Philadelphia: W.B. Saunders, 1993. pp 459–464.

11. 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.

12. Goodheart, George J. Applied kinesiology 1979 workshop procedure manual. Detroit: privately published, 1979.

13. Schmitt, Walter H., Jr. A neurological basis for the effects of cranial manipulation. Proceedings of the ICAK-U.S.A. Volume 1, 1999–2000. p. 133–135.

14. Gangemi, Stephen C, A Newly Discovered Muscle-Organ Relationship: The Pectoralis Minor and the Parotid Gland. Submitted for inclusion in Proceedings of the ICAK-U.S.A. Volume 1, 2005–2006.

15. Schmitt, Walter H., Jr. Centering the spine functional neurological and biochemical considerations. in Collected papers of ICAK - summer 1987. Shawnee Mission, KS:International College of Applied Kinesiology, 1987

16. Schmitt, Walter H., Jr. Where is the triad of health? Proceedings of the ICAK-U.S.A. Volume 1, 1996–97. p. 83.

17. Schmitt, Walter H., Jr. The somatic window on neurological function, part 3. mesencephalic transneural degeneration: the cause of many TMJ problems and bilateral joint problems. Proceedings of the ICAK-U.S.A. Volume 1, 2003–2004. p. 227–233.

18. Goodheart, GJ, Zinc and Cranial Faults. Nutri West company literature. 1985.

19. Gershon, Michael D. The second brain. New York: HarperCollins, 1998.

20. Callahan, R.J. How executives overcome their fear of public speaking and other phobias. Wilmington, De: Enterprise pub., inc., 1987

Adult Attention Deficit Disorder and Learning Disabilities

Paul T. Sprieser, D.C., DIBAK

Abstract

The clinical observations of the learning disability cranial fault and its association to adult attention deficit disorders and other cognitive, perceptive, memory and emotional disorders.

Key Indexing Terms: Learning disability, cranial fault, applied kinesiology.

Introduction

wenty years ago I wrote a research paper on my clinical observation of a completely new cranial fault that was published in the ICAK Collected paper of the winter 1984. This paper was accepted for publication in Chiropractic Economics in two parts because of its length in May/June, and the November/December issues of this chiropractic journal.

There is currently resurgence in interest particularly in the adult form of A.D.D. and learning disability as evident in recent months in articles published in New York Times Magazine section 7/18/2004 article "The Workers Are Restless." Also in the New York Times-Heath & Fitness section of 7/8/04, "Two Types of Brain Problems Are Found to Cause Dyslexia" and another issue "Biology of Dyslexia Varies With Culture, Study Finds." Also in U.S. News and World Report the cover article Living With Adult A.D.D. on 4/26, 2004.

I decided to review my Learning Disabilities paper and bring the information up to date with current information by doing computer web search ant NIH and CHADD and to my surprise this twenty-year-old paper was quite up to date.

When the original paper was written I did not know if the cranial fault that I discovered was diagnostic or therapeutic, and so the paper was an overview of all current theories of that period with a limited application of applied kinesiology techniques. The original research was written before Attention Deficit Disorder became a specific clinical disorder. Although this paper was more about this condition in children the fact that more then half the children with these problem's will become adults with this problem.

Discussion

What I am doing is redoing the original ICAK Collected Paper 1984 winter with the past 20 year of research information that I have learned from practice. The following is the original paper with new information to bring this information into the twenty-first century.

Any parent, teacher, or doctor who has had the opportunity of dealing with a child with learning disabilities knows the frustration of all involved especially that of the child.

Having a brother with a very definite perceptual problem and a child with a slight one, I have a great interest in this area. Over the last few years I have treated over 100 children with this problem. (Now twenty years after this paper was written this number is has become closer to 1,500 hundred both children and adults). It appears as though learning disabilities are becoming increasingly common, or certainly we are now more cognizant of this condition.

Learning disabilities can be classified into various categories, such as: 1.) Visual perception, 2.) Auditory perception, 3.) Verbal and Written, 4.) Mathematical, 5.) Dyslexia reading, 6.) General Organization, and 7.) Hyperactivity this may be a component in any of the preceding.

These children show many predictable characteristics, leading me to believe there may be a number of common denominators in the formation of this problem. Could the cause involve neurotransmitters (such as catecholamine, dopamine and serotonin), allergy, etc., or perhaps a structural transmission problem in the brain such as the corpus callosum, dural tension, or CSF flow rate?

It should be stated here a perceptual problem or learning disability is not due to organic brain dysfunction, but rather the ability of brain and nervous system to integrate, transmit or receive: it is thus hampered and does not function properly due to some unknown reason.

Let us now examine the function of the right and left-brain hemispheres as taken from Robert E. Ornstein's *The Psychology of Consciousness*.[1] The left brain is 1.) Analytic, 2.) Logical thinking, 3.) Verbal, 4.) Mathematical functions, 5.) Linear time oriented, 6.) Process information sequentially, 7.) More predominantly analytic and sequential in its operation mass and 8.) Singular and concentrated.

The right brain is: 1.) Holistic mentation, 2.) Orientation in space, 3.) Artistic endeavors, 4.) Crafts, 5.) Body image, 6.) Recognition of faces, 7.) Process information diffusely, 8.) Responsibilities demand a ready integration of man inputs at once and 9.) More simultaneous in mode of operation.

Dr. Goodheart describes the functions of the right and left hemispheres in the 1980 Workshop Manual[2] as follows: Left-brain is logical, rational, reasonable, sensible, practical and predictable. Right brain is non-logical, non-sensible, non-practical, and non-predictable and under certain conditions can be clairvoyant, clairaudient, intuitive and tonal.

In examining the allergy, or "chemical" aspect of this problem. I referred to *Mental and Elemental Nutrients* by Carl C. Pfeiffer, Ph.D., M.D.,[3] in which he shows a possible allergic and chemical connection for learning disabilities. These might possibly fit our present electron posing theory: the following is taken form the section titled "Cerebral Allergy."

Medical workers have long known that the obvious allergic child can have an allergy-tension-fatigue syndrome, which results in a lack of interest in learning and thus decreases learning ability. When they are tested and the offending allergen is removed, these young patients improve remarkably in respect of all their symptoms, which can range from hyperactivity to somnolence, with headaches and bellyaches in between. The foods that may most commonly precipitate symptoms are milk, wheat, beef, bananas, chocolate and sugar.

The symptoms shown by the child may be those that would previously have produced a diagnosis of minimal brain dysfunction, namely:

1. Specific learning disability: Not reading at his age level, poor spelling, difficulty with arithmetic or abstractions, poor visual-motor coordination, mentally dormant.
2. Perceptual-motor deficits: Poor painting, writing or drawing; poor copying of simple designs.
3. General coordination deficits: Clumsiness or awkwardness.

4. Hyperkinesis: Constantly active, flitting form one object or activity to another, restless and fidgety, voluble uninhibited speech, disorganized thinking.
5. Impulsivity: Unrestrained in touching objects, especially in a new environment, unrestrained speech (even to being insulting), antisocial behavior and nonconformity with school, family, socially.
6. Emotional lability: Irritable and aggressive with rapid swings from temper displays to passivity easily panicked by minimal stress.
7. Short attention span on any object or subject, and ease of distraction, especially from a subject that does not arouse great interest.
8. Equivocal neurological signs: Transient eye muscle paralysis, poor finger coordination, and mixed and confused laterality slow speech development or speech defect.
9. Abnormal electorencephalogrms.

Fortunately, each child does not show all these symptoms or findings, but any one of these findings may be allergic in origin. Since the allergen may be either of the inhalant or food type, considerable testing may be necessary before the culpable agent is found. If the child is allergic to all things tested, as is frequently the case, then adequate nutrition with trace elements and vitamins may be helpful."

When I wrote this paper it was before the works of Walter H. Schmitt, D.C., showing the correlation of muscle testing as a tool to detect food and other allergens. He describes the use of the amino acid histidine as a challenge to identify if allergies are present and need to be tested for.[4]

In a paper presented at Palmer College of Chiropractic in April 15 & 16, 1989, he showed that muscle testing for food allergen could be compared at 90.5% with RAST testing.[5]

Other studies that point to allergies and nutrition in connection with learning disabilities that deserve mentioning are the following: "School Problems and Allergies," Janice Havard, M.S., Journal of Learning Disabilities. This study points out that children labeled in school as hyperactive, language or learning disabled, lazy, minimally brain injured, or emotionally disturbed, may in fact have a health problem. Symptoms may be manifestations of allergic conditions: allergies should be considered in children who are not succeeding in school or in interpersonal relations.[6]

"Nutrition, Metabolism, Brain Function and Learning" by Robert E. Buckley, M.D., Academic Therapy states that many conditions that are named neurological handicap, learning disorder, minimal brain dysfunction are in fact disturbances found in allergic disorders while others have their cause in the metabolizing glucose. A Florida study of maladapting children found that forty percent of them had a flat oral glucose tolerance test, with hypoglycemic reaction during the test.[7]

In Dr. Feingold's study, "Hyperkinesis and Learning Disabilities Linked to the Ingestion of Artificial Color and Flavors," he showed the connection between food additives and their effects on learning the hyperkinetic and perceptually impaired children. Dr. Feingold's findings showed improvement in school performance when these substances were eliminated from the child's diet.[8] The offending agents he was reporting on were dietary salicylates and artificially added colors, and flavors, and preservatives. Follow up controlled studies since 1982, the latest being 1997, have found validity to elimination diets in only a small subset of children.[9, 10, 11, 12] However, applied kinesiology muscle testing has shown a statically significant as demonstrated by Dr. Schmitt study giving us a relative simple method of diagnosing the food sensitivities.[13] The use of (NAET) allows us to change the allergies and food sensitivities.[14]

Nambudripad Allergy Elimination Technique or NAET is an offshoot of applied kinesiology discovered by a Californian who had food allergies and had a reaction to carrots. Her husband, an acupuncturist, treated her for her reaction while she had some carrot under the nail and the allergy completely cleared. Out of this observation came a system of desensitizing the patient and eliminates the allergy. This is done by subjecting

the patient to the offending agent and then muscle testing which causes a weakness to the indicator muscle. The doctor then finds a place on the spine that is on the bladder meridian called the associated point to a specific meridian that neutralizes the weakness. This point is then tapped for one minute and then a series of taps are applied to large intestine (Li-4) on the right to Li-4 on the left then down to Liv-2 on left to Liv-2 on right, and then back to Li-4 on right. This corrects the circuit imbalance and the allergy is cleared if the patient avoids the irritant for the next 24 hours.[15]

Let's now look at the structural aspects that may play a part in learning disabilities. As a chiropractor, I am fully aware that structure and function go hand in hand. In neurological science it is seen that the structure of the nervous system does dictate the way it will or does work.

In applied kinesiology, we are aware of cranial faults, but could there be a fault or faults that have evaded our discovery? This certainly seems to be a possibility. I can now say emphatically that after more then twenty years of following this cranial fault that it does exist and is connected to all learning disabilities, memory and perceptive problems. It may be the cause the mediator or the effect of this problem.

In doing research on a new cranial fault that seemed to have a specific relationship to learning disabilities, I was forced to change my original premise because the dural attachments did not connect, as I had originally believed.

In reviewing the dura mater and its relationship to the corpus callosum, I found some of the descriptions a bit sketchy and difficult to follow. I felt that it would be important to understand this anatomical entity better, however, because of our use of the reciprocal membrane tension in making cranial corrections.

My particular interest was drawn to the Falx Cerebri because of its location between the right and left cerebral hemispheres. I felt that it might have an effect upon the corpus callosum, the connecting bridge of fifty million nerve cells that link the right and left sides of the brain. Since we are very familiar with left and right brain activity in applied kinesiology and the characteristic of each side, it seemed a good place to begin my research.

I thought that if there were to be a mechanical tension change, or perhaps a laxity in the falx cerebri, it could change the transmission of information between the right and left hemispheres. This could be causing the learning disability or perceptual problems, which, we see so frequently today.

If this fault did exist, it could be used as an early screening test for pre-school children in order to get them early help which is so vital for the child's success in school and life in general.

The connection by way of the arachnoid trabecula to the pia mater that attached and adheres to all the brain surfaces and goes into the fissures. Quoting from Fundamentals of Neurology By Gardner, "just internal to the dura is a thin membrane of reticular fibers, the arachnoid, the outer and inner aspects of which are lined by flat and oval cells. The potential space between the arachnoid and the dura mater is termed the subdural space. Around the brain, a delicate network of connective tissue trabeculae connects the arachnoid to the innermost meningeal layer, the pia mater. The pia mater is a delicate membrane of reticular and elastic fiber that is closely applied to the brain and the spinal cord. The outer part of the pia mater consists of loosely arranged connective tissue."[16]

Correlative Neuroanatomy states that the falx cerebri, a crescent-shaped extension of the dura mater, projects into the longitudinal cerebral fissure.

The choroid plexus starting in the lateral ventricle forms cerebrospinal fluid. The roof of these ventricles is formed by the corpus callosum. So a change of the internal pressure might interfere with the integration of information in the learning disabled child. There is also the possibility this slight stricture at the interventricular foramen of Monroe could cause a damming up of the CSF and change the neural conductivity of the corpus callosum.[17]

From the Ciba Collection; The Nervous System: The arachnoidal villi (pacchionian bodies), cluster-like projection of the dura, protrude in the super saggital or transverse sinus. These appear about the age of seven and increase in number and size until adult life. They push their way into the sinus, thin out the dura and inner table of the skull. Their mesothelium serves as the pathway for the fluid into the venous system. The pia mater, a very fine membrane rich in minute blood plexuses and hemispheres is attached laterally and posteriorly to the transverse sinus. Anteriorly it is attached along the superior border of the petrous portion of the temporal bone and to the posterior clinoid process of the sphenoid bone, leaving a narrow space for the superior petrosal sinus. It slopes upward toward the midline where it is continuous with the falx cerebri and then forms the straight sinus. Its free border extends from the junction with the free border of the flax cerebri to the anterior clinoid process.

The falx cerebelli is a small triangle process of dura, which is attached to the lower division of the ventricles crest on the inner surface of the occipital bone with its free border projecting into the posterior cerebella notch between the two cerebella hemispheres.

The diaphragma sellae connects the clinoid attachments of the two sides of the tentorium cerebelli. It forms a roof over the hypophysis lying in the sella turcica. A circular opening in the center which allow passage of the infundibulum, is surrounded by the circular or intercavernous sinus.[18]

From Guyton's Physiology-cerebrospinal fluid: The entire cavity enclosing the brain and spinal cord has a volume of approximately 1650 ml., and about 135 ml. of this volume is occupied by cerebrospinal fluid. Fluid surrounds the brain and spinal cord and pressure of the fluid is regulated at a constant level.

The major function of the cerebrospinal fluid is to cushion the brain within its solid vault. The brain and the cerebrospinal fluid have approximately the same specific gravity, so that the brain simply floats in the fluid.[19] Other possible functions of CSF might be as a nutrient broth or a lubricant to prevent friction in the spinal cord and nerve root during flexion and extension motion in the spinal column. Another function that was pointed out by the Russian neuro-physiologist, A.E. Sparansky in his book, The Spine In Health And Disease. He showed he could stop the progress of distemper in dog by injecting the vaccine (serum) directly into the spinal CSF of the dog showing symptoms of this disease.

The mesothelium cells are associated with the arachnoid and cover the brain intimately, following the invaginations of all sulci and conformations of the gyri. By its various invaginations it helps form the tela choroidea, the chorid plexuses of the lateral, third and fourth ventricles.

The arachnoid granulation reabsorption of CSF into the venous return could be hampered by change in tension of the falx cerebri which contain the superior saggital sinus in which the arachnoid granulation are contained, and could change the flow rate at this region.[20]

Quoting from Gray's Anatomy: "The cisterna venae magnae cerebri occupus the interval between the splenium of the corpus callosum and the superior surface of the cerebellum. It reaches in between the layers of the tela choroidea of the third ventricle and the superior surface of the cerebellum. It reaches in between the layers of the tela choroidea of the third ventricle and contains the great cerebral vein of Galen.

The falx cerebri is a strong membrane extending down into the longitudinal fissure between the two cerebral hemispheres. It is attached to the skull bone along the midline of the inner surface of the cranial vault from the christa galli to the internal occipital protuberance where it becomes continuous with the tentorium cerebelli. At this attachment it is separated from the outer layer of dura, leaving space from the superior saggital sinus. The inner free margin of the falx contains the inferior saggital sinus.

The tentorium cerebelli is a transverse shelf of dura mater separating the cerebellum from the occipital part of the cerebral dural membrane."[21]

CSF continually exudes from the surface of the choroid plexus and is not exactly extracellular fluid.

The cuboidal cells of the chorid plexus actively secrete sodium ions (+) that are positively charged to the CSF which inturn pulls the chlorine ions (-) into the CSF. This creates an osmotic pressure in the ventricles of 160 mm Hg greater than that of the blood plasma, which causes the flow into the ventricles. This could substantiate the idea that the CSF might act as a neurotransmitter system possible allowing communications through this fluid medium.

The normal CSF pressure lying in a horizontal position is 130 mm of water, or 10 mm of Hg. It could also be as low as 70 mm or as high as 180 mm and be considered normal.

The presence of cranial faults could alter the osmotic pressure slightly and cause a slower or greater flow rate at the ventricles. This in turn could effect neural transmission between the right and left hemispheres over the corpus callosum, because as little as 3 mm of Hg pressure have been shown to change the neural transmission rate in the foramena compression studies.[22]

In Craniosacral Therapy, By John E. Upledger, D.O., he describes what sparked his interest in the cranial plate and dural movement. "Our own interest in the craniosacral concept came about quite by chance. I (Upledger) first became involved during surgical procedure in 1971. I was assisting a neurosurgeon in the removal of an extradural calcification from the posterior aspect of the dural tube in the mid-cervical region. Our goal was to remove the calcified plaque without incising or disrupting the integrity of the dura mater. My task was to hold the dural membrane still with two pairs of forceps while the neurosurgeon removed the plaque without cutting or damaging the underlying dural membrane. But the membrane would not hold still. I was embarrassed because I could not carry out such a simple task. The fully anesthetized patient was in a sitting position. I had no difficulty in reaching or seeing the operative field there was no excuse.

It became apparent that the movement of the dural membrane was rhythmical at about 8 cycles per minute. This rhythmic activity was independent of the patient's breathing and cardiac rhythms. It was another physiological rhythm. It appeared to be an ebb and flow of the fluid, which is contained within the dural membrane. Both, the neurosurgeon, the anesthesiologist, nor I had ever observed this phenomenon before. My curiosity was piqued. I could find no information in conventional medical or physiology literature."

From the same text, Dr. Upledger states: "You can use the ventrical falx and the dural tube system to influence the straight sinus system, which, in turn, exerts an influence upon the leaves of the tentorium cerebellil, and of course, converse is true; you can use the cerebella tent to influence the vertical dural membrane system. As you will note in the following illustrations, the falx cerebri attach at the glabella and the frontal and ethmoid bones, and posterior attachments as the straight sinus and the internal occipital protuberance, a swell as the inferior attachment around the foramen magnum, then attaches at C2-C3 and free dural tube still it reaches the second sacral section."[23]

The following illustrations were taken from John E. Upledger, D.O. and Jon D. Vredevoogd, M.F.D. text Craniosacral Therapy with permission for the publisher Eastland Press.

Dysfunctions of the Craniosacral Dural Membrane System

Illustration 6–8 **Venous Sinuses within Falx Cerebri**

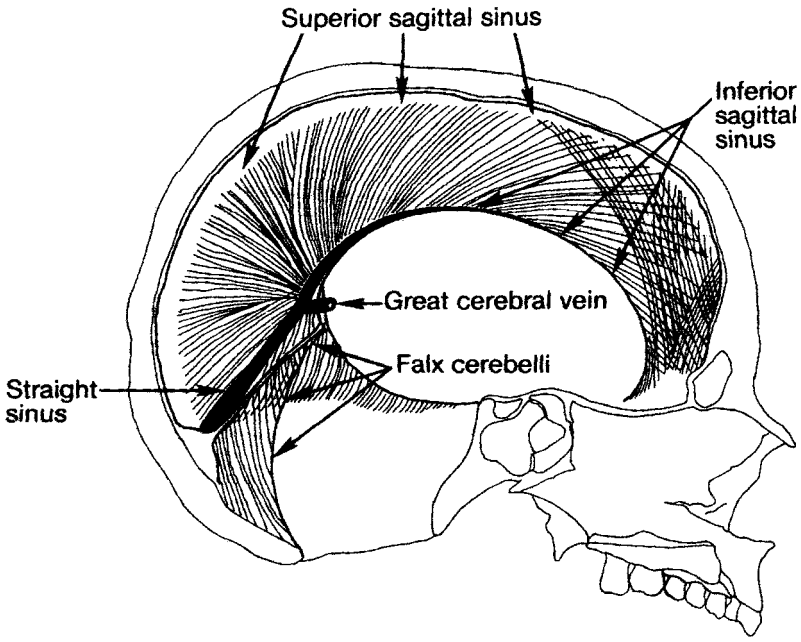


Illustration 6–9 **Formation of the Straight Venous Sinus**

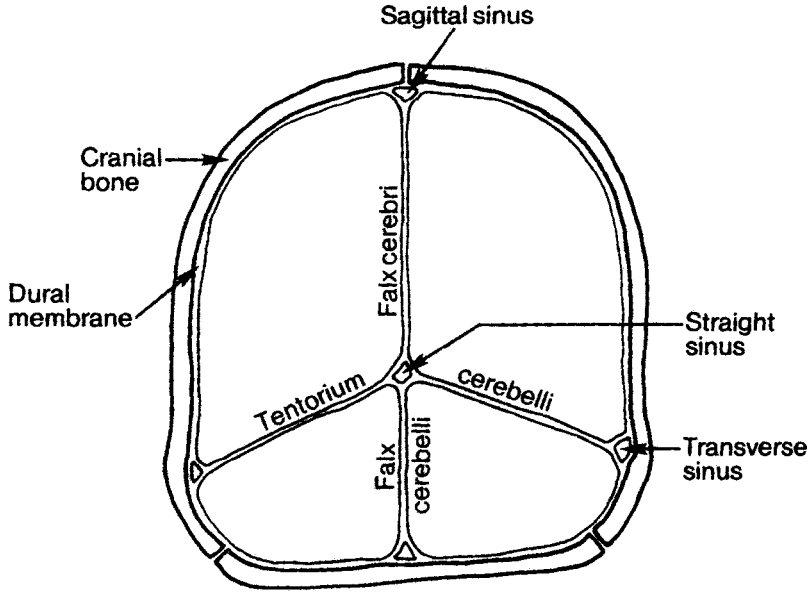


Illustration 6–21 **Effect of Occipital Traction on the Dural Tube and Sacrum**

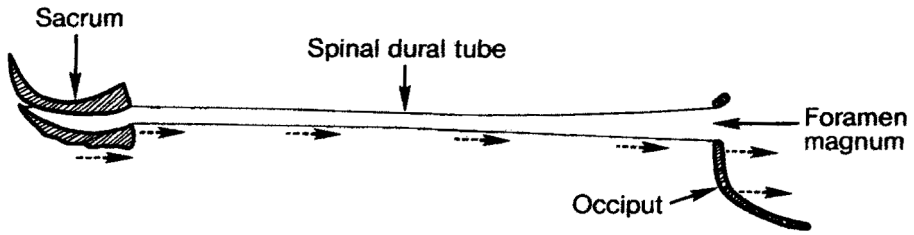
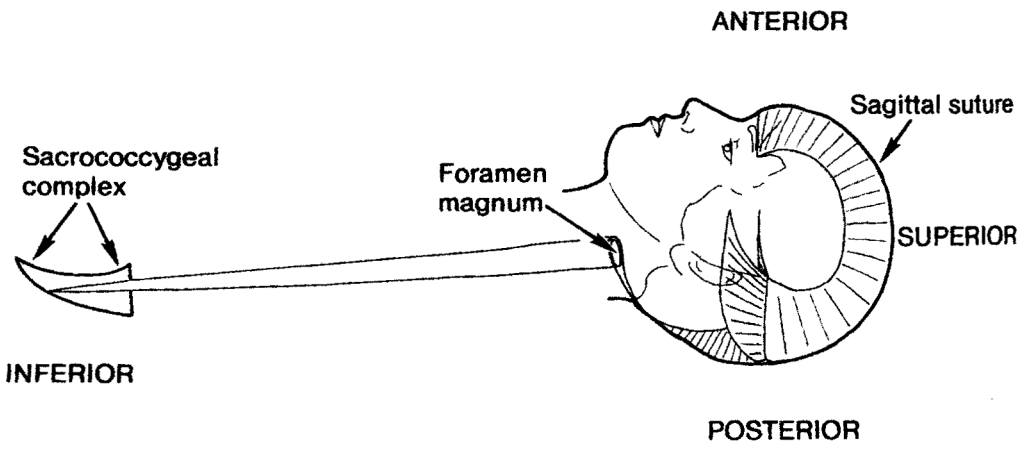


Illustration 6–13 **Anterior-Posterior and Superior-Inferior Axes of the Dural Membrane System**



To substantiate the premise of cranial bone motion this study by Ernest W. Reizlaff, Ph.D., The Structures of Cranial Bone Sutures: The general pattern of the suture was similar to that reported by Pritchard et al. In each of the samples studied the sutures displayed five distinct layers of cells and fiber between the articulating edges of the bones. The outer most layer is a zone of connective tissue, which bridges the suture, and is designated "the sutural ligament." Two layers appear to be continuous with that of the periosteal layer; the sutural ligament is found on both the outer and inner surfaces of the suture. The space between the ligaments is loosely filled with fibrous connective tissue.

The reticular connective tissue portion is seen in the central space, blood vessels and nerve fiber are evident. The function of the nerve fibers is not known but it is possible that they may be involved in the physiological effects of cranial therapy.

Anther study that shows the motion of the cranial plates is a study, Examination of the Cranial Rhythm in Long Standing Coma and Chronic Neurologic Cases, Z Karni, J.E. Upledger, D.O.: The hydraulic contents are subjected to the pulsatory motion of the arterial system, the venous system and the pulmonary system which transmits its effects to the dura mater through the vertebral connections along the cervical section of the spinal column. The lateral displacements, which all these systems induce upon motion, the nature of which depends upon the fluid properties and on the mechanical behavior of the container.

Using a dial gauge measurements which, the tip of the gauges is tightly compressed against the parietal bones, yielded values of 10 to 25 microns side displacement of the bones.

The normal range of frequency of the cranial rhythm is 6 to 12 cycles per minute (cpm). This rate is slower than the respiratory rate in the relaxed state by almost a third. The normal amplitude, identified with the lateral displacement of the parietal bone, may reach 1 to 1 ? mm. Of the various techniques, strain plethysmography by means of high extension, electrical resistance strain gauges prove more sensitive and effective.[24]

In the study, Diagnosis and Treatment of Temporparietal Suture Head Pain, John E. Upledger, D.O., Ernest W. Retozolff, Ph.D., and Jon D. Vredevoogd, M.F.A. By the use of modified staining techniques, the authors have been able to demonstrate the presence of viable myelinated and unmyelinated nerve fibers, nerve receptors endings, a potentially functional vascular network, and collagen elastic fiber complexes with the adult human cranial suture. We have demonstrated that these structures frequently penetrate the sutural bone margins and traverse from the diploe into the suture and vice versa. There is also evidence to suggest that some of the intrasutural vascular and neural structures may arise from the intracranial meninges.

The significance of these findings is simply that now the human cranial suture maybe considered as a functional anatomical complex capable, therefore, of dysfunction resultant to various imbalances, stress and trauma.

The specimen studies were taken from living adult skulls at the time of neurosurgical craniotomy. Hence, these tissue studies resemble more closely the in vivo circumstance.

The presence of nerve endings in the sutural tissues may act as monitor for the meningeal cycle of 6 to 12 cpm. This cycle is probably due to the increase production of CSF, which in turn causes slight expansion of the sutures. This then causes a neural signal to the chorid plexus to slow the CSF production till the arachnoid granulation can reabsorb it into the venous return.

This would account for the meningeal movement observed by Dr. Upledger and would also account for the presence of nerve endings in the sutures.[25]

While reading, The Brain-The Last Frontier, Richard M. Restak, M.D., I read about a study done by Dr. E. Roy John, Ph.D. a researcher at New York University Medical School. Dr. Restak stated the Dr. John found, by using evoked potential studies of learning disabled children, that abnormalities were noted in the right brain for children with math deficiencies, and abnormalities in the left brain for verbal and language. I wondered why math, a left side function, so I read Dr. John's paper, Neurometrics, and another paper titled Comparison of Neurometric and Psychometric Test. In these tests, there were 118 normal and 57 learning disabled. The initial discriminant accuracy was 93 percent for the neurometric and 76 percent for the psychometric indices.

Aside from their bias with respect to age, IQ, culture, and relation between these psychometric measures and brain dysfunction may merely reflect the fact that performance on these tasks is heavily dependent upon skills which are difficult for the learning disabled child to acquire.

The major clusters contained over 64.3 percent of the learning disabled children but only 20 percent of the normal children, thus replicating our original finding that normal and learning disabled children differ markedly with respect to certain neurometric EEG indices. Note that 87.5 percent of the learning disabled children, but only 8 percent of the normal children, displayed abnormal EEG or AER asymmetry, or both, with 71 percent of learning disabled children, but 0 percent of normal children, showing AER asymmetry consistent with our findings of marked AER asymmetry.

The most striking feature of the results is the high percentage of learning disabled children who displayed multiple types of dysfunction in multiple regions. A remarkable feature of these findings is that information processing in children with a particular type of learning disability seems to reflect a general operational defect, independent of the specific information content of the input revealed as a distinctive spatiotemporal pattern.[26]

This study that I found again emphasizes the need for chiropractic care in the newborn and infants, particularly those chiropractors versed in applied kinesiology. The Study is titled, "Diagnosis of Vestibular Disorders in the Learning Disabled," Julio B. De Quiros, M.D., Ph.D. Due to the length of this study I will use only the highlights in this paper.

Children with vestibular disorders and related postural disturbances constitute a large segment of the population described as learning disabled. Vestibular disorders identified in newborn infants can be substantiated in children and adults. Early identification of vestibular disorders could alert physician, clinicians, and educators to the need for modification in the child's learning environment. Appropriate modifications could help to negate the influence of postural disturbances on the development of efficient functional systems.

Vestibular disorders can be diagnosed medically within hours after birth. However, the learning disabilities associated with vestibular disturbances frequently are not identified until "soft signs" lead to a diagnosis of "minimal brain dysfunction" when the child starts school. The need for a definitive medical diagnosis at an early age led me, in 1958, to begin to study postural disturbances of newborn infants and then to follow the subsequent development of these infants. This study was carried out between 1958 and 1967 at the Research Medical Center at the University of the Museum in Buenos Aires, Brazil and consisted of 1,902 children. The data indicated the existence of a syndrome, the main characteristic of which is the following:

1. vestibular areflexia in response to the caloric test (irrigation of the ear with cold or warm water, which is able to elicit a vestibular oculomotor reflex called "provoked nystagmus" rapid and slow movements of the eyes,
2. delay of motor development,
3. walking instability,
4. delay speech development.

The conclusions of the studies were: vestibular disorders and postural disturbances can produce learning disabilities associated with motor skills, the acquisition of language, and the development of normal competencies in reading-writing. When vistibular disorders are identified during infancy, parents, physicians, clinicians, and educators are alerted to the need for an early intervention program.[27]

With advent of applied kinesiology, there are many techniques that have had an influence on this vestibular mechanism such as the organizational reflexes (cloacal, righting, vestibular, tonic neck reflexes) other specific findings are the sacral wobble and the counter torque of the atlas/occiput. Other factors that we will check for are Temporomandibular joint dysfunction and cranial faults especially those that effect the temporal bone.[28, 29, 30]

Another study that we in applied kinesiology are very familiar with is the work of John Ott and effect of light on health titled "Influence of Fluorescent Lights on Hyperactivity and Learning Disabilities." During the first five months of 1973, the Environmental Health and Light Research Institute conducted a pilot project in four first grade windowless classrooms of a school in Sarasota, Florida. In two of the rooms, the standard cool-white fluorescent tubes and fixtures with solid plastic diffusers remained unchanged. In the other two rooms, the cool-white tubes were replaced with full-spectrum fluorescent tubes that more closely duplicate natural daylight. Lad foil shields were wrapped around each end of the tubes where the cathodes are located. Aluminum "egg crate" diffusers with an additional grounded aluminum screen grid replaced the solid plastic diffusers in these latter rooms. A dramatic improvement in behavior was demonstrated in hyperactive children.[31]

Treatments

There are two ways that treatments should be evaluated: (1) standard scientific procedure or (2) limited case studies or testimonials. The scientific approach involves testing different treatment in controlled conditions; there should be sufficient subjects to allow the researchers to be comfortable with the findings. Different research groups should repeat these studies and the finding should be published.

Other methods that should be mentioned that have shown some promise in learning disabilities include Interactive Metronome Training, a computerized version of a simple metronome. The individual "keeps the beat" with the hand or feet, there is auditory feedback to show how well the individual does to match the beat; this improved motor planning and timing skills.

Sensory integration training therapy this is delivered by occupational therapists and is not specific for learning disabilities such as ADD or AD/HD, but is designed for intervention for brain overload by sensory input.[32, 33, 34]

EEG biofeedback or neurofeedback is an intervention for AD/HD a method is based upon studies showing low levels of arousal in frontal brain areas in individual with and without this condition.[35] This treatment has been in use for more then 25 year with reported good results from parents with children with this condition.[36]

Medically, the treatments have been similar for ADD and AD/HD with the use of stimulants that are amphetamine in nature; the go products such as (Adderall, Concerta, Dexedrine, Ritalin). The only new drug being used Strattera that is not a stimulant that effects the neurotransmitter norepinephrine and dopamine. If the parent of a child with ADD or AD/HD takes this route, the medication can be tested both against the cranial fault (LD), and in general for compatibility with the child.

Other learning disabilities that may stand along on may be combined with ADD and AD/HD:[37]

1. Dyscalculia-a problem in doing mathematics.
2. Dysgraphia-inability to write properly.
3. Dyslexia –a problem with reading.
4. Dyspraxia-a problem with motor skills due to pain.
5. Information processing disorders (visual and auditory).
6. Language-Based learning problems.

Methods

When I first started my search, I felt that the most logical place to begin was at the crossroad of the right and left cerebral hemisphere: the corpus callosum. The nearest point to this that I could reach was the palate or the roof of mouth.

The next step was to be able to identify this fault, if it did indeed exist. As we know the sphenobasilar fault was identified with therapy localization of both thumbs on the cruciate suture of the hard palate. This contact would cause a strong indicator muscle to weaken.[38]

I found a fault that therapy localized with both index fingers in the mid-palate over the cruciate suture (only simultaneously); it also TL with the two ring fingers (which is very difficult to use), but not to any other fingers. It would be negative to TL if the index finger were placed on the opposite side of the hard palate over the cruciate suture. In other words, the right index finger placed on the left side of the cruciate suture and visa versa.

This fault did not negate to any respiratory pattern, but did require inspiratory assist to correct.

Findings

My original study consisted of 42 patients, 34 males ranging in age from 8 to 25 years, and 8 females, 9 to 18 years of age. All were classified and known to have learning disabilities or perceptual problems.

My control group consisted of 250 patients, 175 male-ages 8 to 31 years and 75 females, 10 to 22 years of age. This group did not show this specific fault. The control group was questioned carefully to determine that none had any type of learning problems.

At present, 20 years later I have seen this fault on at least 1500 patients. What I have learned over the past five years about this fault, that when I found it even if the patient does not have any learning disability they will have some memory or perception problems.

Procedures and Correction

The learning disability group was checked for all known cranial faults. These were recorded and then correction was made for these faults. I then rechecked by therapy localization with both index fingers against the roof of the mouth over the cruciate suture. I still found a positive localization causing a strong indicator muscle (tensor fascia lata) to weaken.

The one unique feature of this fault was that a respiratory assist (inspiration) is required for correction, but does not negate the positive therapy localization.

The correction is done directly over the cruciate suture in the midline of the palate using the index finger. Pressure is directed toward the vertex of the skull at CV 21. The other hand exerts pressure toward the palate over the saggital suture toward the cruciate suture. This is done on inspiration using 3 to 4 pounds of pressure repeated 4 to 5 times, rechecked with therapy localization to see if the correction had negated the positive finding. The saggital suture is rechecked to make certain it is not jammed during the correction.[39]

Conclusion

When I originally wrote this paper I was not certain whether this correction would improve learning performance in children with learning disabilities. However, twenty years later I can definitely say that this does improve ability to learn. What must be kept in mind is that there are other variables that can effect the outcome in learning disabilities.

1. Length of time the learning disability has been present.
2. The amount of schoolwork that the child has fallen behind in which in many cases is in terms of years.
3. The motivation of the child to want to learn.
4. The length of time before the school's "child study team" diagnosed the learning disability.
5. Parental support, or lack of it.
6. Remediation program in the school and in the home environment.

These are just some of the factors that made assessing the results very difficult at the point that this paper was originally written. However, after twenty years of observing this fault in children with a learning disability, as well adults with this problem, I have found that this fault is both diagnostic and its correction was of definite therapeutic value and does show a marked improvement in the ability to learn.

What should be remembered is that children with ADD and ADHD will be diagnosed in 2 to 5 percent of children between the ages 6 and 16; approximately 80 percent are boys. These symptoms will persist into adulthood in 30 to 50 percent of cases.[40]

Dr. Goodheart commented to me about a year ago that this fault and information was a sleeper and probably has gone unnoticed by most of the ICAK member. This was because at the time of my original paper 1984, Dr. Carl W. Ferreri had written his book *Break Through for Dyslexia and Learning Disabilities* and developed the Neurological Organization Technique (NOT).

Dr. Goodheart's observation of the fault and it's correction as he stated it to me was that "when he made the correction in his patients who were actors or musicians it made it much easier for the to learn difficult scripts and complicated musical scores."

To complete the remainder of what I wrote in the original paper as it appeared in 1984 is the following. I have written to both Drs. E. Roy John and John E. Upledger for any other ideas on verification of the fault with learning disabilities correction to improve learning performance.

Dr. John never answered my request for assistance. Dr. Upledger, however, suggested that I treat only one group and keep one group untreated as a control and observe the results.

As I have mentioned, there are many variables that make this empirical method ineffective in both diagnosing the need and observing the results.

Dr. John's evoked potential method seemed to be more objective in its findings as a method of diagnosing the presence of a learning disability and comparing the presence of the specific cranial fault to that abnormality in the evoked potential. This method could also prove whether or not the correction to this fault would change this evoked potential abnormality.

I am planning further studies in this area, both using double blind methods to see if I can diagnose the learning disability without any prior knowledge of the students being tested, and I will report my finding at a later date.

This being the later date, in fact twenty years later I can report that it does hold true that this fault can represent the presence of a learning disability, but also ties to a number of other memory problems.

To report on a recent case less then six weeks ago I had a high school senior who has been a patient for the past two years. She came in recently for an ankle problem, however, during testing I found evidence of this fault present and she said that she had A.D.D. I took care of her ankle problem and on the follow up visit she mentioned that she took her SAT for the second time.

She mentioned that the room in which the test was being given had the school band playing nearby and everyone in the room was distracted except her. The most notable change besides not being distracted was her test scores increased by more then 230 points. This finding is way beyond what is normal expected for taking the test the second time.

I am arranging to conduct this correction on other students with similar problems who have taken a previous SAT exams which will give a base line score, that will allow me to know if the correction had some bearing on the improvement noted.

References

1. Ornstein, Robert, E., Ph.D., *The Psychology of Consciousness*, The Viking Press, New York, 1972.

2. Goodheart, George, J., D.C., Applied Kinesiology Workshop Manual, Private Publication, 1980.

3. Pfeiffer, Carl, C., Ph.D., M.D., *Mental and Elemental Nutrients*, Keats Publishing Inc. 1975.

4. Schmitt, Walter, H., D.C. The Use of Antronex and Histidine as Screening Tools for Food and Other Allergies, The Collected Paper of International College of Applied Kinesiology, Private Publication, winter 1983, p 173.

5. Schmitt, Walter, H., D.C., Correlation of Applied Kinesiology Muscle Testing Findings With Serum Immunoglobulin Levels for Food Allergies, Paper presentation At Palmer College, Second Symposium on Nutrition and Chiropractic, April, 1989.

6. Harvard, Janice, M.S., School Problems and Allergies, Journal of Learning Disabilities, Vol. 6, No. 8, Oct. 1973.

7. Buckley, Robert, M.D., Nutrition, Metabolism, Brain Function and Learning, Academic Therapy, Vol. XII, No. 3, Spring 1977.

8. Feingold, Benjamin, F., M.D., Hyperkinesis and Learning Disabilities Linked to the Ingestion of Artificial Food Colors and Flavors, Journal of Learning Disabilities, Vol. 9, No. 9, November 1976.

9. Feingold, Benjamin, F., M.D., *Why Your Child is Hyperactive*, Random House Publishers, New York.

10. Wender, E.J., The Food Additive-Free Diet in the Treatment of Behavior Disorders, Journal of Developmental and Behavioral Pediatrics, No. 7, 1986, pp. 735–742.

11. Baumgaertel, A. Alternative and Controversial Treatment for Attention Deficit/Hyperactivity Disorder, Pediatric Clinics of North America, Vol.46, 1999, pp. 977–992.

12. Worlraich, M.L., Lindgren, S.D., Stumbo, P.J., Stegink, L.D., Appelbaum, M.I., and Kiritsy, M.C., Effects of Diet High in Sucrose or Aspartame on the Behavior and Cognitive Performance of Children, New England Journal of Medicine, Vol. 330, pp. 301–307, 1994.

13. Schmitt, Walter, H., D.C. A Pilot Study Showing Efficiency for Applied Kinesiology Muscle Testing Findings With Serum Immunoglobulin Levels for Food Allergies, Collected Paper of ICAK, Private Publication, 1988 Winter pp. 383–391.

14. Cutler, Ellen, W., D.C., *Winning the War Against Asthma and Allergies*, Delmar Publishing, 1998.

15. Cutler, Ellen, W., D.C., *Winning the War Against Asthma and Allergies*, Delmar Publishing, 1998.

16. Gardner, Ernest , M.D. Fundamentals of Neurology, W.B. Saunders Company, Phil., 1963.

17. Chusid, Joseph, M.D., Mc Donald, Joseph, J., M.D., Correlative Neuroanatomy and Functional Neurology, Lange Medical Pub lication, 1964.

18. Netter, Frank, H., M.D., The CIBA Collection of Medical Illustration, The Nervous System, 1964.

19. Guyton, Arthur, C., M.D., Textbook of Medical Physiology, W. B. Saunders Company, 1966.

20. Guyton, Arthur, C., M.D., Organ Physiology, Structure and Function of the Nervous System, W. B. Saunders Company, 1976.

21. Gray, Henry, Anatomy of he Body, Lea & Fabiger, 100th Edition, Phil, 1965.

22. Guyton, Arthur, C. M.D., Organ Physiology, Structure and Function of The Nervous System. W. B. Saunders Company, 1976.

23. Upledger, John, E., D.O., F.A.A.O., and Vredevoogd, Jon, D., M.F.A., Craniosacral Therapy, Eastland Press, Chicago, IL 1983.

24. Retzlaff, Ernest, W. Ph.D., Michael David, D.O., Roppel, Richard, Ph.D., and Michell, Fred, D.O., The Structures of Cranial Bone Sutures, The Journal of The American Osteopathic Association, Vol. 75, 1976, pp. 607–608.

25. Upledger, John, E., D.O., F.A.A.O., Retzlaff, Ernest, W., Ph.D., and Vredevoogd, Jon, D., M.F.A., Diagnosis and Treatment of Temporoparietal Suture Head Pain., Osteopathic Medicine, July 1978, pp. 19–26.

26. John, E., Roy, Ph.D., Neurometrics, Science, Vol. 196, No.4297, June 24, 1977.

27. De Quiros, Julio, B., M.D., Ph.D. Diagnosis of Vestibular Disorders in the Learning Disabled. Journal of Learning Disabilities, Vol. 9, January 1967.

28. Walther, David, S., D.C. Applied Kinesiology, Vol. I Basic Procedures and Muscle Testing, SYSTEMS DC, Pueblo CO, 1981.

29. Walther, David, S., D.C. Applied Kinesiology, Vol. II Head, Neck and Jaw Pain and Dysfunction-The Stomatognathic System., SYSTEM DC, Pueblo, CO 1983.

30. Walther, David, S., D.C. Applied Kinesiology, Synopsis, 2nd Edition, SYSTEM DC, Pueblo, CO, 2000.

31. Ott, John, N., Sc.D. Influence of Fluorescent Light on Hyperactivity and Learning Disabilities, Journal of Learning Disabilities, Vol. 9, Aug./Sept., 1976.

32. Chabot, R.J., M.D., and Serfontein, G., M.D., Quantitative Electroencephalographic Profile of Children With Attention Deficit Disorder, Biological Psychiatry, Vol. 40 pp. 951–963.

33. El-Sayed, E., M.D., Larsson, J.O., M.D., Persson, H.E., M.D. and Rydelius, P.A., M.D., Altered Cortical Activity in Children with Attention Deficit/Hyperactivity Disorder During Attentional Load Task, Journal of the American Academy of Child and Adolescent Psychiatry, Vol. 41, 2002, pp. 811–819.

34. Clark, A.R., M.D., Barry, R.J., M.D., McCarthy, R., M.D., and Selikowitz, M., M.D. Age and Sex Effects in the EEG: Differences in Two Subtypes of Attention Deficit/Hyperactivity Disorder, Clinical Neurophysiology, Vol. 112, 2001, pp. 815–826.

35. Lahey, B.B., Ph.D., and Kazdin, A.E., Ph.D. Advances in Clinical Child Psychology, Plenum Press, New York

36. Monastra, V.J., Ph.D., Monastra, D.M.,Ph.D., George, S., Ph.D. The Effects of Stimulant Therapy, EEG Biofeedback, and Parenting Style on the Primary Symptoms of Attention-Deficit/Hyperactivity Disorder, Applied Psychophysiology And Biofeedback, Vol. 27, pp. 231–249.

37. Ferreri, C.W., D.C., and Wainwright, R.B., D.C., Break Through for Dyslexia and Learning Disabilities, Exposition Press, Pompano Beach, FL, 1984.

38. Sprieser, Paul, T., D.C., DIBAK, Learning Disabilities, Collected Paper of International College of Applied Kinesiology, Private Publication, winter 1984, pp. 265–291.

39. Sprieser, Paul T., D.C., DIBAK, Learning Disabilities, Part I & II, The Digest of Chiropractic Economics, May/June 1987 pp. 34-39, and Nov./ Dec. 1987 pp. 20–25.

40. Rothenberger, Aribert, M.D., Banaschewski, Tobias, M.D., Informing the ADHD Debate, Scientific American MIND, Vol. 14, No. 5, Jan. 24, 2005.

Gastroesophageal Reflux Disorder and Hiatal Hernia, A Universal Problem

Paul T. Sprieser, D.C., DIBAK

Abstract

The exploration of possible causative factors for (GERD) gasroesophageal reflux disorder and hiatial hernia. Both problems and their accompanying symptomatic patterns are caused by a structural failure of muscular systems yet the medical approach seems to be addressed with chemical means.

Key Indexing Terms: Gastroesophageal reflux disorder (GERD), hiatial hernia, applied kinesiology.

Introduction

Have you ever wondered why heartburn and GERD seemed to be at epidemic proportions? You cannot turn on a television or radio or pick up a magazine without being assaulted with ads for antacids such as Tums, Mylanta, Alka-Seltzer, Maalox, and Histamine H_2 receptor antagonists like Zantac, Tagament, Pepcid, Prilosec, Nexium just to name a few.

You might believe that everyone has these problems and to some extent there is some truth to this because we have all had at least one episode of heartburn. However national support groups and other source say that at least 44% of otherwise healthy adult Americans suffer from heartburn at least once a month and 7% suffer this symptom at least once a day this would amount to 25,000,000. About 2% also suffer from GERD, which increased markedly after age 40. This information was obtained in a national survey of heartburn across America done by the Gallup Organization in 1988, but these figures are probably higher today.[1]

What could be the common denominator be, that cause this to become such a common problem? This paper will attempt to give a rational reason for the complaint.

Discussion

Starting with the hiatial hernia the causative factors are divided into congenital or secondary to trauma, but most often the cause is unknown. There are two types the first is sliding that we look at.

The sliding variety with a portion of the stomach slid up through the diaphragm show up on x-ray in 40% of the population. In Boyd's Pathology it was stated that on autopsy that this was found in 70% of patient over 70 years of age.[2] To diagnose this disorder with a barium swallow the patient placed inverted on the Trendelenburg table, however pressure might be needed to make it visible.

Most of these variety are asymptomatic and those with gastroesophageal reflux in a few patients is doubtful if the hernia causes the reflux it also causes chest pain. The second type is known as a paraesophageal hiatial hernia and can incarcerate and strangle the stomach causing GI hemorrhage.

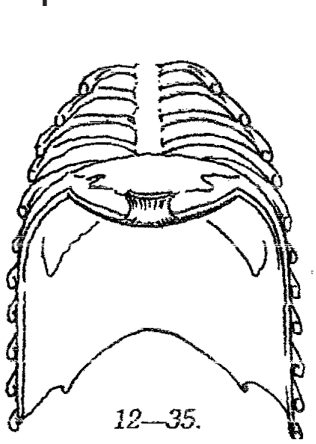
Gastroesophageal reflux is caused by incompetence of the lower esophageal sphincter factors that contribute to the competence of the sphincter the following are the four factors: l. intrinsic sphnicter pressure, 2. Angle of the cardioesophageal junction, 3. Action of the diaphragm, 4. When up right gravity.[3]

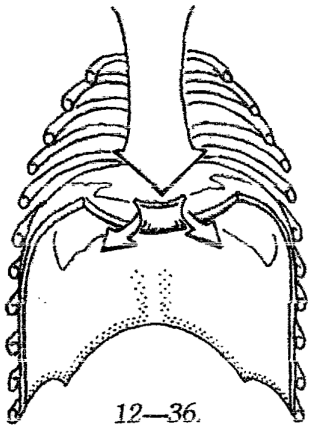
Symptoms are heartburn with or without gastric contents into the mouth. Esophagitis and peptic esophageal stricture, ulceration, dysphasia with solid food in stricture formation. With ulceration localized pain at the xiphoid or high substernal region.

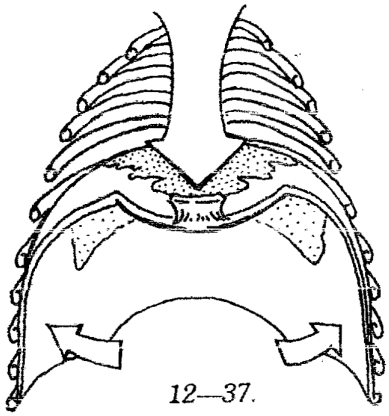
The mechanism of respiration shows 70% of the expansion and contraction of the lungs are in the anterior/posterior movement of the rib cage and 30% movement of the diaphragm.[4]

We must look at the muscles of inspiration and expiration to see what part of this mechanism can play. Inspiration has the following muscles involved in this action in order of importance, diaphragm, external intercostal, sternocleidomastoid, scapular elevators plus anterior serratus, scalenes, and the extensors of the spine. Expiration has the abdominal, internal intercostal, and posterior inferior serratus. The two most important muscles involved in this syndrome are the diaphragm and the abdominal.

Principally the diaphragm causes normal inspiration. This is a bell shaped muscle and when the fiber contract in causes the pull to be downward. Ordinarily expiration is passive, when the diaphragm relaxes the elastic structures of lung, chest cage, and abdominal cause the diaphragm to move upward (Fig. 12-35).
With permission from David S. Walther, D.C. *Applied Kinesiology-Synopsis 2nd Edition*.

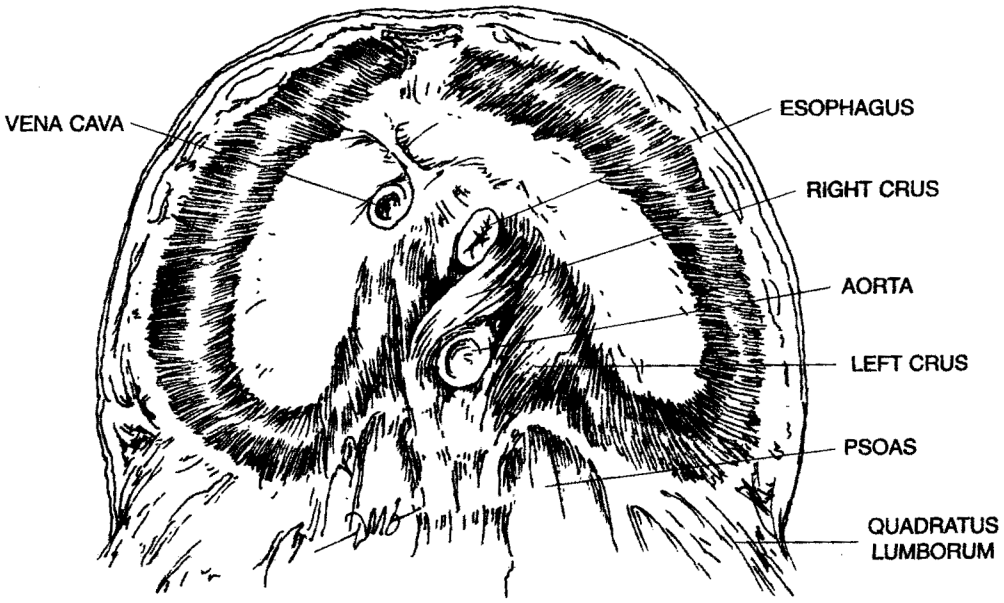

12—35.


12—36.


12—37.

The most critical part of the diaphragm is the crura that appear to assist the cardiac sphincter of the stomach to remain competent and acts as a secondary sphincter. If we look at the diaphragm from below we can see the muscular fibers forming the esophageal hiatus originate form the central tendon and the left leaflet and cross over to the right side and insert and join the right crus and insert into the bodies of L1-L2-L3. There is an opening on the right known as the medial lumbocostal arch through which the right psoas passes (Fig. 12-31).[5]
With permission from Dr. David S. Walther, D.C., *Applied Kinesiology-Synopsis 2nd Edition*.

12–31 **Inferior view of the diaphragm**



The right muscle fiber comes from the right central tendon and encircles the esophagus on the right and passes down and inserts into the left crus that attach to the upper L1-L2 vertebral bodies. So the esophageal hiatus acts to assist the smooth muscle sphincter of he esophagus. The resting pressure in the gastroesophageal sphincter is normally no more then 40 cm of water.[6]

Some etiological reasons for the heart burn and gastric reflux can be over weight, poor posture, diet, and food allergies.

The hiatal hernia and reflux esophagitis (GERD) the slit like opening bound by muscle bundle from the right crus bound in place by the phrenoesophageal ligament. This can be enlarged or torn leading to 75% of sliding variety and about 25% of rolling on paraesophageal mucosal inflammation in the terminal esophagus last 1 to 5 cm.

The cause of the hiatal hernia can be congenital or traumatic enlargement. Influenced by relation of ligaments and skeletal muscles increased into abdominal pressure and caused by pregnancies, advanced age, obesity, ascities, and chronic coughing. Not all-sliding hiatal hernia has reflux as their main symptom. The most important factors are the tone of the vestibular internal sphincter, crura of the diaphragm external sphincter, and the oblique angle of the insertion of the esophagus into the stomach producing a flap valve formation and the formation of rosette by the redundant mucous membrane. This creates a zone of high pressure that prevents the gastric content reflux thought the internal sphincter. Causative factors can be prolonged vomiting due to illness or from eating disorders such as bulimia. Other factors effecting this are is prolong use of stomach tube, surgery that effects the esophagus crura and scleroderma.[7]

Additional consideration pharynx tunica muscularis contains skeletal muscles and the lower end of the esophagus near the stomach is smooth muscle that responds to peristalsis action of swallowing. The muscles surrounding the upper portion of the esophagus are normally in a state of tonic contraction and act as an additional sphincter. The lower 2 to 5 cm. above the cardiac orifice is smooth muscle of the esophagus is normally in a state of tonic contraction and acts as a sphincter. It usually takes 5 to 10 second for food to move from the mouth to the stomach during swallowing.[8]

Other interesting facts of the digestive process that 2/3 of the saliva is produced by the submandibular glands and a total amount produced is between 1 to 1 1/2 liters a day. The gastric glands of the parietal cells (oxyntic) cells produce the HCL which is important for protein digestion and the overall acidity of the stomach is approximately ph of (0.8) which act to kill bacteria that enter the digestive system.[9]

The digestive enzyme of pepsinogen, that optimally works in and acid environment, and is necessary for their digestion and utilization of three amino acids tryptophane, phenylalaine and tyrosine.[10] The proper acidity of the stomach can that be responsible for the use of the amino acid tyrosine and production of hormones thyroid and the adrenal system and could play a part in depression because tryptophane is needed to produce serotonin.

Final consideration is the amount of gastric juice produced which, is approximately 3 liters a day and diet effects it production. A typical meal will cause the production of 700 ml. and it is caused by both neural stimulation and hormonal secretions.[11]

The symptoms that these two condition cause are the following: 1.) Sour or bitter taste in mouth, 2.) Stomach fluid in mouth during sleep, 3.) Hoarseness in voice, 4.) Repeatedly feeling a need to clear the throat, 5.) Difficulty in swallowing food or liquid, 6.)Wheezing or coughing at night, 7.) Symptoms worsen after eating, bending, or lying down. 8.) Lower dorsal pain near the tenth dorsal.[12]

Method of Correction: AK has had much success with dealing with either of these two problems. One the main reasons is the both the Hiatal Hernia and GERD are both mechanical failures of muscular structures that allow for all the symptoms that the patient experiences (page 577-Fig. 12–32, 12–33, 12–34, 2–58). For correction of the hiatal hernia manual pressure is applied to the upper left quadrant of the abdomen two inches lateral of linea alba near the area of the first tendinous inscription of the rectus abdominis muscle. This is accomplished usually with the patient either standing or on a HI/LO table at raised angle of 50 degrees this uses gravity to assist the correction. Pressure is applied over the stomach during the inspiration phase and forceful tug is applied as the air is expired. This uses the natural movement of the diaphragm to assist the correction. The application of this traction pull correction usually is applied four or five time to get adequate correction.

The hiatal hernia is therapy localized with both hand fingertips in the area of the rectus abdominis first tendinous inscription just below the costal cartilage making sure to avoid the neurolymphatic reflex point of the adrenal gland. Another method to be used in evaluation can be a challenge of the left upper quadrant pushing the stomach back and up into the esophageal hiatus. Both methods will cause a strong indicator muscle to weaken.

Treatment to the neurolymphatic reflex (NL) for the diaphragm, which is the entire length of the sternum, should be stimulated and checked with TL to the sternum and also temporal tap to make sure it is sufficiently treated. There is also a posterior NL reflex point at the level of T10 transverse on the right.[13]

The neurovascular reflexes (NV) for the diaphragm are three in number the anterior is at the bregma at the junction of the saggital and coronal suture the posterior reflexes are two located the junction of the lambdoidal and saggital sutures and one inch above on the saggital suture.[14]

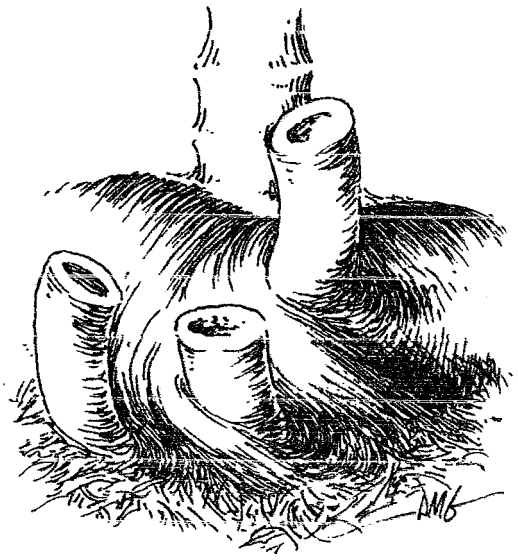
Another factor that should be checked is the stress receptor on the head, which is located in the center frontal bone between the glabella it lies horizontal plane. There are two hand stress receptors on the hands one for inspiration diaphragm in the opponens pollicis along the palm surface just above the wrist. The other hand stress receptor is also on the palm surface at the base of the middle and ring finger.

Reactive muscle should also be checked the classic pattern to the diaphragm is the psoas, but other muscles may play a part in reoccurring hiatal hernia pectoralis minor, abdominal and quadratus lumborum. This is done by TL to the original point in the upper right and left quadrant of abdomen and challenging each muscle to see if it weakens a strong indicator muscle.[15]

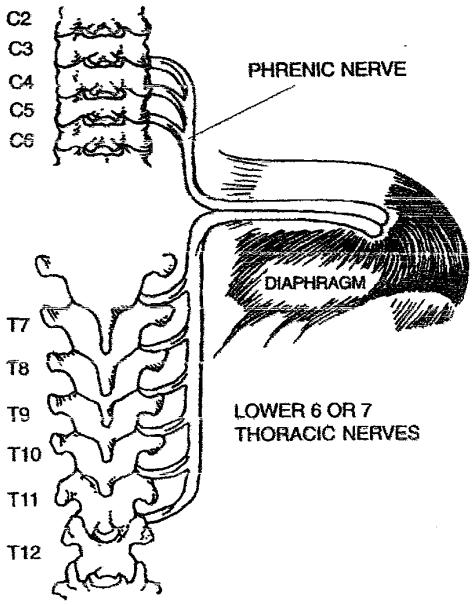
Finally the fixation pattern at the transitional region of the dorsal-lumbar region must be evaluated. This can be tested by challenge, of this region or a bilateral weakness of the lower trapezius muscle. This association is important because of the position of the entry of the esophagus into the stomach at the area of T10 through the esophageal hiatus and the attachment of the right and left crus of the diaphragm. The mechanical stress caused by this factor can lead to the heartburn or even gastric reflux.

The following illustrations with permission from David S. Walther, D.C., *AppliedKinesiologySynopsis 2nd Edition*. Illustration (12–32), (12–33), (12–34) and (2–58).

12–32 **Superior view of the diaphragmatic apertures.**

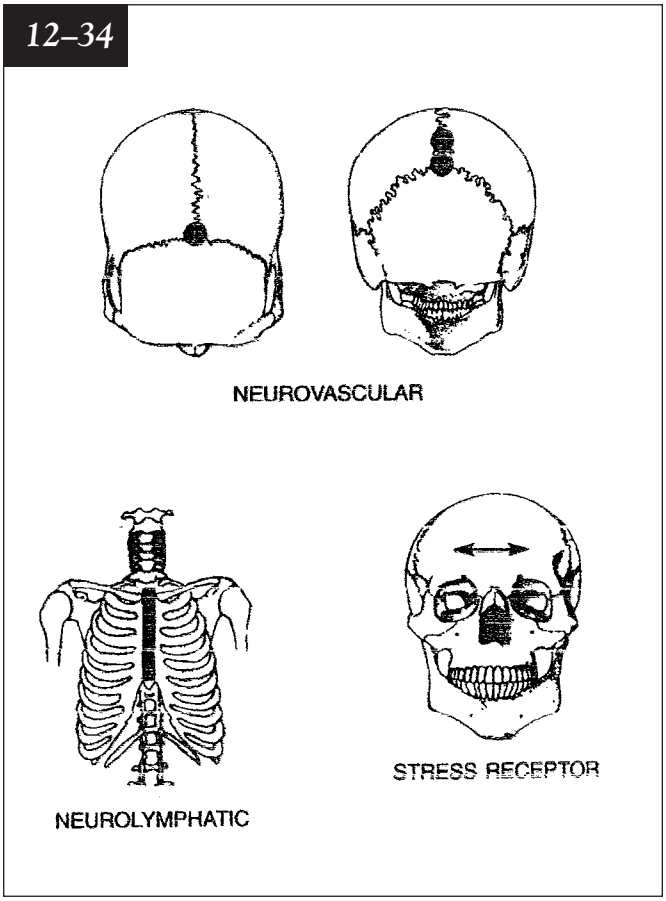


12–33 **Schematic of diaphragmatic nerve supply.**



Diaphragm Pattern: This is a factor that has come to light in the past five or six years because of Dr. Goodheart understanding of work of Dr. Fulford. The observation that about 70% of the population have a restriction of the ribcage when pressed along the lateral border in the mid-axillary line from the right to the left side. This finding was further correlated with passive ROM in the seated position with patient's feet resting on the floor. This group of patients would rotate the torso easily from the right to left but would show a restriction when rotated from left to the right.[16]

This would go back even further into a Japanese researcher Isogai who observation that people walk with a longer stride to one side.[17] Dr. Goodheart noted these observation to be correct and that this would cause problems that are know as dural torque. This finding can be challenged by placing the block under the femoral head on one side and the shoulder on the opposite side on a supine patient and testing a strong indicator muscle. The side of the over stride with the block under the femoral head will cause a weakness to the strong indicator muscle.[18]



This finding I correlated to the Yaw#2 pattern to the left, with the patient supine the pelvis is forward on the right.[19] Dr. Goodheart correlated this to a weakness that could show when using a psoas crura origin test developed by Dr. Alan Beardall only on the right side.[20] This pattern when carried forward would show as a hypertonic psoas on the right due to the muscle weakness pattern of the Yaw#2, with a weak psoas left and weak latissimus dorsi no the right.[21]

The weakness of the left psoas and the hypertonic right psoas will lead to a change in normal respiratory movement of the diaphragm also allowing for the resistance to movement of the ribcage seen from the right to the left side.[22] Add to this the factor that most patients over stride with the right leg, which creates the patterns, I have just described.

There seem to be a universality of over striding with the right leg this is due to dominance of right handedness that exists in 85% to 87% of the general population. The next factor that Dr. Goodheart had discovered was derived from the research of Dr. Fred Illi at National College that was presented in his monograph published in 1951.[23] This information formed the basis of what is know as PLUS Technique. This is an acronym for the muscles that weakened with the spine is flexed approximately 35° and it also occurs during extension of the spine. The muscles that weaken that PLUS stands for are the piriformis-right, latissimus dorsi-left, upper trapezius-left, and Sternocleidomastoid-right. This diagnostic test, that helps, to uncover hidden problems that may exist in weight bearing position. If these muscles don't weaken in flexion and extension seated or standing it simply mean other factors still exist that require correction that have been missed.[24]

The fact that the weakness of the piriformis and SCM only weaken of the right even in the left handed patient. I believe is why almost all patient seem to have the hypertonic psoas on the right and that yaw #2 will be found on the left with the pelvis forward on the right in the transverse plane. This observation seems to exist in 99% of the population if people or examined randomly. This observation I have followed for the past 24 years, but specially over the last six year with total number of patient being observed of over 15,000 patient visits.[25]

The ribcage restriction is TL with the thumb of one hand in the umbilicus and finger of the other on the linea alba just below the xiphoid process. This may require breath holding (B&H) technique to uncover. Dr. Goodheart has further refined the observation with B&H in full inspiration, full expiration and also in half inspiration and half expiration. This may be needed to get TL or to see the restriction in the rib cage when manually challenging for the right to the left.

Incorporation of this corrective technique, which I will briefly describe, will greatly help improve the symptoms of GERD. The correction is simply applying pressure with the thumb of one hand pressing it anterior to posterior with slight cephalad direction. With the other hand finger tips being pressed into abdominal obliques under the tenth rib. Dr. Goodheart suggests that the female patient is treated with the doctor on the

left side and the male patient is treated with the doctor on the right side applying pressure. The pressure is kept on in a steady force observation the relaxation and sinking in of the hand on the abdominal oblique becoming easier.

When the correction is completed the positive TL with is negated and a great improvement in the elasticity and flexibility can easily be observed. There will also be an increase in vital capacity of 15% to 20% the blood oxygenation will increase to the normal level of 98%.

Conclusion

While conventional treatment may be necessary on the short term basis, that is the use of antacids such as Tums or acid blocking drugs such as Nexium. This will allow the inflammation or ulceration of esophagus to heal on the long-term basis the continued use of these drugs will prevent the proper digestion of foods especially protein and will also interfere with the absorption of calcium.

The improper protein break down will also lead to antigen-antibody reactions which can lead to allergic reaction that may even be the cause of the heartburn or gastric reflux. By treating the structural problems and subluxation and nutritional support we can give much greater relief of the patient's symptoms and prevent the return of this problem. If the patient is over weight then a diet should also be instituted to gain better control of these symptoms.

Reported on the morning news TODAY PROGRAM 10/27/04 that the chronic use of antacids and acid blocking drug can lead to pneumonia because the acidity of the stomach acts to kill pathogenic bacteria that in reflux disorder can be inhaled from the regurgitation into the lungs. As I have said a point well taken.[28]

References

1. Gallup Organization-National Survey of Heartburn Across America, 1988.

2. Boyd, William, M.D.-Boyd's Pathology, Lippincott, Williams & Wilkins, 1992.

3. Beeson, Paul, B. M.D. & Mc Dermott, Walsh, M.D.-Textbook of Medicine, 11th Edition, W.B. Saunders Co., Phil. 1963-p. 875.

4. Gray, Henry-Anatomy of the Human Body, Lea & Rebiger, Phil., PA, 1965-p. 452.

5. Ibid, Gray, Henry-Anatomy of the Human Body, Lee & Rebiger, Phil, Pa, 1965-p. 450.

6. Spence, Alexander, P., Ph.D. & Mason, Elliott, B., Ph.D.-Human Anatomy and Physiology, 3rd Edition, Benjamin/Cummings Publishing Co., 1983-p. 627.

7. Beeson, Paul, B. M.D. & Mc Dermott, Walsh, M.D.-Textbook of Medicine, 11th Edition, W. B. Saunders Co., Phil 1963-p. 875.

8. Spence, Alexander, P., Ph.D. & Mason, Elliott, B., Ph.D.-Human Anatomy and Physiology, 3rd Edition, Benjamin/Cummings Publishing Co., 1983-p. 626.

9. Guyton, Arthur, C., M.D.,-Textbook Of Medical Physiology, 3rd Edition, W. B. Saunders Co, Phil 1966-p. 897.

10. Spence, Alexander, P., Ph.D. & Mason, Elliott, B., Ph.D.-Human Anatomy and Physiology, 3rd Edition, Benjamin/Cummings Publishing Co., 1983-p. 626.

11. Spence, Alexander, P., Ph.D. & Mason, Elliott, B., Ph.D.-Human Anatomy and Physiology, 3rd Edition, Benjamin/Cummings Publishing Co., 1983-p. 629.

12. Beeson, Paul, B. M.D. & Mc Dermott, Walsh, M.D.,-Textbook of Medicine, 11th Edition, W. B. Saunders Co., Phil 1962-p. 875.

13. Goodheart, George, J., D.C.- Applied Kinesiology Workshop Manual, Private Publication, 1972-p. 20.

14. Walther, David, S., D. C.-Applied Kinesiology, Synopsis, 2nd Edition, Systems DC, Pueblo, CO-2000-p. 577.

15. Walther, David, S., D.C.-Applied Kinesiology, Synopsis, 2nd Edition, Systems DC, Pueblo CO-2000-p. 581.

16. Goodheart, George, J. D.C.-The Approach to Understanding the Function of the Diaphragm-ICAK notes from summer meeting 2004-pp. 1–15.

17. Goodheart, George, J., D.C.-Applied Kinesiology Workshop Manual, Private Publication, 1982-pp. 24–30.

18. Goodheart, George, J., D.C-Applied Kinesiology Workshop Manual, Private Publication, 1983-pp. 1–39.

19. Sprieser, Paul, T., D.C.-Yaw #2 Muscular Pattern-Collected Paper of ICAK-winter 1982-p. 255.

20. Beardall, Allan, D.C.-Clinical Kinesiology Vol. #1-Lower Back and Abdomen, Private Publication-1980-p. 47.

21. Sprieser, Paul T., D.C.-The Relationship of Switching to the Yaw #2 of the PRY-T Technique, Collected Paper of ICAK, 2003-2004-pp. 243–247.

22. Goodheart, George, J., D.C.-Applied Kinesiology Workshop Manual, Private Publication 1983-pp. 1–39.

23. Illi, Fred, W., D.C.-The Vertebral Column-Life-Line Of The Body, National College Of Chiropractic, Chicago, Ill., 1951.

24. Goodheart, George, J., D.C.-Applied Kinesiology Workshop Manual, Private Publication-1988.

25. Sprieser, Paul T., D.C.-The Relationship of Switching to the Yaw #2 of the PRY-T Techniques, Collected Papers of ICAK, 2003–2004-pp. 234–247.

26. Goodheart, George, J., D.C.-Seminar and Notes for "HOW TO FIND IT & HOW TO FIX IT SEMINAR-PART II-Nov. 1 & 2, 2003.

27. Goodheart, George, J., D.C.-Seminar and Notes for "SEEING IS BELIEVING SEMINAR-April 3 & 4, 2004.

28. Laheij, Robert, Ph.D, Stricker, Bruno, M.D., Ph.D., and Jansen, Jan, M.D., Ph.D. Risk of Community-Acquired Pneumonia and Use of Gastric Acid-Suppressive Drugs, Journal of American Medical Association, Vol. 292, No.16, October 27, 2004, Pages 1955–1960.