INTERNATIONAL COLLEGE OF APPLIED KINESIOLOGY U.S.A.

Experimental Observations of Members of the ICAK

Volume 1, 2010-2011

Fifty-Second Collection of the Proceedings of the Annual Meeting

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

Experimental Observations of the Members of the ICAK

Volume I, 2010-2011

Proceedings of the Annual Meeting

Professional Applied Kinesiology™

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

Experimental Observations of the Members of the ICAK

Volume I, 2010-2011

Proceedings of the Annual Meeting

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Message from the Chairman

David Leaf, D.C., DIBAK

For 34 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. I would like to offer a special thanks to Drs. Allan Zatkin, David Engel, and Janet Calhoon for all their help during the review process, and to Dr. Bart Stark, our Publications Chair. We look forward to seeing you at the Annual Meeting, June 3-6, 2010 in Los Angeles, CA.

Introduction

This fifty second collection of papers from members of the International College of Applied Kinesiology®-U.S.A. contains 36 papers written 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 dialogue, creative thinking and critical review among its members; thus, review in this instance is not blinded. These papers are distributed only to the members of the ICAK-U.S.A. for general comment and 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 more in-depth study and scientific investigations, as well as spawn new areas of research. Such is to inspire 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) and the editor(s). The ICAK-U.S.A. disclaims any responsibility or liability for such material.

The current ICAK-U.S.A. Status Statement appears in *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. Whenever possible, all papers should be supported by statistical analyses, literary references, and/or any other data supporting the procedure.

The ***Proceedings of the ICAK-U.S.A***. is 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) Review comments on papers published in Division II. These particular submissions are intended for constructive review. Opinions or editorials with negative connotations only may be rejected.

Manuscripts are accepted by the ICAK-U.S.A. for publication with the understanding that they represent original unpublished work. Delivery of a manuscript to the ICAK-U.S.A. Central Office does not imply it will be published in the Proceedings. Manuscripts are reviewed by the Proceedings Review Committee and authors will be notified in a timely manner of their manuscripts acceptance or rejection. The author may appeal any paper rejected to a separate 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 any employer if the submission represents a "work for hire." Upon such submission, it is to be understood 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 agreement may result in the cancellation of the ICAK-U.S.A.'s consideration to publish.

Continuing call for papers includes:

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

Hypotheses—projections from previous observations 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 identification of ambiguities, and the delineation of areas that may constitute hypotheses for further study. Meta-analyses are 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 i.e., the comparative evaluation of two or more case reports.

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 with documentation that such procedures have been fully understood. Photographs or artistic likenesses of subjects are publishable only with their written consent or the consent of a legal guardian; the signed consent form, specifying any special conditions (e.g. eyes blocked off), must accompany manuscript.

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; other identifying information about a patient's personal history and characteristics should be disguised.

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 prepared 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.

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 requested to submit final manuscripts via email to icak@dci-kansascity.com or on computer disc (CD) to 6405 Metcalf Ave., Suite 503 Shawnee Mission, KS 66202. Each manuscript file should be titled with the author's last name and the manuscript tile. All manuscripts must be submitted in Microsoft Word.

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 and key words, 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, 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 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 (i.e., position papers, historical treatises).

Please visit the following link online for helpful information on structured abstracts: 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:
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, why it was done and what could be done to address shortcomings or gaps in what we have learned from what 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, 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 could 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 appendices.

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 the 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 alluded in this section; however, they are more formally presented in the section to follow.

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, as readers will 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 use the notation "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 more accurate since it is customarily peer-reviewed. 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 omitted: e.g., 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;16:24-5.

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 (e.g., 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 they are reproduced for publication all details will be clearly discernible; rough sketches with freehand or typed lettering are not encouraged. All illustrations should be submitted embedded in the manuscript document in the appropriate place.

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 a written permission letter from the copyright holder to reproduce the material. Permission is required, regardless of authorship or publisher, except for documents in the public domain*.

Public Domain:** Includes works with expired copyrights, US government work, or when author dedicates their work to public domain.*

Manuscript Submission Summary

Manuscript components

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

- Manuscript electronically via email of CD (The author should be sure to retain the original file in case of loss of the submission copies in transit.)
- Release form (signed by all authors, and by employer if study was a work for hire).
- Permission 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.

Submission Instructions

The manuscript should be emailed to the Central Office at icak@dci-kansascity.com. The Release Form should be completed and signed then fax to 913-384-5112 or mailed to:

The ICAK-U.S.A. Central Office
6405 Metcalf Ave, Suite 503
Shawnee Mission, KS 66202

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 J. 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 AK 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 (microavulsion 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 pre-conceived impressions regarding the test outcome
- Non-painful contacts -- non-painful 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 a 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 AK 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. Updated May, 2001.

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Division I

Informative Papers

What Your Brain Might Say if it Could Speak

Michael Allen, D.C., DIBAK, N.M., D.A.B.C.N., F.A.C.F.N.

Key Indexing Terms
Brain, Functional, Neurology, Aerobic, Diet, Exercise, Vitamins, Eating, Lifestyle, Health

Discussion

Developing good health is not only about nutrition. It is not all about pills one can take to feel better. The "pill for every ill" mindset that believes pills make everything work better can eventually lead to trouble further down the road of life. While proper nutrition is not something that just happens, neither is good health. Both require awareness and determination. Eating right and taking supplements are good practices, but there is another aspect that needs as much attention – neurological health. It is often taken for granted because few people (or doctors) know what to do about it. The nervous system is the controller of all body functions and that includes influencing how all the tissues use the nutrients they get. And when it comes to brain health, neurology is king!

Who Else Wants A Healthier Brain?

"Mobility": the sixth sense, equal to the other five. OK, well maybe movement is more a result of the senses than it is a sense in itself, but nonetheless movement brings sensation to the cortex. People do not think of their ability to move until it breaks down or their bones get too thin to bear their weight. Muscle soreness, fatigue, and various aches and pains can keep one sidelined sometimes for days at a time.

A recent radio talk show challenged me. The host asked, "If your brain could speak, what might it say?" While he talked about his agenda, I was coming up with all sorts of things I have "heard" patient's brains say. I came up with so many answers that I hardly knew where to start.

Oh, the ideas that went through my head. There were so many! When it came right down to it, three possible answers summed up the question of brain-speak, four total: *"I can't breathe!", "I can't take it anymore!", "I lost the words!",* and *"Stop the static!"*

The brain's primary goal is survival. It must endure and that requires available oxygen and plenty of energy in the form of glucose delivered to the tissues by the blood. In addition, the brain must have a specific type of nerve input to complete all its requirements to function properly and ensure survival. Meeting all the brain's needs makes it happy.

"I Can't Breathe!"

The brain contains the most oxygen-sensitive tissues in the whole body. It has to be able to breathe. But how does the brain get oxygen if not through the lungs? We will discuss the importance of getting blood to the brain and how that helps the brain to breathe. No other human tissue is as sensitive to its oxygen levels as the brain. Without access to available oxygen, the brain tissues deteriorate quickly.

The cerebellum and cortex need oxygen to meet the metabolic demands to process the myriad information that they receive. The cerebellum interprets input solely from muscles then sends its analysis to the thalamus, while the thalamus gets input from everywhere – muscles ligaments, joints, skin, etc. All the incoming signals that ultimately reach the brain blend in the thalamus and then get sent to the cortex to generate a motor response. The inputs from the cerebellum and thalamus have to work together, and without oxygen their functions break down quickly. They can do their work if their oxygen levels are functionally low but if oxygen levels are left too low for too long a period, their behaviors falter, hastening aging and senility, and – if the oxygen levels drop below normal levels even for only a few short minutes – death.

Without a doubt, a breathing brain needs the right nutrition. It also needs neurological input that helps the sensitive receptors read the blood's oxygen and carbon dioxide levels in strategic areas. One of these receptor-types is found in the neck. If the signals to it are chaotic, then the brain might get the wrong information making blood vessels be larger or smaller than they should be, and that can compromise the blood flow to the brain.

"I Can't Take It Anymore!"

When the brain says that it can't take it anymore, it speaks of the toxic insult of poor nutrition and incomplete elimination that hinders tissue quality and clogs neurological communication channels. Toxins build up for many reasons, but most of the time it is because of dietary or metabolic error, or from unnecessary drugs and medications.

When poisoning a brain, timing is everything. For centuries, scientists have said "the dose makes the poison," meaning any chemical can be toxic if you eat, drink or absorb too much of it.

Balanced tissues use nutrients as they should and unbalanced tissues use them incorrectly. While the first might represent the smooth flow of metabolic cycles from one point to another, the second might cause the accelerated use, or conversely, the backing up of those same metabolic cycles, and that leads to toxic buildup.

Consider that the metabolic channels are like rivers flowing through the body. Just as a river flows from one place to the next, metabolic products flow from one molecule into another until they are finally eliminated from the body. And just like a river, these metabolic flows can get dammed up creating a pooling upstream and a trickling downstream. This is another example of an increasing trend of toxicity.

A basic chiropractic tenant is that "too much or not enough nerve energy is disease." Well, too much or not enough of anything is not good. Even too much water can lead to death.

Tryptophan morphs into serotonin and then into melatonin in the presence of certain metabolic conditions, but it can also change into niacin given a different set of conditions. Those conditions depend

upon the type of stimulation, the metabolic need, and the presence (or absence) of essential components to drive the metabolism in a specific direction. The direction the metabolism flows is dependent upon many different factors.

As another example, “trans” (bad) fats change the nerve cell membrane, and that alters the way nerve cells transmit their signals. Changing a nerve cell's structure alters its ability to transmit a nerve signal and/or the way nutrients are able to get into or wastes get out of the cell. Therefore, trans fats hinder cellular performance, and those cells can suffer for a long time.

An article from the American Journal of Clinical Nutrition (*Can nutrient supplements modify brain function?)* found that chemistry and function of both the developing and mature brain are influenced by diet. As a result, investigators, physicians, and regulatory bodies have supported and encouraged the use of proper nutrition in the treatment of disease.

In order for it to work at a very high level of efficiency, the brain’s metabolic rate has to be quite high. And it takes the right amounts of its essential components.

Typically, if a nutrient works well for a specific condition, people are given more of it to hopefully get better results. People start taking the nutrient themselves when they have symptoms because of the benefit they received when they or someone they know took it when they had a similar condition.

Eventually, nutrients can be found in their purified state (which essentially means that their active ingredient is distilled or fractionated from the original molecule). The essential and more properly unknown factors that make the purified product do its job are removed. In many cases that same unknown factor are the same ingredients that modify the effects of the purified factor. What used to be a nutrient is now a purified substance – vitamin, mineral, cofactor, etc – which is even more potent than nature ever intended or than the body knows what to do with, and that can lead to unanticipated adverse effects.

Self medication is a big aspect of many people’s symptoms because they think they know what they are doing for themselves but they actually have no way of knowing what they are doing unless they have symptoms. Actually, most people are unaware that many of their supposed symptoms are secondary to their naked nutrients, and they seek professional care for what could be managed by simply changing their diet away from the fractionated supplements and back to supplying their body with the essential nutrients that had been taken away.

The brain has a very high metabolic rate. It consumes more glucose and oxygen than any other organ. Since any metabolic process generates wastes, the brain is especially susceptible to toxicity if the wastes are not removed quickly. Therefore, the brain’s environment must be clean for it to work at its highest level.

If toxins linger in the brain too long, they cause their own brain problems. Retained wastes and toxins clog up the system generating more wastes and toxins, and inefficient purging hampers toxin breakdown. The same is true when the nutrients that would otherwise control a metabolic path are removed or missing from foods or a vitamin’s potency increases because it has been purified via distillation or

Toxins that linger begin to stimulate their own metabolic paths that eventually lead to more complicated problems.

fractionation.

The liver stores many nutrients, and it is the major organ of detoxification. It processes metabolic wastes and pollutants from the blood that comes to it from all over the body. Generally, the liver has two detoxification phases. Phase one breaks toxins apart, and phase two combines the end products of the first phase with other molecules in such a way that they can easily be separated again for elimination. If the first phase of detoxification is successful but the second phase gets plugged up, the toxins from the first phase can get back into the system where their effects are often more toxic than the original products. So, it makes sense to carry the detoxification process through to completion.

The catecholamines are the "fight-or-flight" hormones that are released by the adrenal glands in response to stress. The most abundant catecholamines are epinephrine (adrenaline), norepinephrine (noradrenaline) and dopamine, all of which come from the amino acids phenylalanine and tyrosine. The opioid peptides mimic the effect of opiates in the brain. Opioid peptides may be produced by the body itself, for example endorphins, or be absorbed from partially digested food. Brain opioid peptide systems are known to play an important role in motivation, emotion, attachment behavior, the response to stress and pain, and the control of food intake.

A diet that is fiber-poor, mineral-deficient, and refined carbohydrate rich tends to cause liver congestion. Symptoms like skin problems, poor eyesight, hair loss and hemorrhoids can be related to a stagnant and congested liver.

The kidneys and skin eliminate toxins from the blood, too. In fact, the skin is the largest eliminative organ of the body and it is made up of the same basic tissue as the nervous system – surface ectoderm. Touching the skin causes immediate and profound nervous system changes. So too, keeping the skin clean does the same to the nervous system.

"I Lost the Words!"

Brain tissues speak in *"Neurologese"* - a complicated language that mixes neurotransmitters and neurochemicals as "words." The conversation from one part of the brain to another requires the right timing if they are to be "heard." Therefore, the "words" and the timing of their delivery keep the brain from getting confused. If the brain's "words" get confused, then the brain loses track of what it is saying and how it says it. A good analogy might be that your brain's natural dialect of neurologese changes to something foreign.

While neurotransmitters come from within the nerve cell itself, neurochemicals exist outside the cell, relaying, amplifying, and modifying signals between one neuron and another. Neurochemicals include neurotransmitters (and other molecules like neuroactive drugs, i.e., catecholamines and opioid peptides), making up the language of the brain.

Getting a neurotransmitter from one nerve cell to another is like watching horses race at the track. When that bell rings the horses take off for all they are worth in order to be the first one to cross the finish line. Neurotransmitters act in much the same way. When they are stimulated, off they go to be the first to reach the next nerve causing it to send its

signal. If the right neurotransmitter gets there first then the designed function is maintained, but if there is not enough of that neurotransmitter or if the wrong one gets there first the wrong thing can happen. That can cause confusion.

While we know certain drugs can change the way the brain works, it is actually because they change the neurotransmitters in the tissues. Scientists are finding that they can also influence brain function by managing the way the brain makes proteins from amino acids. A brain's diminished ability to make proteins affects the response to neuroactive drugs, while an increased protein synthesis increases the response to the drugs.

When the brain has trouble with its neurochemical balance (its "words"), chaos erupts. Neurochemical balance – their production and persistence – is dependent upon the right kind of nerve signals. As nerve signals change, so do the ways the neurochemicals are used and that changes the person and who they are. Neurotransmitters are influenced by physical stimulation and nutrition, leading to functional brain changes.

In a recent lecture tour to Russia, one doctor was surprised that eliminating static in the human nervous system eliminated the need for particular nutrients.

This illustrates how an imbalanced nervous system accelerates or slows the consumption of particular nutrients. There may be no need for supplementation if the nervous system works according to its original design, and/or balancing the nervous system makes those nutrients available again, further indicating that there may be no need for supplementation.

Stop the Static!"

The first three brain-speak topics deal with the brain's metabolic issues and nutritional needs. However, when it comes to the brain's function the scenario changes. The brain is dependent upon nerve signals from joints and muscles that are balanced, reciprocal and free from noise. Anything less than that is pathological.

The nerve signals that reach the spinal cord and brain must be free from interference. To be healthy and for their signals to be crystal clear, nerve signals must be static-free or at least the static noise must be minimal.

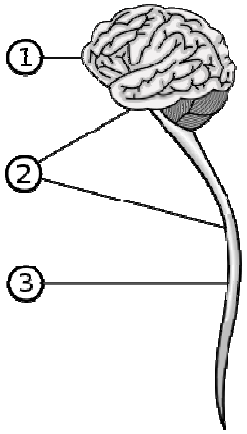
While the nutritional components of brain function cannot be denied, they provide the substrate for nervous system function, but the brain's primary stimulation comes from body structures rather than nutrients. Nutrients provide for the brain tissue performance but joint signal turns on the brain centers that are designed to control all areas below it. Sometimes that control is to make things happen and other times the controls keep things from happening. And other times the brain focuses performance by inhibiting a first signal so a subsequent signal can work.

Patterns of neurological conflict tend to be inherent to the structure. When one muscle works harder than another that supports the same function it can lead to imbalance. That structural dysfunction hinders the way the nerves send their signals, and that affects the brain; that is nerve static. Nerve static displays itself with inflammation, swelling, dysfunction, joint breakdown, and eventually pain. Static reveals its presence in other ways, too. Each static pattern can be understood by a doctor who practices applied

Your brain has to be aerobic in order to work at its optimal level. Anything less is pathology.

kinesiology as functional neurology.

Neurological static is always present. It is a product of our physical world. So in order for the nervous system to work according to its efficient design, the effects of the static must be purged or at least minimized. When static is involved with the production of pain, the static is called nociception (it is the neural process of encoding unconscious pain). A nociceptor is a sensory receptor that reacts to potentially damaging stimuli by sending nerve signals to the spinal cord and brain. Nociception is to the nervous system what inflammation is to immunity. Both are necessary to proper function but each should be controlled.



A diagram showing the Central Nervous System:
1. Brain
2. Central nervous system; Brain and Spinal Cord
3. Spinal cord

More visibly, neurological static displays itself in abnormal muscle function. It shows up in gait problems, postural distortions, or joint dysfunction and it jeopardizes the nervous system's stability; structural problems lead to neurological static. When muscles test weak when they should be strong and/or stay strong when they should be weak, that's neurologese for ***HELP!*** No wonder the brain wants to stop the static!

Summary

Simply providing oxygen to the lungs does not make your body use it as it should or the brain use it any better. Further, eating the right foods or giving the right vitamins, or taking the right drugs does not guarantee that your brain will work any better. The brain's environment has to be kept free of toxins. It also needs to be able to communicate within itself and with all other areas of the body as if it were done all at once. The central and peripheral nervous systems (the whole nervous system) are designed to keep all the internal communications together and categorized according to their original design. The trouble comes when these systems get out of step one with the other.

Detoxification is no assurance of tissue health if it is not done to completion. The conversion of one product into its metabolites needs to reach its natural end. If the process is interrupted before it is finished, the end result is often not good. The lingering byproducts are often more toxic than the original toxin. When they remain in the system they perpetuate the havoc.

Finally, the brain needs to be free from static interference. Its signals must be free to work according to their original design if the system is to be optimal. Persistent neurological static leads to brain confusion.

The human brain is highly dependent upon a fervent metabolism, a clean environment, the ability to communicate with itself and the rest of the body, and crisp, clean incoming and outgoing nerve signals. Its performance is only as good as how it processes the

signals that come to it. When the brain's needs are met - blood, oxygen, glucose, and a specific kind of nerve input - the nervous system functions according to its original design. When it works right, its tissues are strong, resistant, and resilient. A healthy brain leads to health and wellness, and a slowed biological clock. That is youthful health.

Surprise Cause of Recurring Problems

Janet Calhoon, D.C., DIBAK

Abstract

Objective

To describe the use of applied kinesiology in uncovering the cause of recurring cranial faults and other problems caused by restricted skull motion induced by spring loaded eyeglass frames.

Clinical Features

72 year old man presents with antalgic posture and low back pain.

Intervention and Outcome

Applied Kinesiology methods were utilized to diagnose and treat this patient with a successful outcome.

Conclusion

Applied Kinesiology methods and change of eyeglass frames were successful in resolving cranial faults and reducing low back pain.

Key Indexing Terms

Applied Kinesiology, External Frontal Fault, Nasosphenoidal Fault, Category 1 Pelvis, Fixation

Introduction

We have all had many experiences of fixing patients, observing the change in the patient's posture, and knowing the patient was better when they left our offices. Many of us have also had the occasional experience of seeing a patient return on the next visit, looking and feeling as though he/she had never been treated, a frustrating event for the patient and the doctor. It then becomes the doctor's responsibility to answer the question Dr. Goodheart taught us to ask, "Why is that?" This paper looks at one such case.

Discussion

A 73-year old man presented with a complaint of low back pain of four years duration. The MRI report stated, "No significant spinal stenosis. Moderate to severe right neural foramina narrowing present at L4-5 secondary to facet degeneration changes and broad-based disc bulge." His history was significant for asthma, benign essential hypertension, chronic renal failure, coronary artery disease, glucose intolerance and osteoarthritis.

Examination revealed an antalgic posture with thoracolumbar forward flexion of 45º. Thoracolumbar range of motion was: forward flexion 90º, extension 20º, left lateral flexion 30º and right lateral flexion 20º.

He showed a bilaterally positive Fabere Patrick Test. Left hip abduction was 55º and right hip abduction was 50º. His bicep, tricep and patellar reflexes were normal on the right and diminished on the left. His blood pressure seated was 144/88; supine 134/74 and erect 150/84. The systolic blood pressure measured on his left calf was 220 and 130 on his right calf.

Manual muscle testing demonstrated bilaterally inhibited lower trapezius muscles and bilaterally inhibited gluteus maximus muscles, indicators for thoracolumbar fixation and upper cervical fixation respectfully. Applied kinesiology evaluation also revealed a category 1 pelvis, a nasophenoidal fault and a right external frontal fault.

I observed that his glasses fit so snugly they indented his skull at both temple pieces. I instructed him to have the glasses adjusted prior to his next treatment.

On that initial visit, using Applied Kinesiology Techniques, I corrected the cranial faults, the category 1 pelvis, thoracolumbar fixation and upper cervical fixation. Post treatment his posture improved, the forward flexion was reduced to 15º and the systolic readings on his calves were 220 on the left and 180 on the right.

At his next visit, two weeks later, he said he was feeling “some better” with less back pain. However, his posture looked very much as it did when he presented initially to my office. Many of the same findings had returned, the upper cervical fix, the nasophenoidal fault, the external frontal fault and the category 1 pelvis. I again noticed how the temple pieces on his glasses made deeply indented marks on his head. He commented that he had not found the time to have them adjusted.

Using Applied Kinesiology Techniques, I corrected again the right external frontal fault, the category 1 pelvis, the thoracolumbar fix and the upper cervical fix. His posture improved, his low back pain diminished and he generally felt better.

One week later, I saw him the third time. Again he had not had his glasses adjusted. Standing, his forward lean had increased to 55º, he complained his low back pain was worse with walking. I found a thoracolumbar fix, an upper cervical fix and a right external frontal fault. After treatment, I demonstrated to the patient that everything was fixed. I asked him to put on his glasses, and I was able to show him everything we fixed returned.

When I looked at his glasses, I saw the temple pieces were attached with springs to give a very snug fit and maintain constant pressure against his skull. The patient was very satisfied with his glasses because they didn’t slide down his nose, a problem he had experienced previously. I emphatically recommended that he obtain a different frame before his next visit.

Three weeks later he returned; his forward flexion on presentation that day was 20º. He was wearing new glasses and the frames were not spring loaded. I treated him that day, asked him to put his glasses on and everything stayed fixed. He is an older gentleman with some chronic health issues. However, from the time he got new glasses, we were able to help him progress forward each visit to his satisfaction as well as mine.

Conclusion

After I saw the effect of spring loaded eye glass frames on this gentleman I had several other patients present with similar frames. They have, at least in my area, become very popular because it solves the problem of glasses sliding down the nose. However, they cause a host of other problems, each set unique to the individual wearing them. For each patient, it was impossible to maintain a correction and have the patient progress forward, until the frames were changed.

We all remember Dr. Goodheart teaching us to examine patients in the postures in which they live and work. He jokingly, yet seriously, said the patient doesn't spend his/her day on a chiropractic table. Many of our patients do wear glasses all their awake hours. It is important for us to take glasses into consideration when observing patients, as well as tight head bands, shoes and other apparel. Consider your patient's environment as he/she functions each day. When something seems not quite right, or you see the same thing repeatedly, listen to Dr. Goodheart asking, "Why is that?"

References

1. Schmitt, Walter H. and McCord, Kerry M., Quintessential Applications, A(K) Clinical Protocol, 2nd Edition, 2009, HealthWorks!, P.O. Box 530186, St. Petersburg, FL 33747, pp. 10, 11, A25, 28, A26

2. Walther, David S., Applied Kinesiology, volume 1, 2009 ICAK-U.S.A., Shawnee Mission, KS 66202, pp. 85-90

3. Walther, David S., Applied Kinesiology, volume II, 2009 ICAK-U.S.A., Shawnee Mission, KS 66202, pp 184-186, 211-213

4. Walther, David S., Applied Kinesiology, Synopsis, 2nd Edition, 2009, ICAK-U.S.A. Shawnee Mission, KS 66202, pp. 110-112, 394, 395

Association of Lower Iliac Fixation with a Bilateral Inhibition of the Upper Trapezius Muscle

Eugene Charles, D.C., DIBAK

Abstract

Clinical observation that a fixation of the lower sacroiliac joint can result in bilateral inhibition of the upper trapezius muscles. If replicated, lower sacroliliac fixation can be added to the previous nine spinal fixations and the upper trapezius included as one of the fixation muscles.

Key Indexing Terms

Applied Kinesiology, Manual Muscle Testing, Lower Sacroiliac Joint, Spinal Fixations, Upper Trapezius

Introduction

It has been clinically observed that fixations of the spine, including the occiput, sacrum and iliac cause inhibitions of specific muscles, usually in a bilateral pattern.1 Presented is a previously unmentioned bilateral inhibition of the upper trapezius muscle as a consequence of a fixation at the lower sacroiliac joint.

Discussion

An established patient continued to present with chronic neck pain and a corresponding bilateral inhibition of the upper trapezius muscles. Previous attempts to facilitate the muscle were unsuccessful and/or short-lived.

Realizing that other bilateral inhibitions have a related spinal fixation, I reasoned that possibly this patient's persistent upper trapezius inhibition pattern was resultant of a fixation, perhaps as a variant of other established fixations.

The patient proceeded to therapy localize the spine in hopes of negating the inhibition. When this was fruitless and therapy localizing the sacroiliac joint at the usual posterior superior iliac spine/sacral area (usually resulting in an inhibition of the upper trapezius' neighbor, the nearby neck extensor) I entertained the possibility that a fixation of the lower sacroiliac joint (lower sacral ala and the posterior inferior iliac spine.) could be the causative agent.

Therapy localization over the lower SI joint dramatically facilitated his upper trapezius bilaterally. After challenging the joint for direction a two handed thrust to separate the

joint was performed. Manual muscle testing demonstrated facilitation of the previously inhibited muscles.

Conclusions

In addition to the previously documented nine fixation patterns and their resultant muscle inhibition patterns, I propose adding that a lower iliac fixation can result in a bilateral inhibition of the upper trapezius muscles.

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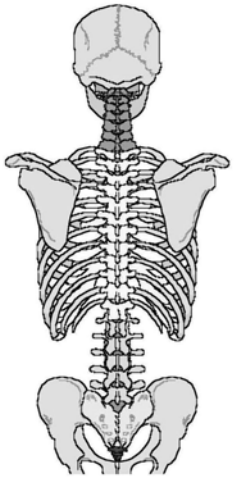
TREATMENT - Physical

- Occiput fixation: Bilateral Psoas
- Upper Cervical: Bilateral Gluteus Maximus
- Mid-cervical: Bilateral Popliteus
- Cervical-thoracic junction: Bilateral Deltoid
- Thoracic: Bilateral Teres Major



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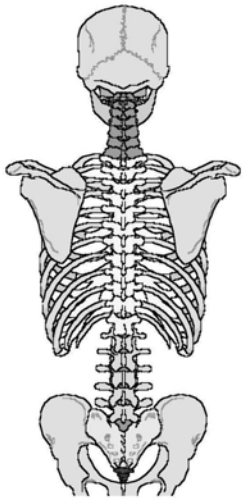
TREATMENT - Physical

- Thoraco-lumbar junction: Bilateral Lower Trapezius
- Lumbar: Bilateral neck extensors testes together
- Iliac Fixation: Unilateral neck extensors
- Sacral fixation: Bilateral neck extensors
- Lower Iliac Fixation: Bilateral Upper Trapezius



References

1. Illustration from AK The First Visit Tying Together The 100 Hour Course With Advanced Techniques.

2. Goodheart, George The Law Of The Ligaments, The Digest of Chiropractic Economics November/December 1973.

3. Charles, Eugene AK The First Visit Tying Together The 100 Hour Course with Advanced Techniques. Charles Seminars.

Ileofemoral Ligament Technique

Kenneth Feder, D.C., DIBAK

Abstract

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

Anatomy

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

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

Structure

Arising from the anterior iliac spine and the rim of the acetabulum, the ileofemoral ligament spreads obliquely downwards and lateralwards to the intertrochanteric line on the anterior side of the femoral head. It is divided into two parts or bands which act differently. The transverse part above is strong and runs parallel to the axis of the femoral neck. The descending part below is weaker and runs parallel to the femoral shaft. As the lateral portion is twisted like a screw, the two parts together take the form of an inverted "Y". It is intimately connected with the joint capsule, and serves to strengthen the joint by resisting hyperextension. Its upper band is sometimes named the iliotrochanteric ligament. Between the two bands is a thinner part of the capsule. In some cases there is no division and the ligament spreads out into a flat triangular band which is attached to the whole length of the intertrochanteric line.

Procedure

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

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

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

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

Correction

1. The patient is supine.

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

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

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

5. Raw bone used as nutritional support.

Alternative Correction

1. Patient is in standing position.

2. Contact involved ileofemoral ligament.

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

Conclusion

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

References

1. Goss, M. Gray's Anatomy, Lea & Febiger, Twenty Ninth American Edition.

2. Hertlins and Kessler. Management of Common Musculoskeletal Disorders, Physical Therapy Principles & Methods, 2nd Edition. J. B. Lippincott

3. Schafer, R.C. Clinical Biomechanics, Musculoskeletal Actions & Reactions, Baltimore, Williams & Wilkins, 1983

4. Sewell, M. E. "Ligaments involved in Lumbosacral Sprain and Their Conservative Management." ACA Journal of Chiropractic, 1970, E, S 36-39.

Methylxanthine Toxicity Syndrome

Michael P. Lebowitz, D.C. and Ami D. Kapadia, M.D.

Abstract

The methylxanthine family of compounds consists of caffeine, as well as theobromine, theophylline and paraxanthine. Caffeine is the most consumed stimulant in the world, and thus could have far-reaching effects on health. Although the beneficial as well as detrimental effects of caffeine have been long-debated, some well-defined short and long-term deleterious effects of this stimulant are clearly evident. Through Applied Kinesiology, it is possible to identify those individuals whose health is being compromised by consumption of any members of the methylxanthine family. Once this problem is identified, a specific diet along with nutrients can be prescribed to help the patient detoxify and achieve improved health by avoiding these substances.

Key Indexing Terms
Methylxanthine(S), Caffeine, Theobromine, Theophylline, Paraxanthine, Dimethylxanthine, Trimethylxanthine, Green Tea, Tea, Coffee, Cola, Chocolate, Cocoa

Introduction

Caffeine, Paraxanthine, Theobromine and Theophylline

Caffeine is the most consumed, socially-acceptable stimulant in the world. Approximately 90% of adults in the world consume caffeine in their daily diet. More than 150 million people in the US drink coffee on a regular basis, averaging 2 cups a day, which is the equivalent of 280 mg/day of caffeine. (1)

Caffeine, as well as theobromine, paraxanthine and theophylline, are part of the methylxanthine family and can be labeled as psychoactive stimulants. These substances in varying amounts and complexes are found in coffee, tea, chocolate, cola, yerba mate and guarana. (2)

Coffee contains caffeine and theophylline, but no theobromine, while tea and chocolate are higher in theobromine. Tea actually contains more caffeine then coffee but since it is brewed weaker, the average cup of tea has less than the average cup of coffee.

The prevalence of coffee shops and promotion of chocolate as a “health food” in our society and worldwide may be a result of the high addiction rate that humans have towards methylxanthines.

Caffeine Biochemistry and Pharmacokinetics

Caffeine is metabolized in phase 1 liver detoxification by the cytochrome P450 oxidase enzyme system (the 1A2 isozyme) into the following compounds with approximate percentages: paraxanthine (84%), theobromine (12%) and theophylline (4%).

Caffeine is readily absorbed in the GI tract after oral administration. It's bioavailability is almost 100% through oral administration.(3) The average half life of caffeine is 5 hours, with a range of 3-7 hours.(4) Defects in the CYP1A2 enzyme can be associated with impaired caffeine metabolism and a prolonged half life. (5) There are also genetic polymorphisms in the CYP1A2 pathway that could explain some of the varying effects of caffeine on different individuals. For example, a study of 120 healthy volunteers found that CYP1A2 activity, gender, and smoking influenced whether or not individuals experiences toxic effects of caffeine. Females and nonsmokers who had experienced toxic effects of caffeine were found to have lower CYP1A2 activity compared to females and nonsmokers who did not experience toxicity symptoms. (6)

Once it is absorbed through the GI tract, and enters the bloodstream, caffeine's main effects occur through its action as an antagonist of adenosine receptors (blocks adenosine receptors) in the central and peripheral nervous systems. The caffeine molecule is structurally similar to adenosine, and binds to adenosine receptors on the surface of cells without activating them. Caffeine acts as a competitive inhibitor of adenosine and results in stimulation of excitatory neurotransmitters. (4)

Symptoms associated with too much caffeine (too much ingested or impaired breakdown of it) include: headache, anxiety (including generalized anxiety disorder), depression, panic attacks, tremors, insomnia, nervousness, irritability, muscle twitching, chronic or acute pain,and GERD. (2)(7) Both acute and chronic ingestion of caffeine influences mood and cognition. (2)(7) In addition, heavy coffee (>2 cups/day) intake may trigger coronary and arrhythmic events in susceptible individuals. (8)(9) Finally, it has been shown that excess caffeine consumption (>200 mg/day) during pregnancy may increase the risk of miscarriage. (10)

The other related methylxanthines

Theobromine

While theobromine and caffeine are similar, theobromine is weaker in both its inhibition of cyclic nucleotide phosphodiesterases and its antagonism of adenosine receptors. Therefore, it can be postulated that theobromine may have a lesser, but still significant, impact on the human central nervous system. While theobromine is not as addictive as caffeine, it may contribute to chocolate addiction.

Theophylline
In susceptible individuals, theophylline can cause nausea, diarrhea, increase in heart rate, arrhythmias, and CNS excitation with resultant headaches, insomnia, irritability, dizziness and lightheadedness.

Paraxanthine
Paraxanthine is not produced by plants and is only observed as a metabolite of caffeine in animals. After caffeine intake, approximately 84% of the original compound is demethylated at the 3-position to yield paraxanthine, making paraxanthine the chief metabolite of caffeine.

Testing

On food sensitivity testing via Applied Kinesiology, I find chocolate, coffee and green tea show up (give an abnormal muscle test response) only on occasion: 10%, 5% and 2%, respectively. I know that caffeine is not great for burned out adrenals, which are so common in our society; so where does the truth lie?

AK as well as caffeine biochemistry have yielded some interesting answers. **Just like with solanines, you are getting many false negatives by not testing the chemical components directly.**

Because caffeine is metabolized into theobromine, paraxanthine and theophylline, any abnormal muscle test (hypo or hypertonic) to any of the substances would implicate all of the substances as possible sources. For example, a negative response to caffeine but a positive response to theobromine would implicate caffeine also, since the caffeine in coffee is partially metabolized into theobromine after ingestion. The metabolism of caffeine to theobromine may be fully functional, but the breakdown of theobromine at the next step may be impaired. This could result in the build up of theobromine in the system with resulting problems, despite no direct theobromine consumption.

I have found in some people, only one of the four substances either causes a strong muscle to weaken or a weak muscle to become hypertonic (pectoralis sternal and subscapularis are preferred due to their relationship to the liver and heart). Either weakening of a strong muscle or a muscle becoming hypertonic is considered to be a positive test (problematic response). A positive response can be due to overconsumption or hypersensitivity or a compromised phase 1 detoxification system. We have found about 40% our patients exhibit a positive test (hypo or hyper) to at least one of the 4 substances. In about 90% of these patients, the test was normalized by the herb phyllanthus fraternus, reishi, carbonized bamboo, or certain B-vitamin complexes. In many of these cases, avoiding coffee, tea, yerba mate, chocolate, guarana, acai and cola for 1 month while taking the appropriate remedies normalizes the test and the person can re-introduce the “foods” in moderation after that. They may or may not have to stay on the supplementation. A small percentage of people cannot re-introduce methylxanthines and need long term avoidance. People that refuse to avoid the foods should take maintenance doses of the appropriate remedies.

Personal Observations

The most common symptoms I see are locked up joints ("I woke up and my neck won't turn despite no trauma"), being prone to musculoskeletal injuries, sleeplessness, anxiety, cardiac symptoms (palpitations etc), adrenal weakness (mid afternoon fatigue, postural hypotension, etc), hemorrhoids and varicose veins.

Conclusions

The methylxanthine family of compounds is a common source of musculoskeletal and endocrine complaints. Applied Kinesiological testing with the actual methylxanthines avoids many of the false negatives found by just testing the "food" substance.

References

1. Kabagambe EK, Wellons MF. Benefits and Risks of Caffeine and Caffeinated Beverages. http://utdol.com. 2009.

2. Davidek, J, editor. Natural Toxic Compounds of Foods, Formation and Change During Food Processing and Storage. Boca Raton, Florida: CRC Press; 1995.

3. Fredholm BB, Battig K, Holmen J, Nehlig A, Zvartau EE. Actions of Caffeine in the Brain with Special Reference to Factors that Contribute to its Widespread use. Pharmacol Rev 1999; 51; 83-133

4. Giardina E. Cardiovascular Effects of Caffeine. http://utdol.com. 2009.

5. Cornelis MC, El-SOhemy A, Kaagambe EK, Campos H. Coffee, CYP1A2 Genotype, and Risk of Myocardial Infarction. JAMA 2006; 295: 1135-1141.

6. Carrillo JA, Benitez J. CYP1A2 activity, Gender and Smoking, as Variables Influencing the Toxicity of Caffeine. Br J Clin Pharmacol 1996; 41: 605-608.

7. Broderick P, Benjamin AB. Caffeine and Psychiatric Symptoms: A Review. J Okla State Med Assoc. 2004; 97 (12): 538-42.

8. Cannon ME, Cooke CT, McCarthy JS. Caffeine-Induced Cardiac Arrhythmia: An Unrecognized Danger of Healthfood Products. Med J Aust 2001; 174:520-1.

9. Chopra A, Morrison L. Resolution of Caffeine-Induced Complex Dsrhythmia with Procainamide Therapy. J Emerg Med 1995; 13:113-117.

10.Weng X, Odouli R, D-K Li. Maternal Caffeine Consumption During Pregnancy and the Risk of Miscarriage: A Prospective Cohort Study. A J Obstet Gynecol 2008; 198: 279.e1-279.e8.

Solanine Toxicity Syndrome

Michael P. Lebowitz, D.C. and Ami D. Kapadia, M.D.

Abstract

Alpha-solanine is a chemical compound/toxin found in the *Solanaceae* or nightshade plant family. The members of this plant family that are consumed by humans include: tomatoes, potatoes, eggplant, peppers, paprika, tobacco, gogi berries and ashwagandha. Through the research of a horticulturist named Dr. Norman Childers and others, it has been shown that alpha-solanine can have many detrimental effects on the health of humans. Through Applied Kinesiology testing, it is possible to determine which individuals are affected by this toxin. It is then possible to prescribe a specific type of diet along with certain nutrients to help rid the body of accumulated solanine in an effort to re-establish health.

Key Indexing Terms
Solanine, Alpha Solanine, Glycoalkaloid, Solanaceae, Nightshade(S), Norman Childers, Arthritis

Introduction

In the 1950's, Norman F. Childers, a professor of horticulture at Rutgers University, began to investigate the relationship between nightshade plants and arthritic as well as other medical conditions. His work involved examining naturally occurring toxins in food plants. Childers specific interest in the nightshade group of plants stemmed from his own experience with consumption of members of the *Solanaceae* family, or nightshade family of plants. This group of plants includes tomatoes, potatoes, eggplant, peppers (all except black and white pepper), paprika, gogi berries and tobacco, as well as other plants that are not generally part of the human diet. Childers noticed that his diverticulitis symptoms, and later his arthritis pain, disappeared when he avoided the nightshade foods. After some of his colleagues and acquaintances had similar relief of chronic symptoms by following the "nightshade-free" diet, Childers decided to recruit volunteers across the country to try his diet through small recruitment advertisements. In 1977, Childers and one of his students, Gerald M. Russo, published the first edition of *The Nightshades and Health*, which describes this association between nightshades and various chronic health ailments such as structural and arthritic type conditions. Childers includes numerous abstracts in his book that describe the effects of nightshade plant consumption on livestock, such as: gait abnormalities, weakness, osteopetrosis, calcinosis and arterial calcification. He also includes several detailed case reports from the human correspondents who followed his prescribed diet, and their experiences involving relief of chronic ailments. (1)

The compound/chemical thought to be responsible for the potential harmful effects of plants in the *Solanaceae* family is alpha-solanine. As discussed above, the members of this plant family that might practically affect us (much of this family is not consumed by humans) are tomatoes, potatoes, eggplant, peppers (all except black and white pepper), paprika, tobacco, gogi berries and ashwagandha. The amount of solanine present in the above foods varies tremendously depending on growing conditions, time harvested, storage conditions, cooking techniques, etc. Much of the academic work can be credited to Dr. Norman Childers as described above, who has been researching nightshades for about 50 years, in farm animals, as well as through dietary modification techniques with human correspondents across the country.

Historically, most solanine containing foods were not considered edible before the 1800's (except in some parts of South America). In fact, as late as the 1850's, most Americans considered potato as a food for animals rather than humans. The Farmer's Manual from this time period recommended that potatoes, "be grown near the hog pens as a convenience towards feeding the hogs."(2) Even foods like kim-chee did not have peppers in them 100 years ago but just utilized a salt brine. Solanine containing foods were mainly used for witchcraft a few hundred years ago, not regular ingestion. Now it is rare for people to go a day or even a meal without some form of tomatoes, potatoes, peppers, etc.

Solanines are not water soluble, are not destroyed by cooking and are not broken down inside the body but must be excreted as alpha-solanine. (1) (2) Different people have different degrees of sensitivity to them, and different efficiencies in being able to excrete them. How or in what way they will affect you will be a matter of genetics, as well as lifestyle and nutritional status. If you test positive for this problem, the probability is very high that at least one of your parents will have the same condition. The average daily intake of alpha-solanine is approximately 13mg and the average daily excretion is 5% the first day and 1-2% daily thereafter, with a half-life of about 1-2 months. Considering that is for one day's dose, it is estimated that the average body burden is at least 50mg.(2) It can be much higher in people who consume large amounts of these foods on a daily basis. There has been no definite established "safe" or "toxic" level of solanine consumption because of the variations in individual sensitivity and capability to excrete this chemical.(1) Alpha-solanine is stored in most organs (with a special affinity for the thyroid gland) as well as most soft tissue including skeletal muscle. (1) (2)

Alpha-solanine is classified as a neuro-toxin. Interestingly most "foods" that contain alpha-solanine also contain at least 5 other neurotoxins including atropine and nicotine. Acute solanine poisoning can happen from ingesting green or sprouted potatoes or green tomatoes, with symptoms including: cramps, nausea, diarrhea, headache, dizziness and sleepiness. (1) (2)

We are more concerned with "chronic poisoning", which we are calling Solanine Toxicity Syndrome, or STS. Solanine acts as an acetyl cholinesterase (AChE) inhibitor (similar to Malathion, Parathion and other "nerve gases"), allowing acetylcholine to build up in the synapses. (2) Acetylcholine (Ach) is a chemical neurotransmitter that is released

by neurons in the peripheral and central nervous systems. AChE is the enzyme that breaks down ACh, resulting in the subsequent detachment of this neurotransmitter from the post-synaptic neuron. However, solanine inhibits AChE, which effectively results in continued attachment of ACh to the post-synaptic neuron. This continued attachment in turn leads to disruption of normal nerve impulse transmission.(2)

Most of the studies involving the ill-effects of chronic solanine ingestion involve animals. There have been numerous incidences of poisonings in cattle, pigs, sheep and goats feeding on shoots of the tomato, on potato vines, potato peelings and sprouts, and plant tops of several wild species of the nightshade plant family. (1)

However, Childers did record the case histories and results that a nightshade free diet had on over 400 correspondents that chose to try his diet in an attempt to relieve their chronic arthritis. The following is a summary of the report issued by Dr. Childers and one of his colleagues:
-Total of 434 correspondents who returned questionnaire issued related to diagnosis of arthritis and their willingness to attempt elimination of nightshades as a form of treatment
-Of those rigidly on the diet, 94% had complete or substantial relief of symptoms
-Of dieters with occasional “slip”, 50% had complete or substantial relief of symptoms
-Overall, 68% had complete or substantial relief

Childers’ case histories do illustrate the fact that transgressions in the diet can often result in recurrence of symptoms.

In addition to resulting in osteoarthritis, on a practical level, it is theorized that nightshades can do the following in sensitive patients: 1) act as an endocrine disruptor especially to the thyroid, 2) cause chronic joint pain, all forms of arthritis, joint inflammation (this is due to solanine’s ability to remove calcium from the bones and deposit it in any weak or genetically predisposed area of the body), 3) for the same reason it can be a major contributor to osteoporosis (since it removes calcium from the bones) and arteriosclerosis (it can deposit the calcium in the blood vessels), 4) “leaky gut” as well as IBS, 5) appendicitis, 6) birth defects including spina bifida, 7) depression, 8) migraines, 9) can greatly interfere with calcium and vitamin D absorption, despite supplementation. Regarding interference with calcium/vitamin D absorption and osteoporosis, one hypothesis involves the possibility that solanine may be used by the body in place of the active form of vitamin D3, thus resulting in altered bone growth. (2)

Testing

In muscle testing we are only as good as taking a great history, knowing what to test and how to interpret the test, testing accurately and without bias, having top quality test vials (if applicable), and top quality therapeutic remedies (if applicable). In the case of solanines, I found many people who benefit from nightshade avoidance did not show muscle inhibition when tested on individual nightshade foods.

STS appears to affect about 1/3rd of the chronic patient load of my practice and other doctors with whom I have shared this technique.

Again most of the patients that "suffer" from STS do not routinely weaken on manual muscle testing of tomato, potato, peppers, etc. They may weaken on them sporadically but not each one and not every time, so it is very easy to miss the problem.
The extent of the problem became obvious when I was able to obtain a vial of alpha solanine.

At this point I am screening all patients for STS, because recognizing it can really help your patients' health both short term and long term. STS can be a major causative factor in a significant number of your patients and I have seen numerous resolutions of chronic conditions upon finding and treating it.

1. Test your patient on oral insalivation of homeopathic alpha solanine (1x) . It may cause universal muscle weakness or only weaken when the patient is therapy localizing an area of chronic subluxations or pain. Sometimes it causes universal muscle hypertonicity (muscles will not turn off when approximating spindle cells).

2. Check homeopathic acetylcholine orally in the same fashion. You don't need the acetylcholine to confirm- the solanine will do by itself.

3. Also, if a patient is on calcium or vitamin D supplementation, see if they no longer strengthen on them if you test solanine simultaneously. This is most often the case, and the supplements will typically yield little or no results on that patient, unless the solanines are removed from their diet.

4. Often patients' positive on any of the above steps will not weaken on individual solanine foods (though they need to avoid them).

5. If step 1 is positive, see if it is negated by Thera Supreme (Mid American Marketing 1-800-922-1744). This will negate it in about 80% of cases. In the vast majority of cases, no other supplements will negate it and neither will any reflexes, acupuncture points, adjustments or desensitization procedures. Thera Supreme is a supplement I formulated and later modified for the solanine sensitive patient.

6. Have the patient stay off nightshades (potato, tomato, eggplant, pepper, paprika, tobacco, ashwagandha, gogi) until alpha solanine no longer tests positive. This could take weeks or months or could be permanent. They should read labels carefully and have 100% avoidance for optimal results (if a label says "spices" and doesn't say what kind assume it has paprika or red pepper). Once alpha solanine no longer tests positive (on subsequent visits), if the patient wants to, you can have them add some nightshades back into their diet for 3 straight days and then retest. If alpha solanine continues to test negative- they can eat the foods in moderation but keep re-checking each visit. If

positive- it is probably permanent. I have found (in almost all cases) that if alpha solanine tests positive after a few weeks of avoidance that careful questioning of the patient will reveal that compliance has not been 100%.

7. Thera Supreme appears to help rid the body of solanine. We give 3 scoops a day for the first 2 bottles and then decrease to 1 scoop daily. The best I can theorize is that the Thera helps the body get rid of stored solanines faster. It does not fix the problem but allows symptoms to resolve faster. On patients that can't be compliant with the diet, it lessens (though does not eliminate) the effects of solanine.

Personal Observation

I had the opportunity to test some professional ballplayers that needed "Tommy John" surgery and they all showed STS and I personally think that STS made them more injury prone. I have seen it be positive on almost all arthritics as well.

In one patient, correcting STS resulted in alleviation of strong suicidal tendencies.
On other patients, it has eliminated post surgical wrist swelling in one, greatly decreased disabling shoulder and neck pain in another (this pain was unresponsive to multiple surgical interventions), chronic bilateral knee pain scheduled for 2 surgeries was eliminated, a juvenile RA patient became asymptomatic, etc. The results have been exciting but in some ways I think the results are as important in asymptomatic patients as it can help prevent arteriosclerosis, osteoporosis and many other conditions in certain patients (though of course you can't prove that).

Our new 2009 DVD goes into this technique in more detail as well as all the other parts of our protocols to treat "difficult patients". You may also want to read more about solanines in books by Norman Childers or Michael Fowler.

Conclusions

Solanine toxicity is an important finding previously missed in most Applied Kinesiological exams that should be screened on almost every patient with chronic health problems. Avoidance of solanine containing foods is a key variable in chronic pain, subluxations, arthritides, etc.

References

1. Childers, NF, Russo, GM. The Nightshades and Health. 1st Ed. Somerville, New Jersey: Somerset Press, Inc.; 1977.

2. Fowler, M. Nightshade Free Pain Free! Michael Fowler; 2007.

3. Lebowitz M, Wilson J. Correcting Chronic Health Problems DVD set. 2009.

4. Lebowitz M. Biomagnetic Kinesiology Testing. Collected Papers of the International College of Applied Kinesiology-U.S.A. Vol. 1. 1991-1992.

Hearing Loss Post Meniere's Syndrome and Hearing Recovery - A Case Study

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Abstract

A case study of the hearing problem medically diagnosed as profound and severe loss with the treatment plan of cochlea implants to restore hearing loss. The utilization of Applied Kinesiology diagnostic and treatment techniques in this case, which led to normal restoration of hearing.

Introduction

Meniere's Disease or Syndrome is a disorder characterized by recurrent prostrating vertigo, sensory hearing loss and tinnitus, associated with generalized dilation of the membranous labyrinth (endolymphatic hydrops). The etiology is unknown with the pathophysiology poorly understood. The attacks appear suddenly and last from a few minutes to 24 hours and gradually subside.

The attacks have nausea and vomiting as one of the symptoms along with a feeling of fullness or pressure in the affected ear. The hearing in the affected ear tends to fluctuate and usually worsens over the years, and tinnitus usually worsens after an attack of vertigo. It usually effects one ear only, with both ear being involved in about 10% to 15% of patients.[1]

The National Institute of Health (NIT) states the Meniere's disease is associated with changes in fluid volume within the portion of the inner ear know as the labyrinth. It can cause the membranous labyrinth to balloon or dilate, which is known as endolymphatic hydrops. Several possible causes that are being evaluated are noise pollution and viral infections, as well as biological factors.[2,3]

Case History

Patient (MB) is currently a 60-year old female, who was first evaluated in 7/26/05 for severe hearing loss. MB is a first cousin that was relating her hearing loss problems at family reunion in the Pocono, PA in 2005. She stated that her hearing had deteriorated so badly that she now had to use hearing aid in both ears and she was also resorting to lip reading to understand what people were saying.

Patient stated the hearing problem started about 12 years before with seasonal allergies

and sinus congestion with more problems in the right ear. She also had a history of Meniere's Disease in the right ear with the initial occurrence in 1986.

Other factors she had were hypoglycemia diagnosed with 6-hour glucose tolerance test in her early adult life. She was diagnosed with a gastric ulcer in her thirties and had breast cancer at age 42 with a bilateral ductal carcinoma and lobular carcinoma treated with bilateral mastectomy. In the past few years (2007) she had skin testing for food and environmental allergens showing strong responses to (trees: birch+3, white ash+3, maple and hickory +2), (weeds: march elder+3, ragweed, English plantain+2 and Lamb'sQuarter+2), (animal: cat+3), (Inhalants-dust mite F & P +3) and (foods: orange, filbert/hazelnuts, white potato, mushroom, garlic, shrimp).

Another major health issue has been gastroesophageal reflux disorder requiring her to take a number of different medications to suppress her stomach acid production. Her original allergist believed GERD was a major cause of the hearing loss because of the irritation to the pharyngeal region and the effect on the Eustachian tubes of the ear.

Findings

The initial examination was conducted on 7/26/05 showed the following structural findings: hip high on the right, shoulder and occiput high on the left a classic category one postural pattern. Range of motion cervical spine showed restriction to lateral flexion both right and left no pain noted in movement. Dorsal/Lumbar range of motion showed only slight restriction to flexion no pain noted.

Blood pressure was 132/90 seated and 128/89 standing which is positive a Raglan's effect, pulse rate was 71 seated and 76 standing. Blood oxygen level measured with an oximeter reading of 94%. Otoscopic examination of external acoustical meatus revealed on obstructions with some fluid build up behind both tympanic membranes. Reflexes showed biceps and triceps absent on both right and left. Radial, Extensor carpi and digits reflex normal both right and left and patella reflex is normal left and diminished on the right –1.

Applied Kinesiology findings-neurological disorganization positive both K27 and crossed K27. Cranial faults examination revealed Sphenobasilar right inspiratory, Frontal left external, Universal, Inspiratory assist on left, Learning Disability fault. Ileocecal valve syndrome was positive open variety, also present was meridian imbalance showing excess energy in the kidney (K) and diminished energy in circulation/sex (CX).

Fixation pattern present at time of examination upper cervical C1 right posterior, mid cervical C4 left posterior, Transitional T1 left posterior and T12 posterior left. The pelvic region showed Category #1 on the left AS. Also present was Pitch in flexion, Yaw #1 on the right and Yaw #2 on the left. Subluxation T1-PR, C5-PL and T5-anterior. HO meridian pattern showed B-54 positive and anterior atlas displacement. Also present was Hiatal Hernia and Diaphragm fixation pattern on the right.

The most critical finding in my opinion in this case was Pectoralis Minor weakness both right and left which was positive to retrograde muscle testing. Both Pectoralis Minor with weak to MMT and also showed the presence of positive pincer palpation (Travell), Jones trigger point and Anatomy Train-Thomas Meyers Functional Lines involved on both the right and left.[4,5] This finding was the one consisting finding that was present on three examinations 7/26/05, 7/10/07 and 7/18/09.

Patient obtained audiometry examinations from ENT and Allergy Associates-Hearing Services. These tests are preformed at what is considered normal speech levels of 250-8000 Hz, although the human ear can detect frequency form 20-20,000 Hz. The following scales will give you the normal ranges and hearing loss ranges.[7]

Degrees of Hearing Loss

1. Normal hearing (0-25 dB)
2. Mild hearing loss (26-40 dB)
3. Moderate hearing loss (41-55 dB)
4. Moderate-severe hearing loss (56-70 dB)
5. Severe hearing loss (71-90 dB)
6. Profound hearing loss (>90 dB)

These are three separate Audiometry tests showing the following findings:

12/26/07	Right ear 95-80 dB	profound to severe loss
before	Left ear 70-55 dB	moderate to severe loss
10/29/08	Right ear 105-75 dB	profound to severe loss
before	Left ear 80-40 dB	severe to moderate loss
10/07/09	Right ear 105-70 dB	profound to severe
after	Left ear 40-20 dB	mild to normal

Tympanogram findings

Tympanogram represents the volume of air displaced by movement of the eardrum or tympanic membrane. The examination is performed with a probe inserted into the external ear canal. A 226-Hz tone is transmitted though the probe, and the compliance or movement of the tympanic membrane is measured while the external canal pressure is varied. The peak movement is recorded and the external canal volume is also measured.

The normal middle ear pressure gradient is found in the range of +100 mm H_2O to -150 mm H_2O. The compliance peak usually shows a displacement of 0.2 to 2.0 mL of air measured on the vertical axis and the horizontal axis is the amount of pressure in the middle ear (+100 mm to –150 mm H_20).[8]

12/26/07 reading showed right ear –50 and left ear –95.
10/07/09 reading showed right ear –90 and left ear –65.

Outcome

During the initial phase of care I treated MB on only three visits of 7/26, 8/2 and 8/9/05 and she reported very good response and no major hearing problems for two years. She then experienced a gradual loss of hearing the culminated her returning for care starting with an examination and treatment on 7/10, 7/16 and 7/24/07 while she had some improvement it was not a drastic a change as she first experienced.

I believed she only did three visits at a one-week interval because this is what worked during initial phase of care so she decided this was the proper way to deal with this condition. When she returned this year 2009 I explained that she should continue care not only after she had an improvement but to maintain the improvement.

She returned for care on 7/18/09 a little more then two years since her previous course treatment. I did a complete physical and AK examination and treated all abnormal finding including NAET allergy treatment combined with HO point meridian therapy. The allergy treatments were for both food and pollens. Treatments were done on 7/18, 7/23, 7/25, 8/11, 8/17, 9/29, 10/27 and the last visit on 12/8/09.

Her hearing had significantly improved so she no longer needed hearing aids and will not require the cochlea implant. She also stated that changes in weather no longer have any effect on her acuity as it had done in the past.

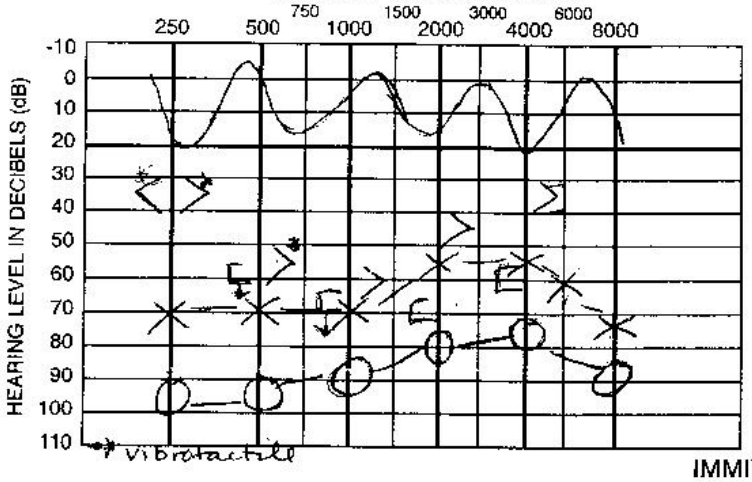
ENT ALLERGY ASSOCIATES, LLC

AUDIOLOGY TESTING
DIZZINESS EVALUATIONS
HEARING AID DISPENSING

- 385 Prospect Avenue, 2nd Floor, Hackensack, NJ 07601 • 201-883-1062
- 177 North Dean Street, Englewood, NJ 07631 • 201-567-2771
- 1211 Harnburg Tpke., Suite 205, Wayne, NJ 07470 • 973-633-0808
- 690 Kinderkamack Rd., Suite 101, Oradell, NJ 07649 • 201-722-9850
- 1001 Clifton Avenue, Clifton, NJ 07013 • 973-777-5151

[redacted] M[redacted] — Patient's Name
Dr. [redacted] — Referred by
12-26-07 — Date
[redacted] CCC/ — Audiologist

FREQUENCY IN Hz re: ANSI 1989



inserts

KEY	EAR RIGHT (RED)	EAR LEFT (BLUE)
A/C	O	X
A/C NO RESPONSE	♀	♀
A/C MASKED	△	□
A/C/ MASKED, NO RESPONSE	♀	♀
BC	<	>
B/C NO RESPONSE	♀	♀
B/C MASKED	[	]
B/C MASKED, NO RESPONSE	♀	♀
SOUND FIELD	S	
SOUND FIELD, NO RESPONSE	♀	
SOUND FIELD AIDED	A	

SPEECH AUDIOMETRY

	SRT	WRS	LEVEL
RIGHT	75	40%	95dB (m)
LEFT	60	88%	85dB

IMMITTANCE EVALUATION

	TYMPANOMETRY TYPE	TYMPANOMETRY MEP	ACOUSTIC REFLEX THRESHOLD 500	1000	2000	REFLEX DECAY	(IPSI) CONTRA
RIGHT	A	-50	1M	→			
LEFT	A	-95	M	→			

COMMENTS: Pt has Rexound ITE HA (AS)

AUDIO: (R) profound essentially snhl
(L) severe-moderate essentially snhl

SPEECH:
(R) SDS poor
(L) SDS good

IMMITTANCE: type As

RECOMMENDATIONS: ENT
new HA (AS)

Rev 4/07

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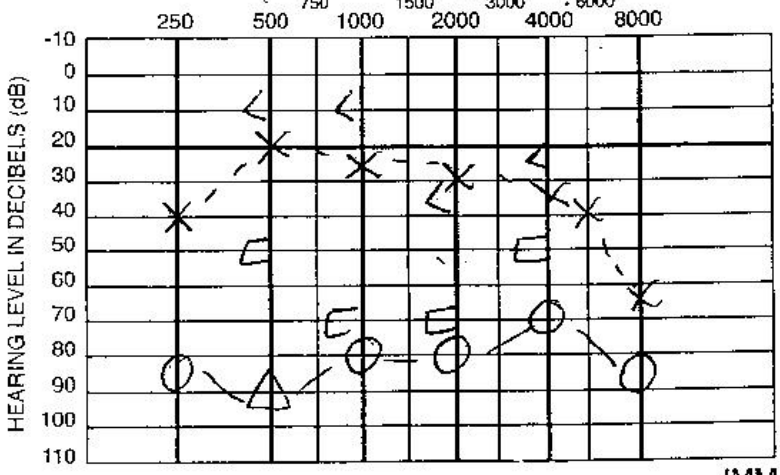
- 56 Union Avenue, Suite 1 · Somerville, NJ 08876 · 908-722-1022
- 79 Hudson Street · Suite 303 · Hoboken, NJ 07030 · 201-792-1109

B[redacted], M[redacted] — Patient's Name
Dr [redacted] — Referred by
10-7-09 — Date
E[redacted] Aud — Audiologist
8-1-49 — Patient's D.O.B.

This is an Audiological Examination and is NOT a Medical Examination.

FREQUENCY IN Hz re: ANSI 1989



☐ Headphones
☒ Inserts

KEY	EAR RIGHT (RED)	EAR LEFT (BLU)
A/C	O	X
A/C NO RESPONSE	♀	X
A/C MASKED	△	□
A/C/ MASKED, NO RESPONSE	♀	♀
BC	<	>
B/C NO RESPONSE	♂	X
B/C MASKED	⊏	⊐
B/C MASKED, NO RESPONSE	♂	♂
SOUND FIELD	S	
SOUND FIELD, NO RESPONSE	S	
SOUND FIELD AIDED	A	

SPEECH AUDIOMETRY

	SRT	WRS	LEVEL	
RIGHT	80	[redacted]	64%	105/70
LEFT	30	100%	70	

IMMITTANCE EVALUATION

	TYMPANOMETRY TYPE	MEP	ECV	ACOUSTIC REFLEX THRESHOLD 500	1000	2000	REFLEX DECAY	IPSI CONTR
RIGHT	A	-90	1.4	DNT	→			
LEFT	As	-65	1.0					

COM- Pt reports improved hearing AS post Chiropract visits

AUDIO: AS: significantly improved ↑ borderline normal/mild SNHL 250Hz - 4K sloping to mod-severe @ 8K.
AD: severe SNHL

SPEECH: SD excellent AS, poor AD

IMMITTANCE: Tymps WNL

Discussion

There are a number of factors the first is chiropractic spinal manipulation of the cervical region could effect directly or indirectly by way of the superior cervical sympathetic ganglion with feedback from vertebra C1-C2-C3-C4 to the Eustachian tube and middle and inner ear structures.[9]

The second factor was that of cranial faults that were treated and corrected that make up the hearing mechanism and have effects on the CSF and the production and reabsorption of perilymph and endolymph of the cochlear and vestibular labyrinth.

The third factor, which I believe, is the crucial one in this case was the lymphatic system and the drainage of the head region. In a text Applied Anatomy of the Lymphatics stated that the superior deep cervical nodes which are found posterior upper cervical region are made of (10 to 15nodes). Collect lymphatic capillary drainage from cranium especially the pharynx, tympanum, Eustachian Tube, inner ear. It is also stated that the middle ear drains lymphatics, which pass out through the external auditory canal to join the superficial lymph glands of the neck. The inner ear communicates with the subdural and subarachnoid space of the brain and there is connected to CSF filtration and return system and would be effected by cranial faults.[10,11,12]

The final and forth factor is the general lymphatic system and it collection return for the entire body is to the right and left subclavian veins. The thoracic duct drains from the lower extremities and abdominal cavity and the left arm and the neck to the left subclavian vein. The right lymphatic duct drains the right thorax, right arm, neck and head.

The importance of this factor was acknowledge by Dr. Goodheart in the 1979 Workshop Manual the article titled, “Role of Pectoral Traction in the Treatment of Lymphatic Flow Disturbances was sent to him by George V. Kroll, DO.[13] This was the basis for the diagnostic muscle testing with the patient body placed head downward on the treatment table at approximately 20 degree decline known as Retrograde Lymphatic Technique.[14]

Since my cousin MB had a bilateral mastectomy and some lymph node removal from the axillary region it seemed logical to me that this could very well is the major cause of her hearing loss. The surgery would have left tissue changes and weakness in the pectoral muscle group especially the Pectoralis Minor that over lies the subclavian vein and the subclavian lymph trunks. If the venous or the lymphatic return is slow from the neck and head region then the middle and inner ear may back up causing the hearing loss either by tympanic membrane motion restriction or changes in the endolymphatic pressure in the cochlea.

Conclusion

The factor of a disturbance in lymphatic flow was the one constant that was present in all three examination and treatment programs and when it was corrected and stabilized her

symptoms improved. The reason I so strongly believe that this a major causative factor is MB also suffers with gastroesophageal reflux (GERD) that Dr. Goodheart shown a restriction in lower ribcage movement from the right to the left but not from left to right. This would change the vital capacity and cause a slight reduction in blood oxygen level. The diaphragm acts by way of it respiratory action to assist the movement of lymph return to the subclavian vein.

I demonstrated this factor in my research paper titled Gastroesophageal Reflux Disorder and Hiatal Hernia, A Universal Problem and was a contributing factor that played a role in this case hearing loss.[15]

Many patients with these factors could be greatly helped with their hearing loss if these underlying problems are corrected.

Reference

1. Berkow, Robert, M.D., The Merck Manual of Diagnosis and Therapy, Merck Sharp and Dohme Research Laboratories, 15th Edition, 1987.

2. Beeson, Paul, B., M.D., McDermott, Walsh, M.D., Cecil-Loeb Textbook of Medicine, 11th Edition, W.B. Saunders Co., 1963.

3. National Institute on Deafness and Other Communication Disorders, National Institute of Health, NHI Web site, 2001.

4. Travell, Janet, G., M.D., Simons, David, G., M.D., Myofacial Pain and Dysfunction-The Trigger Point Manual, Willimas & Wilkins, 1983.

5. Myers, Thomas, W., LMT, ARP, Anatomy Trains-Myofacial Meridians for Manual and Movement Therapists, Churchill Livingstone, 2001.

6. Goodheart, George, J., D.C., Applied Kinesiology-Workshop Procedure Manual Private Publication, 15th Edition, 1979. Pages 1-12.

7. Walter Joseph Kutz, M.D., Mullin, Ginger, Au.D, Campbell, Kathleen, Ph.D, Audiology, Medscape-Center for Disease Control Commentary Series, Web site June 25, 2008.

8. Bredfeldt, Raymond, C., M.D., An Introduction to Tympanometry, American Family Physician, Dec. 1991, Vol. 44, No. 6, Pages 2114-2118.

9. Chusid, Joseph, G., M.D., McDonald, Joseph, J., M.D., Correlative Neuroanatomy and Functional Neurology, Lange Medical Publication, 12th Edition, 1964, Page 110.

10. Gray, Henry, Anatomy of the Human Body, Lea & Febiger, 27th Edition, 1965, Pages 785-786.

11. Millard, F.P., D.O., Walmsky, A., G., D.O., Applied Anatomy of the Lymphatics, 1922, Kessinger Publishers, 2004.

12. Spence, Alexander, P., Ph.D., Mason, Elliott, B., Ph.D. Human Anatomy and Physiology, 2nd Edition, The Benjamin/ Cummings Publishing, 1983, Pages 542-547.

13. Zink, Gordon, D.O., Lawson, William, B., Ph.D., The Role of Pectoral Traction in The Treatment of Lymphatic Flow Disturbances, Osteopathic Annals, Vol. 6, No. 11, Nov 1978.

14. Walther, David, S., D.C., Applied Kinesiology, Synopsis 2nd Edition, ICAK-U.S.A., 2009, Pages 539-541.

15. Sprieser, Paul, T., D.C., Gastroesophageal Reflux Disorder and Hiatal Hernia, A Universal Problem, Collected Paper of International College of Applied Kinesiology-U.S.A., Private Publication 2005-2006. Pages 209-217.

The Importance of Recognizing the Yaw #2 Skeletal Distortion Pattern in Chiropractic Practice

Paul T. Sprieser, D.C., DIBAK

Abstract

The recognition of the almost universal skeletal torque pattern that is a manifestation and cause of dural torque irritation, however this simple pattern, is often seen but seldom recognized or corrected in the practice of chiropractic.

Introduction

This structural pattern was first discussed by Dr. George J. Goodheart in the 1979 Workshop Procedural Manual under the heading of "Ocular-Basic Technique.[1] He came upon this information in a book titled "Awareness Through Movement"[2], which showed the importance that eyes played in coordinating the musculature of the body and its effect on the neck muscle. This is known as the "visual righting reflexes". A secondary system of reflexes that was connected to the sacrum known as "cloacal reflex", which must be in synchronization meaning that the head and sacrum must be level in order to provide maximum neural communications.

These observations were developed further by Dr. Goodheart and appeared in the 1980 Workshop Procedural Manual as P.R.Y. Technic which is an acronym for Pitch, Roll and Yaw and subtitled (Labyrinth and Righting Reflexes).[3] These terms are aeronautical for deviation from a flight path about an aircraft's vertical, longitudinal or lateral axis. This paper will only address one aspect of this distortion Yaw #2, which consists of deviation along a vertical axis of the body. This pattern is seen as a rotation of the pelvic girdle forward on the right and backward on the left through the transverse plane and the shoulder girdle rotation forward left and backward on the right.

The diagnosis and correction of the yaw #2, pattern that exists in 9 out of every 10 of our patients is vital to the correction of subluxation, prevention of fixations and correction of neurological disorganization/switching and the general improvement of neural communication of the central and peripheral nervous systems.

This is a central theme that includes at least two of the chiropractic colleges. The first is Palmer College and this came about from B.J. Palmer's interest in F. Matthias Alexander an actor of his era. Alexander developed the technique as a personal tool to alleviate breathing problems and hoarseness that solved his inability to pursue a career as a

Shakespearean actor. B.J. Palmer interests in the Alexander Technique was due to the importance of the anatomical structure of atlanto-occipital joint that became known as Hole-In-One or (HIO) specific upper cervical technique.

Logan Basic Technique came about for the observations of its founder Hugh Logan, D.C. interest in the sacrum as the foundation of the vertebral column. If it sacrum was not level it would effect the vertebra above that rest on the sacral base.

What the both techniques have in common is its dural connection from it firm attachments at the foramen magnum and C1-C2-C3 (Palmer) and the sacral dural attachment in the sacral canal on the anterior wall at S2 and filum terminale to the coccyx (Logan).[4,5]

Discussion

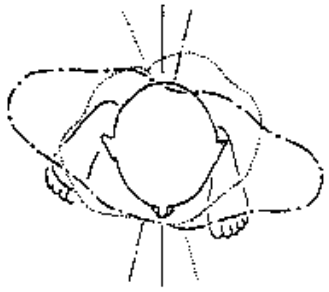
I have been using the P.R.Y. Techniques since 1980 and have been evaluating this on every new patient since its inception. Over the past 15 years I have evaluated not only on all new patients, but also on every patient and on every visit. I became aware the Yaw #2 pattern would not stay corrected very long. Whereas the Pitch, Roll, and Yaw #1 could be corrected on the first visit and this might last as long as six month to a year. I had learned from Dr. Goodheart to always question when I didn't understand what I observed, is to ask, **"why is that"?**

Over the years of observing the persistence and reoccurrence of the Yaw #2 pattern in general population of patients. I tried to find a way to get greater and longer lasting correction of the pattern. Since 1982 till 2006 I authored 7-research paper about Yaw #2 distortion and additional 5 papers that are results of this chronic pattern.

The importance of a longer lasting correction is that it is Yaw #2 is the main cause of neurological disorganization also know as switching[6], chronic and persistent subluxation, fixations and (GERD) Gastroesophageal Reflux Disorder

Method

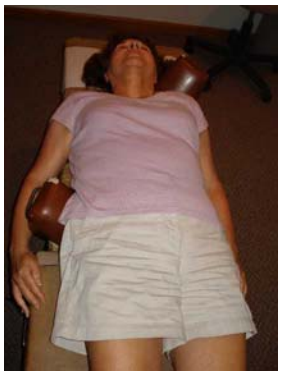
First visual observation of the pattern can be made with the patient standing and be seen from the rear as well as the front. Making sure the patient's feet are place on a parallel line to one and other and about a shoulder width apart. What will be observed is the pelvis will be held forward on the right with rare exception.



6—72. Superior view revealing modular displacement about the vertical axis of the body.

Second if a patient is lying supine on a table observe the area of the (ASIS) anterior superior iliac spine. It will be higher towards the ceiling on the right and lower on the left towards the floor. If viewing with the patient prone the left ilium near the (PSIS) posterior superior iliac spine will be higher from table towards the ceiling and the right will be lower towards the floor. On the more corpulent patient especially supine the doctor will have to press down over both (ASIS) to confirm the distortion.[8]

If you use manual muscle testing (MMT) an appropriate indicator muscle such as Tensor Fascia Lata (TFL) or Pectoralis Major Sternal or Clavicular (PMS or PMC) will be used to test. A challenge using a pair of S.O.T blocks will be done in the supine patient one block is under the right ilium pushing it further into distortion the other block is under the left shoulder region near the glenoid cavity this cause both are to be distorted toward the ceiling.



In AK this is called body into distortion or (BID) and will cause a strong indictor muscle to weaken using either upper or lower extremity indicator muscle that are not on the block.

If you start with the patient in the prone position then the S.O.T. blocks would be placed under the left ilium at the ASIS pushing it toward the ceiling the other block is placed under the right shoulder joint pushing it towards the ceiling. The indicator muscle used in the instance is the hamstring on the side on the block or the right hamstring.[9]

Correction is simply to push the high ilium down to the floor either with the patient supine or prone. If you had a pelvic drop section this can be used. If you don't you can use the S.O.T. block on the lower side to raise up and gently with a broad contact on the high side thrusting it towards the floor about for or five times.

Visually observe that the pelvic is now more level if you use MMT then replace the blocks in the challenge position, which should no longer weaken a strong indicator muscle.

Findings

I have been examining and treating patient with the skeletal distortion pattern since 1980 and to say modestly that I have checked this at least 75,000 times would be not be an exaggeration. You are probably wondering why I stated the pelvic is forward on the right and backward on the left will be the most easy to observe this torque distortion. This pattern of torque will be found, in at least 9 out of every 10 patients you treat either. The reason is the predominance of right handedness and left brain dominance exists at a minimum of 85% to 89% of any group examined. Add to the fact that everyone weighs more on the right due to the fact that the largest organ of the body, the liver is on the right. Finally there is a consistent muscle imbalance manifested with the hip flexor Psoas muscle found weak on the left and hypertonic on the right which pull the pelvis into the yaw position.[10]

Conclusion

Adding this simple observation and correction to your chiropractic treatment plans is easy and will add no appreciable time needed to treat your patient. You will find that it pays back in dividends of allowing the body to self correct and improve your patients general health and wellbeing. Just give it a try.

References

1. Goodheart, George, J.,D.C.,-Applied Kinesiology Workshop Procedure Manual, Private Publication, 15th Edition, 1979, pages 24-26.

2. Feldenkrais, Moshe-Awareness Through Movement, Harper and Rowe, 1972.

3. Goodheart, George, J., D.C.,-Applied Kinesiology Workshop Procedure Manual, Private Publication, 16th Edition, 1980, pages 1-13.

4. Sprieser, Paul, T., D.C.-The Relationship of Rocker Action to PRY-Technique as a Method of Diagnosis of Specific Dural Lesions, The Collected Papers of The International College of Applied Kinesiology-U.S.A., Private Publication, 2001, pages 47-48.

5. Sprieser, Paul, T., D.C.-Dural Torque and its Association to Pitch Pattern of the PRY-Technique and Improvement in Range of Motion, The Collected Papers of The International College of Applied Kinesiology-U.S.A., Private Publication, 2002, Pages 193-195.

6. Sprieser, Paul, T. D.C.-The Relationship of Switching to the Yaw #2 of the PRT-Technique, The Collected Papers of The International College of Applied Kinesiology-U.S.A., Private Publication, 2003, pages 243-247.

7. Sprieser, Paul T. D.C.-Gastroesophageal Reflux Disorder (GERD) and Hiatal Hernia a Universal Problem, The Collected Papers of The International College of Applied Kinesiology-U.S.A., Private Publication, 2005, pages 209-217.

8. Walther, David, S., D.C.-Applied Kinesiology, Synopsis, 2nd Edition, ICAK-U.S.A., 2000, pages 213-217.

9. Walther, David, S., D.C.-Applied Kinesiology, Synopsis, 2nd Edition, ICAK-U.S.A., 2000, pages 213-217.

10. Sprieser, Paul, T., D.C.-Yaw #2 Muscular Pattern, The Collected Papers of The International College of Applied Kinesiology-U.S.A., Private Publication, Winter, 1982, pages, 255-258

11. Image 1 - Reproduced with kind permission from Dr. David S. Walther-Synopsis 1988

Sound Localization and Reflex Changes in Head Posture

How Did Harvey Lillard Get Back His Hearing?

Kurt A. Vreeland, D.C., DIBAK, DABCN

Abstract

The story of Harvey Lillard might seem miraculous, but functional neurology has the explanation. Applied kinesiology is a method of using muscle testing and therapy localization to unveil altered eye, head and neck relationships. A component of human hearing is a built in sound localization system, much like that of the bat or the barn owl detecting prey in complete darkness. Persons devoid of sight also use this system to "see", by using clicks and timing the reflected sound. Chronic changes in the perception of where a sound comes from causes action potentials, and hence, plasticity to develop in the sternocleidomastoid and the complexus musculature. This paper reveals a simple, fast and effective way to determine cause and effect of the aforementioned muscle imbalance based on the central integration of this sound-detecting portion of hearing. To say it another way, head tilts and rotations causing chronic altered neck mechanics and head and eye relationships are many times secondary to the perception of the direction that sound comes from.

Introduction

The human neuraxis contains a complex sound localization system. The hair cells in the cochlea transduce mechanical input into electrical responses. The brain interprets this noise to make sense of our world. The entire auditory system superimposed on the central integration, and the individual human experience in its entirety, is majestic, and well beyond the scope of this paper. This paper will delineate the two separate and distinct pathways of how sound integrates into the neuraxis. One being for actual hearing; the kind of hearing we all think about, while the other is vestibular in nature. The focus of this paper is the relationship of sound detection to the vestibular system, and its relationship to posture, especially head posture, and the relationship to Applied Kinesiology. This paper will elaborate on the myogenic vestibular evoked potentials or (MVEP) or vestibular evoked myogenic potentials (VEMP) and their relationship to the chronic tone of the cervical musculature. It will also elaborate on the hearing pathways from the cochlea to the primary auditory cortex, with special attention to the superior olivary (SO) complex and its' role in sound localization.

Vestibular component pathway: Afferent and efferent (simplified)

The VEMP/ MVEP response is elicited by loud clicks or tone bursts in the ear and recorded at the sternocleidomastoid muscle. It is a vestibular-neck, termed vestibulo-colic

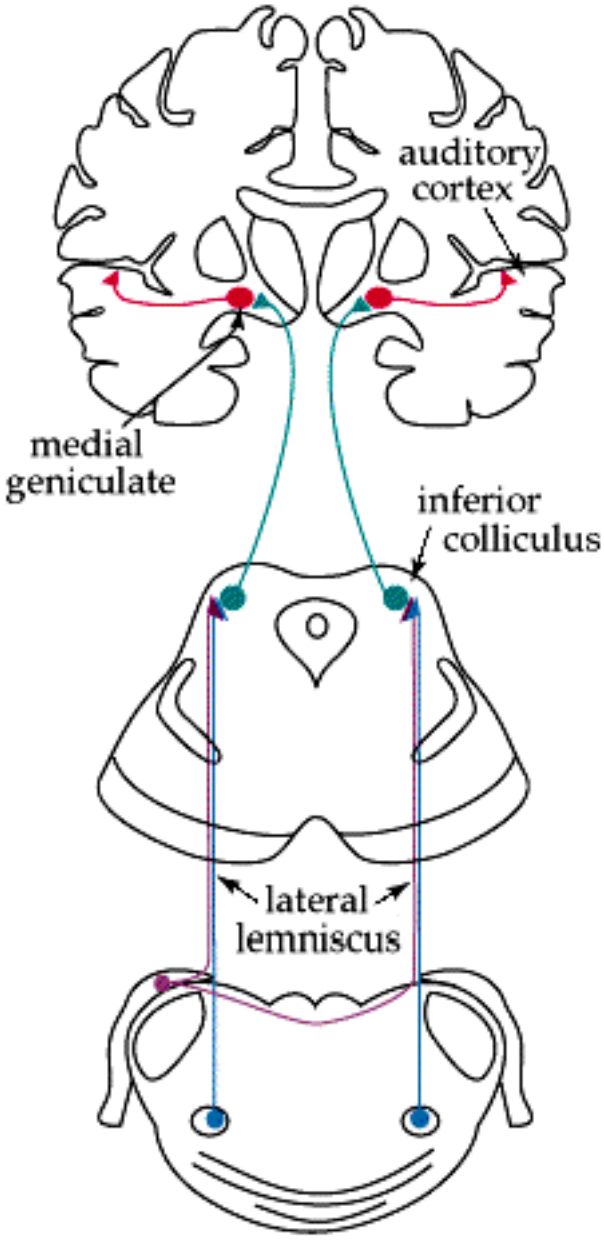
reflex whose afferent limb arises from the acoustically sensitive cells in the saccule. Its signals are conducted via the inferior vestibular nerve to the vestibular nuclei, mainly the caudal medial vestibular nucleus. To be more precise, the caudal portion of the medial vestibular nucleus. (MVN). The MVN has reciprocal connections to the vermis, the nodulus, and the midline structures of the cerebellum (where afferents from spinal joints and muscle spindles also integrate). The descending output from the MVN travels in the vestibulospinal tracts to orient head position. Since this reflex arises from the saccule, not the cochlea, it is vestibular in nature. Therefore involved in posture regulation, gravaceptive orientation and gaze stabilization, especially in conjunction with the central integration of the midline cerebellum. Since this particular reflex is vestibulo-colic (vestibulo-neck) it is concerned with the orientation of the head in space. Patients with profound hearing loss and even agenesis, or surgical ablation of the cochlea maintain a VEMP response. Those patients with vestibular nerve section (vestibular neurectomy) do not have a VEMP response. This is proof that this vestibular response is pure and separate from hearing***. In other words, the cochlear and vestibular divisions of crainal nerve VIII carry very different and distinct afferents to orient head posture.***

The saccule detects acceleration, deceleration, up and down, and side-to-side movement. The patient with a disturbance in this system will complain of a feeling of **lateral pulsion**, like being pushed to one side or the other, or, forward or backward movements. It is important to keep this in mind because it is a primary reason for head posturing distortions, head perturbations, tremors and titubations. Patients with various head posturing have these issues, especially, the patient with head forward posturing, or head tilts or rotations to one side or the other.

Other symptoms of this patient would include lateral pulsion, forward or backward movements in the Romberg's position. They will have chronic joint dysfunction in the upper cervical region. They will have chronic recidivism in therapy localizing the upper cervical segments. They will complain of all the consequences of upper cervical joint dysfunction. They will have neck problems. They will have autonomic imbalances related to the Nucleus of the Tractus Solitarius (NTS) i.e. digestive, respiratory, and or cardiac disturbances. They will also be cognizant of the fact that they perceive motion in the Romberg position. All of the other symptoms arise from these neural pathways.

Hearing component pathway: afferent and efferent (simplified)

The pathways for sound detection originate in the sensory receptors in the cochlea, at the level of the ponto-medullary junction. The axons form the cochlea terminates in the cochlear nuclear complex. From the cochlea nuclear complex there are *three* main pathways "north" to the primary auditory complex. *One* is via the dorsal acoustic stria. The *second* is the intermediate stria. The *third* is the ventral acoustic stria or trapezoid body. It is the third(or ventral acoustic stria, or the trapezoid body) that we will focus on, for this is the pathway involved in sound localization. The trapezoid body projects to the medial and lateral divisions of the superior olive. It is these nuclei in the superior olive that are involved in sound localization. The cell bodies of the superior olive in tern project their axons to inferior colliculus in the mesencephalon via the lateral lemniscus.

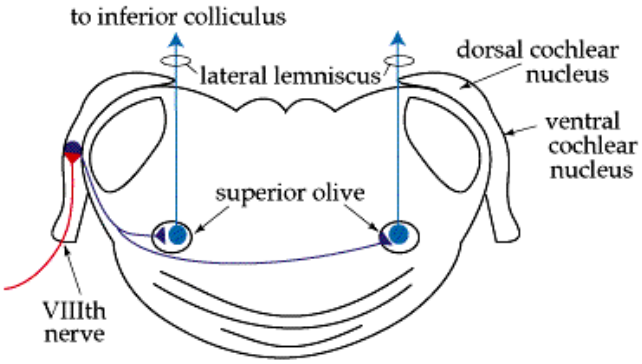


From the inferior colliculus axons travel to the medial geniculate of the thalamus. The axons from the medial geniculate then terminate in the primary auditory cortex, area A1, or areas 41 and 42 of Brodmann in the superior temporal gyrus.

Hearing is considered mainly a bilateral event, in that, the afferents from the cochlea bifurcate in the trapezoid body, and has interconnections along the way to the primary auditory cortex. The unique feature, (*which has been overlooked clinically, except maybe accidentally by D.D. Palmer),* **is the ability of the medial superior olive to localize sound along the azimuthal or horizontal axis.**

It is obvious when using therapy localization that the detection of, or more importantly, the perception of the detection of, sound has immense consequences in head posturing. For example, sound produced in the mid-sagittal plane will reach both ears at the same time. On the other hand, a sound produced closer to one ear or the other, will cause a time delay in reaching the more distant ear.

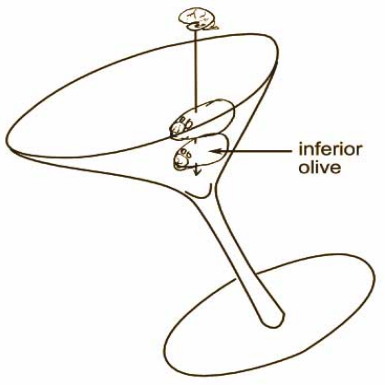
The *gist* of this paper is, that the central integration of the superior olive, the inferior colliculus, the medial geniculate, or the auditory cortex is ***not*** exactly the same from side to side, and in fact, seldom is the same. For these nuclei receive inputs from many other sensory systems. In fact sound perception is like all other somatosensory inputs. We, (that is if we examine) almost without exception, find discrepancies in bilateral blood pressures, pupil reaction times, pupil sizes, blind spots, muscle tone, reflexes, and the list goes on. The alteration in the perception of a sound source will cause reflexogenic head adjustments to help localize that sound source. The **medial superior olive** thus localizes



sound by comparing the delay of sound, from one side to the other. The **lateral superior olive**, on the other hand, uses intensity cues to localize the sound source. The difference between the medial and lateral superior olive's ability to localize sound is in the receptor physiology and central integration of the afferent input, and is nothing short of glorious. It is well described in detail in any good neurology text.

The post-synaptic cells of the superior olive are found in the inferior colliculus of the mesencephalon. This is part of the tectal region of the mesencephalon. The descending output from this tectal region is carried via the tecto-spinal pathways to orient the head in space by adjusting the tone of cervical musculature.

{*The inferior colliculus also receives somatosensory input, which it in turn relays it to the thalamus, and then to the cortex. The central integrated state of these mesencephalic and thalamic relays will determine the perceptual experience of sound to the individual. The central integration, or the resting membrane potentials of these relays is based on their pre-synaptic input. There is a probability that the Chiropractic profession was founded on this exact principal. It was fortunate for Harvey Lillard that DD Palmer had the skill to alter these membranes potentials by adding somatosensory input either to the inferior colliculus, to the cerebellum, to the thalamus, or to all structures involved. This added input likely allowed these relays to come to threshold and fire on to the primary cortex where a sound became apparent.}*

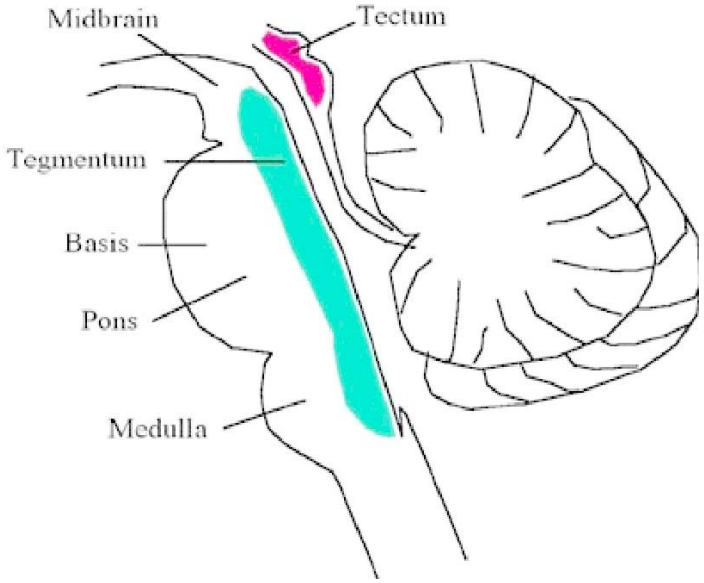


The above is what happens under normal circumstances. Unfortunately our patients do not present under normal circumstances, for if they were normal, there likely would be no reason for them to present at all.

Most patients with persistent posture changes involving head position, tilts, rotations, etc. have *primary* alterations in central neural circuitry. This asymmetry in the output of central motor systems is many times the primary cause of muscle imbalance. In some cases, the muscle, as the final recipient of a neuronal frequency of firing is merely responding normally to the central generator. Over time plastic changes in neuronal and muscular function produce chronic and adaptive alterations in head and eye position. The reverse can also be true, in that, aberrant ocular alignment will cause compensatory head positioning. [*The primary ocular distortions are well beyond the scope of this paper, but will likely be the subject in the future]*.

The purpose of this paper is to alert the clinician to the abnormal muscular responses to sound detection; these are actually normal responses to an underlying abnormal central integrated state (CIS). This means that the resting membrane potential at the superior olive, the inferior colliculus, the thalamus, or the auditory cortex, is closer, or further away, from threshold, on one side compared to the other. The brain's perception of the

sum of all this integration causes the output through the **tectospinal** or the **vestibulospinal** reflexes to change the tone of the neck musculature, in this case to look toward the sound source. [Just as a barn owl using sound detection would do while hunting in the dark]. In fact this is common practice in blind persons using auditory clicks, or the tapping of a cane to "recognize" objects, and orient themselves to an environment. *Functional MRI's (f MRI's) of non-sighted individuals reveal activation of the visual cortex to sound, and in a sense blind persons actually "see" these objects.*



In fact a click in the right ear causes an action potential in the left SCM and the right complexes musculature. The reverse is true for the left ear. So we have two pathways delineated above. Both of which will cause head posturing alterations. One involves the vestibular system, and has powerful connections to the midline cerebellum. The receptor is the saccule and the final destination is the cerebellum, and the vestibulo-colic reflex. The other involves a complex sound detection system. It compares the timing and intensity of sound from one side to the other and adjusts head posture accordingly. It arises from receptors in the cochlea with the primary auditory cortex the final destination. It is mainly the primary auditory pathway that we will be discussing. The VEMP is a different pathway but seems to have some redundancy.

This has enormous implications in several clinical scenarios:

The patient with headache, neck pain or dysfunction, especially without a history of frank trauma.

The patient with hearing loss, (hyperacusis) increased hearing, or in migraines, tinnitus, or Menerirs's disease

The learning disabled, with dyslexia or any other "auditory processing disorder".

The mesencephalic patient who is in a constant state of being hyper- vigil, producing an increased sympathetic drive, with all of the consequences of too much sympathetic activation.

The patient with autonomic disturbances of the NTS with dysregulation of the gut or heart.

My last two papers introduced (ICAK Vol 1,2 2009-2010) and detailed the concept of fatigability of neuron pools in the mesencephalon producing end organ and muscle dysfunction. These muscle weaknesses are not the typical AK on /off scenario, but are a produced as a consequence of fatigability of central pools of neurons. Since the inferior colliculus is part of the mesencephalon, using the following technique along with the previous described techniques will enhance your ability to stabilize the mesencephalic output. Stabilizing this output will, in turn, stabilize the Intermediolateral cell column (IML) and the sympathetic responses (real or perceived) from the external environment in your patient. The VEMP portion of the reflex is vestibular in nature and loops through the cerebellum. I always use this in conjunction with the tectal and the tegmental responses (ICAK Vol 1,2 2009-2010) and find it increases the benefits in balancing the sympathetic/parasympathetic output, and helps the patient exponentially.

Method

Do all the usual and customary tests for hearing and vestibular imbalance. Make sure you visualize the tympanic membrane to be normal. Infection or obstruction of any kind will render invalid results. One does not necessarily need audiometric testing. The bedside tests of Weber, Rhine, rubbing your fingers together, or having the patient listen for the ticking of your watch, are good for our purpose here. The most important feature is for you to compare *minimal* activation from one side to the other, as you would do for all clinical signs. Most physicians will simply ask the patient if they can hear it or not, but will fail to compare minimal differences from one side to the other, or will use maximum activation of the whole system. For instance, yelling in a patient's ear with a bullhorn "*can you hear me now*"? This does a grave injustice to the patient for it misses important clinical data. In other words the difference between the two sides *is* the critical clinical data. For example, if the patient can hear my watch ticking 12 inches from the right ear, but 24 inches from the left ear, they have an imbalance. This means that the patient has a change in the CIS of the central systems that are involved in hearing or the detection of sound. These changes in the CIS will be expressed in the vestibulo-colic reflex and the neck muscles to adjust the head accordingly. It is common, but abnormal to see these differences in patients.

When you do Romberg's test look for lateral or A-P sway as soon as the patient closes their eyes. The initial direction of movement of the patient *is* the crucial clinical data. It is reproducible, and gives added value and meaning to the Romberg's test. If your patient has a reproducible initial movement, such as an initial sway to the right, the moment they close their eyes, not the corrective sway, which will compel the patient to sway to the left to keep stable, then this patient has a vestibular imbalance, plain and simple.

Therapy localize to the upper cervical segments. If there is no inhibition of a muscle, place a click (I use a series of clicks from a metronome purchased at the local music store) about one meter in front of the patient in the sagittal plane. Make sure the click is directly in front of the patient, (in the sagittal plane) not off to one side or the other, for this defeats the very nature of the test. All the while the patient is maintaining the TL on

the upper cervical segments. It works best with one finger on the occiput, one on the atlas, and one on the axis on each side. If the TL to the upper cervical spine causes the test muscle to be inhibited after the clicks, there is an aberrant influence to the cervical musculature through the vestibulospinal or tectospinal reflex. This is causing altered head posture and chronic joint dysfunction. The reverse scenario also works well here. If you start with a positive TL to the upper cervical spine place a series of clicks off to one side and the other side. Retest the previously inhibited muscle. If a series of clicks to either one side or the other causes the muscle to become facilitated, the patient has upper cervical joint dysfunction **secondary** to an altered central integrated state of central generators producing muscle imbalance causing aberrant head posturing.

This would indicate that it would *not* be appropriate to adjust an effect of muscular imbalance but would be best to treat the central causes. That way we get exponential benefits in the brain.

Treatment

If the patient got strong, with the click to the right side, they need activation of the left SCM, and the right semispinalis capitus or the right complexus musculature.

Step 1. Reset the muscle spindles of the left SCM by activating the 1a's and group II's causing excitatory post- synaptic potentials (EPSP's) centrally. (*There is a bias for the 1a's and the group II's to fire greater when the muscle is placed under a concentric load).* Have the patient turn their head, against your resistance, to the right, or toward the side that the click produced facilitation of the test muscle. In order to add together, or summate EPSP's have the patient move their eyes to the right. (Turning the eyes to the right excites the left SCM). Repeat having the patient turn their head and move their eyes to the right at least 5 times, sometimes 10 or 15. (More chronic cases will need more treatment. I always instruct patients in home care, to build plasticity in the deficient system).

Step 2. Place the right SCM under eccentric load by trying to push the patient's chin to the right while they resist (the patient is actually pushing their chin /head to the left thereby activating the right SCM and left complexes, **but** in an eccentric fashion. the patient is allowing the chin to actually move to the right.) (*This eccentric load places a bias on the Golgi Tendon Organ GTO and activates the 1b's to inhibit the right SCM centrally).* So in effect, you are exciting the left SCM and the right complexus, and inhibiting the right SCM and the left complexus. You accomplish this both mono-synaptically by direct activation and by reciprocal di-synaptic means. Have the patient, as above; move the eyes to the right to further excite the left and inhibit the right SCM.

Reverse the entire scenario above if the patient got strong with a click in the left ear.

Perform the above procedures (step 1 and 2) until the patient no longer TL's and/or more importantly, until the patient's hearing is equal bilaterally.

Retest the patients hearing by comparing the subtle differences from side to side. Almost without fail you will be able the change/balance the patient's hearing. (They will now appreciate the rubbing of your fingers equally on the right and the left and to the same distance). They will also be more stable in the Romberg's position. It is a good practice to demonstrate this to your patient, so they can appreciate the wonder of Chiropractic, AK, and Functional Neurology, as did Harvey Lillard.

References

1. Kendel Eric, R, Schwartz, James, H, Jessel, Thomas, M. Principles of Neural Science 4th Edition.

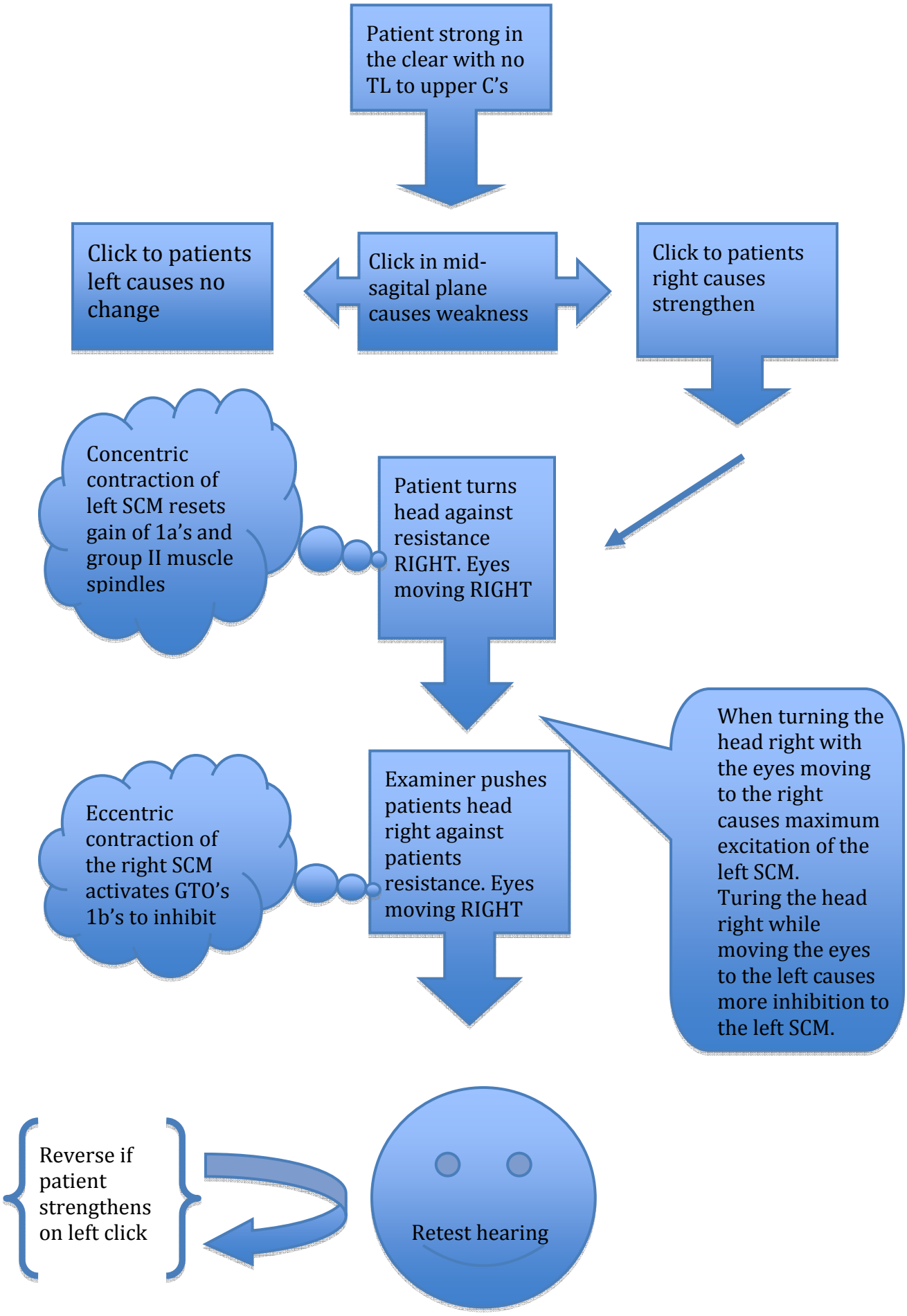
2. Beck Randy, W, Functional Neurology for Practitioners Of Manual Medicine.

3. Leigh, John, R, Zee, David, S, The Neurology of Eye Movements 3RD Edition

4. Vreeland, Kurt, A, The Sartorius Muscle as an Assessment Tool for Evaluating the Intermediolateral Cell Column (Part 1) International College of Applied Kinesiology-U.S.A. Vol 1 2009-2010

5. Vreeland, Kurt, A, The Subscapularis as an Assesment Tool for Evaluating the Intermediolateral Cell (Part 2) International College of Applied Kinesiology-U.S.A. Vol 1 2009-2010

6. Nolte, John, The Human Brain an Introduction to its Functional Anatomy. 5th Edition


Patient strong in the clear with no TL to upper C's
Click to patients left causes no change
Click in mid-sagital plane causes weakness
Click to patients right causes strengthen
Concentric contraction of left SCM resets gain of 1a's and group II muscle spindles
Patient turns head against resistance RIGHT. Eyes moving RIGHT
When turning the head right with the eyes moving to the right causes maximum excitation of the left SCM. Turing the head right while moving the eyes to the left causes more inhibition to the left SCM.
Eccentric contraction of the right SCM activates GTO's 1b's to inhibit
Examiner pushes patients head right against patients resistance. Eyes moving RIGHT
Reverse if patient strengthens on left click
Retest hearing

Division II

Critical Review

The Prevalence of Sensitivity to Wheat Based on Available Laboratory Data

Donald C. Baker, D.C., NMD

Abstract

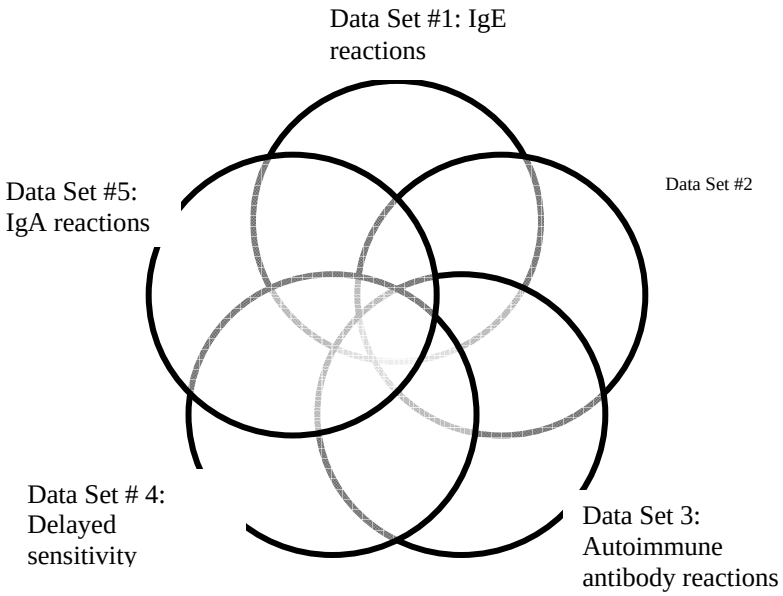
Based on a review of available scientific data, the prevalence of sensitivity to wheat in the population cannot be precisely determined. However, the prevalence appears to fall within a range from 29% to 80%.

Key Indexing Terms
Gluten, Wheat, Applied Kinesiology, Prevalence, Celiac, Gliadin

Discussion

Examining the prevalence of sensitivity to wheat determined by laboratory testing and comparing this to the prevalence of sensitivity to wheat via AK procedures can be informative. If both laboratory methods and AK methods are accurate, then the percentages of positive individuals (testing sensitive to wheat) determined by either method should be roughly in agreement. This paper will examine the first part of this question -- the prevalence of sensitivity to wheat as determined via laboratory testing.

We will use the term “sensitivity” as a broad and general term to include any and all adverse reactions to wheat. This would include but not be limited to IgE, IgG, or IgA reactions, delayed sensitivity reactions, and reactions stimulatory of autoimmune antibodies. An individual who is sensitive to wheat may be sensitive via one of these routes or via more than one. Hence, the Venn diagram below which depicts groups of people with various types of sensitivity to wheat shows overlapping zones between and among these various routes of sensitivity.



Venn Diagram depicting groups of people with various types of sensitivity to wheat.

Data Set #1 - The prevalence of IgE reactions to wheat : Innomata reports prevalence data from several different studies of IgE reaction to wheat. [1] Prevalence ranges in these studies from 0.5% to 9%. For example, one study from Stockholm reported that 4% of 2336 four-year-old children were found to have a wheat IgE reaction.

Data Set #2 - The prevalence of IgG reactions to wheat: No reliable data could be found on the overall prevalence of IgG reactions to wheat.

Data Set #3 - The prevalence of autoimmune antibody reactions due to wheat: No reliable data could be found on the overall prevalence of all autoimmune antibody reactions due wheat. However, data are available on one such disease condition, Celiac Disease. Celiac Disease (CD) is perhaps the most well-known autoimmune condition caused by wheat. CD affects at least 3 million Americans (1% of the population). [2] Other studies have indicated that autoimmune antibody reactions due to wheat may be important in many other conditions ranging from Hashimoto's thyroiditis to diabetes. [3] Enterolab [4] finds that 77% of people with autoimmune diseases are also sensitive to wheat, however, it does not follow that wheat is necessarily the cause of these autoimmune diseases or the cause of autoimmune antibody production.

Data Set #4 - The prevalence of Delayed Sensitivity reactions to wheat: No reliable data could be found on the overall prevalence of delayed sensitivity reactions to wheat.

Data Set #5 - The prevalence of IgA reactions to gliadin in wheat: Gliadin is the protein group in wheat most implicated in diseases like CD. Consequently serum antigliadin antibodies have been used as one of the ways to look for wheat sensitivity problems. Indeed, in recent years serum antigliadin antibody testing has become one of the most common forms of laboratory testing done for wheat problems. Relevant data [4] on serum IgA antibody testing are as follows:

a. Serum IgA antigliadin antibody tests are positive in 76% of CD patients.
b. Serum antigliadin antibody tests are positive in 9% of patients with microscopic colitis.
c. Serum antigliadin antibody tests are positive in 11% of normal people with no specific symptoms or syndromes.

IgA antibodies protect body surfaces that are exposed to outside foreign substances. The intestines are one of these surfaces. It is logical to presume that antigliadin antibodies might be found more frequently if intestinal or fecal testing could be done rather than serum testing for these antibodies. Indeed, Enterolab has developed IgA testing methods for fecal samples. Their data [4] using fecal antigliadin antibodies, as compared to serum antigliadin antibody testing, are as follows:

Test Subjects	% positive in Serum antigliadin antibody tests	% positive in Fecal antigliadin antibody tests
CD patients	76%	100%
Microscopic colitis patients	9%	76%
"Normal" people	11%	29%
People w/ autoimmune disease	--	77%
People w/ IBS	--	57%
Family members of those with CD	--	79%
All persons tested at Enterolab [e-mail 9/5/09]	--	approximately 80%

Several comments are in order relative to these IgA test data:

a. Serum IgA antigliadin antibody tests produce large numbers of false negative results. Notice, for example, that even in confirmed CD patients, serum testing falsely indicates that only 76% of these people are sensitive to wheat. Then, notice that compared to fecal IgA testing, the serum IgA testing is markedly inferior when testing people with microscopic colitis. Finally, notice that serum IgA has many false negative findings in the normal population.

b. The lowest rate of positive findings for wheat sensitivity using the fecal IgA antigliadin antibody test is in "normal" people. Enterolab defines "normal" as people without specific symptoms or syndromes. In this group, 29% are sensitive to wheat.

c. The people at Enterolab tell us that they have not done a detailed analysis of all their data but they estimate that using their latest testing techniques, approximately 80% of people tested are positive for fecal IgA antigliadin antibodies. They want us to understand that the population being tested may be skewed as compared to the general population (sick people usually request medical tests; not "normal", healthy people). [e-mail from Enterolab, 9/8/09]

Most of the data presented here was developed by Enterolab in Dallas, Texas. Enterolab was established by Dr. Kenneth Fine. Dr. Fine is a gastroenterologist, graduate of the University of Missouri school of medicine, and the author of numerous research articles in the field of gastroenterology.

Conclusions

(1) Data are insufficient to conclusively identify the prevalence of wheat sensitivity.

(2) Data reported above can be used to identify the lower limits of the prevalence of wheat sensitivity. The key numbers in this regard are those determined by Enterolab in their study of the normal population (those people without specific symptoms or syndromes) versus the population with disease. First, Enterolab finds evidence that the diseased population has higher prevalence of wheat sensitivity than the normal population. Secondly, Enterolab finds that the normal population without specific symptoms or syndromes has a prevalence of wheat sensitivity of 29%. Thus, the data indicate that at the lower limit at least 29% of the population is sensitive to wheat.

(3) The current medical laboratory test which will identify wheat sensitivity most often is the fecal IgA antigliadin antibody test.

(4) The overall prevalence of wheat sensitivity is far higher than 29% for several reasons:

a) People in Data Sets 1, 2, 3, and 4 that do not concurrently have fecal IgA antigliadin antibodies would be additive to the lower limit of 29%.

b) The "normals" used in the Enterolab study were people with no specific symptoms and no syndromes (healthy people). In the United States, the CDC estimates that 133,000,000 people suffer with at least one chronic disease. We know from Enterolab data that "sick" people have higher prevalence of wheat sensitivity. Therefore, if a random sample were taken from among all the people in the United States, including those with chronic disease, the percentage found sensitive to wheat would be higher than 29%.

c) The latest and best laboratory test for wheat sensitivity appears to be the fecal IgA antigliadin antibody test. Using the latest fecal IgA antigliadin antibody testing, people requesting this test (presumably "sick" people) are showing a prevalence of wheat sensitivity of approximately 80% (note: exact counting of positive and negative test results and calculation of percentage positive at Enterolab has not been done).

In summary, laboratory data indicate the prevalence of sensitivity to wheat ranges from about 29% to 80%. The data indicate that about 29% of "healthy" people are sensitive to wheat and that about 80% of "sick" people are sensitive to wheat.

References

1. Inomata, Naoko, "Wheat Allergy", Current Opinion in Allergy and Clinical Immunology 2009, 9:238–243

2. Celiacdisease.net. (n.d.) Celiac Disease Facts and Figure. Retrieved October 20, 2009 from http://www.celiacdisease.net/assets/pdf/CDCFactSheets%20FactsFigures %20v3.pdf

3. Ventura, A., Neri. E., Ughi C., et al, "Gluten-dependent diabetes-related and thyroid-related autoantibodies in patients with celiac disease." *J. Pediatr* 2000; 137: 263-5.

4. Fine, Kenneth, (n.d.). Early Diagnosis of Gluten Sensitivity, Retrieved October 20, 2009 from https://www.enterolab.com/StaticPages/EarlyDiagnosis.html

The Prevalence of Sensitivity to Wheat in Patients with Chronic Health Problems – Based on Applied Kinesiology Testing

Donald C. Baker, D.C., NMD

Abstract

A retrospective study examining the files of 100 patients tested for wheat sensitivity found that 85% were sensitive to wheat as determined by applied kinesiology (AK) testing techniques. The patient population studied consisted primarily of people with chronic health problems.

Key Indexing Terms
Wheat, Gluten, Applied Kinesiology, Prevalence, Gliadin, Sensitivity

Discussion

Examining the prevalence of sensitivity to wheat determined by laboratory testing and comparing this to the prevalence of sensitivity to wheat via AK procedures can be informative. If both laboratory methods and AK methods are accurate, then the percentages of positive individuals (testing sensitive to wheat) determined by either method should be roughly in agreement. This paper will examine the second part of this question -- the prevalence of sensitivity to wheat as determined via AK testing.

In this retrospective study, 100 patient files of one AK practitioner (this author) wherein wheat had been tested using applied kinesiology techniques were examined. Files were selected at random. Only those files wherein testing had been conducted within the last 2 years (using the latest testing techniques) were used; older files were not included in the 100 cases studied. AK testing techniques used are described in detail in reference [2]. These testing methods consisted of several prerequisite steps to correct dysfunctional signals from stress receptors, acupuncture points, neurolymphatics, and referred pain zones before wheat is tested. Unless these prerequisite steps are employed, large numbers of false negative results will be obtained in AK testing for wheat sensitivity.

The majority of the people in these files had originally presented with chronic health problems -- since this is the nature of this medical practice. These range from more serious chronic health issues such as diabetes, lupus, colitis, and ataxia to less serious chronic conditions such as dyspepsia, fatigue, migraine, and weight gain.

Results

This study indicates that 85% of patients studied were sensitive to wheat based on testing via applied kinesiology methods.

In another article by this author [1], the various laboratory testing methods for wheat sensitivity have been discussed. The most sensitive laboratory testing methods yet developed appear to be those from Enterolab in Dallas, Texas. The results of this study are relatively consistent with laboratory results from Enterolab. No detailed counting of positive and negative test results has been done by Enterolab personnel as of this writing. However, the people at Enterolab generously volunteered that in their patient population approximately 80% of people tested are positive for fecal antigliadin antibodies (i.e., wheat sensitivity).

	AK testing methods	**Enterolab fecal testing methods**
% wheat sensitive	**85%**	**80%**

In both the patient population studied above via AK testing methods and in the Enterolab patient population, data are skewed toward people who are "sick." It would be reasonable to expect that testing the general population would yield a lower percentage showing sensitivity to wheat since many of these people are "not sick." Indeed, other data from Enterolab indicates that this may be the case. [3]

Conclusions

Based on AK testing of a patient population consisting primarily of people with chronic illness, 85% were found to be sensitive to wheat. These results are relatively consistent with the most up-to-date laboratory testing methods.

References

1. Baker, D., The prevalence of sensitivity to wheat based on available laboratory data, ICAK-U.S.A. Proceedings (in press)

2. Baker, D., Wheat/Prolamin Sensitivity, ICAK-U.S.A. Proceedings (in press)

3. Fine, Kenneth, (n.d.). Early Diagnosis of Gluten Sensitivity, Retrieved October 20, 2009 from https://www.enterolab.com/StaticPages/EarlyDiagnosis.html

Wheat/Prolamin Sensitivity

Donald C. Baker, D.C., NMD

Abstract

Wheat/prolamin sensitivity is an important factor in chronic disease. This paper reviews the impact of wheat/prolamin in chronic disease and discusses laboratory and AK methods for testing and treating wheat/prolamin sensitivity.

Key Indexing Terms
Gluten, Prolamin, Applied Kinesiology, Autoimmune, Chronic Illness, Celiac

Discussion

Part I in this paper will discuss the significance of wheat/prolamin sensitivity in chronic disease. Part II will discuss testing methods (laboratory and AK methods) with emphasis on an improved AK method for testing. Also included are comments on treatment.

Part I: <u>The Negative Health Consequences of Wheat and Similar Grains</u>

Celiac disease (CD) is a disease of the small intestines that was first associated with the ingestion of wheat by research conducted in the 1940's. It is said to be present in 0.5-1.0% of the American population. Symptomatic celiac patients may have failure to thrive (in children), abdominal pain and bloating, fatigue, and chronic diarrhea.

CD is an auto-immune disease, i.e., the immune system is signaled to produce antibodies which result in damage to the small intestines. Gluten, a protein within the endosperm of wheat, is thought to be the substance responsible for turning on this abnormal immune system response. More specifically, gluten is composed of two primary groups of proteins, the gliadins and the glutenins; it is the gliadins that have been implicated in most of the scientific research as triggering the abnormal immune system response.

Wheat is a member of the grass family. Other members of the grass family also are associated with CD, including rye and barley. Oats had been thought to be associated with CD but in more recent research, the connection with CD has not been shown. Some authors believe that once CD is present, oats may exacerbate the condition. Yet another important study found that most sources of oats are contaminated with wheat, rye, or barley residues.

Some people develop CD early in life and others develop it later in life. One theory suggests that triggers are necessary to activate or create CD in susceptible individuals. Thus, a person may be free of disease for many years, be subjected to certain conditions that trigger the disease, and then develop CD. The triggers for the disease are not known, however theories as to the triggers include viruses and *Candida albicans*.

The common diagnostic methods used for CD involve biopsy of the small intestines and/or blood testing for antibodies (IgA antibodies against endomysium (EMA) or tissue tranglutaminase (TTG)). It is the antibody findings in CD which characterize CD as an autoimmune disease. It should be noted that in some cases of CD, these antibodies are not found. Research in the last few years shows these blood tests for celiac disease are producing a large percentage of false negative results.

There is no effective treatment for CD other than the avoidance of offending grains. Most CD patients return to normal with the avoidance of wheat, rye, barley and similar grains. Some do not.

A number of other diseases have been associated with wheat and its grass relatives. One of these is the itchy skin disease know as dermatitis herpetiformis. Elimination of wheat and similar grains usually results in dramatic improvement or elimination of the condition. But, less well-known and much more compelling is the story of Hashimoto's thyroid disease.

Thyroid disease is wide-spread in our population, particularly among middle-aged women. Since the thyroid gland is responsible for establishing metabolic rate, subnormal thyroid function can lead to weight gain, fatigue, hair loss, constipation, depression and a host of other symptoms. Hashimoto's thyroid disease is an autoimmune disease of the thyroid. Some authors have suggested that Hashimoto's thyroid disease represents over 50% of all thyroid disease.

When Hashimoto's thyroid disease is suspected, two blood tests can be done to measure thyroid antibodies: thyroid peroxidase (TPO) antibodies and thyroglobulin (TG) antibodies. A positive result for either of these antibodies indicates that the immune system has become dysfunctional with respect to the thyroid

Hashimoto's thyroid disease usually is a slowly progressing disease. As with many organs in the human body, considerable damage can occur to the thyroid gland and yet the gland can still produce adequate thyroid hormone. Thyroid antibodies (TPO or TG) may be present, but the measurements of thyroid output (tests such as T4 and TSH) can be within normal limits. There comes a point, however, where the damage done to the thyroid is so great that adequate hormone production is no longer possible—and measurements of T4 and/or TSH become abnormal.

Could it be that Hashimoto's thyroid disease, an autoimmune disease, also is associated with wheat? To study this, researchers placed 13 children who tested positive for TPO antibodies on a wheat-free/gluten-free diet. After 6 months. only 10 of the 13 tested positive for TPO antibodies. After 12 months, 6 instead of 13 tested positive for TPO antibodies. After 24 months, 2 instead of 13 tested positive for TPO antibodies. [1]

In the same study, the pancreas and antibodies related to Type 1 diabetes were examined. In 11 children with pancreas-related antibodies, after 6 months of a wheat-free/gluten-

free diet, 5 instead of 11 were positive for antibodies. After one year, none were positive for pancreas-related antibodies. [1]

What other illnesses and conditions are related to the consumption of wheat and similar grains? An excellent treatment of this question was presented in Reference 2. Reference 2 lists over 50 diseases and conditions that have been associated in the scientific literature with consumption of wheat and related grains. A few of these are listed below. Notice that the conditions range across the entire spectrum, from simple headaches and dyspepsia to more serious conditions such as multiple sclerosis, dementia, and diabetes. It is also instructive to note that many of the conditions are autoimmune diseases.

Anxiety and Depression
Arthritis
Ataxia
Brain Atrophy
Brain White-Matter Lesions
Systemic Lupus Erythematosus
Cerebellar Atrophy
Crohn's Disease
Dementia
Scleroderma
Dyspepsia
Epilepsy
Sarcoidosis
Headaches/Migraines
Irritable Bowel Syndrome (IBS)
Infertility/Miscarriage
Type I Diabetes Mellitus
Multiple Sclerosis
Pancreatic Disorders/Exocrine
Polymyositis
Thyroiditis

Recognize that illness can have more than a single cause. Gluten-related grains are but one cause for some of these illnesses. For example, headaches and migraines have many causes, with gluten-related grains being only one of these causes.

The full extent to which wheat and other grains contribute to illness and disease remains under study. Some of the diseases and conditions related to wheat require several factors (wheat being only one) to come together before the disease or condition manifests.

Laboratory studies provide very interesting data on the prevalence of wheat related sensitivities. Enterolab reports concerning their laboratory tests of fecal IgA antigliadin antibodies as follows: [3]

1) 77% of people with autoimmune disease are positive for fecal IgA antigliadin antibodies.
2) 76% of people with microscopic colitis (a disease of the colon) are positive for fecal IgA antigliadin antibodies.
3) 57% of patients with irritable bowel syndrome-like abdominal symptoms are positive for fecal IgA antigliadin antibodies.
4) 29% of people (N=227) with no symptoms (no currently identified health problems) are positive for fecal IgA antigliadin antibodies.
5) A detailed retrospective study has not been done of all people (sick and not-so-sick) tested via fecal IgA antigliadin antibody testing methods. However, it is estimated that 80% of all people tested via the most recent fecal testing

> methods are positive. Note that this number is skewed toward "sick" people since it is mostly "sick" people who seek testing. [e-mail from Enterolab, 9/5/2009]

These data are extremely alarming. First, let's look at autoimmune disease. There are millions of Americans with autoimmune disease. Most have no idea that their disease may be caused by or related to wheat and related grains. These data show that at least 77% (maybe more because of the limitations of testing) are sensitive to wheat.

Now examine the data for microscopic colitis. Because of outdated medical information, many people with microscopic colitis have been told by their physicians that the disease has nothing to do with food. Yet, these data show that at least 76% are sensitive to wheat. In a later phase of this study 19 of 25 patients with microscopic colitis had their chronic diarrhea resolve completely with a grain-free diet; 5 of the 25 had their chronic diarrhea notably improved; 1 did not.

Then examine the data for the very common condition known as Irritable Bowel Syndrome. The data show that at least 57% of these people are sensitive to wheat -- and yet most doctors continue to ignore food in treating this condition.

In the healthy general population, how many millions are symptom-free right now but slowly developing disease? Many chronic diseases are slow to develop, sometimes requiring years to manifest. These laboratory test data indicate that 29% of "healthy" people may be starting the process of developing disease due to wheat sensitivity.

As has been noted, the protein groups in wheat that make dough have its sticky character are called gliadins and glutenins. Consequently, one definition of gluten (let's call it Definition 1) would be a combination of gliadin and glutenin proteins. Anything that contained gliadins and glutenins could be said to contain gluten.

Some people use the term "gluten" to refer to the proteins in grains that are clearly established to cause celiac disease. Using this definition (let's call it Definition 2), wheat, rye, barley, would be said to contain gluten. In many scientific papers and non-scientific publications you will see references to the "gluten" contained in wheat, rye and barley.

Other scientific and non-scientific papers refer to grains such as wheat, rye, barley, oats, millet, sorghum, corn and rice as containing gluten in various amounts (we will call this Definition 3).

So here we have at least three different definitions of gluten -- or 3 different statements as to which grains contain gluten. What we should do is look more closely at the similarities and differences between these grains.
In this paper, we will use the first of these definitions (Definition 1) because we think it is the most informative and the most scientifically rigorous. Gluten we will define as consisting of the glutenin and gliadin proteins in wheat. Of these two, it is the gliadin

that most concerns us. The gliadin proteins are the ones most implicated in disease. But what exactly are these gliadin proteins?

Gliadins are examples of prolamins, proteins that contain high concentrations of the amino acid proline. There are other prolamins besides gliadin. Rye, barley, oats, corn, rice, sorghum, and millet contain similar but different prolamins. For example, one scientific reference states,

> "Barley (*Hordeum vulgare)* and rye (*Secale cereale)* are closely related to wheat and are classified in the same botanical Tribe, the Triticeae. Although it is not possible to make gluten from these species, both contain groups of prolamins, called hordeins, in barley and secalins in rye, which are related to gliadin and glutenin components of wheat. Neither of these grains contains gliadin. But, both of these grains contain protein groups that are very similar to gliadin." [4]

Prolamins are proteins in cereal grains and seeds containing high concentrations of proline (and glutamine) that are insoluble in water, but which generally are soluble in high concentrations of alcohol. Strictly speaking then, rye and barley don't contain gluten (based on Definition 1) because they do not contain gliadin.

It is clear that health issues related to grains are not limited to wheat. For example, rye and barley are clearly linked to celiac disease. We should focus our attention not only on gliadin and wheat, but also on the other grains that contain similar prolamins.

The names of the prolamins found in various cereal grains are as follows:

Grain	Prolamin contained in the grain
Wheat	gliadin
Rye	secalin
Barley	hordein
Oats	avenin
Millet	panicin
Sorghum	kafirin
Corn	zein
Rice	orzenin

All of these grains are in the Gramineae family (grass family). Some are more closely related to wheat than others (see below). Rye and barley are the most closely related to wheat and can very commonly be a factor in chronic illness. By contrast, rice contains only small amounts of prolamins and is more distantly related to wheat.

Grain	Belongs to the tribe:
Wheat	Triticeae
Rye	Triticeae
Barley	Triticeae
Oat	Aveneae

Millet	Paniceae
Sorghum	Andropogoneae
Corn	Andropogoneae
Rice	Oryzeae

We are faced with a situation where wheat and other grains can be the cause or a factor in very serious chronic illness. At the other extreme we have people who are not sensitive at all. And, to make the situation more complex, we have gradations between these extremes where people are sensitive to grains but without the most serious of the diseases we have discussed.

Some people are sensitive only to wheat, rye, and barley. Some are sensitive to wheat, rye, barley, oats, millet, and sorghum. And, some people are also sensitive to corn or rice. These people have a prolamin problem, not just a gluten problem.

Part II: <u>Testing for sensitivity to wheat/prolamins</u>

Laboratory Testing

Only in the last few years has a laboratory test for wheat sensitivity become available that appears relatively accurate. This is the fecal antigliadin antibody test available from Enterolab.

One difficulty with laboratory tests relates to the history of testing for celiac disease. Yes, we are concerned with people who have celiac disease but we are also concerned with the vastly larger number of people who do not have celiac disease but have other diseases associated with sensitivity to wheat/prolamins. The gold standard of testing for celiac disease has been the intestinal biopsy to look for villous atrophy. Other tests related to celiac disease include:

1) <u>Serum IgA anti-endomysial antibody test:</u> This is a blood test used for people who have celiac disease. It yields a positive result when there has been substantial damage to the small intestines. It is negative when there is not significant damage to the small intestines. Therefore it is of limited value for us in evaluating sensitivity to wheat in non-celiac wheat and grain-related chronic illness because the test will usually be negative. It is also of limited value in detecting celiac disease at its early stages when malabsorption and abnormal intestinal permeability is occurring in the small intestines, but wide-spread damage to intestinal villi has not yet occurred. The test can be of value in monitoring patient compliance with a wheat/prolamin-free diet.

2) <u>Serum IgA or IgG tissue transglutaminase antibody test</u>: This is a blood test used for people who have celiac disease. It yields a positive result when there has been substantial damage to the small intestines. It is negative when there is not significant damage to the small intestines. Therefore it is of limited value for us in evaluating sensitivity to wheat in non-celiac wheat and grain-related chronic illness because the test will usually be negative. It is even of limited value in

detecting celiac disease at its early stages when malabsorption and abnormal intestinal permeability is occurring in the small intestines but wide-spread damage to intestinal villi has not yet occurred. The test can be of value in monitoring patient compliance with a wheat/prolamin-free diet.

3) Serum IgA antigliadin antibody test: This test is considered to have greater sensitivity as compared to the two tests mentioned above. However, as you will see in the data presented below, the sensitivity of this test is very poor compared to the fecal IgA antigliadin antibody test (also discussed below).

Beyond the tests shown above for celiac disease, other tests have been used over the years for wheat/prolamin sensitivity. Two of these are as follows:

4) Salivary IgA antigliadin antibody test: Some laboratories now are specializing in salivary testing -- particularly for free-fraction hormone testing. Salivary testing can identify some people with sensitivity to wheat, however, the sensitivity of these tests appears to be low as compared to the fecal IgA antigliadin antibody test discussed below.

6) IgE allergy tests for wheat: IgE testing can identify some patients who are allergic to wheat. However, IgE reactions are not the mediator of the disease process in most people who are sensitive to wheat. Therefore, this test is of very limited value for our purposes.

In the last few years, Enterolab has developed fecal IgA antigliadin antibody tests. These tests are conceptually based on the idea that since IgA antibodies to gliadin are produced in the intestines, antibodies to gliadin might best be detected if tests for antibodies in the intestines could be done (rather than from the blood). When fecal and serum IgA antigliadin antibody tests are compared in the same groups of people, far better sensitivity is found using the fecal IgA tests, as follows: [3]

Test Subjects	% positive in Serum antigliadin antibody tests	% positive in Fecal antigliadin antibody tests
CD patients	76%	100%
Microscopic colitis patients	9%	76%
"Normal" people	11%	29%

Notice than even in confirmed celiac disease patients, the serum IgA antigliadin antibody test was negative (i.e., wrong) 24% of time. Therefore, we conclude that the serum test is not even adequate in all cases of celiac disease. In other disease conditions, as the data illustrate, it is also greatly inferior to the fecal test.

Applied Kinesiology Testing

At some point many years ago, it was realized that results obtained by this author using basic AK procedures were probably under-predicting the number of people with wheat/prolamin sensitivity. This led to an effort to determine why this might be the case and to improve AK testing procedures.

The AK Synopsis states that an important initial step in an AK evaluation is the Injury Recall Procedure (IRT). [5] Schmitt and others have elaborated the wide array of applications for the IRT procedures, including prior physical injuries, neurolymphatics, and TMJ procedures.

If you view the AK patient as analogous to a digital computer, some of the corrections we make early in a session (such as via IRT procedures) might be thought of as correcting computer functions so that results obtained subsequently would be more accurate. Indeed, the procedures that will be recommended here are consistent with this concept.

The mediator for most (and maybe all) of the diseases and conditions related to wheat sensitivity is the small intestine. If you test using the IRT procedure and apply it to the small intestines, this is what you will find:

1. If you apply the IRT procedure to the quadricep stress receptor (recall quadricep is small intestine-related), you will find that many times the test is positive.
2. If you apply the IRT procedure to SI 4 (the source point for the SI meridian), you will find that many times the test is positive
3. If you apply the IRT procedure to SI 3 (the tonification point for the SI meridian), you will find that many times the test is positive
4. If you apply the IRT procedure to SI 1 and SI 19 (the beginning and end points) simultaneously, you will find that many times the test is positive.
5. If you apply the IRT procedure to the anterior neurolymphatic for the quadriceps muscle, many times the test is positive.
6. If you apply the IRT procedure to the posterior neurolymphatic for the quadriceps muscle, many times the test is positive.
7. If you apply the IRT procedure to the referred pain zone for the small intestines (around the umbilicus), many times the test is positive.

Correction can be made to each of the findings in 1-7 by applying the standard IRT procedures of the AK Synopsis. If wheat or other prolamins are tested following these steps, higher percentages of positive results will be found

The process noted above is analogous to resetting circuit breakers in an electrical circuit. Each of the items 1-7 is analogous to a circuit breaker. Once these “circuit breakers” are reset, it appears that recognition of substances harmful to the small intestines is much improved. It is as if this system is “on” and “monitoring” when its circuit breakers are reset.

The order listed for items 1-7 is useful. For example, sometimes items 2-7 will not show as needing correction unless item 1 (stress receptors) is first corrected using the IRT correction procedure. As you try these procedures you may also see that correction to acupuncture points before neurolymphatics sometimes is necessary in order for neurolymphatics to test positive. The order of posterior and anterior neurolymphatic testing and correction usually is not important.

Be aware that the IRT correction procedures can be applied to items 1-7 without first doing the IRT testing of these items. However, since the testing provides useful information to the doctor about the status of the system, you will usually want to do the testing.

In some cases other related corrections are necessary. You can use your AK skills to identify these in stubborn cases. For example, sometimes the IRT procedure needs to be applied to the quadriceps muscles itself. You may also find it necessary on occasion to apply the IRT procedure to the anterior and posterior abdominal muscle neurolymphatic reflexes (since these are related to small intestines). Sometimes you may find it necessary to apply the IRT procedure to the sagittal suture (this suture has been found in AK studies to be related to the small intestines).

When you have finished the IRT procedure and begin testing prolamins, you must consider that on some occasions testing will cause hypertonicity rather than inhibition of muscle response. Be aware of this possibility and check for it when necessary. It will happen less often when you make the IRT corrections first.

This procedure for enhancing the accuracy of testing for wheat allows you to test for sensitivity to the other prolamins more accurately. Most of the time barley and rye will test just like wheat; if a person is sensitive to wheat, they are most likely sensitive to barley and rye also. Many times a person sensitive to wheat will test as sensitive to oats (and even oats marketed as uncontaminated with wheat, so-call “gluten-free” oats). Many people will test as sensitive to sorghum and millet. Some people will test sensitive to corn and some will test sensitive to rice.

The ability to test the other grains containing prolamins (those other than wheat) is one of the advantages of AK testing over the laboratory test of fecal IgA antigliadin antibodies (which test only wheat). The laboratory test provides no information concerning the other grains. Being able to help patients determine the full scope of their grain sensitivity problem is very helpful.

Other existing AK procedures were examined before settling on this approach to testing. For example, the various “switching procedures” were studied and questions of hypertonicity when testing wheat were examined. No other procedures have yet been found that are adequate prerequisites for testing for wheat sensitivity.

Using this procedure, a retrospective study of 100 patients tested over the last 2 years with AK procedures was conducted. This study is discussed in more detail separately. [6]

Eighty-five percent (85%) of those tested were found sensitive to wheat. By way of comparison, laboratory testing of a similar population finds approximately 80% sensitive to wheat. [7] Both populations were skewed toward a higher percentage than might be observed in the general population because people with chronic illness tend to have higher rates of sensitivity to wheat and these people also seek naturopathic care and lab testing more frequently than people who are healthier.

The concept for the procedure describe herein can be applied to other systems (e.g., large intestines, stomach, liver, etc.). For these other systems, use the applicable stress receptors, acupuncture points (source, tonification, beginning and end points), neurolymphatics, and referred pain zones. You will find that after making the necessary IRT corrections, your findings when testing factors relevant to those system will be improved. Furthermore, if you first make all the necessary IRT system corrections to all systems requiring corrections, the accuracy of all your subsequent testing of stressors and nutritional supplements will improve.

Patients with prolamin sensitivity are advised to completely avoid the prolamins to which they are sensitive. Since this often is very difficult for patients, it is helpful if the doctor can provide guidance on how a person can do this while still finding satisfying foods to eat.

Therapies directed toward "resetting" the nervous system so that these foods will no longer be a problem generally are not effective; they may appear initially to be helpful, but "resetting" procedures do not hold. This may be because for these foods the problem is usually autoimmune in character rather than an allergic reaction.

Procedure

To improve the accuracy of testing for wheat/prolamin sensitivity:

1. Test the quadricep stress receptors using the Injury Recall Test (IRT) procedure. Make the IRT correction if positive.
2. Test SI 4 using the IRT procedures and correct if positive.
3. Test SI 3 using the IRT procedures and correct if positive.
4. Test SI 1 and SI 19 simultaneously using the IRT procedures and correct if positive.
5. Test the anterior neurolymphatic for the quadriceps muscle using the IRT procedures and correct if positive.
6. Test the posterior neurolymphatic using the IRT procedures and correct if positive.
7. Test the referred pain zone for the small intestines (around the umbilicus) using the IRT procedures and correct if positive.

8. Test for sensitivity to wheat and other prolamin containing grains. As always when testing for sensitivity to a substance, you must consider that on some occasions exposure to the substance will cause hypertonicity rather than inhibition of muscle response. Be aware of this possibility and check for it.

These steps for the small intestines are basic. To further improve the accuracy of testing, apply the same procedure to all other systems (adrenals, thyroid, pancreas, LI, stomach, etc) before testing for wheat/prolamin sensitivity.

References

1. Ventura, A., Neri. E., Ughi C., et al Gluten-dependent diabetes-related and thyroid-related autoantibodies in patients with celiac disease. *J. Pediatr* 2000; 137: 263-5.

2. Helms, S. Celiac Disease and Gluten-Associated Diseases, *Alternative Medicine Review*, 2005, Volume 10, Number 3, 172-192.

3. Fine, Kenneth, (n.d.). Early Diagnosis of Gluten Sensitivity, Retrieved October 20, 2009 from https://www.enterolab.com/StaticPages/EarlyDiagnosis.html.

4. Marsh, M., Celiac Disease: Methods and Protocols, (Totowa, NJ, Humana Press, 2000), p. 57.

5. Walther, D, Applied Kinesiology Synopsis, 2nd Ed (Shawnee Mission, KS, ICAK-U.S.A., 2009), p. 184.

6. Baker, D., The Prevalence of Sensitivity to Wheat in Patients with Chronic Health Problems -- based on Applied Kinesiology Testing, ICAK-U.S.A. Proceedings, (in press)

7. Baker, D., The Prevalence of Sensitivity to Wheat based on available Laboratory Data, ICAK-U.S.A. Proceedings, (in press)

Analysis of Central Nervous System ATP Production with Applied Kinesiology Manual Muscle Testing

Richard Belli, D.C., DACNB

Abstract

Applied Kinesiology manual muscle testing provides a tool for evaluating ATP producing pathways in the central nervous system. Specifically, unilateral supraspinatus inhibition provides a means for evaluating central nervous system ATP pathways. There are reflexes associated to glycolytic pathways, the Krebs cycle, and the electron transport chain that provide useful a window for evaluation. Methods and protocol for evaluating and correcting dysfunction of ATP production pathways are described.

Key Indexing Terms

Applied Kinesiology (AK), Supraspinatus, ATP production

Introduction

Adenosine triphosphate (ATP) has many important roles in the central nervous system. In addition to providing high energy phosphate bonds for cellular energy it has other functions. ATP modulates communication among neurons and between neurons and support calls called glia. Signaling by ATP and its breakdown product adenosine is also involved in sleep, memory, learning, movement and other brain activities. All considered ATP is a major player in the health and function of the central nervous system.

The brain relies almost entirely on glucose for fuel, which is ultimately converted to ATP by three different but coupled pathways. These include anaerobic glycolysis when oxygen is not adequately available, or aerobic glycolysis, the Krebs cycle and electron transport chain when adequate oxygen is available.

In the Applied Kinesiology literature the supraspinatus muscle is associated to brain function.1 Based on recent findings by the author, unilateral supraspinatus inhibition implies that there is dysfunction of one or more ATP production pathways in the brain. It has also been observed by the author that unilateral inhibition of the supraspinatus can be temporarily facilitated by ingesting a small amount of sugar, or breathing pure oxygen. These observations have led to the hypothesis that unilateral supraspinatus inhibition is associated to dysfunction of one or more of the central nervous system ATP production pathways.

It has also been observed by the author that when sugar facilitates the supraspinatus muscle, so does therapy localization to the brain neurolymphatic reflex (NL) located at the posterior lateral aspect of C-1 vertebra.2 Additionally, the author observed that when

oxygen facilitated the supraspinatus muscle either therapy localization to the posterior tubercle of C-1 or the heart NL would facilitate.3 Since re-breathing also facilitates when the heart neurolymphatic TL's it suggests dysfunction of the Krebs cycle.4 This leaves the posterior tubercle of C-1 as the associated point for the electron transport chain.

The unilateral relationship between the supraspinatus may be explainable with the hemisphericity that humans acquired from our brachiating ancestors that swung from the trees. Grasping one limb after another requires the arms to act independently instead of in unison. Since brachiating may be done for extended periods of time may explain the relationship of ATP production and unilateral inhibition.5

Clinical trials revealed treatment methods for each of the three reflexes and normal facilitation for the supraspinatus muscle. When the supraspinatus muscle is facilitated by therapy localization (TL) to the brain glycolytic pathway NL on the posterior lateral aspect of the atlas it can be corrected by injury recall to the maxilla bone. The author speculates that this particular correction affects the hypothalamus, thus the glycolytic pathways. Furthermore, when the supraspinatus is refacilitated by TL to the posterior tubercle of C-1 it can be corrected by visceral neurolymphatic treatment. And finally, when the supraspinatus is facilitated by TL to the heart NL, IRT to the mastoid process is the method of correction.

The oxidative ATP pathways are easily uncoupled by inflammatory cytokines and free radical activity. Visceral dysfunction in particular may be producing cytokines that are uncoupling the electron transport chain and Krebs cycle. Visceral cytokine uncoupling explains why neurolymphatic treatment is one of several effective modalities used in correcting ATP pathway dysfunction. Nutrient deficiency or over exercising may cause excess free radical build up and uncoupling. Lifestyle changes in both exercise and eating habits will be necessary to maintain long term correction.

The purpose of this paper is to present techniques in which dysfunctional ATP production pathways can be identified and corrected using Applied Kinesiology manual muscle testing and natural therapies.

Discussion

High metabolic activity and high oxygen consumption characterize cerebral metabolism. A high constant supply of energy is necessary for the support of neural and neurologic function. These vital energy-dependent processes include the establishment of membrane potentials, maintenance of transmembrane ionic gradients, membrane transport, and the synthesis of cellular constituents such as proteins, nucleic acids, lipids and neurotransmitters.

The energy needed is supplied in the form of high-energy phosphate bonds from ATP, which is synthesized in the brain, as in other organ systems, through glycolytic pathways, the Krebs cycle, and the electron transport chain.6 Glucose is the basic substrate for brain metabolism. The central nervous system uses virtually no fat for energy. The vertebrate brain is completely dependent on glucose for energy metabolism.7 In the presence of oxygen, oxidative phosphorylation is by far the most important mechanism of

ATP synthesis in the central nervous system.8 Oxidative phosphorylation takes place in glycolytic pathways as well as the Krebs cycle. ATP is synthesized in the mitochondria of the neuron via the process of the Krebs cycle and oxidative phosphorylation in the cytoplasm via glycolysis.9

Studies have shown that the ATP producing capability of the neuron operating at a basal rate is operating at near maximal capacity of the oxidative Krebs cycle and electron transport pathways. When the neuron undergoes activity it needs to utilize the glycolytic pathway for ATP production or a short term supply of work energy.10 Therefore, when the subject has an immediate problem to solve, a relatively short term increase of ATP production for the task may temporarily come from the oxidative glycolytic pathway, but when the brain is doing mundane work, maintenance, or just idling the lions share of ATP comes from the slower responding, yet more efficient Krebs cycle and electron transport chain.

The glycolytic pathways are highly dependent on B-1, B-2, and B-3. The Krebs cycle is dependent on multiple B-vitamins, manganese, and lipoic acid for proper function. Function of the electron transport chain depends on Iron, coenzyme-Q10, and B-vitamins and copper. Any of the above mentioned cofactors that are deficient or inadequate can dramatically affect ATP production.

Inflammatory cytokines and free radical activity are known to uncouple ATP production pathways. In addition to supplying vitamin and mineral cofactors for treatment, it may be necessary to address inflammation and free radical pathology. One of the most common sites of uncoupling cytokine production is the large intestine. When the large intestine is involved, there is usually an issue with dysbiosis or leaky gut. Other viscera can also be involved, but much less frequently than the GI tract. Chronic soft tissue inflammation from injury or immune system dysfunction can also be a source of uncoupling chemicals, making it important to address the immune system and other sources of inflammation. And finally, free radical activity can come from simple deficiency of anti-oxidants, and over exercising.

Procedure

ATP production dysfunction is determined to exist when there is unilateral inhibition of the supraspinatus muscle. It is important to differentiate between bilateral and unilateral inhibition. Bilateral indicates a different dysfunctional process that is addressed in another paper. Once unilateral supraspinatus inhibition has been ascertained, therapy localize the brain neurolymphatic reflex at the posterior lateral aspect of C-1, if the supraspinatus is refacilitated it indicates glycolysis dysfunction. Proceed to perform IRT to the maxilla bone. If TL to the brain neurolymphatic reflex does not facilitate TL the posterior tubercle of T-1. If TL to the posterior aspect of T-1 facilitates proceed to TL visceral NL's until one is found that facilitates the supraspinatus. And finally, if TL for glycolytic and oxidative phosphorylation pathways do not facilitate, TL the heart NL. If the heart NL facilitates TL the mastoid processes. If a mastoid process facilitates correct with IRT. If supraspinatus inhibition is not present and dysfunction is suspected, the glycolytic pathways can be challenged by having the patient do a mental task such as a

math problem. The oxidative pathways are easily challenged by having the patient hold their breath. These challenges are very important and effective in bringing out hidden dysfunction.

- **Clinical procedure outline**
  - Unilateral supraspinatus inhibition
    - Anaerobic pathways
      - Brain NL TL at lateral aspect of C-1 facilitates supraspinatus
        - Bilateral Maxilla IRT to correct
    - Oxidative phosphorylation
      - Brain stem NL TL at posterior tubercle of C-1 facilitates supraspinatus
        - Treat NL that abolishes TL
    - Krebs cycle
      - Heart NL TL facilitates supraspinatus
        - Treat cranial fault that abolishes TL, usually mastoid process
  - Challenge ATP pathways by having the patient do a math problem in their head and/or having them hold their breath 20-30 seconds

- **Nutritional Considerations**
- B-vitamin and mineral cofactors
  - B1, B-2, B-3
  - Manganese
  - Iron
  - Copper
  - Lipoic Acid
  - Coenzyme Q-10
- Antioxidants
  - C
  - E
  - Glutathione
- Products that inhibit CNS inflammation

Conclusion

It has been observed that correcting ATP production in the central nervous system has profound effects in many aspects of brain and central nervous system function. Even very minor shifts away from normal levels of ATP production can have significant effect on mood, mental energy, productivity and overall sense of well being. These techniques for evaluating and correcting ATP production have provided a source of profound positive feedback from patients. Applied Kinesiology manual muscle testing provides a valuable tool for evaluating and correcting central nervous system pathways.

References

1. Walther, DS. Applied Kinesiology Synopsis. 2nd. ed. Shawnee Mission, KS. ICAK-U.S.A.; 2009, pp. 348

2. Walther, DS. Applied Kinesiology Synopsis. 2nd. ed. Shawnee Mission, KS. ICAK-U.S.A.; 2009, pp. 348

3. Walther, DS. Applied Kinesiology Synopsis. 2nd. ed. Shawnee Mission, KS. ICAK-US.A.; 2009, pp. 353

4. McCord KM, Schmitt WH. Quintessential Applications A(k) Clinical Protocol. 1st. ed. St. Petersburg, FL: Health Works!; 2005. Sections 12.

5. Sweeney, Michael S. Brain The Complete Mind. Washington D.C.: National Geographic; 2009. pp.70

6. Westmorland, Barbara F. Medical Neurosciences, 3rd. ed. Boston: Little Brown; 1994. pp. 286

7. Shepard, Gordon M. Neurobiology. 3rd.ed. New York: Oxford University Press; 1994. pp.189

8. Salway, JG. Metabolism at a Glance. Malden MA.: Blackwell Science; pp.12

9. Beck, Randy W. Functional Neurology for Practitioners of Manual Therapy, 3rd. ed. Edinburgh: Churchill Livingstone; 1998. pp.58

10. Beck, Randy W. Functional Neurology for Practitioners of Manual Therapy, 3rd. ed. Edinburgh: Churchill Livingstone; 1998. pp.59

Evaluating the Extra Thalamic Cortical Modulating System with Applied Kinesiology Manual Muscle Testing

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Abstract

The author has observed that patients frequently therapy localize to all B&E acupuncture head points. The author postulates that multiple head point TL indicates dysfunction of the Extra Thalamic Cortical Modulating Systems. There are six neurochemically distinct extra thalamic projection systems that reach the cerebral cortex monosynaptically without a relay in the thalamus. The Extra Thalamic Cortical Modulating Systems modulate synaptic functions of the cortex. The Extra Thalamic Cortical Modulating Systems is one of many possible avenues available for identification and correcting neurotransmitter dysfunction.

Key Indexing Terms

Applied Kinesiology (AK), Neurotransmitter Evaluation, Acupuncture B&E Points

Introduction

The author observed that a group of patients would present with therapy localization (TL) to all acupuncture B&E points on the head (head points). This particular therapy localization would disrupt the normal muscle inhibition pattern of gait. In other words, normally inhibited gait muscles become conditionally facilitated (disrupted gait). The head points are significant in this matter, because they are associated to the major neurotransmitters.1

However, it is not logical that all the neurotransmitters associated to the head points are deficient, therefore, it has been postulated that there is a problem with modulation. This brings to light the one system in the brain that would have such an effect, it is commonly referred to as the Extra Thalamic Cortical Modulating System (ETCMS).2 The ETCMS is a group of sub cortical nuclei that have projections to virtually all cortical neurons, modulating them. Interestingly all of the nuclei in the ETCMS correspond to the acupuncture head points.3,4 Additionally, the nuclei of the ETCMS are major production areas of the involved neurotransmitters.5 With that in mind it has been postulated that when dysfunction of the ETCMS is revealed, it may be an indicator of inadequate production of the involved neurotransmitter.

Dysfunction of neurotransmitters is related to production, receptor site sensitivity, or metabolization. Clinical testing ruled out issues with receptor site sensitivity and metabolization, leaving only issues with neurotransmitter production as a possibility in ETCMS dysfunction.

The Extra Thalamic Cortical Modulating System is actually made up of six systems arising from small nuclei or regions of the brain stem and forebrain which project neurons directly to the cerebral cortex without first synapsing in the dorsal thalamus. These projections are often incorrectly referred to as part of the reticular activating system. All six of these extra thalamic projection systems appear to modulate cortical activity. Accordingly, the phrase extra thalamic cortical modulating systems is a better and more accurate description of these systems than the more restrictive phrase “reticular activating system”.6

It has been well established that neurotransmitter production in the central nervous system is compartmentalized and localized.7 Therefore, a neurotransmitter may be normal in one functional area and low in another. Furthermore, that same neurotransmitter may have receptor site sensitivity problems in another, and in yet another be antagonized by a neurotransmitter that is not being properly metabolized. These factors make lab testing for neurotransmitter levels very unreliable.

In an attempt to determine a treatment method for a dysfunctional ETCMS, the author had a serendipitous discovery that voluntary lateral eye movement, saccades, disrupted gait. Voluntary lateral eye movement involves significant cortical activity especially in an area referred to as the frontal eye fields.8 The frontal eye field is believed to be a center for voluntary movements of the eyes independent of visual stimuli. These conjugate eye movements are called “movements of command”, since they can be elicited by instructing the patient to look right or left.9

Apparently lateral eye movement increased cortical activity enough to challenge for adequate modulation from the ETCMS. Further testing demonstrated that TL to one set of head points would normalize gait that has been disrupted from lateral eye movement. This finding would later be determined to be the head points that need be treated to clear TL to all of the head points and normalize lateral eye movement. With these observations in mind the author postulated that treating this primary set of head points corrected the dysfunctional ETCMS and indicated which neurotransmitter is not being adequately produced.

Clinical nutritional correction can involve vitamin and mineral cofactors, as well neurotransmitter support products that commonly contain amino acids and synergistic herbs. There are many well researched and effective neurotransmitter products currently on the market that should be considered.

The purpose of this paper is to present techniques in which dysfunction of the Extra Thalamic Cortical Modulating Systems can be identified and corrected using Applied Kinesiology manual muscle testing and natural therapies.

Discussion

There are over sixty neurotransmitters and neuropeptides found in the cerebral cortex. Of the neurons intrinsic to the cortex the majority are either GABAergic, or glutaminergic. Working in conjunction with this there is a system of extra thalamic projections from lower brain centers that actively modulate cortical synaptic activity. If for some reason there is disruption of glutamate and GABA function and synaptic activity is altered, the extra thalamic cortical modulating system (ETCMS) will attempt to shift cortical synaptic activity towards normal. In an attempt to modulate cortical function other areas of the central neuraxis may be disrupted and misleading neurotransmitter findings may appear.

Glutamate and GABA are the primary neurotransmitters for most neurons native to the cortex. All the efferent neurons, either those projecting to other cortical areas or those projecting to sub cortical areas of the CNS are excitatory and utilize glutamate. Cortical inhibitory signals are generated by GABAergic interneurons. Therefore, overall excitability and inhibition may be controlled by the state of glutamanergic and GABAergic cortical neurons. Inhibitory signals are essential for processing information at the level of the cortex. Nearly thirty percent of cortical neurons use GABA as primary neurotransmitter. Both GABAergic and glutaminergic neurons have fast responding receptors in the cortex. This limits all rapid central signaling to glutamate and GABA. The rest of the neurotransmitters use second messenger coupled receptors which are slow response, and modulation.

Many neurotransmitters are found at the level of the cortex, but they are within nerve terminals of the neurons that project to the cortex from the brain stem and basal forebrain. Most are neurons that belong to the ETCMS. This is a system of chemically defined neurons with each area using only one of the following neurotransmitters, norepinephrine (NE), seratonin (5-HT), dopamine (DA), histamine, acetylcholine (ACh), and gamma-aminobuteric acid (GABA). With the exception of GABA, no neurons intrinsic to the cortex use these neurotransmitters. These chemically defined groups of neurons do not always correspond to the more traditionally anatomically defined brain stem nuclei. These groups of neurons, from six distinct systems, project mono-synaptically to the cerebral cortex without passing through the thalamus, thus the description, extra thalamic cortical modulating system. The synaptic activity of the ETCMS alters the membrane properties of the post synaptic cortical neurons. All six modulate cortical activity by highly divergent single neurons projecting to large far reaching areas of the cortex. Branches of these projecting neurons terminate in nearly all the cortical laminae. The ETCMS does not send specific signals to the cortex, but plays a role strictly of modulation. Ultimately the ETCMS affects the magnitude of response of the cortical neurons to afferent input. Anatomically the ETCMS is located in three areas of the reticular formation, two areas of the hypothalamus and one area of the basal forebrain. These areas include nucleus basalis of the forebrain-ACh, locus ceruleus of the pontine reticular formation-NE, midbrain raphe nucleus-5-HT, ventral midbrain-DA, and two areas of the hypothalamus-histamine and GABA.

In an effort to maintain cortical homeostasis, the ETCMS constantly modulates the cortical response to afferent input. If there is a disruption of the GABA-glutamate balance

there will be altered response and a need for modulation. Conversely, if the ETCMS is not properly modulating there will be false signs of GABA and glutamate dysfunction.

All the areas of the ETCMS project rostral as well as caudal. In the ETCMS's effort to modulate the cortex, the ETCMS may inadvertently facilitate or inhibit caudal areas. This can lead to dysfunction of many other aspects of the neuraxis. When excessive modulation is required it may tax areas of the ETCMS, depleting neurotransmitters, giving the false impression that additional neurotransmitter activity to this area is needed when in reality correction of cortical neurotransmitter activity is what is needed. Addressing GABAergic and glutaminergic activity first avoids false positives for neurotransmitter activity and wide ranging dysfunction of the neuraxis.

Procedure

The procedure is normally done after AK procedures related to injury recall are done. The patient is put in the gait position (one leg advanced forward) and a muscle that would normally be inhibited in the walking gait mechanism, for example, a contralateral extensor such as the latissimus muscle is tested for normal inhibition. Once it has been established that the gait mechanism is functioning normally the patient is instructed to move the eyes laterally several times then bring them back to center. This must be done voluntarily and not following an object, as smooth pursuit introduces more lower brain centers such as the cerebellum.10 If after lateral eye movement the normally inhibited gait muscle strengthens or becomes conditionally facilitated TL GV-15 located just below the spinous process of C-2.11 If GV-15 TL re-facilitates the conditionally inhibited gait muscle it implies that there is ETCMS dysfunction. The next step is to TL the head points until one is found that normalizes gait. Treat by tapping the bilateral set of head points 60-90 times.

Because ETCMS dysfunction is a problem with synthesis of neurotransmitters, nutritional support should always be considered. Take into consideration the vitamin and mineral cofactors as well as many of the fine products on the market for neurotransmitter support

Clinical procedure outline

- Gait is disrupted after voluntary eye lateralization to both sides then back to center- patient looks side to side several times-do not have patient track something
    - GV-15 TL normalizes gait
        - TL for head points that normalize gait
            - Treat by tapping head points that TL normalized gait 60-90 times or until gait is normalized
                - Test vitamin and mineral cofactors and neurotransmitter support supplements associated to the involved head point and related neurotransmitter
    - It is clinically effective to perform nutritional testing before correction, that way if it returns ETCMS dysfunction returns the cofactors can be easily provided

Conclusion

Neurotransmitters are so functionally compartmentalized in the human nervous system that it leaves no known lab testing method effective in determining neurotransmitter dysfunction in all areas.

Evaluation and correction of ETCMS dysfunction provides a method of determining neurotransmitter dysfunction in an area of the nervous system that has a significant functional effect.

It is only Applied Kinesiology manual muscle testing that can provide a simple tool for evaluating and correcting dysfunction of the ETCMS to make such a functionally significant area of the human nervous system.

References

1. McCord KM, Schmitt WH. Quintessential Applications A(k) Clinical Protocol. 1st ed. St. Petersburg, FL: Health Works!; 2005. Sections 4b.

2. Burt, Alvin, M. Textbook of Neuroanatomy. Phileadelphia:W.B. Saunders publishing; 1993, pp. 459-464

3. Burt, Alvin, M. Textbook of Neuroanatomy. Phileadelphia: W.B. Saunders publishing; 1993, pp.460

4. McCord KM, Schmitt WH. Quintessential Applications A(k) Clinical Protocol. 1st ed. St. Petersburg, FL: Health Works!; 2005. Sections 4b.

5. Burt, Alvin, M. Textbook of Neuroanatomy. Phileadelphia: W.B. Saunders publishing; 1993, pp.329

6. Burt, Alvin, M. Textbook of Neuroanatomy. Phileadelphia: W.B. Saunders publishing; 1993, pp.460

7. Kharrzian, Datis. Neurotransmitters and the Brain Manual. Sponsored by the post graduate department of the University of Bridgeport and Apex Energetics. 2008

8. Burt, Alvin, M. Textbook of Neuroanatomy. Phileadelphia: W.B. Saunders publishing; 1993, pp.329

9. Carpenter, M, B. Core text of Neuroanatomy, 3rd. ed. .Baltimore: Williams & Wilkins; pp.385

10. Their P, Uwe J. The Neural Basis of Smooth-Pursuit Eye Movements, Current Opinion in Neurobiology 2005; 15:645-652

11. Walther DS. Applied Kinesiology Synopsis, 2nd ed. Shawnee Mission, KS: ICAK-U.S.A.; 2009. pp. 371.

Analysis of the Brain's Default Mode System with Applied Kinesiology Manual Muscle Testing

Richard Belli, D.C., DACNB and Lawrence Calderon, D.C., MS

Abstract

The brain has a system of slow wave production referred to as the default network. The default network is most active when the brain is doing nothing in particular. Malfunction of the default mode system is associated to at least several neurological maladies. Applied Kinesiology manual muscle testing provides a unique system of evaluating and correcting dysfunction of the default mode system.

Key Indexing Terms
Default Mode, Default Network Applied Kinesiology (AK), Manual Muscle Testing

Introduction

Networks are the in thing for brain scientists. One of the most important, mysterious and well-connected networks of all is called the default mode network. The default mode network is responsible for what the brain does when it is doing nothing in particular. The Brain's default mode network is a series of connected areas that work hardest when most of the brain is at rest. Scientists believe the default network has two major hubs, one in the posterior cingulated cortex and one in the medial prefrontal cortex. In actuality, the default network is the brain's hardest working system. When the brain is at rest there are a series of slow waves that are coordinated fluctuations in activity that slightly stimulates neurons when they aren't active. These syncopations or slow waves continue even while people are asleep, under anesthesia or comas.

Default mode system activation may lead to day-dreaming and mind-wandering. If the default mode system is dysfunctional, it can lead to problems with attention. Evidence suggests that a malfunctioning network is involved with diseases and disorders as diverse as Alzeheimner's, autism, depression, post-traumatic stress disorder, Tourette syndrome, attention-deficit/hyperactive disorder, and maybe may others.

Scientists found that having subjects let their mind wander increased activity in the parts of the brain they refer to as the default mode system. The authors have observed that there is a distinct muscle inhibition pattern that often presents when a patient is asked to close their eyes and let their mind wander. The particular muscle inhibition pattern is bilateral supraspinatus. In Applied Kinesiology (AK) the supraspinatus has been established to be associated to brain function.1 The authors have also observed that when the bilateral supraspinatus inhibition is present, there will be a positive therapy

localization (TL) to the bilateral emotional neurovascular reflexes on the forehead and the external occipital protuberance.

The authors speculate that activity of the default mode system is in sort of a normal channel. In other words the slow waves that are emitted stay within a specific range. If the default mode system is too active the baseline will be above this range, and if it is functioning at too low a level it will be below the range. When the patient performs a mental task such as a math problem, the default mode system has to shut down and get out of the way so they can focus on the task. If the system is functioning too high, then it doesn't go low enough to get out of the way and causes dysfunction. If the default mode system is functioning too low, having the patient think about something in past history, such as a third grade friend, which normally causes increased activity, will not bring the it into the normal functional range, again resulting in dysfunction.

The authors have also observed that when the appropriate mental task is performed, not only will the supraspinatus muscle remain inhibited it will also disrupt normal gait inhibition and facilitation patterns. This particular observation has provided a tool to evaluate and correct default mode dysfunction.

The authors have found that the bilateral supraspinatus inhibition pattern associated to default mode dysfunction does not usually show up without provocation. Provocation or exposure of default mode dysfunction is easily done by having the patient simply close their eyes and let their mind wander. If there is default mode dysfunction a approximately 20 seconds of quiet mind wandering will result in bilateral supraspinatus inhibition.

Clinical trial and error testing has enabled the authors to develop a simple and effective method for correction. Once the bilateral supraspinatus pattern has been ascertained, the patient either does a math problem in their head or thinks about ancient personal history. One of these two challenges will disrupt the normal gait pattern (gait pattern disruption is indicated by changes in the normal inhibition and facilitation pattern of walking gait). Using a conditionally facilitated gait muscle (one that would normally be inhibited) TL the B&E acupuncture points on the head that are associated to neurotransmitters, until a bilateral set are found that normalize the disrupted gait pattern.2 Once these head points have been located, correction is done simply by tapping bilaterally 60-90 times.

The purpose of this paper is to present techniques in which dysfunction of the brain's default mode system can be identified and corrected using Applied Kinesiology manual muscle testing and natural therapies.

Discussion

A default mode of functioning was initially inferred on the basis of observations that certain areas of the brain consistently decrease activity when subjects engage in goal-directed tasks as compared to simply resting quietly with the eyes or visually fixating.3.. When neuroscientists use PET and fMRI scanners to image brain activity they have the subject lie with the brain at rest with their eyes closed or stare at a dot to establish a base line. To tell which areas of the brain become more active during a mental task, scientists compare activity during the task with the established base line. Marcus Raichle of

University of Washington in St. Louis, and others saw that every time a person engaged in a mental activity such as memorizing a list of words, a collection of brain regions consistently decreased activity compared to their resting levels.4 Raichle hypothesized that the network is more active when the brain is at rest and has to dial back its activity to let people concentrate on specific tasks.5 Only when people recall autobiographical memories or imagine alternative situations is the network more active than it is at rest, which is a very important point for clinical assessment.

Michael Greicius a neuroscientist at Stanford University found that while volunteers had their eyes closed ant thought of nothing in particular that the blood flow in parts of the brain implicated in the default network rises and falls in slow by synchronized waves.6 During a monotonous task the default mode sometimes stirs, drawing away a person's attention. Not only is the default network involved mind-wandering, it also distracts executive areas of the brain, so that people aren't even aware that their mind has wandered off task.

Slow yet coordinated fluctuations in activity bind the network together. The syncopations continue even while people are asleep, under anesthesia or in comas.7 The fluctuations that move through the network are one cycle every 15-20 seconds. Most conscious thought happens in split seconds, so it is thought that that the slow pulses are for synapse maintenance. Functional MRI demonstrates a signal in the frequency range of 0.1 HZ that appears to reflect a spontaneous fluctuating neuronal activity that exhibits striking patterns of coherence within known brain systems even in the absence of observable behaviors associated with those systems. Additionally these patterns of coherence are remarkably consistent among individual as well as across subject groups.8

The main areas of the default system are the posterior cingulated cortex, the percuneus, and the medial prefrontal cortex. The medial prefrontal cortex is involved in imagining, thinking about yourself and "theory of mind". The percuneus and the posterior cingulated cortex are involved in pulling personal memories from the brain's archives, visualizing your self doing various activities and describing yourself. Together these areas give you a sense of who you are. Considering the anatomical areas involved in the default mode explains the points of anterior and posterior therapy localization, the emotional neurovascular reflexes and external occipital protuberance.

Procedure

Rarely the patient will display bilateral supraspinatus inhibition in the clear or without provocation. However, most often supraspinatus inhibition will have to be elicited by having the patient rest with eyes closed and mind wandering. If default mode dysfunction is present it usually only takes about 20 seconds of mind wandering to provoke bilateral supraspinatus inhibition. Once the bilateral supraspinatus inhibition has been elicited, put the patient in the lying gait position and check for normal inhibition and facilitation patterns. Next have the patient perform a math problem in their head, if the normal gait pattern is disrupted use the disrupted pattern to TL head points until a pair that normalizes gait has been found. If the math problem does not disrupt gait, have the

patient try to remember ancient past history such as an event from childhood. Again check the gait pattern and follow the same procedure. Once the head points have been localized, tap them bilaterally 60-90 times. Retest the supraspinatus inhibition for normal facilitation.

The predominant head points are associated to default mode dysfunction are LI-20/GABA if a math problem disrupts gait and B-1/serotonin if ancient history disrupts gait. Nutritional considerations are directed towards the involved neurotransmitters, which includes cofactors and nutrients directed at production.

- **Clinical procedure outline**
  - Test both supraspinatus muscles after having the patient close their eyes and letting their mind wander for 10-20 seconds
    - Put patient into the lying gait position and test for normal pattern
      - A mental task such as doing a math problem in their head disrupts gait
        - Treat by tapping head points that normalize gait 60-90 times, usually LI-20/GABA
      - Thinking of past or ancient history disrupts gait
        - Treat by tapping the head points that normalize gait 60-90 times, usually B-1/serotonin
    - Supraspinatus muscle should now be normally facilitated and gait pattern normalized
    - Address nutrition associated to involved neurotransmitters

Conclusion

To the best of our knowledge, with the exception of Applied Kinesiology, there are currently no other practical clinical methods available to evaluate and correct dysfunction of the brain's default mode system. Considering the maladies that have been potentially connected to default mode dysfunction, the ability of Applied Kinesiology to treat default mode dysfunction leaves us with potential to positively affect the health of many individuals who suffer from pathologies associated to default mode dysfunction.

References

1. Walther, DS. Applied Kinesiology Synopsis. 2nd ed. Shawnee Mission, KS, ICAK-U.S.A, 2009, pp.348

2. McCord KM, Schmitt WH. Quintessential Applications A(k) Clinical Protocol. 1st ed. St. Petersburg, FL: Health Works!; 2005. Sections 4b.

3. Raichle, Marcus E., Snyder, Abraham Z., A Default Mode of Brain Function: A Brief History of an Evolving Idea, Neuroimage 2007

4. Hesman Sae, T. You are Who You Are by Default, Science News. July 18, 2009. pp16-20

5. Hesman Sae, T. You are Who You Are by Default, Science News. July 18, 2009. pp16-20

6. Hesman Sae, T. You are Who You Are by Default, Science News. July 18, 2009. pp16-20

7. Hesman Sae, T. You are Who You Are by Default, Science News. July 18, 2009. pp16-20

8. Raichle, Marcus E., Snyder, Abraham Z., A Default Mode of Brain Function: A Brief History of an Evolving Idea, Neuroimage 2007

Clinical Applications of the Horizontal Tonic Labrynthine Reflex: An Update on the "Roll" Component of PRYT

James P. Blumenthal, D.C., DACBN, FACFN

Abstract

As Applied Kinesiology (AK) has evolved into Professional Applied Kinesiology (PAK), the neurological and biochemical underpinnings of classic AK/PAK therapies are coming into clearer focus. Combined with the addition and expansion of related techniques, this has inspired re-examination and updating of terminology and explanations and a renewed understanding of several of the foundational tools and techniques which have kept AK/PAK at the forefront of integrative medicine for the past 46 years.

Research and development of one of these techniques, Retained Neonatal Reflex Therapy (RNR) ®, has generated an opportunity to address these topics.
Tonic Labrynthine Reflexes have been considered by Applied Kinesiology since AK's early days. On reviewing these techniques in light of current knowledge in functional neurology, a new set of tests and treatment protocols has emerged which can be used to neurologically integrate them.

Key Indexing Terms
Horizontal Tonic Labrynthine Reflex, TLR, Retained Neonatal Reflex, RNR, Retained Primitive Reflex, RPR, Vestibular Nerve, CNVIII, Vestibular-Ocular, Labrynthine, Histamine, Methylhistamine, Visual Righting Reflex, Vertigo, Autonomic Nervous System, ANS, Sympathetic, Parasympathetic, Histamine, H1, H2, H1/H2, Spleen, Stomach, Professional Applied Kinesiology™, AK, PAK®

Introduction

Over the past several years, as Applied Kinesiology (AK) has been evolving into Professional Applied Kinesiology (PAK), our understanding of the neurological and biochemical underpinnings of classic AK/PAK therapies have been coming into clearer focus. Combined with the addition and expansion of techniques under the rubric of our Craft, this has inspired re-examination and updating of our terminology and explanations and a renewed understanding of several of the foundational tools and techniques which have carried AK/PAK to the forefront of integrative medicine over the past 46 years.
In the process of contributing to the research and development of one of these techniques, Retained Neonatal Reflex Therapy ® (RNR)[1], I have had the opportunity to confront such an area.

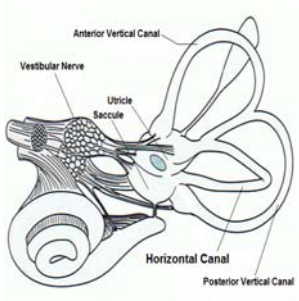
Tonic Labrynthine Reflexes have been an area of consideration in Applied Kinesiology since the early days of AK. Dr. Walther described Labrynthine Reflexes, Neck Righting Reflexes, and Visual Righting Reflexes in the earliest pages of "Basic AK Testing and Treatment Procedures" in the AK Synopsis[2] and then presented PRYT Technique a few pages later[3] . Reviewing these techniques in light of what is now known about functional neurology, we can see the close relationship between them. Furthermore, a new set of tests and treatment protocols has emerged which may be applied to them.

For several years, RNR has concerned itself with the effects of non-integrated Tonic Labrynthine Reflexes (TLRs), particularly the sagittal and lateral (or coronal) TLRs. These can be associated with the Pitch and Yaw components of PRYT respectively. Likewise, they can be associated with specific activation of the vestibular semi-circular canals. The "Roll" component, neglected by RNR until recently, has now been elucidated. It is referred to as the Horizontal TLR and corresponds to activation of the horizontal semicircular canals and the vestibulo-ocular reflexes, as described by Kandel and Schwartz[4].

Semi-Circular Canals and Vestibulo-Ocular Reflex

A brief review of semicircular canal anatomy and physiology may be helpful along with a summary of the vestibulo-ocular reflex. The vestibular labyrinth houses five types of receptor organs:

- Clusters of hair cells which transduce mechanical stimuli into electrical "receptor potentials"
- Utricles and saccules which detect linear accelerations created by gravity and body movements
- Semi-circular canals which detect angular accelerations, caused by rotation of the head or body
- The vestibular nerve (CNVIII) which carries sensory information from the vestibular organs



The 3 semi-circular canals are named for their physical appearance. They depolarize or electronically fire when specific body or head movements cause the endolymph within them to move opposite the direction of the head's motion, bending the hair cells which line the walls of the canals and selectively depolarizing the nerves at the base of each hair cell. Because the hair cells only depolarize in one direction and hyperpolarize in the other, head movement will most commonly depolarize or activate the canals on either the left side or the right side, but rarely on both sides simultaneously.

Movements can be described as either linear (body and head moving in the same direction) or rotational (around an X/Y/Z orthogonal axis)[5] motion. Most rotational head movements involve activation of at least two of the three canals. The simplest movement, vis a vis the canals, is pure lateral rotation of the head (rotating right or left around the Y axis in the axial plane) which only depolarizes the ipsilateral horizontal canal. This is the same 'head vs. body' motion involved in the Roll portion of PRYT. We have non-volitional vestibular reflexes which are used to compensate for the movement of our head and to manage our perception of motion-in-space. These include the *vestibulo-ocular reflex* which allows us to keep our eyes still during head movement

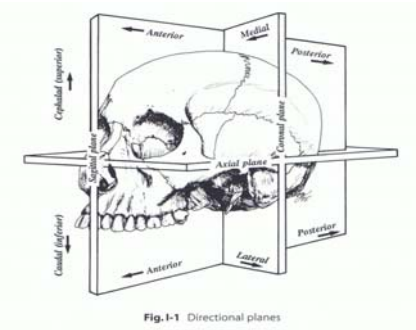


Fig. I-1 Directional planes

and the *vestibulospinal reflex* which the skeleto-motor system uses to compensate for head movement. Both of these reflexes are involved with aspects of PRYT as described by Walther, however only the vestibulo-ocular reflex is primarily involved with the TLRs as considered by RNR.

According to Kandel and Schwartz, there are three different vestibulo-ocular reflexes which arise from the three major components of the labyrinth:

1. The *rotational vestibulo-ocular reflex* compensates for head rotation and receives its input predominantly from the semi-circular canals
2. The *translational vestibulo-ocular reflex* compensates for linear movement
3. The *ocular counter-rolling response* compensates for vertical head tilt [6]

Phylogenetically, it is believed that we developed vestibulo-ocular reflexes as a fundamental survival mechanism to enable us to keep our eyes focused on prey as we chased it down instead of having our eyes travel up and down or side to side with each phase of gait, which would have severely reduced our prospects of chasing down a meal.

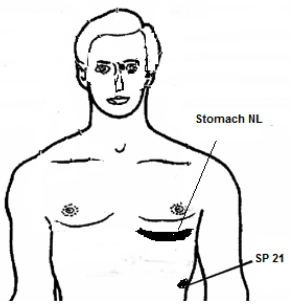
AK/PAK Correlations and Reflexes

In the process of teaching, using, and researching the Retained Primitive Reflexes, I noticed that corrections for sagital (Pitch) and coronal (Yaw) TLRs had been worked out, however the horizontal (Roll) TLR had not. As a student of chiropractic neurology, this appeared to be an unfortunate oversight. Dr. Goodheart frequently exhorted us to "look with eyes that see " and , as Providence would have it, a parade of patients presented with just the symptoms of geocentric rotational vertigo that I was looking for to work out the horizontal TLR. One of the tests for the horizontal vestibulo-ocular reflex taught in the Carrick neurology program is to have the Patient focus their eyes on an object in front of them while rotating their head left and right as the doctor observes for ocular, motoric, or postural deviations. These signs can be quite subtle and indistinct to any but the most experienced observer. By coupling the neurological challenge with standard indicator muscle testing, the resulting autonomic shift becomes far more evident to the majority of trained observers, especially to PAK providers.

Building on the work of two of my AK mentors, Drs. Christopher Astill-Smith and Keith Keen, I was able to expand this to an Applied Kinesiology test. Dr Astill-Smith used his impressive knowledge of biochemistry to correlate body reflexes with excesses and deficiencies of several neurotransmitters[7]. Dr. Keen is the founder of RNR® and the developer of much of what we currently know about identifying and treating retained primitive reflexes. In an earlier paper[8], I correlated each of the RPRs with neurotransmitters which were then found to coincide with Dr Astill-Smith's correlations. Using the same processes which made the earlier correlations possible, we were able to identify the testing protocol, associated body reflex points, correction protocol, and neurotransmitter associated with the Horizontal TLR, as follows.

Testing

1. Identify a convenient normotonic indicator muscle[9].
2. Have the patient rotate their head fully to the left and test for weakening of the indicator muscle.
3. Have the patient rotate their head fully to the right and test for weakening of the indicator muscle.
4. If the indicator muscle weakens in either steps (2) or (3) above, identify and correct the cervical or upper thoracic lesion.
5. Assuming that there was no indicator muscle change in either



steps (2) or (3) above, have the patient fix their eyes on an object in front of their face while fully rotating their head left and right.
6. If the indicator muscle weakens in either direction, that is a positive finding.
7. This test should be performed both seated and supine.

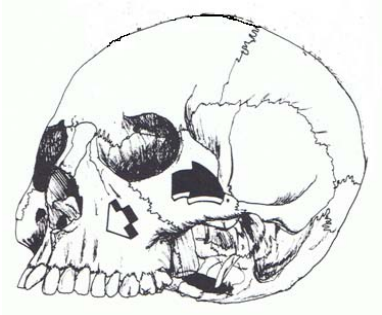
Body Reflexes
The horizontal TLR will therapy localize to either or both SP21 and the stomach neurolymphatic (NL) area under the left breast.

Neurotransmitters
The neurotransmitter correlation for Horizontal TLR is methylhistamine, which is synthesized as follows:

Histamine-n-Methyltransferase (HNMT) +
Mono- or Di-amine oxidase (MAO or DAO)
Histamine (H1, H2) ⟶ ***Methyl-histamine***
Methylcobalamin, methionine,
Mg, Zn, R-5-P (B2), Cu, Fe

There are four known histamine receptors at this time (H1 through H4). For the purposes of this paper, we are only interested in H1 and H2 histamine receptors which recognize the histamine that is metabolized through HNMT and MAO/DAO enzymes to Methylhistamine. This metabolite of histamine is related to both the spleen and stomach reflex organ systems. H1 histamine receptors in the spleen participate in the anaphylactic barrage, a classic expression of the allergic fight-or-flight response characteristic of the sympathetic portion of the autonomic nervous system. H2 receptors in the stomach are responsible for signaling the parietal cells to produce and release hydrochloric acid. Digestion is a primary expression of the parasympathetic portion of the autonomic nervous system. Hence, the Horizontal TLR appears to represent an interface between both aspects of the ANS.

Correction
Once the Horizontal TLR has been identified, correction entails releasing a jammed zygomatico-maxillary suture with respiration assist.
1. Have Patient hold their head in the direction which weakens the indicator muscle as in (6) above while maintaining their field of gaze directly forward (anteriorly).
2. Alternately therapy localize the left and right zygomatico-maxillary sutures to find the one which TLs.
3. Find the phase of respiration which cross therapy localizes.
4. Adjust the appropriate zygomatico-maxillary suture in the phase of respiration which XTL'd as illustrated[10].
5. Retest the Horizontal TLR, which may occasionally show up on the opposite side. If it does, fix what you find as described above.
6. Perform the Habituation Correction and the NOW Point Correction from RNR as described by Dr. Keith Keen[11] and taught by Dr. Keen and myself to lock in the fix.



Discussion

The Horizontal TLR has proven to be valuable in a number of applications. We have used it to alleviate geocentric rotational vertigo, to improve digestion and reduce allergic responses (often along with ICV or hiatal hernia issues), and in conjunction with the Fear Paralysis Reflex (release of vagus perineural fascial adhesions). We have found it active on patients from 2 years old to elderly adults. It may be present in younger children however we have not yet overcome the challenge of testing this reflex in children younger than 2 years old. It seems to represent a meeting of the sympathetic and parasympathetic autonomic functions and may reveal more significant applications from this perspective. As we have only been working with the Horizontal TLR as a Retained Primitive Reflex for the past 6 months or so, any feedback will be most welcome.

I would like to dedicate this paper to the memory of my friend and mentor, Dr. Bert Hanicke (1928-2010), without whose guidance and support I would not have become a chiropractor, much less an applied kinesiologist. Thanks, Bert. Merry meet and merry part and merry meet again.

References

1. Retained Neonatal Reflex Thearapy (RNR) is the internationally trademarked name of the AK-based retained primitive reflex (RPR) treatment procedures which have been developed by the multicenter and multidisciplinary VMP group, headed by Dr. Keith Keen out of Sydney, Australia

2. Walther, David S., Applied Kinesiology Synopsis Vol.1, pp 164-165, ICAK-U.S.A, 1988

3. Walther, pp 177-182

4. Kandel ER, Schwartz JH, Jessel TM, Principles of Neural Science, 4th Ed., McGraw-Hill, 2000, pp 801-814

5. Illustration from Pick, Marc G, Cranial Sutures: Analysis, Morphology, and Manipulative Strategies, Eastland Press 1999, p xvi. Used with permission.

6. Kandel and Schwartz p. 809
   Astill-Smith, Christopher. Biochemistry of Emotion and Physiology of Learning, Lecture notes, Los Angeles, November 15, 2003

7. Blumenthal, James P, "Correlation of Retained Primitive Reflexes (RPRs) with Acupuncture Meridian Beginning and Ending (B&E) Points and Neurotransmitter Ascriptions", ICAK Collected Papers, 2008, pp 237-240

8. Schmitt, Walter H, “What to do When you Can’t Find a Weak Muscle”, Ski With Wally Seminar notes, Snowmass, CO March 2007

9. Illustration from Pick p 204. Used with permission.

10. Keen, Keith, Retained Neonatal Reflexes Manual, Self-published, 2008, pp.18-21 and 30

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Competitive Muscle Integration Balance

John Erdmann, D.C., DIBAK

Key Indexing Terms
Applied Kinesiology, Dr. Goodheart, Neurology, Physiology, Muscle Testing, Muscle Correlations, Synergistic and Antagonistic Muscles, Spinal Thalamic Tract, Muscle Facilitation

Discussion

Muscle correlations between normal and abnormal physiology and neurology have been the foundational observation since 1964 when Dr. Goodheart made his first observation of this imbalance noticing an abnormal postural winging of the scapulae. In Dr. Goodheart's case comparatively and in normal physiology the scapulae is adhered to the ribcage in a flat planar with the ribcage position being stabilized chiefly from the Serratus Anticus and Subscapularis muscles.

Muscle testing was born when Dr.Goodheart was able to isolate the Serratus Anticus sufficiently from other muscles and the opposite normal appearing side and test for strength differences.

Since that time, many neurological and physiological relationships have developed and broadenned the diagnostic window. For example, synergistic and antagonistic relationships that "should exist" in normal physiology, facilitation and inhibition from golgi tendon organs, deep tendon reflexes and more, gait analysis relationships, tonic labrynth, injury recall.

The focus point of my research presented below, is to find specific core observations that when brought to more normalcy will balance many other systems and window of the body.

For my research presented here I tested and treated 40 individuals. Ancillary observations were made in most cases using blind spot mapping and degrees of nystagmus present.

While normal, graceful movement is never performed with simultaneous firing of synergistic and antagonistic muscles in equal competition, this procedure is to do just that. The practitioner can illicit a simultaneous test in a quadrant (upper or lower, right and left extremity) expecting equal balance and stand off power from both opposite muscle movements.

First, we should pretest all muscles in our test for individual normal muscle testing tone grade 4, normal muscle facilitation.

In this example, we will test a Rectus Femoris and a Hamsting of the same leg and at the same time with gentle but equal force. The expectation is for a cancellation of clear movement in either direction of the femur and leg. Of course, consideration would be given to test under the strength /fatigue potential of the weakest muscle.

This exercise is then performed on the other leg side and then in the upper extremity using conveniently a Pectoralis Clavicular and Triceps group muscle together on the same arm and then the next arm.

Patient familiarity and clear instruction is initially a consideration. However, in practice the learning curve is quick for patients to follow.

When a competitive muscle group is found to clearly over power its' antagonistic counter part or in other words move the limb in a particular direction I am concluding that we have found improper neurological response.

More research needs to be done to theorize the actual deficit whether it be a local muscle fiber or somewhere along the spinal thalamic tract or even higher in the nervous system. It is my observation, that local input somewhere near the same extremity has reversed these findings in most of my cases. The correction of these findings has specifically impacted balance, blind spot mapping, and nystagmus for the better.

Procedure

1. Test for other findings: IE. ROM, Palpate, Blind spot, Nystagmus, Postural deviations

2. Check both Rectus Femoris, hamstrings (supine), Pectoralis major, and triceps (elevated) for normal facilitation.

3. Simultaneously test both opposing muscles of an extremity, then the next extremity, and so on.

4. If a finding of over and under facilitation is found (a muscle is able to overpower it's counter part) then Therapy localize or challenge related or near bye structures to find a negation of the undesired response.

5. After correction, re-evaluate other findings.

Acknowledgments

George Goodheart, D.C., DIBAK; David Leaf, D.C., DIBAK

References

1. Walther, David S., Applied Kinesiology, Synopsis 2nd ed., 2009 ICAK-U.S.A., Shawnee Mission, KS 66202.

2. Leaf. David, Applied Kinesiology Flow Chart Manual, 3rd ed. 1995.

G.A.L.T Related "Tennis Elbow"

John Erdmann, D.C., DIBAK

Abstract

This paper reviews the association between a common tennis and other elbow injuries and diseases associated with the gut. Acupuncture meridian treatment points along with chiropractic and diet/ lifestyle are discussed in the treatment of the intestines.

Key Indexing Terms

Lateral Epicondylitis, GALT, Goodheart, Applied Kinesiology, Chiropractic, Acupuncture, Gut Conditions, Ulcerative Colitis, Irritable Bowel, Chrons Disease, Acid Reflux, Bowel Problems, Colitis

Discussion

GALT or Gut associated (altered) Lymphatic tissue supports 70% of the immune response via T cells and IGA secreting cells. Normal intestinal mucosa contains abundant immunoglobulin A (IgA)-secreting cells, which are generated from B cells in gut-associated lymphoid tissues (GALT). We know that dendritic cells (DC) from GALT induce T cell-independent expression of IgA and gut-homing receptors on B cells.

Commonly diagnosed and increasingly epidemic is IBS or Irritable Bowel Syndrome, leading to Ulcerative Collitis, and Chrons disease. Cystitis is another complication increasingly more diagnosed as well as Colon cancer and polyp production.

All this “GLOOM and Doom,” and your patients could be giving you the early warning symptoms years or months earlier!

Acupuncture LI11 is the tonification point conveniently located at the “Tennis Elbow” or hot spot. There will be a very tender point running along or over some aspect of the radial head or lateral distal humerous. In Applied Kinesiology an imbalance in Supinator and Pronator muscles are often observable through muscle testing, as well as knee related Adductor muscles.

First we start North on our compass; evaluating the stomach function primarily for hypochlorydia, chronic anti-acid use, persistent gas and bloating. If undigested routing food is allowed to be pushed into the small intestine, gut flora will be altered along with inflammatory and immune reactions. The lymphatic tissue will swell and become more toxic and sluggish. Poor bowel evacuation will become more chronic, reliance on fiber, magnesium, and other laxatives is very common at this juncture.

The large intestine is nearing the final exit where the GALT is more highly developed. Water permeation and transport / elimination is the primary job. This stage of breakdown usually puts a higher demand on the kidney's to secrete more ADH to act upon the renin

and angiotensin system. Night time urination, constipation, elevated blood pressure are associated.

Goodheart noted LI4 (sedation) treatment point for cystitis over 35 years ago, he coined the “Ammonia Sniff test” was associated with increase need of organic minerals high in potassium. This is a test convenient for the practitioner to perform where the patient sniffs a sample jar of clorox and there is an observable weakness of a previous strong muscle. Different physiology books vary in intestinal PH, but the range is higher than the stomach somewhere between 5.8 and 6.8. Good Flora or "bugs," are known to proliferate in ideal PH, where the reverse is likely true of bad bugs. Correcting PH levels at all “cellular level” and organ sites are critical overall patient health. Conjunctive acupuncture/ acupressure, chiropractic and chemical protocols lead to the best results when treated together.

Thorough observation and treatment should be given to elbow and arm muscles as well as the Adductor and ligament interlink areas. The Differential analysis and complications can arise with local and systemic inflammation, poor stomach, gut, immune and thymus, and simple joint misalignment. In re-aggravation cases, proper technique for the sports enthusiast or ergonomics should be considered. Jeffrey Bland, PHD has coined a "Four R" process for supporting proper gut function: Remove offending, repair, restore and reinoculate

Summary

Patients present with everyday problems and symptoms that under more examination may lead to bigger, more important health determinants and future diseases. In the above case, the well known and studied Chinese acupuncture treatment points at the elbow lend some investigative responsibility to inquire into GALT and other gut issues and questions.

Acknowledgments

Jefferey Bland, Ph.D.; George Goodheart, D.C., DIBAK

References

1. Guyton, Arthur C., Guyton's Physiology Text.

2. Bland, Jeffery, Eclectic Works.

3. Walther, David S., Applied Kinesiology, Synopsis, 2nd ed., 2009 ICAK-U.S.A., Shawnee Mission, KS 66202.

G.A.L.T Related "Tennis Elbow"

John Erdmann, D.C., DIBAK

Additional Cervical Correlations

Timothy D. Francis, D.C., FIACA, DIBAK, MS, DHM

Abstract

Muscle dysfunction has traditionally been correlated throughout the body by Applied Kinesiology (AK) methodology due to cervical spine subluxation/fixation patterns. This paper adds additional muscle dysfunction correlations observed by this author.

Introduction

The cervical spine has seven times the mechanoreceptors than anywhere else in the spine. Therefore proper alignment and motion is of utmost importance for optimal body function. Previous subluxation/fixation patterns observed by this author will be reviewed as well as new observations for cervical subluxations.

Discussion

Fixations are a locking together of two or more vertebrae producing a predictable bilateral muscle weakness pattern. According to Leaf "Fixations are the body's attempt to stop dural torque." In a previous paper by this author entitled Additional Fixation Patterns the following patterns have been observed.

Cervical Spine Area	Bilateral Muscle Dysfunction
1. Occiput	Upper Trapezius
	Finger Flexors
2. Occiput/Atlas	Unilateral Hamstrings (Supine)
3. Upper cervicals	Triceps (Long Head)
	Subclavius
	Sartorius
	Inferior Gemelus
	Popliteus
4. Mid-cervicals	Biceps (Short Head)
	Opponens Pollicis
5. Lower Cervicals	Wrist Flexors
	Brachioradialis
	Quadratus Femoris
	Teres Minor

These additional patterns follow the same rules for diagnosis and treatment as the original observations by Goodheart.

There is yet another bilateral muscle dysfunction pattern previously discussed in a paper by this author entitled Spinal Subluxation/Bilateral Muscle Syndrome Correlations.

These particular muscle dyspoiesis only displays via manual muscle testing (MMT) when both the right and left sides are tested simultaneously. For example, the right middle deltoid tests strong in the clear as well as the left middle deltoid. However, when tested together simultaneously they test weak. These cervical patterns are as follows.

Cervical Vertebrae Dyspoiesis	Simultaneous Muscle
1. Atlas	Teres Major
2. Fifth Cervical	Middle Deltoids Latissimus Dorsi
3. Seventh Cervical	Sternocleidomastoid

Unilateral muscle correlations was discussed by this author in a paper entitled Spinal-Rib Subluxation/Muscle Syndrome Correlations. Occular lock patterns were also discussed as noted below.

Cervical Spine	Muscle/Syndrome
1. Inferior Occiput	Ocular lock pattern with the eyes held in a straight superior direction Hiatal hernia Rib cage torque Anterograde lymphatic
2. Lateral Occiput	Ocular lock pattern with eyes held up and out on ipsilateral side Hyoid dysfunction
3. Second cervical	Ocular lock pattern with eyes straight to ipsilateral side Hyoid dysfunction
4. C6/7 Counter rotation	Sartorius/Gracilis Category II

The new additional cervical corrections are not related to a specific cervical vertebral level but rather to a specific type of subluxation. These are listed in the chart below.

Cervical Subluxation	Muscle Dysfunction
1. Anterior Cervical (C2-C7)	Finger Flexors
2. Lateral Cervical	Finger Abductors (Index-Middle-Ring)
3. Posterior Cervical	Finger Extensors

The anterior cervical will usually only therapy localize (TL) on the anterior portion of the vertebrae on the side of anteriority. This may be corrected manually with a push or pull procedure depending on how the vertebrae challenges and may be performed in the supine, prone, or seated patient position.

A lateral cervical may only TL on the lateral portion of the cervical vertebrae. A piece of paper is placed between the index/middle finger and/or middle/ring finger with the wrist in a neutral position and the elbow flexed to a perpendicular position. The subject is asked to hold the paper tightly between his/her fingers. If the manual correction is performed correctly, the subject is able to grasp the paper without letting it slip through the fingers.

For the posterior cervical subluxation, the elbow is bent perpendicular, the wrist is in neutral, and the hand is extended to full open position. The examiner braces the front of the hand and attempts to flex the fingers into a fist.

Conclusion

These three patterns are valid for the second to the seventh cervical and give the practitioner as well as the patient an immediate reliable feedback system as to the efficacy of the manual adjustive procedure.

Resources

1. Francis, Timothy D., Structural Corrections For Eyes Into Distortion Patterns. Collected Papers of the Members of the I.C.A.K., Vol. I Summer (1990-1991).

2. Ibid., Spinal-Rib Subluxation/Muscle Syndrome Correlations. Experimental Observations of the Members of the I.C.A.K., Vol. I (1999-2000).

3. Ibid., Spinal Subluxation/Bilateral Muscle Syndrome Correlations. Experimental Observations of the Members of the I.C.A.K., Vol. I (2000-2001).

4. Ibid., Additional Fixation Patterns., Experimental Observations of the I.C.A.K., Vol. I, (2001- 2002).

5. Ibid., The Occiput/Atlas Fixation., Experimental Observations of the I.C.A.K.-U.S.A., (2009- 2010).

6. Leaf, David, Applied Kinesiology Flow Chart Manual, 3rd Edition. Privately Published. (1995).

Identifying and Correcting Sleep Disturbances Related to Nocturnal Glucose Regulation

Stephen C. Gangemi, D.C., DIBAK

Abstract

Sleep disorders are said to affect approximately 20 percent of the US population[1] with problems ranging from insomnia, restless sleep, waking up at specific times, sleep apnea, and a host of others. There are many etiologies for each sleep disturbance such as hormonal imbalances and emotional problems. Often the reason an individual wakes up throughout the night is due to a spike in either cortisol or epinephrine as a result of a sudden drop in blood sugar levels or a drop below normal (hypoglycemia). Though it is well known that one of these two hormones is responsible for the disruption of sleep, it is often difficult to verify, as rarely can a patient be treated during the actual sleep cycle. However, using manual muscle testing (MMT) along with specific challenge procedures, one can often identify the reason for the sleep disruption and therefore the treatment can be tailored towards its improvement and, often, correction. This author has found a specific procedure to implement during the neurological examination process which will identify and treat such sleeping disorders.

Key Indexing Terms
Glucose, Glycogen, Cortisol, Epinephrine, Nocturnal, Sleep, Manual Muscle Testing (MMT), Glycogenolysis, Gluconeogenesis

Introduction

Blood sugar levels are monitored by cells in the pancreas called the Islets of Langerhans. When blood glucose levels fall too low, the body must increase this level via two main mechanisms. The body may convert stored glycogen into glucose (glycogenolysis), which occurs in the muscle and liver tissue, or it may generate glucose from some non-carbohydrate carbon substrate such as amino acids, glycerol or lactate. This process is called gluconeogenesis and occurs primarily in the liver.

Epinephrine and glucagon promote glycogenolysis which increases plasma glucose levels by breaking down liver glycogen.[2] Due to its origin in the adrenal medulla and its affect on the sympathetic nervous system, an increase of epinephrine will have a negative effect on restful sleep. Epinephrine excretion has been shown to have a positive correlation with percent of waking time during bed-rest in the daytime and at night.[3]

Cortisol, produced in the adrenal cortex, will increase protein catabolism which in turn frees amino acids to be used for gluconeogenesis. The excretion of cortisol is synonymous with a stress response, therefore, like epinephrine, it will also negatively affect sleep.

The liver is the main target organ involved in both the epinephrine effect of glycogenolysis and the cortisol effect of gluconeogensis. Therefore, the status of the liver must be observed when investigating sleep problems.

Discussion

There currently are various methods to help identify problems that may occur when a patient is sleeping, yet the examination is occurring at a different time; one such procedure is the Then & Now Technique.[4] Additionally, the patient may simulate REM sleep by rolling their eyes in a circle; muscle inhibition of a previously facilitated muscle often indicates a problem. Many times a challenge is needed during this REM sleep test to see a muscle change response and indicate a problem. The challenge is typically something that would over-stimulate the nervous system, such as cortisol, epinephrine, emotional or physical stress or a dietary stress such as sugar, caffeine or a food allergy/intolerance. However, often when something is expected to occur during the examination, based on the patient's history and symptoms, correlating such with a muscle test may not be evident. Additionally, as noted above, the liver plays an important role in sleep issues due to the influence of cortisol and epinephrine on the organ in blood sugar regulation. According to acupuncture meridian therapy, the liver's horary period is 1am-3am. This is when the organ is at its highest energy level. While many sleep disturbances do occur at this time due to certain stresses on the liver, some do not, so the liver must be challenged in a different way, in order to "smoke-out" a hidden sleep problem that may not be seen otherwise.

When there is a drop in the blood glucose level at night, the liver must work to bring the level back up. This is accomplished by either pulling glycogen from the liver and converting it into glucose (glycogenolysis), which is done via epinephrine, or by converting amino acids into glucose (gluconeogenesis), which results in an increase in cortisol output.

A doctor can test one of the liver related muscles, either the pectoralis major sternal or the rhomboid, while challenging the patient with either cortisol or epinephrine as the patient simulates REM sleep by closing the eyes and rolling them in a circle. This will verify if the patient is awakening at night due to a dysglycemic or a hypoglycemic state as a result of an increase in either cortisol or epinephrine. Furthermore, the reason for the spike in either hormone can be identified by challenging various stressors, such as dietary offenders, emotional issues, other hormonal imbalances or medication/supplemental intolerances. After the stressor is determined, challenging with various supplements to help regulate either epinephrine or cortisol, as well as nutrients, which will help support or overcome any other stressors and will prove to be very useful.

Procedure

1. Test a liver related muscle, (rhomboid or pectoralis major sternal division). The pec sternal is easier to use and should be strong with normal autogenic inhibition - (shortening of the spindle cell weakens the muscle).
    a. Have the patient close the eyes and slowly roll them in either direction, (REM sleep). If this produces a change (weakening) in the muscle being tested, the doctor must correct according to other procedures which will not be addressed in this paper.
    b. With the patient's eyes open, challenge the patient with cortisol by using either oral nutrient testing of homeopathic 6X cortisol or vigorously rubbing the adrenal Chapman's Reflexes (CR) for 2-3 seconds. If this produces a change (weakening) in the muscle being tested, the doctor must correct according to other procedures which will not be addressed in this paper.
        i. If this produces no change, then perform the muscle test again, challenging cortisol <u>simultaneously</u> with the REM sleep eye movement. If this results in a weakening of the pec sternal, go to 2. This is a positive test indicating that the patient is experiencing nocturnal gluconeogenesis.
    c. Challenge the patient with epinephrine by using either oral nutrient testing of homeopathic 6X epinephrine or asking the patient to squeeze the hands into a fist as hard as possible for 2-3 seconds. If this produces a change (weakening) in the muscle being tested, the doctor must correct according to other procedures which will not be addressed in this paper.
        i. If this produces no change, then perform the muscle test again, challenging epinephrine <u>simultaneously</u> with the REM sleep eye movement. If this results in a weakening of the pec sternal, go to 2. This is a positive test indicating that the patient is experiencing nocturnal glycogenolysis.

2. The treatment is to provide more parasympathetic activity to the liver, which is accomplished by rubbing the CR of the liver while either cortisol or epinephrine stimulation is introduced. However, before this is corrected, the doctor may wish to find an offender/stressor that may be driving the increased epinephrine or cortisol output, as well as investigate any possible supplements which may help the patient deal with the hormonal stress output or offender/stressor.
    a. To investigate common offenders, the oral homeopathic solution, if used, should be removed from the tongue. Next the doctor should check for various offenders that may be suspected to weaken the pec sternal during the REM sleep challenge. Common offenders are the following: caffeine, sugar, food allergies/intolerances, medications, other hormones (such as estrogen, testosterone, thyroxine), and emotional stress (patient should therapy localize to the stomach neurovascular points). This procedure is done by challenging with the offender/stressor while the patient is rolling their closed eyes and the doctor is testing the pec sternal.

b. Next, the doctor should check to see if any supplementation may help the patient recover from the sleep disorder faster.
    i. If cortisol showed weakening during the testing, the doctor, while performing the REM challenge test (patient's eyes are closed and rolling and the doctor vigorously rubs the adrenal gland CRs so oral testing of the nutrient may be performed), should check the following nutrients: B1, B2, B3, B5, Phosphatidylserine, choline, vitamin E, vitamin C, and magnesium. Supplement as indicated.
        2 Additionally, protein intake should be assessed as levels may be low or inadequate
    ii. If epinephrine weakened during the testing, the doctor, while performing the challenge test (patient's eyes are closed and rolling and they are asked to squeeze their hands into a fist as hard as possible for 2-3 seconds), should check the following substances: B2, B5, B12, selenium, zinc, glucuronic acid, cysteine, glutathione, and copper. Supplement as indicated.
        2 Additionally, carbohydrate intake should be assessed as liver glycogen levels may be low

Conclusion

Nocturnal hypoglycemia is a common problem that results in a disrupted sleep cycle. The resultant hormonal spikes of either cortisol due to gluconeogenesis from amino acids converting to glucose or epinephrine due to glycogenolysis will awaken the individual. Testing the various substances and/or hormones via the liver related muscles during a simultaneous REM sleep and hormonal challenge is the key to a proper diagnosis and treatment. Adjusting the patient's diet, either throughout the day by providing adequate and healthy levels of carbohydrate or protein or by identifying and correcting common offenders, often proves to be useful in dealing with this problem. Supplementation of various nutrients described when indicated to help break down epinephrine and cortisol will also benefit the patient.

References

1. National Institutes of Health. National Center on Sleep Disorder Research. 2003.

2. Costill D, Kenney L, Wilmore J. Physiology of Sport and Exercise. Champaign, IL: Human Kinetics; 2008. p.68-9.

3. Mori K, Nishirara K. The Relationship Between Waking Time and Urinary Epinephrine in Bed-Rested Humans Under Conditions Involving Minimal Stress. Int J of Psychophysiology.1988 Jun;6(2):133-7.

4. Walther DS. Applied Kinesiology; Synopsis 2ed. Shawnee Mission, KS: ICAK-U.S.A.; 2009. p. 282-3.

Identifying and Treating the Primary Problem in Patients

Stephen C. Gangemi, D.C., DIBAK

Abstract

Using manual muscle testing (MMT) as part of the neurological exam is a powerful tool physicians have to assist the diagnosis of many ailments. Pinpointing health processes gone awry or being able to clearly define the etiology of a particular health problem is a difficult task. While this may be an understatement, unfortunately, this leaves many physicians treating symptoms and not the root cause.

Over time, and as an ailment progresses, patients tend to develop compensations of various degrees, both physically and chemically. These compensations are the body's way of dealing with the problem by adapting to change, so the person can essentially "live to fight another day." Unfortunately, these compensations merely act as a "smoke screen" for the physician and must be further investigated in order to determine what is contributing to or maintaining the problem at its origin.

Specific testing procedures must be employed, as described herein, by the physician to resolve compensations, to address/treat the health issue at its core/root and to prevent "painting over rust." As success of treatment is not just determined by whether or not a physician is able to identify indicators that arise during the treatment process but by whether or not one can discern the nature of a particular indicator, e.g. is it part of the overall health problem or is it simply an indication that the patient has mal-adapted to a specific offender over time.

Key Indexing Terms
Applied Kinesiology, Manual Muscle Testing (MMT), Acupuncture Meridian Therapy, Chapman's Reflex (CR), Spinal Subluxation, Injury Recall Technique (IRT)

Introduction

Applied kinesiology (AK) is a diagnostic system which employs manual muscle testing (MMT) with other standard methods of diagnosis. AK muscle testing is performed using specific muscle tests which provide a real-time sensory-motor (input-output) response. This combined with a thorough patient history, orthopedic and neurological testing, and often laboratory work, can render a thorough understanding of a person's health problems and guide treatment. AK is diagnostic; it is not a treatment procedure. A skilled AK physician will employ various treatment methods such as chiropractic manipulation, cranial techniques, myofascial release or trigger point therapy, acupressure, nutritional or dietary changes/counseling, exercise therapies, and emotional techniques, as well as other

procedures to help the patient restore his or her health. The treatment is tailored to fit the individual patient based on the examination.

Patients compensate for problems by developing structural (musculoskeletal) and chemical (nutritional, hormonal, inflammatory) processes of varying degrees. Over time, most will compensate more for these compensations and essentially "add more layers to the ever growing onion." Compensations are the body's way of managing or coping with the problem by adapting to change—the body is more concerned with today and tomorrow than it is about survival and health next year. However, compensations muddy the water for the physician and must be dealt with to see what is truly at the core of the problem.

Often, physicians tend to treat symptoms based on the patient's reports, leaving the true root problem untouched. It is important to remember certain training or diagnostic/examination procedures can lead physicians to believe they are treating the root problem. How do physicians really know if they are treating the patient's problem and not just the multitude of adaptations or compensations the patient has developed? One would have to agree to some extent that it may be impossible to *always* know *all* the time; however, the more skilled and thorough the physician is in his or her examination, the more one reduces the chance of this occurring.

Physicians look at indicators when treating a patient to determine if their treatment protocol is successful, or not. The beauty of MMT is that the physician can quickly see if the treatment procedure employed is right for the patient, as the neurological response is immediate. This does not mean, however, that compensations are not also being addressed; one could simply be removing the indication that there is a problem, but not addressing it at all. For example, if the physician determines there is a spinal subluxation at a certain level of the spine, how does one know adjusting that subluxation is the root cause of the ailment (or part of the ailment), and not just the body's indication that there is a problem? Clearly, there are other factors, which is why this example is referred to as a subluxation-complex, but what part of the complex should be treated? AK physicians pay specific attention to the muscle imbalances involved in such a process and often the reason for the subluxation is due to those muscular imbalances. Additionally, through the understanding that muscles have a viscerosomatic relationship, these imbalances are often due to a problem with a specific organ. Does the organ dysfunction create muscle imbalances and those muscle imbalances create the spinal subluxation, or does it start at the spinal subluxation? Do they treat the chicken or the egg (the spine or the organ or the muscle)? This is left for the physician to decide. Understanding whether they are treating a compensation and indication of a problem or actually the root problem is paramount.

Discussion

There are several factors which must be considered in order for the physician to be more certain they are addressing the primary cause, or causes, of the patient's health issues. That is not to say that compensations are not to be addressed. Actually, compensations

which the body has developed often need to be identified and treated accordingly so the physician may have a clearer picture of what is the true underlying condition.

The majority of compensations developed by a patient will be seen as injury patterns during the examination procedure and must be treated with injury recall technique (IRT).[1] The most certain way to tell if an injury pattern needs to be addressed is when the muscle does not strengthen with spindle cell activation. However, many times it may strengthen the weak (inhibited) muscle even though there is injury affecting the dysfunction process somewhere in the body. Therefore, taking further steps described by this author in a previous paper will lead to the discovery of these injuries and subsequently to their correction.[2]

Cranial faults are also common components of the adaptation processes. They must be addressed either by manual correction, IRT correction, or via their relationship to their respective immune system viscera.[3]

A muscle tested through a standard Type I MMT (physician initiated), which is the primary way to test via AK methods, may be inhibited for several reasons, i.e. an organ dysfunction, a meridian imbalance or a nutritional/chemical component. Often, in the case of an organ dysfunction the organ's Chapman's Reflex (CR) will therapy localize (TL) indicating that the organ needs some form of treatment. The major question that arises when the muscle-organ (viscerosomatic) relationship is identified is how does the physician truly know if this is a primary issue, either entirely or at the current time of treatment, or just an adaptation process? This author submits that there are several ways to verify if the organ should be treated, because if the physician just assumes that a positive TL to the organ CR is enough to address the organ, he or she will often be misguided in their treatment and treat around the problem rather than the root cause of the issue.

One way to verify if the organ should be treated at the time is to use a Type III MMT (patient initiated submaximal MMT)[4] and perform the opposite action to the organ's visceral referred pain area (VRP) than which was first seen. In other words, if there is a weak muscle and the physician rubs over the VRP of an organ (creating more parasympathetic activity) to verify if this is something that should be treated at the time then the opposite should hold true – the physician should use a different muscle (a strong indicator) and using a Type III MMT, the physician should create a sympathetic response over the organ's VRP, which is done by pinching that VRP. A weakening of this muscle during the Type III test would confirm the Type I finding.

**In essence: Type I MMT weakness > physician rubs over VRP = strong; then Type III MMT of a strong muscle should become weak when the physician pinches over the same VRP. If the Type III test does not support the Type I testing response then it is not time for the organ to be treated, and the physician should investigate why that is. Often there is an injury, cranial fault, TMJ or tooth problem, or an immune system issue that needs to be addressed first.

Another way to verify if it is time to treat the organ is to check the acupuncture meridian alarm point for the organ. The alarm point is very effective, particularly as a diagnostic tool. The alarm point for the organ, (there are twelve, so not all organs have an alarm point), should TL and either strengthen the muscle related to the organ dysfunction or weaken another indicator muscle.[5]

A third approach in the verification process is to check for any other parasympathetic or sympathetic activity which may be affecting the organ in question. This can be performed by having the patient gaze at a point in the distance or retract their jaw (either of which will simulate more sympathetic activity) or by asking the patient to gaze at the nose (cross-eyed) or protrude their jaw (either will simulate more parasympathetic activity). If either test changes the MMT response then that indicates that more sympathetic or parasympathetic activity is influencing that muscle/organ in question from another viscerosomatic relationship. For example, if the patient has a weak latissimus dorsi and there is a positive TL to the pancreas CR, (the muscle strengthens), the physician may think that there is a need to address the pancreas. However, if the patient stares at the nose (without the TL to the CR) and this strengthens the latissimus dorsi, then this would indicate there is another organ in need of more parasympathetic activity, which is affecting the latissimus muscle and the pancreas. Therefore, the pancreas is not the primary organ and should not be addressed at that time in the treatment process. The physician should proceed by rubbing over the VRPs of other organs (parasympathetic effect) to see which one strengthens the latissimus and then continue based on that finding.

Cranial faults, temporomandibular joint (TMJ) dysfunction, tooth problems, and especially injuries, (actual injuries the patient has sustained as well as injuries that have occurred from compensations), can certainly be a primary problem, but are often secondary to another issue that needs to be corrected. As described in a previous paper by this author, TMJ problems are a common finding with dysglycemic patients, which comprises a vast majority of the population.[6]

Using the above procedures to verify if one should be treating a certain organ or other point at a certain time in the treatment process will also have a dramatic effect on the number of spinal subluxations which need to be corrected. As Walter H. Schmitt, D.C. describes, many subluxations or misalignments which appear to be in need of a correction are actually uncoupled, and that needs to be addressed before adjusting, or even adjusting at all.[7] Using the weight bearing techniques to check for hidden injuries as previously described and the sympathetic and parasympathetic procedures, as well as determining any TMJ or tooth involvement and using pre-test imaging to screen for cranial faults[3] will significantly decrease the amount of adjustments the patient needs. In this author's experience, it will often eliminate the need for them entirely.

Conclusion

A physician's success when treating a patient is largely determined by his or her skill in differentiating between what *is* a compensation and what *is not*. Essential to the

physician's treatment process is being led by certain indicators as well as changes, signs, and symptoms which the patient experiences and presents with during the visit. Treating only an indication that there is a problem is ultimately a disservice to the patient as his/her improvement will eventually unwind resulting in the same problem or a variation thereof on subsequent visits. Basically, the physician will be pushing the health problem around and the patient will see little or no lasting results. Compensations do need to be addressed, but the physician must discern between what is a compensation and what is the actual problem. By following the procedures presented in this paper, physicians can have a better understanding when a procedure should be performed and when something requires further investigation, should the verification processes not support the findings present at that time.

References

1. McCord KM, Schmitt WH. Quintessential Applications: A(k) Clinical Protocol. 2nd ed. St. Petersburg, FL: Healthworks!; 2009. step #4.

2. Gangemi SC. Faster and More Efficient Ways to Identify Hidden Injuries and Diaphragmatic Problems. In: Proceedings of the ICAK-U.S.A.; 2009-2010.

3. McCord KM, Schmitt WH. Quintessential Applications: A(k) Clinical Protocol. 2nd ed. St. Petersburg, FL: Healthworks!; 2009. steps #9-11.

4. Schmitt WS, Yanuck, SF. Expanding the Neurological Examination Using Functional Neurological Assessment: Part II Neurologic Basis of Applied Kinesiology. Int J Neuroscience 1999;97:97-8.

5. Walther DS. Applied kinesiology; Synopsis 2ed.. Shawnee Mission, KS: ICAK-U.S.A., 2009. p. 274.

6. Gangemi SC. The Dysglycemia Test and its Connection to Temporomandibular Joint Dysfunction and Tinnitus. In: Proceedings of the ICAK-U.S.A.; 2009-2010.

7. McCord KM, Schmitt WH. Quintessential Applications: A(k) Clinical Protocol. 2nd ed. St. Petersburg, FL: Healthworks!; 2009. step #29.

Enhancing Athletic Performance by Predicting Fatigue and Preventing Muscle Failure

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Abstract

Preventing muscle fatigue and increasing human performance, whether for the average person or a world-class athlete, is a considerable topic of investigation. Often, muscle fatigue occurs with no clear antecedent, typically sooner than expected, and always sooner than desired. If an individual, or even a specific muscle, is not experiencing any muscle inhibition (weakness) during the examination process, it is very difficult, if not impossible, to determine what may be done to prevent that weakness from occurring once exercise begins. What if there was a way to determine what would fatigue before it actually did? This author submits a way to challenge the body and test to see if prediction is possible, and, if so, determine to what extent prevention is possible or at least significantly delay fatigue onset.

Key Indexing Terms

Glucose, Branched Chain Amino Acids, BCAAS, Protein, Glycogen, Cortisol, Manual Muscle Testing (MMT), Gluconeogenesis

Introduction

Muscle fatigue has been shown to occur for many reasons – depletion of ATP, depletion of glycogen, dehydration, lactic acid accumulation, generalized physical fatigue, cramping, mental fatigue - the list goes on. The quest for athletes to push their physical performance to new levels is understandably under constant scrutiny; more strength and speed is of utmost importance. Dietary considerations are well known to have a major impact on performance, not only before and during exercise, but also immediately after. An increasing amount of research emphasizes post exercise carbohydrate and protein intake is paramount given their impact on performance.

The average 150-pound endurance athlete can store approximately 450g (1800Kcal) of glycogen/glucose while the average 120-pound female can store slightly under 400g (1550Kcal).[1] The intensity and duration of exercise will determine how much glycogen is being depleted, and therefore, how much eventually needs to be replaced. Protein requirements vary for individuals, but they are significantly increased in athletes. The current protein guidelines for endurance athletes are 1.6 to 1.7 g/kg for strength athletes and 1.2 to 1.4 g/kg for endurance athletes.[2] However, some studies suggest higher levels in endurance athletes, up to 15% of caloric expenditure.[3] For a 150lb male endurance athlete burning 4,000 Kcal a day, that would equate to 2.2g/kg of protein. Nitrogen

balance is the main factor affecting how much protein any person needs, athlete or not. Nitrogen balance refers to the body's state of either having enough protein available for the body to use or having a lack of protein for the body's use. When protein is broken down and used as energy, whether to repair, rebuild, or fuel the body, nitrogen is released, and an excess accumulation will overburden the body, resulting in ammonia toxicity. An individual with ammonia toxicity will experience physical and mental fatigue – an overall lack of motivation. Ammonia toxicity can be screened by performing the "Ammonia Sniff Test" as developed by Walter H. Schmitt, D.C.

During the recovery phase following any training regime or competition, there is a time considered as the "window of opportunity" where an athlete can recover faster than if he or she was to wait. The window is said to be open for approximately 60 minutes post-exercise and the focus should not be just on hydration, but also on protein and carbohydrate intake particularly. Approximately 100g of carbohydrates and 25g of protein are needed within the first hour after exercise (the actual amount depends on body weight, activity duration and intensity). This ratio of 4:1 is said to nearly double the insulin response, which results in more stored glycogen. Carbohydrate-protein (CHO-PRO) supplementation is more effective to rapidly replenish glycogen levels than just a regular carbohydrate supplementation post-exercise.[4] CHO-PRO supplementation has also been shown to improve exercise endurance during a second bout of exercise performed on the same day.[5] Glycogen stores are quickly replenished in depleted muscles (and to some extent the liver) and amino acids are readily available to repair any tissue damage, particularly those caused by cortisol's influence of converting amino acids (specifically branch chain amino acids) to glucose for fuel through the process of gluconeogenesis.

Amino acids are oxidized as substrate during prolonged exercise and both endurance and resistance training increase skeletal muscle protein synthesis and breakdown in the post-exercise period. During intense exercise workouts lasting longer than three hours, as much as ten percent of energy may come from protein. The branch chain amino acids (BCAAs) leucine, isoleucine, and valine are said to be the most important for recovery than any of the other amino acids. BCAAs make up about one-third of muscle's protein and enhance endurance by conserving glycogen, maintaining muscle mass, power and endurance during exhaustive bouts of exercise. Furthermore, BCAAs have been shown to help maintain immune status and therefore reduce overtraining.[6]

The BCAAs are catabolized by the glucose-alanine cycle, resulting in a net increase in glucose for anaerobic muscle contraction. The non-essential amino acid, alanine, is formed after glucose is converted to pyruvate in the liver, kidneys and muscle tissue. This cycle also serves to help prevent the buildup of toxic ammonia molecules, as previously mentioned in this paper. This is primarily a cortisol stimulated pathway, resulting in gluconeogenesis.[7]

Of the three BCAAs, leucine is thought to be the most important as it is essential to amplify the signal for protein synthesis at the level of peptide initiation. Studies have shown that feeding amino acids or leucine soon after exercise suggest that post-exercise

consumption of amino acids stimulates recovery of muscle protein synthesis via translation regulations.[8]

Discussion

Manual muscle testing (MMT) is based on a change in muscle function. Whether a facilitated (strong) muscle becomes inhibited (weak) or a weak muscle becomes strong, the physician is using this testing method as part of the neurological exam to aid in diagnosing processes gone awry in the patient's body. However, this review proposes there may exist times when MMT is not entirely sufficient in detecting a change in the muscle function. In other words, it is impossible for a physician to determine if a truly strong muscle is "even more strong" or an already weak muscle is "even more weak." Yes, there are varying degrees of inhibition and facilitation, but often a weak muscle will only respond favorably (by strengthening) if there are some positive or beneficial stimuli introduced. For example, this may be a need for a nutrient or correction of some physical or emotional problem.

Likewise, a strong muscle will often only weaken if there are some negative stimuli introduced. A dietary sensitivity (such as a food allergy) or an injury to a muscle would cause this to occur.

Additionally, muscles are going to respond more favorably to what they are most depleted in. In other words, if there is a need for supplement X, supplement Y, and supplement Z, typically not all three supplements are going to test positive during the same examination procedure. Often the most depleted nutrient will be needed first, even if the others are at unfavorable levels.

However, what if one was to take an already inhibited muscle and challenge it so it is functionally even more inhibited, even though this cannot be measured via MMT? This can be done by introducing some negative stimuli such as a food offender or by directly causing further stress to the muscle. The muscle is theoretically weaker, even though the MMT appears to be the same, and subsequent testing can be performed to see what negates the further muscle inhibition that was introduced.

For instance, consider a patient with bilateral weakness in both their gluteus maximus muscles. Having him/her perform some exercise to further inhibit those muscles, such as deep squats, or even an exercise like plyometric box jumps, is going to make the glut max muscles even more fatigued. The extra inhibition is going to stress the muscles out enough, which in turn will change how they will respond to testing – particularly oral nutrient testing – for the muscle in distress.

This is an important consideration for a couple reasons. First, most athletes do not present under such distress. Sometimes they may, say after an exhausting period of physical activity, but most recover quickly enough that by the time they get into the office, those muscles which were inhibited are already on their way to recovering. In essence, the athlete is pulling stored glycogen, and especially protein sources, to recover as quickly as

they can. Second, the muscle is going to respond differently to any testing if it is stressed out more and tested immediately after, even though it is already weak in both cases. An athlete with weak glutes provides another example. Having him/her perform ten or more deep knee bend jumps is going to further inhibit those muscles and change how they test and what might strengthen them. By having the athlete perform this type of testing, the physician can see two things he or she would otherwise miss. First, the physician will be able to determine what will aid in faster recovery in the weak muscle, not only locally but systemically. The physician will then be able to advise the athlete in regards to dietary changes to prevent the condition from occurring again. The physician is then able to fundamentally predict what is going to happen when muscles are further stressed even though it cannot be measured via MMT. This allows for the significant delay and even the possible prevention of muscle fatigue.

This author has determined that the two major factors contributing to fatigue and poor performance are glycogen depletion and insufficient protein/BCAA supplementation. Once the body depletes glycogen it will need to make glucose from other sources (this is called gluconeogenesis). The most common source comes from breaking down protein (muscle) into amino acids for fuel. Note that this response is due to the glycogen depletion. If the glycogen levels were adequate, then the body would not shift to using protein as fuel. However, even though both glycogen levels and protein levels are insufficient, the body is more concerned about getting the protein levels back up so muscle tissue is spared. But again, this would never occur if glycogen levels were at an optimum.

Clinically, this can be verified. If the physician asks the patient to further stress a muscle that is already weakened, and they are depleted in either glycogen, protein, or typically both, the muscle will then strengthen during oral nutrient testing of some high quality protein source (such as denatured whey protein) or BCAAs. Testing of the protein or BCAAs against the weak muscle without further stressing it often shows no change (no need for supplementation) unless the patient/athlete is severely depleted. In the experience of this author, this is very uncommon. However, positive testing for more protein is more common than one may imagine if the weak muscles are stressed, and in essence, made “more weak.”

Correcting this problem can be simple or complex depending on each individual patient. The immediate need for more protein in the diet is most important. This will get the patient out of any crisis mode. Second, glycogen stores should be assessed, which can be done by looking at the patient’s diet as well as training regime. Third, the training/exercise program should be assessed, with particular attention given to aerobic/anaerobic systems.

Procedure

1. Identify a muscle that is inhibited (weak) and responds to autogenic facilitation (muscle turns on with spindle cell activation)

2. Perform oral nutrient testing using a protein source (preferably denatured, non-hydrolyzed whey protein), BCAAs, and some complex carbohydrate source such as maltodextrin
    a. If any of the three above test positive, investigate the need for such supplementation accordingly
    b. If none tests positive, continue to 3.

3. Have the patient further stress the muscle that is weak to the point where they are at least 80% fatigued. This is easy for some muscles, such as a pectoralis muscle (they can perform multiple push-ups), or rectus/glutes (they can perform deep squats or jumps), but more difficult for others. If you are using a muscle such as a latissimus dorsi and can't have the patient perform an exercise such as pull-ups, you will either need to find a different muscle or use another muscle as an indicator. For example, you can perhaps find an acupuncture meridian alarm point which the patient can therapy localize to and weaken a muscle that can be tested.

4. Immediately after the patient stresses out the weak muscle, test them again on the three substances – the [whey] protein, BCAAs, and the carbohydrate source
    a. A positive test indicates glycogen depletion and the body's shift towards protein as fuel is strengthening on the protein and/or BCAAs. *Note, this author has yet to find a patient strengthen on the carbohydrate during this part of the testing, even though they are theoretically glycogen depleted.
        i. Assess the patient's diet – specifically protein intake (perhaps aim for approximately 2g of protein per kilogram of body weight) and carbohydrate intake
        ii. Assess the patient's exercise intensity and frequency. This can be done by looking at their training log as well as administering specific testing procedures set forth by this author in a previous paper, to assess aerobic, anaerobic, and creatine phosphate pathways.[9]
    b. A negative test is no change in the muscle test, as it was before the muscle was stressed.

** Interesting side-note: Often during a positive test, the doctor will find that if they ask the patient to perform *any* strenuous, power-induced exercise to failure, the *strong* muscle they are stressing will weaken. In other words, if the patient strengthens on protein or BCAAs after the weak muscle is stressed, have him or her perform an exercise such as deep knee jumps to the point of complete failure. (These muscles should have been tested to be strong.) The muscles related to that exercise (specifically the rectus and glutes in this example) will be completely

inhibited, but will strengthen on the protein or BCAA substance already discovered to benefit some other inhibited muscle.

Conclusion

The beauty of employing MMT in the examination of patients is that the physician can specifically identify a problem that an individual may be having and correct that problem through various means. Challenge procedures are often needed to further stress out the patient's nervous system, as often a problem the patient is having may be hiding under many adaptations they have developed or their problem may only show itself under a particular stress. Such is the case when testing and determining the best treatment process for athletes, particularly those involved in endurance events and those at the elite level.

The common way of muscle testing to see a change from negative to positive (weak to strong) or vice-versa is not necessarily going to reveal problems in certain individuals. In athletes who are only experiencing a problem when under a particular amount of stress, that stress must be replicated to some degree in the office setting in order to find a correction and treatment protocol. However, when glycogen levels are depleted and the body is literally using muscle (protein/amino-acids) for fuel, compensations have occurred to such degrees that "smoking out" the problem is often difficult, if not impossible. Challenging a muscle, or group of muscles, to the point of fatiguing them beyond their current state while there is no discernable change in the MMT, and then testing based on that understanding, is a significant new direction in evaluating and treating a patient. Fatigue that is going to occur, but has not yet done so, can be identified and performance can be significantly increased.

The implications which may come forth through this type of testing are not just limited to athletes. Challenging a patient with a substance and seeing no change in the MMT outcome does not necessarily mean there is not a change. Absence of proof is not proof of absence. All it can mean is that there is no change, which can be observed through current methods. If a secondary testing procedure is utilized after the assumed negative response is employed, the doctor can see that a change did occur, identified by the MMT response.

References

1. Sleamaker R, Browing R. Serious training for endurance athletes. Champaign, IL: Human Kinetics; 1996. p.156.

2. Fielding R, Parkington J. What are the dietary protein requirements of physically active individuals? New evidence on the effects of exercise on protein utilization during post-exercise recovery. Nutr Clin Care. 2002;5(4):191-6.

3. Lemon PW, Proctor DN. Protein intake and athletic performance. Sports Med 1991;12:313–25.

4. Ivy JL, Goforth Jr. HW, Damon BM, McCauley TR, Parsons EC, Price TB. Early postexercise muscle glycogen recovery is enhanced with a carbohydrate-protein supplement. J Appl Physiol 2002;93:1337-44.

5. Niles E, Lachowetz T, Garfi J, Sullivan W, Smith J, Leyh B, Headley S. Carbohydrate-protein drink improves time to exhaustion after recovery from endurance exercise. J Exercise Phys 2001;4(1):45-52.

6. Friel J. The triathlete's training bible. 2nd ed. Boulder, CO:Velo Press; 2004. p 264.

7. Di Pasquale MG. Amino acids and proteins for the athlete: the anabolic edge. Boca Raton, FL:CRC Press; 1997. p.85.

8. Layman DK. Role of leucine in protein metabolism during exercise and recovery. Appl. Physiol. Nutr. Metab 2002;27(6):646–62.

9. Gangemi SC. New and updated challenge procedures to assess anaerobic, aerobic, and creatine phosphate pathways. In: Proceedings of the I.C.A.K. - U.S.A. 2009-2010.

Emotional Equation Supplements:
Neutralizing Deep-Rooted Emotional Charges from Past Relationships and Feelings of Victimization

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Abstract

John Diamond, MD, in his work, *Life Energy*, suggested that if mental attitude is changed, stress is prevented, and disease will not occur.[i] Relieving somatic manifestations of emotional stress has been accomplished by tapping acupuncture head points while the patient thinks about an emotionally stressful problem, person, or event. Walter Schmitt, DC, DABCN, DIBAK enhanced this technique with Emotional Recall Quick Fix, identifying acupuncture head points that negated thought induced weakness, and performing Injury Recall Technique, herein referred to as IRT, to the Chapman's Reflex related to the acupuncture head point.[ii]. Applying these techniques to past relationships and experiences of victimization may help to neutralize long-standing and often hidden emotional factors.

Key Indexing Terms
Acupuncture, Meridian, Emotional Recall, Applied Kinesiology, Chapman's Reflexes, Emotional Healing, Emotional Techniques, Relationships

Introduction

Merriam-Webster Online Dictionary defines emotion as a "conscious mental reaction (as anger or fear) subjectively experienced as strong feeling usually directed toward a specific object and typically **accompanied by physiological and behavioral changes in the body"**.[iii] Gregg Braden, in Walking Between the Worlds: The Science of Compassion, writes, "Researchers have now demonstrated to the Western world that human emotion determines the actual patterning of DNA within our bodies… Furthermore, the demonstrations have shown that the arrangement of matter (atoms, bacteria, viruses, climate, even other people) surrounding your body, is directly linked to the feeling and emotion from within your body."[iv] Applied Kinesiology methods and methods of techniques with origins tied to applied kinesiology to affect emotional and emotionally induced physiological states have included psychological reversal, emotional recall, emotional recall quick fix, Neuro-Emotional Technique, and emotional erase[v], all of which include usage of the body's acupuncture meridians.

The combined application of these techniques to past relationships and any sense of victimization may help neutralize deep-rooted emotional charges. In the case of past relationships, as will be discussed in this paper, certain personality characteristics of the

other person in the relationship may trigger a neurological weakness, as seen in manual muscle-testing. A sense of victimization may be broken down into three components – justice, forgiveness, and self-responsibility – and the treatment of each of one these factors is imperative in neutralizing highly charged emotional factors which may trigger physiological breakdown.

Discussion

Long-standing emotional issues may have their origins in either past relationships and/or an experience (past or present) involving the feeling of victimization. In the case of past relationships, it is not merely the experience of the relationship, which causes an extreme ripple effect, but the actual personality traits of the other person involved that has deep emotional effects. Neutralizing (or clearing) these emotional charges, prove an effective supplement in emotional healing techniques as described in my 2008 ICAK Proceedings paper, "Enhancing Emotional Recall Quick Fix: Utilizing Sensory Modalities Beyond the Primary Representational System"[vi] and my 2009 ICAK Proceedings paper, "The Emotional Equation: Raising the Level of Consciousness".[vii] For the biomechanics behind the methodology, please refer to my previous papers.

I. Clearing Past Relationships: Eliminating Personality Attractors and Triggers

Braden comments, "Our relationships serve as direct windows into the thinking, feeling, and emotional postures that we assume in our world."[viii] He adds, "You will only see in others that which the filters of your belief allows you to see."[ix] Additionally, Braden writes, "Once a pattern changes in one relationship, all other relationships that are based in the same pattern benefit from the original change."[x]

List the top 5 people you can think of that, over the course of your lifetime and personal to you (ie not a political, celebrity, or historical figure) that you deem "evil", may have wronged you, treated you poorly, or you simply don't like. Remember that those considered most beloved may also be considered at times to be most evil. Underneath the names of each person, list only what you consider to be their most negative characteristics. List whatever comes to mind.

Ex: John – controlling, hurtful, lazy, unfaithful

Now examine your list. If you cover the names, you may find that regardless of the name, all of your "evil" people share the same negative qualities. They are almost the same person across the board. This may be the reason why many people share having the same relationship, over and over again, despite the change of the significant other.

You may have an emotional charge on each of the qualities listed. This may affect you in several ways:

1) Many authors have postulated, "Like attracts like." Thus, the charge you have on each of these qualities actually attracts it! If you have an emotional charge to a personality

trait such as “manipulative”, you may find yourself repeatedly attracting manipulative people into your life. By eliminating the charges, you may find yourself soon attracting positive people and situations instead of reliving yesterday’s relationships.

2) Each person you meet and situation you encounter must pass through your personal judgmental barrier and judged through your very own personally filtered lenses. Individuals who may actually be beneficial to your experience may not make it through your altered fortifications. By eliminating the charges, you may now allow positive people and situations to enter your life.

3) Each of the negative qualities listed serve as an emotional trigger of sorts. Have you ever had an unpleasant conversation, no matter how brief, and found that it affected your entire day? Have your friends told you that a certain person “brings out the worst in you”? By eliminating the emotional charges, you may help to eliminate the triggers that underlie processes of dis-ease, even if seemingly unrelated.

Step-by-Step Procedure:
Preface: You may choose to give the patient the homework assignment of listing the top 5 “evil” people (or people who have wronged them, rubbed them the wrong way, or for whom the patient has some dislike/distaste), and then having the patient list what he/she the negative qualities of each person. Please remind the patient, that the person considered to be “most loved” (like a parent) may also be on the “most evil” list. Only list the people that have personally affected the patient, as opposed to listing historical, political, or celebrity figures (unless, of course, such a figure actually did indeed personally affect the patient). The patient may then construct a new list, in order of importance, of the most prevalent (most frequently listed) negative qualities, and also include within that list any negative qualities that really strike a chord (even if only listed once) or resonate negatively within the patient. When the patient presents for treatment, you can treat each listed characteristic individually.

You may also do without the homework assignment, as shown below.

1. Patient is supine. Practitioner finds a strong indicator muscle, preferably the Pec Major Clavicular.
2. If the homework assignment listed above has been completed, the practitioner may pick the first characteristic on the list. Otherwise, the practitioner may simply ask the patient to think of someone who, at some point during their lifetime, has either wronged them in some way, shape, or form, or who they dislike, or who they consider to be one of the most evil people during their lifetime, reminding the patient that this person is personal and not a political, historical, or celebrity figure, and may be from any point in their life. The practitioner may also remind the patient that the most loved may also be the most evil (ex: a parent).
    a. Example: Patient thinks of an ex-boyfriend.
3. The practitioner now asks the patient to think of a negative quality of this person, and further asks the patient to now concentrate on the quality, trait, or characteristic, and not the person. Practitioner performs a muscle test (test the

formerly strong indicator muscle) with the patient thinking about the negative personality trait and the strong indicator muscle weakens.
    a. Example: Trait is Hurtful (patient thinks about the quality of "hurtful") and the indicator muscle tests weak
4. Patient continues thinking about personality trait or characteristic that weakens indicator muscle and practitioner taps acupuncture head points until a pair is found that negates weakness.
    a. Ex: Tapping or therapy localizing ST1 negates the weakness.
5. Practitioner may also choose to test the laterality to see if the acupuncture head point is on the left or right side of the head. Positive point on either right or left strengthens weak indicator muscle while patient thinks about stressor.
6. Patient therapy localizes neurolymphatic (Chapman's Reflex) points correlated to coupled meridian acupuncture head points until one point, on either right or left side, is found that negates weak indicator muscle. For example, if therapy localizing ST1 facilitates the indicator muscle, test both the Stomach and Spleen Chapman's (Neurolymphatic) Reflex points. For Triple Warmer meridian, test Thyroid NL point as well as the Thymus point. For Circulation-Sex meridian, test adrenal NL point or reproductive organs NL point.
    a. Ex: Therapy localizing the Stomach Chapman's Reflex strengthens the indicator muscle, while therapy localizing the Spleen Chapman's Reflex does not.
7. Practitioner places patient's hands on indicated Chapman's Reflex point(s). The Practitioner has a choice of tapping the acupuncture head points bilaterally, or tapping the Beginning/End Points on one side only. Neither option has appeared to hold a significant advantage over the other in recent tests. If point in unilateral, then both hands therapy localize the same point. Practitioner taps acupuncture head point 120 times while patient continues to think of negative personality trait that weakens while patient therapy localizes the indicated Chapman's Reflex point(s).
    a. Ex: Practitioner taps ST1 bilaterally 120 times while patient therapy localizes the Stomach Chapman's Reflex, simultaneously thinking about the trait of being hurtful.
8. Practitioner performs IRT to Chapman's Reflex while patient continues to think of negative personality trait that weakened indicator muscle.
    a. Ex: Practitioner performs IRT using talus distraction while patient therapy localizes Stomach Chapman's Reflex while thinking about "hurtful".
9. Practitioner moves patient to a seated position and affirms a strong indicator muscle. The patient Therapy Localizes the alarm point(s) that correspond to the Chapman's Reflex area and the practitioner checks for weakening of the indicator muscle. If there are bilateral alarm points, test each point independently.
    a. Ex: Patient therapy localizes the Stomach alarm point and checks for weakening of the indicator muscle.
10. As the patient continues to Therapy Localize the alarm point which caused weakening of the indicator muscle, the practitioner has the patient recite the correlated Life Energy Affirmations[xi] (listed in the latter part of this paper) as the

practitioner tests for strengthening of the weak indicator muscle following each affirmation. This is performed in a "repeat after me" format.

   a. Example: With the patient Therapy Localizing the Stomach Alarm Point (which caused the indicator muscle to go weak), instruct the patient to "repeat after me", and then recite the following affirmations, testing the indicator muscle after each affirmation is spoken:
      " I have enough."
      "I am enough."
      "I am satisfied."
      "I am thankful."
      "I am tranquil."

11. Upon finding the affirmation that negated the weakening caused by the Therapy Localization of the indicated alarm point, instruct the patient to keep his or her contact on the alarm point, and in a manner similar to that of Dr. Scott Walker's Neuro-Emotional Technique[xii], the patient puts the palm of the free hand on his/her forehead, encompassing the emotional Neurovascular points. The practitioner instructs the patient to SILENTLY REPEAT the affirmation found in the last step.
    a. Example: the patient would have one hand therapy localizing the Stomach alarm point, while the other hand therapy localizes the entire forehead (with the intention of covering both emotional Neurovascular points). The practitioner instructs the patient to now "Silently repeat, 'I am enough. I am enough. I am enough,' over and over again."
12. While the patient is holding both the points on the head and body and reciting the affirmation silently, the practitioner, again in a manner similar to that of Dr. Scott Walker's Neuro-Emotional Technique[xiii] as well as Dr. Victor Frank's Total Body Modification[xiv], using either his/her hands, a double-tipped activator instrument, or an IMPAC Arthro-Stim, stimulates Dr. Frank's TBM Sequence points on the Transverse Processes which correspond to that organ. Stimulate first while the patient's breath is neutral, then again while instructing the patient to breathe in, and again while instructing patient to exhale.
    a. Example, the practitioner adjusts the T8, T10, and lastly T12 transverse processes upon neutral breath, inhalation, and then exhalation (one vertebra at a time) by the patient while the patient silently recites the affirmation of "I am enough."
13. If the practitioner wishes to investigate further, he or she may use investigative techniques used in Neuro-Emotional Technique, in order to find out if there were precipitating events in the patient's lifetime that lead to the block at the indication affirmation. In our example, the patient may ask the body if "I am enough" is the original concept, or if there is was a more original concept. Not wanting to re-present Dr. Walker's original work and claim it as my own, please refer to seminars and manuals by Dr. Scott Walker and *Neuro-Emotional Technique (N.E.T)*.
14. Practitioner may also wish to recheck the original personality trait using different sensory modalities. For more information on checking different modalities/predicates, please refer to my paper in the 2008 ICAK Annual

Proceedings entitled, "Beyond Emotional Recall Quick Fix – Using Representational Systems."[xv]

15. Recheck personality trait to confirm correction.
16. Practitioner may now choose to test a different personality trait, or ask the patient to either think of a different "evil" person, or the same person but a different characteristic/trait that that person has exhibited, and repeat the entire procedure. The practitioner may simply perform a correction on a single trait, or continue to treat as many different traits as desired.
17. Let the patient know that sometimes the effects are not immediate. The patient may feel very subtly more grounded, balanced, relaxed, and/or lighter immediately, but should really notice the effect in about 20-30 minutes. The practitioner may recommend meditation, any type of meditative yoga (this author practices Kundalini Yoga and has found it to provide an excellent adjunct), as well as The Sedona Method®, which helps the patient to actively let go, or release, aberrant thought and belief programs that are slowing down the body's hardware. The patient may even choose to work on releasing whatever thought patterns or affirmations were found in the office visit.
18. This technique may be used more frequently (every other day) in cases of extreme stress. Be forewarned that as this technique inhibits the amygdala, over-utilization may result in a sort of "emotional numbing".

II. Victimization Process

I was working with a client on some emotional releasing, and without her getting into the story, she made a reference to not wanting/being able to forgive someone. Before I could say anything, she attempted to fortify her position by saying, "I have to take my power back. If I forgave him for what he did, I would be giving my power away."

I have often read that a sense of victimization will greatly impede ANY type of healing. In the above story, my client would not forgive the "offender" because, according to her, by not forgiving him, she would prevent giving her power away, and thus she would not be a victim. However, by not forgiving the offender, she was actually giving her power away by allowing a past experience to continually affect her. This person may have wronged her in the past, but exactly who is wronging her NOW?

In order to release any feeling of victimization, three objectives must be accomplished:

1) Letting go of any WANTING to be the victimizer, WANTING justice, vengeance, revenge, to get even, and so forth. (notice the emphasis on "wanting".) It is the "wanting" that often carries the emotional charge.
2) Forgiving all parties involved
3) Taking responsibility for yourself in that situation, in whichever way you can. This is NOT the same as accepting blame. For example, if God forbid I was mugged while jogging in Central Park, I might think, "Next time I run during daylight hours; or maybe I will take a big dog as a running companion, or perhaps run with a group of people; I

don't carry $500 in my wallet with me, etc, etc." Taking self-responsibility actually may be equated with self-forgiveness.

So if my client was agreeable to forgiving the offender, she would actually be enabling herself to release feelings of victimization. However, since she is not ok with forgiveness in this instance, she is actually involuntarily retaining a feeling of victimization, which is counterintuitive to her objective of "taking her power back."

Step-by-Step Procedure:

1. Patient is supine. Practitioner finds a strong indicator muscle, preferably the Pec Major Clavicular.

2. Ask the client to pick an experience that occurred during the course of their lifetime in which they felt victimized. If practitioner wishes, he/she may first treat the entire experience using the method employed in my 2008 ICAK Proceedings paper, "Enhancing Emotional Recall Quick Fix: Utilizing Sensory Modalities Beyond the Primary Representational System."[xvi] This is not totally necessary, as the practitioner may simply move on to treating the three parts of victimization.

    Example: Patient thinks of an experience in which he/she felt like a victim – a break-up (end of relationship), bad business deal, etc., etc.

3. SEEKING JUSTICE - The practitioner now asks the patient IF there is any sense of WANTING to get even, seek retribution, justice, etc., etc. I use the word "If" because there may not always be a sense of wanting to "get even." If there is indeed a sense of wanting justice, vindication, vengeance - test (test the formerly strong indicator muscle) with the patient thinking about wanting to get even, achieve justice, etc., etc. and the strong indicator muscle weakens. If there is no sense of wanting to get even or seek justice, move on to the next part (Forgiveness) of the victimization process.

    Example: Patient thinks of a boyfriend who was unfaithful and stole money, and thinks about wanting to get even

4. Patient continues thinking about personality trait or characteristic that weakens indicator muscle and practitioner taps acupuncture head points until a pair is found that negates weakness.

    Example: Tapping or therapy localizing ST1 negates the weakness.

5. Practitioner may also choose to test for laterality in order to see if the acupuncture head point is on the left or right side of the head. Positive point on either right or left strengthens weak indicator muscle while patient thinks about wanting to get even.

6. Patient therapy localizes neurolymphatic (Chapman's Reflex) points correlated to coupled meridian acupuncture head points until one point, on either right or left side, is found that negates weak indicator muscle. For example, if the Therapy localizing ST1 facilitates the indicator muscle, test both the Stomach and Spleen Chapman's

(Neurolymphatic) Reflex points. For Triple Warmer meridian, test Thyroid NL point. For Circulation-Sex meridian, test adrenal NL point or reproductive organs NL point.

Example: Therapy localizing the Spleen Chapman's Reflex strengthens the indicator muscle, while therapy localizing the Stomach Chapman's Reflex does not.

7. Practitioner places patient's hands on indicated Chapman's Reflex point(s). The Practitioner has a choice of tapping the acupuncture head points bilaterally, or tapping the Beginning/End Points on one side only. Neither option has appeared to hold a significant advantage over the other in recent tests. If point in unilateral, then both hands therapy localize the same point. Practitioner taps acupuncture head point 120 times while patient continues to think of wanting to get even while patient therapy localizes the indicated Chapman's Reflex point(s).

Example: Practitioner taps ST1 bilaterally 120 times while patient therapy localizes the Spleen Chapman's Reflex while thinking about wanting to get even.

8. Practitioner performs IRT to Chapman's Reflex while patient continues to think of negative personality trait that weakened indicator muscle.

Example: Practitioner performs IRT using talus compression while patient therapy localizes Spleen Chapman's Reflex while thinking about wanting to get even.

9. Practitioner moves patient to a seated position and affirms a strong indicator muscle. The patient Therapy Localizes the alarm point(s) that corresponds to the Chapman's Reflex area and the practitioner checks for weakening of the indicator muscle. If there are bilateral alarm points, test each point independently.

Example: Patient therapy localizes the Spleen alarm point and checks for weakening of the indicator muscle.

10. As the patient continues to Therapy Localize the alarm point which caused weakening of the indicator muscle, the practitioner has the patient recite Dr. John Diamond's Life Energy[xvii] Affirmations, as listed towards the end of this paper, as the practitioner tests for strengthening of the weak indicator muscle following each affirmation. This is performed in a "repeat after me" format.

Example: With the patient therapy localizing the Spleen Alarm Point (which caused the indicator muscle to go weak), instruct the patient to "repeat after me", and then recite the following Spleen-related affirmations, testing the indicator muscle after each affirmation is spoken:

" I have faith and confidence in my future."
"I am secure."
"My future is secure."
"I have faith and courage in my future."

11. Upon finding the affirmation that negated the weakening caused by the therapy localization of the indicated alarm point, instruct the patient to keep his or her contact

on the alarm point, and in a manner similar to that of Dr. Scott Walker's Neuro-Emotional Technique[xviii], the patient puts the palm of the free hand on his/her forehead, encompassing the emotional Neurovascular points. The practitioner instructs the patient to SILENTLY REPEAT the affirmation found in the last step.

> Example: the patient has one hand therapy localizing the Spleen alarm point, while the other hand therapy localizes the entire forehead (with the intention of covering both emotional Neurovascular points). The practitioner instructs the patient to now "Silently repeat, 'My future is secure. My future is secure. My future is secure,' over and over again."

12. While the patient is holding both the points on the head and body and reciting the affirmation silently, the practitioner, again in a manner similar to that of Dr. Scott Walker's Neuro-Emotional Technique[xix] as well as Dr. Victor Frank's Total Body Modification[xx], using either his/her hands, a double-tipped activator instrument, or an IMPAC Arthro-Stim, stimulates Dr. Frank's TBM Sequence points on the Transverse Processes which correspond to that organ. Stimulate first while the patient's breath is neutral, then again while instructing the patient to breathe in, and again while instructing patient to exhale.

> Example: the practitioner adjusts the T1, T5, and lastly T9 transverse processes upon neutral breath, inhalation, and then exhalation (one vertebra at a time) by the patient while the patient silently recites the affirmation of "My future is secure."

13. Recheck the sense of wanting to get even to confirm correction. (the patient will often report that there is no more sense of wanting justice.) If negative, continue to next step (Forgiveness).

14. FORGIVENESS – Ask the patient to think about the CONCEPT of forgiving ALL PARTIES involved. Please be sure to let the patient know that you are not asking the patient to actually forgive, but merely to THINK ABOUT (the concept of) forgiving all parties involved. If the patient chooses to think about and correct for each party individually, then you may correct in that fashion. Follow the same correction procedure as indicated in correcting for the wanting to get even.

15. TAKING RESPONSIBILITY FOR YOURSELF – Ask the patient to think about taking responsibility for him/herself. This is not the equivalent to blaming oneself. For example, I usually phrase it in this manner: "In that instance, or a future similar instance, think about taking responsibility for yourself. For example, if god forbid I was mugged while jogging in the park, I may think, 'Next time I jog with a dog at my side', or 'I don't wear my IPOD on my arm', or 'I don't have my wallet bulging out of my sock'. You are not blaming yourself. You are simply taking responsibility for yourself in whichever way you possibly can." Treat the patient using the same procedure for the previous two parts (Getting even, Forgiveness) of the victimization process.

16. Let the patient know that sometimes the effects are not immediate. The patient may feel very subtly more grounded, balanced, relaxed, and/or lighter immediately, but should really notice the effect in about 20-30 minutes. The practitioner may recommend meditation, any type of meditative yoga (this author practices Kundalini Yoga and has found it to provide an excellent adjunct), as well as The Sedona Method®, which helps the patient to actively let go, or release, aberrant thought and belief programs that are slowing down the body's hardware. The patient may even choose to work on releasing whatever thought patterns or affirmations were found in the office visit.

17. This technique may be used more frequently (every other day) in cases of extreme stress. Be forewarned that as this technique inhibits the amygdala, over-utilization may result in a sort of "emotional numbing".

In summation, to facilitate healing, release all related feelings and attachments to victimization. This is accomplished by letting go of wanting vengeance/justice; forgiving all parties involved; and taking responsibility for yourself in that instance. An inability to forgive may actually impede any objective of taking one's power back.

It should be noted that I always use "The Emotional Equation", as described in my 2009 ICAK Proceedings paper entitled, "The Emotional Equation: Raising the Level of Consciousness" as a finishing step or clean-up move.

Life Energy Alarm Points and Affirmations, from Dr John Diamond's *Life Energy*[xxi] and associated Teas and Victor Frank TBM Sequences[xxii]

(Note: This author highly recommends a thorough reading of Dr. Diamond's *Life Energy* in order to fully grasp all concepts instead of merely looking at an outline).

BILATERAL MERIDIANS

LUNG

Negative	Positive
Disdain	Humility
Scorn	Modesty
Contempt	Tolerance
Haughtiness	
False Pride	
Intolerance	
Prejudice	

POSITIVE AFFIRMATIONS:

I am humble.
I am tolerant.
I am modest.

Sequence: Left: T1, T8, L2; Right: T2, T9, L3

LIVER

Negative	Positive
Unhappiness	Happiness
	Cheer

POSITIVE AFFIRMATIONS:

I am happy.
I have good fortune.
I am cheerful.

Sequence: T2, T5, T8

GALL BLADDER

Negative	Positive
Rage	(Reaching out with) Love
Fury	(Reaching out with) Forgiveness
Wrath	Adoration

POSITIVE AFFIRMATIONS

I reach out with love.
I reach out with forgiveness.
I come forward with love and forgiveness.
I adore.

Sequence: T4

SPLEEN

Negative	Positive
Anxiety about future	Faith (about the future)
	Confidence (about the future)
	Security

POSITIVE AFFIRMATIONS

I have faith and confidence in my future.
I am secure.
My future is secure.
I have faith and courage in my future.

Sequence: T1, T5, T9

KIDNEY

Negative	Positive
Sexual Indecision	Sexual Security/Assuredness

POSITIVE AFFIRMATIONS

I am sexually secure.

My energies are balanced. (Note: This affirmation is modified from the original affirmation as listed by Diamond In *Life Energy*: "My sexual energies are balanced."[xxiii])

Sequence: T1, T5, T8

LARGE INTESTINE

Negative	Positive
Guilt	Self-Worth
Obsessional thinking	

(Note: While Diamond does not list Obsessional thinking as a negative emotion, he mentions "People who feel guilty tend to be obsessional."[xxiv])

POSITIVE AFFIRMATIONS

I am basically clean and good.
I am basically clean and pure.
I am worthy of being loved.
I am loveable.*
(* added by this author)

Sequence: L5

MIDLINE MERIDIANS

CIRCULATION-SEX

Negative	Positive
Regret and Remorse	Renunciation of Past
Sexual Tension	Relaxation
Jealousy	Generosity
Stubbornness	Kindness

POSITIVE AFFIRMATIONS

I renounce the past.
I am relaxed. My body is relaxed.
I am generous.
That is done. It is the past. I will let it go and move on in the present.
My mind is wholly disconnected with things of the past.

Sequence: T7, T9, T11

HEART

Negative	Positive
Anger	Love
	Forgiveness

POSITIVE AFFIRMATIONS

I love.
I forgive.
There is forgiveness in my heart.

Sequence: T2, T8, T12

STOMACH

Negative	Positive
Disappointment	Contentment
Disgust	Receiving Enough
Greed	Having Enough
Bitterness	Tranquility
Emptiness	
Deprivation	
Nausea	
Hunger	

POSITIVE AFFIRMATIONS

I am content.
I am tranquil.
I have enough. What I have is sufficient.
I am thankful for what I have now.
I am thankful for having enough now.
I am enough.*
I am satisfied.*
(* added by this author)

Sequences: T8, T10, T12

THYROID/TRIPLE HEATER

Negative	Positive
Depression	Elation
Heaviness	Lightness
Despair	Buoyancy
Grief	Floating
Hopelessness	Hope
Despondency	
Loneliness	
Solitude	

POSITIVE AFFIRMATIONS

I am light and buoyant.
I am buoyed up with hope.
I am hopeful.*
I am lifted up by hope.*

(* added by this author)

Sequence: C1, C4, C7
Teas: Green Tea, Vitality, Ginseng, Horny Goat Weed

THYMUS/TRIPLE HEATER

Negative	Positive
Hate	Love
Envy	Faith
Fear	Gratitude
	Trust
	Courage

POSITIVE AFFIRMATIONS

I have love, faith, gratitude, trust, and courage.
I love.*
I am filled with love.*
I trust.*
I am filled with faith and trust.*
(* added by this author)

Sequence: T9
Teas: Immune System Enhancing Teas

SMALL INTESTINE

Negative	Positive
Sorrow	Joy
Sadness	

POSITIVE AFFIRMATIONS

I am full of joy. (I am joyful)
I am jumping with joy.
I have joy in my life.*
I am lifted up with joy. *
(* Added by this author)

Sequence: L5

BLADDER

Negative	Positive
Restlessness	Peace
Impatience	Harmony
Frustration	Patience
	Serenity
	Calm

POSITIVE AFFIRMATIONS

I am at peace.
I am in harmony.
Dissonances and conflicts within me have been resolved. I am balanced.

Sequence: L5

GOVERNING VESSEL
Negative
Embarrassment

No affirmations listed. Have the patient make up his or her own based on the opposite of the negative attitude.

Sequence: T3, T6 (from Dr. Scott Walker, N.E.T)[xxv]

CONCEPTION VESSEL
Negative
Shame
Shyness

No affirmations listed. Have the patient make up his or her own based on the opposite of the negative attitude.

Sequence: T3, T6 (from Dr. Scott Walker, N.E.T)[xxvi]

When To Use

Dr. Walter Schmitt and Dr. Kerry McCord, in their *Quintessential Applications of Applied Kinesiology Protocol*, put the step of treating the emotional/mental component at the end of their protocol, following all treatment for the organs, but just before dealing with any fascial elements or the actual vertebral or extra-spinal adjustment. However, in this same protocol, the "emotional step" may be performed while correcting cranial-sacral flow disturbances, if it is indeed found that emotional stressors are primary and are playing a role in the dysfunction of the cranial-sacral mechanism and/or the immune system[xxvii]. In either case, correcting neurological injury patterns, neutralizing histamine, allergy, and/or sensitivity reactions, and improving neurotransmitter function should be performed prior to any type of emotional treatment. If a histamine reaction is not neutralized prior to treating emotional factors, deleterious effects, including greater stressful feelings, may occur.[xxviii] For further elaboration of their recommended protocol, please refer to the *Quintessential Applications of Applied Kinesiology* manual or Recorded (video) lectures, available from Applied Kinesiology Study Program. As Dr. Schmitt notes in his newsletter, The Uplink, the "emotional correction" is "NOT an optional step."[xxix]

Ideally, the practitioner performs the Personality Trait and Victimization Process steps following the emotional release of a current or surface stressor, and finishes the emotional treatment using the Emotional Equation. The practitioner may also skip the current stress and jump right to the other techniques (Emotional Equation, if performed, should be performed last). The practitioner may choose to perform other Emotional Techniques as well, depending on the desired length of the treatment session, and the desired portion of the treatment visit devoted to the Mental/Emotional Side of the Triad of Health. This author first checks for and performs The Emotional Recall-Quick Fix using Representational Systems as outlined by this author's paper of that title in the *2008 Annual Proceedings of the ICAK*[xxx] and perhaps exploring the patient's main stressor(s) using Dr. Scott Walker's Neuro-Emotional Technique. This step is then followed by clearing charges on Personality Traits and the Victimization Process, before finally utilizing The Emotional Equation as the very last step of treating the emotional/mental side of the Triad of Health.

Table 1 (located on TABLE page) shows the results of hip flexor ROM (patient supine) of five random patients, one male, four female, age ranges 17 - 50, who were treated consecutively. Measurement of ROM was taken visually with the examiner passively flexing the supine patient's leg at the hip. The patient's baseline ROM was measured, followed by the clearing of two personality factors/traits and the victimization process (all 3 parts). This was followed by the post technique measurement. No other applied kinesiology, emotional, or other techniques were used before or during any measurements. The Emotional Equation was not utilized.

In the findings, there is an increase in ROM measurements bilaterally. Symmetry may not have been realized due to the non-use of the finishing step, The Emotional Equation. While these examples show the release of tension and neurological re-organization in linear terms, what is of greater importance is the non-linear benefit that can be communicated but not measured. Here is a case study of the results of a singular patient treatment session (ROM findings not recorded):

Danna was a self-employed 35 year old woman who had recently met a man. She was convinced that the new relationship would just end in yet another heartbreak. She was even afraid to see him again. She also expressed fear in operating her own business. Her last assistant had lied about being fired (she had quit) in order to receive benefits, and Danna was now involved in a legal wrangle. She was afraid the new business venture would just be another failure. Danna came into the office and said she had been sick for almost the entire month, had digestive difficulties, was extremely stressed, and said all of her symptoms had intensified after she met her new beaux.

After applying two rounds of personality trait factors, as well as the victimization process relating to a past bad relationship experience, and finishing it off with The Emotional Equation, the light came back into her eyes. She was smiling. She contacted me that night to let me know she felt great, that she felt "fully healed", and she had even called her new man and was currently enjoying his company over dinner and having a great time. She

also commented that she had no fears concerning her romantic interest or her business. Her digestive symptoms and headaches had abated.

Conclusion

Clearing personality factors and feelings of victimization (using the 3-step victimization process) brings a new facet to the emotional side of the Triad of Health that may help neutralize deep-rooted and long-standing detrimental emotional factors.

Further studies involving a greater number of measurements taken, testing for the facilitation of previously inhibited muscles, subjective measurement of stress level by the patient, and recording of pH levels may need to be performed. Long-term follow-up of future relationships, or how the patient reacts to similar situations (compared to past instances which produced feelings of victimization) may also prove to be intriguing.

References

1. Diamond, John, *Life Energy*. St. Paul, Minnesota: Paragon Health; 1985. p.7.

2. McCord, K.M., and Schmitt, W.H., *Quintessential Applications: A(K) Clinical Protocol*. St. Petersburg, Florida: HealthWorks!, 2005.

3. Merriam Webster Online. Merriam-Webster 2008.

4. Braden, Gregg, *Walking Between the Worlds, The Science of Compassion*. Bellevue, Washington: Radio Bookstore Press; 1997. P. 161

5. Frank, Victor, *Total Body Modification*, Fort Pierce, Utah: Total Body Modification; 2006

6. Keschner, Matthew G. Beyond Emotional Recall Quick Fix – Using Representational Systems. *Proceedings of the ICAK-U.S.A.*. 2008

7. Keschner, Matthew G. Beyond Emotional Recall Quick Fix – Using Representational Systems. *Proceedings of the ICAK-U.S.A.*. 2009

8. Braden, Gregg, *Walking Between the Worlds, The Science of Compassion*. Bellevue, Washington: Radio Bookstore Press; 1997. P. 127

9. Braden, Gregg, *Walking Between the Worlds, The Science of Compassion*. Bellevue, Washington: Radio Bookstore Press; 1997. P. 129.

10. Braden, Gregg, *Walking Between the Worlds, The Science of Compassion*. Bellevue, Washington: Radio Bookstore Press; 1997. P. 147.

11. Diamond, John, *Life Energy*. St. Paul, Minnesota: Paragon Health; 1985.

12. Walker, Scott, *Neuro-Emotional Technique*. Carlsbad, California: N.E.T., Inc; 1996.

13. Walker, Scott, *Neuro-Emotional Technique*. Carlsbad, California: N.E.T., Inc; 1996.

14. Frank, Victor, *Total Body Modification*, Fort Pierce, Utah: Total Body Modification; 2006.

15. Keschner, Matthew G. Beyond Emotional Recall Quick Fix – Using Representational Systems. *Proceedings of the ICAK-U.S.A.*. 2008

16. Keschner, Matthew G. Beyond Emotional Recall Quick Fix – Using Representational Systems. *Proceedings of the ICAK-U.S.A.* 2008

17. Diamond, John, *Life Energy*. St. Paul, Minnesota: Paragon Health; 1985.

18. Walker, Scott, *Neuro-Emotional Technique*. Carlsbad, California: N.E.T., Inc; 1996.

19. Walker, Scott, *Neuro-Emotional Technique*. Carlsbad, California: N.E.T., Inc; 1996.

20. Frank, Victor, *Total Body Modification*, Fort Pierce, Utah: Total Body Modification; 2006.

21. Diamond, John, *Life Energy*. St. Paul, Minnesota: Paragon Health; 1985. p. 99-191.

22. Frank, Victor, *Total Body Modification*, Fort Pierce, Utah: Total Body Modification; 2006

23. Diamond, John, *Life Energy*. St. Paul, Minnesota: Paragon Health; 1985. P. 128.

24. Diamond, John, *Life Energy*. St. Paul, Minnesota: Paragon Health; 1985. P. 137.

25. Walker, Scott, *Neuro-Emotional Technique*. Carlsbad, California: N.E.T., Inc; 1996

26. Walker, Scott, *Neuro-Emotional Technique*. Carlsbad, California: N.E.T., Inc; 1996

27. McCord, K.M., and Schmitt, W.H., *Quintessential Applications: A(K) Clinical Protocol*. St. Petersburg, Florida: HealthWorks!, 2005.

28. Schmitt, W.H., Emotions and The QA Protocol. *The Uplink* 2007; 39: 1.

29. Schmitt, W.H., Emotions and The QA Protocol. *The Uplink* 2007; 39: 1.

30. Keschner, Matthew G. Beyond Emotional Recall Quick Fix – Using Representational Systems. *Proceedings of the ICAK-U.S.A.*. 2008

Tables

Table 1
Post Technique ROM measured following completion of the combination of clearing two rounds of Personality Traits, and one whole Victimization Process.

Right = Right Leg; Left = Left Leg;

Note: Trial #1 is a male, while the following 4 trials are female, ages 17-50.

	Baseline ROM	Post Technique ROM
1. Right	75 degrees	90 degrees
Left	85 degrees	95 degrees
2. Right	90 degrees	90 degrees
Left	90 degrees	95 degrees
3. Right	90 degrees	95 degrees
Left	90 degrees	100 degrees
4. Right	90 degrees	95 degrees
Left	90 degrees	100 degrees
5. Right	80 degrees	90 degrees
Left	85 degrees	95 degrees

Case Report - The Use of Muscle Testing as an Inexpensive Procedure to Identify Peripheral Nerve Entrapments of the Lower Leg

David Leaf, D.C., DIBAK

Abstract

The use of muscle testing to identify peripheral nerve entrapments in a patient with chronic weakness and pain in the lower leg is presented. It shows the efficacy of using muscle testing as at least a screening test for the presence of peripheral nerve entrapments. Leaving expensive testing for determining resistant cases instead of as a screening procedure.

Discussion

A 55-year-old man of slight build presented with general ache and weakness on the right leg and severe pain under the metatarsals and moderate pain along the lateral aspect of the gastrocnemius. He also reported mild to moderate generalized pain in the right posterior pelvic area with mild radiation up the right lumbar spine. He reported a tendency to trip and an inability to walk for over 10 minutes without a severe ache occurring in the leg,

He had previously been examined for claudication with no signs of vascular problems; x-rays were negative for pelvic, lumbar and foot problems. These were done in California and his insurance carrier would not approve any other care. He then relocated to Massachusetts where he had previously lived and consulted me and a friend who was a physiatrist. Prior care had consisted of trials of antiinflammatories, muscle relaxants and mood elevating medications.

Posturally, he exhibited a right low pelvis, marked ankle pronation and walked with a short stride on the right landing with little dorsiflexion of the foot and no toe off.

Significant examination findings using muscle testing included:

Inhibition, weakness, of the right gluteus maximus, gluteus medius, tensor fascia femoris, hamstrings, peroneus longus, peroneus tertius, flexor hallucis brevis and extensor hallucis brevis.

Treatment

After correcting the pelvic imbalance, the use of a trochanter belt caused an immediate increase in strength of the hamstrings, gluteus maximus and medius to manual muscle testing. The weakness of he peroneus longus and other foot muscle continued. Approximation of the fibular head against the tibia resulted in an increase in strength of the peroneal muscles and a reduction in tenderness in the gastrocnemius.

Before initiating treatment for the lower possible nerve entrapments, he was to be examined fully by the physiatrist for the foot and leg pain. He underwent MRI examinations of his pelvis, lumbar spine and foot and had an EMG evaluation.

The only positive finding was a slowing of transmission of the peroneal nerve.

On return to the office, he told me of the findings and I informed him that that was what had been found.

Approximation of the fibular head and taping to support it resulted in an increase in strength of the peroneus longus and brevis The ache created by palpation over the dorsum of the foot and weakness of the extensor hallucis brevis was negated by posterior pressure applied to the talus. Anterior pressure against the calcaneus resulted in an increase in strength of the flexor hallucis brevis and a decrease in pain at the first metatarsophalangeal joint.

Placing a small support under the second and third metatarsal heads reduced tenderness over the metatarsal heads.

The treatment program set out for this patient included correction of the pelvic misalignment.

The uses of a trochanter belt to approximate the SI joints and relax the piriformis.

Exercises with the trochanter belt to strength the gluteal muscles.

Approximation of the fibular head and the wearing of a brace to support it.

Exercise for the tibialis posterior and the peroneus longus to aid in the support of the metatarsal arch.

Correction of the anterior displacement of the talus and then taping to approximate the distal fibula and tibia

The use of an orthotic to stabilize the calcaneus and support the arches.

At four weeks, the symptom pattern has decreased over 80 percent. By 6 weeks, the patient was able to walk a mile and a half with no pain on a level surface.

Manual muscle testing showed the presence of the peripheral nerve entrapments that only one, which required patient discomfort, that was time consuming and expense was able to identify.

Conclusion

Manual muscle testing used appropriately is a valuable tool to identify peripheral nerve entrapments that are commonly overlooked in the treatment of chronic problems.

Improper Inhibition Patterns in Low Back Injuries

David Leaf, D.C., DIBAK

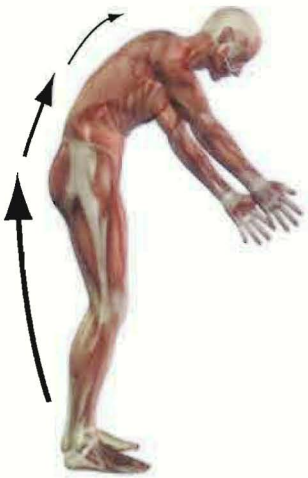
Abstract

Improper muscle firing patterns can be the cause of sudden unexpected low back problems. This pattern is usually overlooked. The sequence of firing depends on the degree of lumbar flexion of the subject.

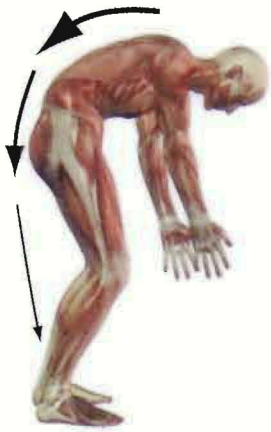
Discussion

Some patient's present with chronic repeated low back injuries, while others in supposedly exceedingly good health suddenly suffer an injury for no apparently reason. The cause of these injuries is many times missed. One of the great problems that we have in treating patients is thinking that we need to just look at individual muscles and not the interactive functioning of the muscles.

Myers, in his Anatomy Trains book, presents a concept of fascia that connects from the foot through to the head in specific patterns. The concept of muscular trains and not just fascia trains must be considered. This paper will discuss a very short section of these muscular trains.



Recent research in EMG activity of going from a position of lumbar flexion to straightening of the spine has shown that there is a muscle sequence firing that occurs that is different depending upon the degree of forward flexion of the subject.[1, 2] At approximately 45° of lumbar flexion, the muscle firing sequence changes.



From past 45° flexion, the muscles fire from superior to inferior. This means that the thoracic and lumbar extensor muscles, including the quadratus lumborum contract prior to the gluteus maximus. This is followed by contraction of the hamstrings and then the gastrocnemius.

When the subject is bent at 45° flexion or less, this muscle firing sequence is completely reversed. The gastrocnemius is followed by contraction of the hamstrings and then the gluteus maximus and then finally the lumbar extensor muscles.

Understanding of this firing sequence is a great input into why people keep injuring themselves.

The following is a short discussion of how this was presented at a number of seminars based on biomechanics.

After correcting all structural problems in the lumbar, thoracic and pelvis, all the muscles are tested to make sure that they're functioning in a normal manner.

The next step is to have the patient activate the posterior chain of muscles.

For those subjects that you suspect injured themselves past 45° of flexion, the patient is asked to raise their head and shoulders with their arms either by their side or in front of them causing contraction of the lumbar and thoracic extensor muscles. This is followed by quickly testing the strength of the gluteus maximus and the hamstrings to see if they are inhibited by the contraction of these extensor muscles.

If one suspects that the subject has been injured at a degree less than 45°, then pressure is applied against the lower leg just proximal to the ankle so that the patient contracts the gastrocnemius and hamstrings and initiates the muscle chain of action from inferior to superior. Following this, a test for the quadratus lumborum and or the gluteus maximus is performed in a prone position

In general, you will find the muscles that fire sequentially after the muscle, which is over contracting, will be inhibited.

Once the muscle that is causing the misfiring has been identified, it is treated in one of two methods. It is treated by the original technique developed by Goodheart of applying pressure to approximate the spindle cells in the belly of the muscle, or a PNF, proprioceptive neuromuscular facilitation; procedure is used repeatedly to accomplish the same result.

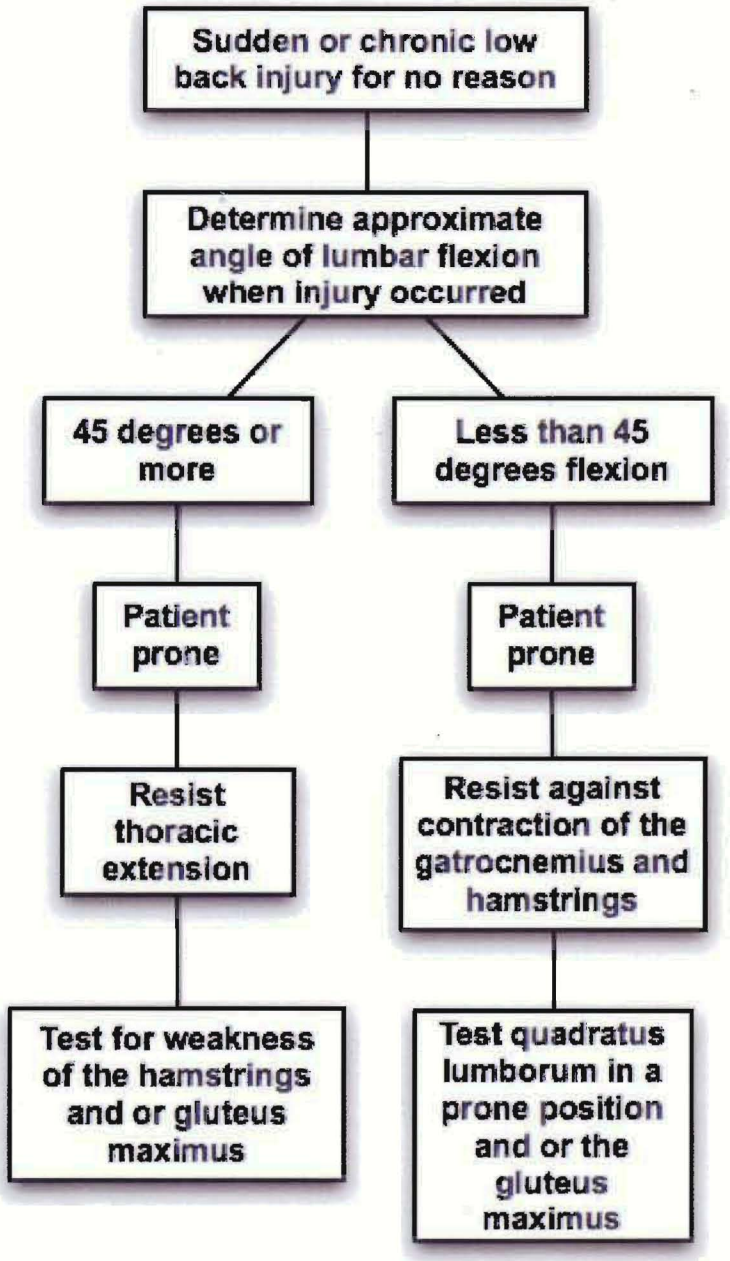
Treatment

The treatment is the easy part, knowing where to apply it is the problem. Muscles are tested for need of strength training and for reactive muscle patterns. If a muscle is found to be over contracting creating weakness of muscles that fire after that muscle, this is a compensation for an under functioning muscle that must be found for a complete correction.

Conclusion

In the classes where this was presented, over 75% of the attendees had a problem with these firing sequences.

This examination procedure should be incorporated in the examination of all patients with low back symptoms.



References

1. Roy AL, Keller TS, Colloca CJ. Posture-Dependent Trunk Extensor EMG Activity During Maximum Isometrics Exertions in Normal Male And Female Subjects. J Electromyogr Kinesiol 2003;13:469-76.

2. Hashemirad F, Talebian S, Hatef B, Kahlaee AH. The Relationship Between Flexibility and Emg Activity Pattern Of The Erector Spinae Muscles During Trunk Flexion-Extension. J Electromyogr Kinesiol 2009;19:746-53.

Muscle Activation Prior to Striking a Golf Ball

David Leaf, D.C., DIBAK

Abstract

The muscles that fire prior to striking a golf ball are described. A testing procedure to identify defects in the firing sequence is presented. This pattern of testing can be used for different activities in daily life and in all sports.

Discussion

In treating athletes, we are confronted with more than testing for weak muscles or short muscles as they affect their activities. Every sport involves a coordinated function of muscles in order to perform a specific task. This same concept is involved in turning over in bed, getting out of cars and performing daily activities. Specific sports activities require consistent firing of muscles to perform properly. This paper is going to discuss the muscles that are active in a specific section of the golf swing. All discussions of muscle action will be described for a right-handed golf swing.

Golf could be described as one of the easiest sports in the world and one of the hardest. The easy part is that it is basically one swing. It is one pattern of muscle activity from beginning to end that is repeated over and over. Tennis in comparison has a number of different strokes that have to be learned. On the other hand, golf is one of the most difficult sports because if there is any muscular imbalance or malfunction anywhere in the coordinated activity of the swing, the swing is imperfect.

The golf swing can be broken down into many sections. These subsections of the swing include the backswing, the beginning of the forward movement of the club, the down swing of the club to approximately the horizontal position of the club with the ground, the acceleration of the club to the point of impact with the ball, and then the early and late fall through.

In this paper, we are going to discuss the coordinated muscle action that occurs in the second half of the downswing. This is the section of the swing where the club head is accelerated to maximally hit the golf ball.

The downswing is where the stored energy that occurs during the backswing is released.

The lower body muscles are especially important here in accelerating the torso and getting the weight of the body behind the swing. The unwinding that occurs during the backswing starts in the lower body and works its way up, all the way to the club at ball strike.

To properly and maximally accelerate the club head, muscle activity has to occur in a coordinated fashion from the legs through the body, shoulders and into the arms.[1-6]

In the lower portion of the body, there is a coordinated activity between the two legs. The weight is shifting from the right to the left leg.

The right foot is stabilized with contraction of the flexor hallucis muscles and the tibialis posterior. On the right leg, the gluteus medius is contracted to accelerate and stabilize the pelvis. On the left leg, the lateral hamstring, biceps femoris, is contracted along with the gluteus maximus.

This is followed by rotation of the chest cage is by the right abdominal oblique and the transverse abdominals.

Consequently, the force is being transferred from the lower extremity to the upper extremity with the right abdominal oblique being the “mid transfer station” of the energy. Though out this stage of the swing, the erector spinae and the quadratus lumborum muscles are active bilaterally. 1

In the upper torso, the pectoralis muscles are highly active during this stage of the swing. These are contracting bilaterally. In the right arm, the serratus anterior contracts to stabilize the scapula against the rib cage. In the left arm, a combination of the levator scapula, the upper trapezius, the supraspinatus, the posterior deltoid and the long head of the triceps are involved.

Farther down the arm, the wrist extensions contract causing movement of the hand locking it into position for impact with the golf ball.

As you can see, there has to be a coordinated contraction of muscles from the feet up the legs through the pelvis with rotation of the torso with stabilization of the thorax. Locking of the scapula against the rib cage follows this and the force is transferred through the arms down through the arm to the wrist to properly hit the golf ball. Any weakness or over contraction of a muscle in his sequence will cause muscular deficits adversely affecting the swing.

The sections of the swing will be broken down for testing of the prime movers, and then testing to see if there is a weakness farther up the chain caused by either the weakness or the over contraction of a muscle in that section of the swing.

Prior to testing each section of the swing, muscles must be tested for their ability to contract in a normal manner.

In the right leg, a critical muscle for rotation of pelvis is the gluteus medius. The subject is asked to apply pressure on the foot as in initiating this portion of the swing and the gluteus medius is quickly tested for strength.

The next critical potential weak link in the swing is contraction stabilization and motion provided by the abdominal oblique. This is accomplished by the coordinated contraction of the abdominal oblique and the transverse abdominal and is stabilized by continual

contraction of the erector spine and the quadratus lumborum muscles. To test for proper muscle activation, the subject shifts their weight from the right to the left and the right abdominal oblique is quickly tested. If this produces inhibition of the abdominal oblique, then there is a defect in the lower extremity or quadratus lumborum causing the improper activation of the muscle.

In the left upper extremity, there are two critical potential weak links. These are the long head of the triceps and the wrist extensors. The subject is asked to stand and then apply pressure against resistance with their arms at the point of impact with the golf ball. The wrist extensors and the long head of the triceps, and possibly the supraspinatus, are quickly tested for improper inhibition.

Using these techniques it is possible to isolate sections of the swing where improper muscle functioning exists. The cause of these improper sequencing can be due to weakness or over contraction of a muscle.

The problem is not only to find the overworking muscle but also to identify why it is performing in that manner. Generally, a muscle that is over contracting is the result of inhibition a synergistic muscle. This over contraction results in improper muscle activation of those muscles that fire sequentially after.

References

1. Adlington GS. Proper Swing Technique and Biomechanics of Golf. Clin Sports Med 1996;15:9-26.

2. Bechler JR, Jobe FW, Pink M, Perry J, Ruwe PA. Electromyographic Analysis of the Hip and Knee During the Golf Swing. Clin J Sport Med 1995;5:162-6.

3. Bradley JP, Tibone JE. Electromyographic Analysis of Muscle Action About the Shoulder. Clin Sports Med 1991;10:789-805.

4. Egret CI, Nicolle B, Dujardin FH, Weber J, Chollet D. Kinematic Analysis of the Golf Swing in Men and Women Experienced Golfers. Int J Sports Med 2006; 27:463-7

5. Pink M, Jobe FW, Perry J. Electromyographic Analysis Of the Shoulder During the Golf Swing. Am J Sports Med 1990;18:137-40.

6. Pink M, Perry J, Jobe FW. Electromyographic Analysis of the Trunk in Golfers. Am J Sports Med 1993;21:385-8.

A New Twist on Origin and Insertion

Brian Llewellyn, D.C., BS

Abstract

In this paper I propose that every muscle has a second set of 'origin and insertion' points that are located directly through the bone to surface on the other side. In the case of a joint, the connective tissue crosses over the joint and then runs through the bone to the opposite side. These points do not replace the traditional O&I points but add another option to treatment in injuries.

Discussion

If one considers the explanation of O&I as the pulling of Sharpie's fibers from the bone, then how far into the bone do those fibers actually penetrate?

If we imagine the patella tendon/ligament to be like a piece of adhesive tape and stick it down on the tibial tubercle, we would not expect there to be a great adhesiveness the first time the quadriceps contract. As with fiberglass laminating, the greatest strength is obtained if the glass fibers are oriented in a way to receive the greatest stress and then bonded within the resin. I maintain the same has happened with these ligamentous/tendon connective tissue fibers.

In the case of the quadriceps insertion, the second O&I contact point becomes the popliteal fossa on the tibia. Additional examples would be the deltoid tendon's second insertion O&I point on the medial aspect of the humerus, the gluteus maximus insertion translates through to be on the medial femur as the reverse is true for the psoas insertion on the upper lateral femur. The psoas origin is interesting as I find it penetrates through the vertebra to the contralateral spinous tips. These contact points are usually quite sore.

Conclusion

It is easy to extrapolate the second O&I points after the above examples. There are some points that are, of course, inaccessible. We must also consider within the concepts of kinematic chains that a multitude of 'adhesion points' may be strained and more than the traditional two at a time.

A common pattern I find is the psoas origin (contralateral spinous tips), the rectus abdominis on a rib (usually 4 or 5), and the psoas and/or gluteus maximus insertion on the femur. These patterns are only marginally corrected with digital pressure. It is far more therapeutic if my prior "Impact Challenge" and "Injury Recall" (see collected papers 2009) are utilized in the correction process.

Seventeen New and Old Functional Tests Related to Soft Tissue Integrity

Scott Monk, D.C.

Abstract

It has long been recommended in applied kinesiology to do whatever you can in the office to recreate the "real world", how patients actually live. Stressing their tissues through standing tests, gait tests, or even having them run in place will often manifest problems that would otherwise go undiscovered. This is one form of functional testing.
In this paper a review of seventeen functional tests all relating to soft tissue integrity is presented as well as a discussion of the physiologic rationale for their validity and purpose.

Key Indexing Words
Soft Tissue Tests (STT), Manual Muscle Test (MMT)

Introduction

Sulfur is one of the most important and overlooked nutrients in the realm of functional medicine. Sulfur is the major detoxifier of the liver. Ten of the fourteen pathways of phase II liver detoxification are sulfur dependent. The form of sulfur used in these pathways can be free-form or as part of the structure of an amino acid such as taurine and cysteine. Without proper sulfur levels detoxification does not occur as designed. Also, sulfur could be thought of as the glue that holds us together. Without sulfur all soft tissues: ligaments, cartilage, collagen, elastin, and fibrilin, would literally begin to fall apart. There are many other nutrients that need consideration concerning the integrity of soft tissues, but sulfur should be first and foremost. Below is a review of the formation of chondroitin sulfate, a glycosaminoglycan (GAG) and critical component of many soft tissues.

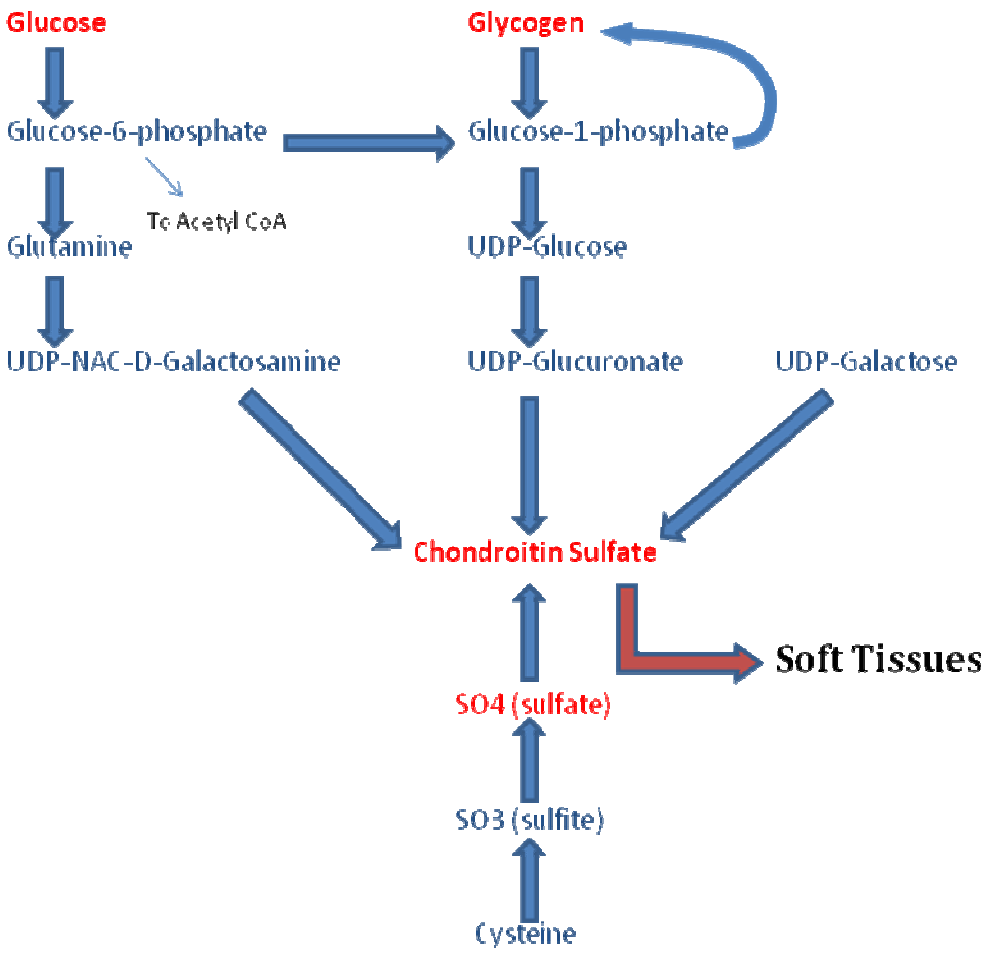


Figure 1: Chondroitin Sulfate Metabolism

Glycosaminoglycans are various polysaccharides which are linked to a molecule of serine. Each polysaccharide or sugar molecule is derived from the glycolytic pathway (from glucose) and connected together to make a GAG. It can be seen from Figure 1, that SO_4 or sulfate is one of three critical precursors to the metabolism of chondroitin (4 or 6) sulfate. The other two are glucose and glycogen (stored glucose). Just from knowing these three key components it can be theorized that if blood sugar management or liver detoxification were compromised, soft tissue formation and repair would suffer. This indeed turns out to be the case. In fact, one issue can often generate the other.

Poor detoxification often results from a poor diet and is then compounded by a disrupted blood sugar system. The pattern goes something like this: A prepubescent female eats the Standard American Diet (SAD) which is full of trans-fats, refined sugars, too many simple carbohydrates and not enough quality protein. This diet and other lifestyle factors associated with living in the modern age, place a strain on regulation and utilization of both glucose and glycogen. Because the primary fuel for the brain is glucose, the body will do what it must in order to first feed the brain. This means that the sympathetic, fight-or-

flight system gets turned on in order to generate glucose from muscle catabolism and never gets turned off. As a result, other glucose dependent systems are sacrificed out of necessity as glucose is scavenged to feed the brain; systems such as those that assemble chondroitin sulfate. The entire endocrine and glucose system is further stressed by a woman's menstrual cycle and the demand by the liver to process excess progesterone, estrogen, luteinizing and follicle stimulating hormones from the time of ovulation to menstruation. The sulfur-depleted liver is soon overburdened by the hormonal barrage, which results in the recognized symptoms of PMS. This process continues for decades. Therefore, it is no surprise that when hormones are out of balance, joint problems usually follow. In fact, it has been the author's experience that most arthritic issues are related to liver abnormalities.

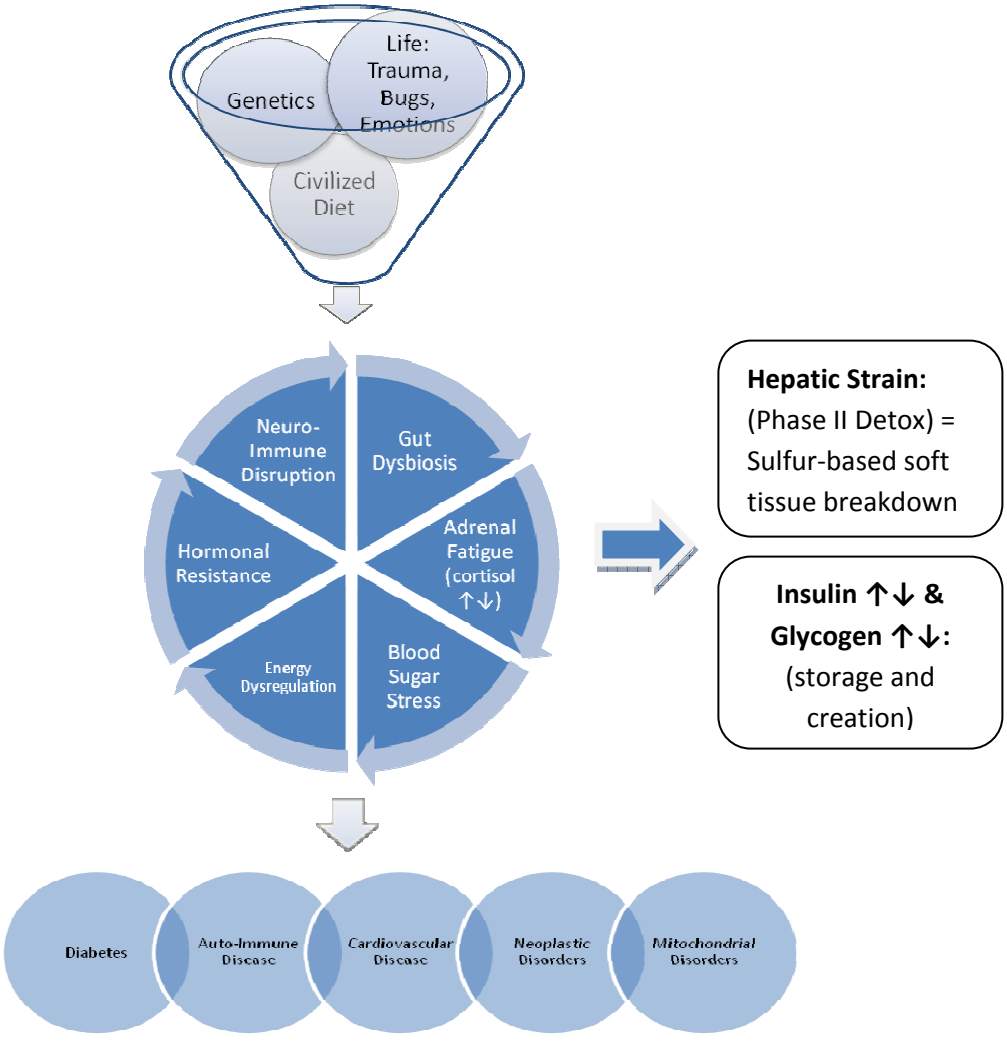


Figure 2: The Path to Functional Illness and Disease

Soft Tissue Tests

Listed below are the functional soft tissue tests. Those with an asterisk are believed to have been discovered by the author.

1. Joint squeeze[1] – *An easy squeeze directly into any joint.* This is one of the most important tests for anyone who complains of joint problems. The answer is often a specific form of sulfur. When someone on their own chooses a supplement for joint pain they are often directed to glucosamine, chondroitin or MSM. These are all forms of sulfur. The answer though is not usually a combination of one of these, but a good dose of just one form. It is no surprise that those with joint problems are also the ones who have hormonal or detoxification (liver) issues. Since the liver requires sulfur for nearly all of its detoxification pathways, the clear connection between these widespread issues is sulfur.

2. Ligament stretch[2] – *Gently stretching the ligaments of the wrist or ankle.* This test often verifies the presence of adrenal gland fatigue and excess Aldosterone, the hormone responsible for sodium retention. The adrenal gland could respond to a variety of substances to aid in healing. Vitamin C, glandular tissue, zinc and again sulfur are all possibilities. High levels of dense protein are often required as well, since adrenal-repairing amino acids come from protein. Additionally, protein's counterpart: the carbohydrates, place a great deal of stress on the blood sugar system and consequently the adrenals as well.

3. Skin squeeze[3] – *Lightly squeezing the skin of the hand (or anywhere else).* A very good test to check for collagen integrity. Collagen is one of the most abundant soft tissue components in the body making up large percentages of skin and bone. Amino acids and sulfur are important for collagen repair as is vitamin A and zinc. [4]

4. Skin stretch and release[5] – *A mild tug of the skin with an immediate release.* This is a test for the integrity of elastin – the substance that helps keep our skin tight and tone. As we age elastin becomes less viable and skin therefore begins to sag. With the loss of elastin on the outer skin, integrity within blood vessels also suffers leading to varicose and spider veins. Elastin is made up of 1/3 alanine, 1/3 glycine, valine and other nutrients. Therefore amino acid levels should always be considered. Elastin requires high levels of antioxidants like A, C and E, also sulfur and proper blood sugar balance (glucose is one of the most important raw materials for all healthy soft tissues). Elastin defects are also related to the hormone progesterone[6].

5. Wrinkle stretch[7] – *Stretching across a wrinkle.* Antioxidants again are an important consideration if this test is positive. Alpha lipoic acid is a common sulfur-containing antioxidant used to repair wrinkles as well as thiamin pyrophosphate.

6. *Muscle squeeze – *Moderate pressure squeezing any muscle.* A very good test to help those who regularly get muscle cramps at rest or with exercise. Mineral

imbalances are the number one reason why people get leg cramps. The most common need is calcium in its correct form, followed by magnesium and potassium.

7. *Muscle stretch - *One of my favorite tests since it is the one that helped me overcome 6 months worth of generalized lower back pain.* If stretching of an uninjured muscle causes consistent tension and/or pain for the patient, then the muscle-stretch test will likely be positive. The test is performed with sustained stretching of the fibers in the belly of any muscle and simultaneously checking a strong indicator muscle. Fat soluble vitamins are usually the answer here. Vitamins E, A and D, in that order are often helpful. Vitamin E must usually be in a mixed tocopherol form, not just alpha-tocopherol.

8. Fat squeeze[8] – *Can you release fat from storage?* That is the question that this test answers. If a strong muscle goes weak with a gentle squeeze to the abdominal tissue then the answer is no. A critical piece of data to have when attempting to help someone lose weight or balance hormones. A specific form of niacin (B3) is critical with this test, although other possibilities exist.

9. *Liver/Gall Bladder touch – *While the patient rests their left hand over the liver neurolymphatic reflex area (under the right breast), the gall bladder point (GB1) at the corner of the eye is touched at the same time.* If this produces muscle inhibition, then the patient is having trouble utilizing fat for fuel after it has been released from storage. The gall bladder's job is to provide bile salts into the digestive system whenever fat is present. Bile, like a grease-fighting detergent, is a fat emulsifier. Without bile, fats do not properly get broken down into smaller units. The gall bladder often needs good fats to stay healthy. The easiest way to upset the gall bladder is to eat too many unhealthy fats. Partially hydrogenated oils are the worst followed closely by canola oil. Yes, canola, the wonder oil that is so heavily touted in the health food stores. Testing GB1 with canola oil in the mouth will produce a surprising number of positive tests. A great all purpose oil is olive. And at higher elevations, black currant seed oil is commonly helpful.

10. Joint Grind[9] – *An easy pressure compressing and slightly rotating a joint.* Within joints is a lubricating fluid called hyaluronic acid. If this test is positive, there is most likely a deficiency of this important substance.

11. *Tissue Tap – *Gently tapping the skin eight or ten times in succession.* This is a good test to perform on patients who bruise easily, have hemorrhoids, nose bleeds, varicose veins or other vascular issues and reveals a deficiency in a necessary antioxidant. Vitamin C with flavinoids, Alaria Supreme[10], and lutein are commonly found.

12. *Vessel stretch – *Gently stretching a superficial vein, usually on the back of the hand.* This is another good test to perform on patients with vascular issues. Antioxidants are the answer here as well. However, when this test is positive,

checking for elevated homocysteine levels is warranted. Homocysteine is extremely inflammatory[11] and indicates a need for methylcobalamin (B12) or folic acid – two common methyl donors.

13. *Nail lift test – *Sustained gently lifting of the nail bed*. Weak or fragile nails are a common complaint among patients. When this test is positive it will often negate with TL to SI 19. Supplementation designed to aid absorption in the digestive tract is often helpful as well as supplying the necessary depleted minerals. Supplements containing slippery elm or marshmallow root have worked well as has Morinda Supreme[12], a good all around anti-fungal developed by applied kinesiologist, Dr. Michael Lebowitz.

14. *Nail pressure – *Repeated pressure of approximately 10 quick squeezes of the nail itself.* In patients with white spots on their fingernails this test should be performed. If positive, it generally indicates a need for a specific form of zinc (citrate if the patient is alkaline, carbonate if they are acidic and picolinate if they are neutral). However, these spots may also be present without a zinc deficiency, but with an imbalance of either iron or copper. These two additional nutrients are on a three-way teeter-tooter with zinc and they should always be evaluated as a group.

15. *Hair Pull – *Sustained gentle pull of the hair on the scalp*. There are many hormonal reasons for hair loss. The author has found hidden thyroid imbalances with most. Folic acid is one of the nutrients that will be required since it is critical for the growth and repair of all tissues. B12 could also be critical here for a variety of reasons.[13]

16. Gum Rub[14] – *Gentle rubbing of the gums*. If this test is positive, folic acid is usually required. Combine this test with other functional tests for folic acid.

17. *Bone stress – *Gentle lateral or cross-pressure and release of any long bone*. If positive, any of the nutrients you would expect with bone formation could be needed: vitamin D, boron, phosphorous, and/or calcium.

Conclusion

Well before the soft tissues are compromised, the hormonal and blood sugar systems were first under stress. When pain becomes a complaint, generally some form of metabolic disturbance has already been present, compromising the integrity of the supporting soft tissues. The soft tissue testing performed with MMT is the only tool that provides an immediate assessment of a patient's structural state. The results of these findings will both lead the doctor into the most appropriate treatment for the patient as well as provide him with an overall assessment as to the severity and chronicity of the underlying metabolic problems.

References

1. Astil-Smith, Chris; http://www.metabolics-seminars.com/manuals/Mod8.pdf

2. The Uplink, Schmitt, Walter; Issue 12, Fall 1998

3. Astil-Smith, Chris; http://www.metabolics-seminars.com/manuals/Mod8.pdf

4. Werner N, Nickenig G., Sex hormones save our skin: the vascular networking of estrogen. Circ Res. 2009 Jan 30;104(2):135-7.

5. Astil-Smith, Chris; http://www.metabolics-seminars.com/manuals/Mod8.pdf

6. Kuś E, Karowicz-Bilińska A. The influence of steroid sex hormones on collagen composition in post-operative wounds after long-term treatment with anticoagulants. Ginekol Pol. 2009 Nov;80(11):814-8. PubMed PMID: 20088393.

7. Astil-Smith, Chris; http://www.metabolics-seminars.com/manuals/Mod8.pdf

8. Ibid

9. Ibid

10. http://www.supremenutritionproducts.com/AlariaSupreme/index.html

11. Williams KT, Schalinske KL., Homocysteine metabolism and its relation to health and disease.: Biofactors. 2010 Jan 20. Department of Food Science and Human Nutrition, Iowa State University, Ames, IA, USA.

12. http://www.supremenutritionproducts.com/MorindaSupreme/index.html

13. Molloy AM, Kirke PN, Troendle JF, Burke H, Sutton M, Brody LC, Scott JM, Mills JL. Maternal vitamin B12 status and risk of neural tube defects in a population with high neural tube defect prevalence and no folic Acid fortification. Pediatrics. 2009 Mar;123(3):917-23. Schools of Medicine, Trinity College Dublin, School of Medicine, Dublin 2, Ireland.

14. Astil-Smith, Chris; http://www.metabolics-seminars.com/manuals/Mod8.pdf

Proprioceptors Dysfunctions P – DTR: Classification, Testing, P – DTR & Tx. Part IV

Jose Palomar, M.D., Orthopedic Surgeon, DIBAK

Abstract

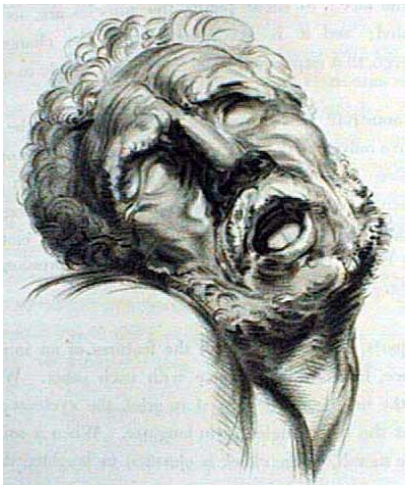
A reclassification of the classical muscle proprioceptor dysfunctions and proprioceptor deep tendon reflex treatment, including additional ways to find these problems and treat them are described. In the Dynamic muscle propioceptors research published in the 2008 proceedings only part of the material was presented. This paper will complete the research.

Key Indexing Words
P DTR (Proprioceptive Deep Tendon Reflex), NeoSpinothalamic Pain Tract/ PaleoSpinothalamic Pain Tract, Nociceptors

Introduction

Classical dysfunctions - Strain/Counterstrain, Myofascial, Reactor/Reactive, Gelosis / Vibration Propioceptor, Origin/Insertion and Meridians have been found to have more varied presentations than previously considered and require re-classification.

All these dysfunctions result mostly from trauma and all are a hyper or hypo response of a normal neurological reflex. The reflexes can show different levels of dysfunction.-Each level has it´s particular properties and tests differently.



Pain Nociceptors Dysfunctions

AK description - There is no AK or non AK description of this condition before.

Physiology

Types of Pain and Their Qualities-Fast Pain and Slow Pain

Pain has been classified into two major types: *fast pain* and *slow pain.* Fast pain is felt within about 0.1 second after a pain stimulus is applied, whereas slow pain begins only after 1 second or more and then increases slowly over many seconds and sometimes even minutes. During the course of this chapter, we shall see that the conduction pathways for these two types of pain are different and that each of them has specific qualities.

Fast pain is also described by many alternative names, such as *sharp pain, pricking pain, acute pain,* and *electric pain.* This type of pain is felt when a needle is stuck into the skin, when the skin is cut with a knife, or when the skin is acutely burned. It is also felt when the skin is subjected to electric shock. Fast-sharp pain is not felt in deeper tissues of the body.

Slow pain also goes by many names, such as *slow burning pain, aching pain, throbbing pain, nauseous pain,* and *chronic pain.* This type of pain is usually associated with *tissue destruction.* It can lead to prolonged, unbearable suffering. It can occur both in the skin and in almost any deep tissue or organ.

Pain Receptors and Their Stimulation

Pain Receptors Are Free Nerve Endings. The pain receptors in the skin and other tissues are all free nerve endings. They are widespread in the superficial layers of the *skin* as well as in certain internal tissues, such as the *periosteum,* the *arterial walls,* the *joint surfaces,* and the *falx* and *tentorium* in the cranial vault. Most other deep tissues are only sparsely supplied with pain endings; nevertheless, any widespread tissue damage can summate to cause the slow-chronic-aching type of pain in most of these areas.

Three Types of Stimuli Excite Pain Receptors-Mechanical, Thermal, and Chemical

Pain can be elicited by multiple types of stimuli. They are classified as *mechanical, thermal,* and *chemical pain stimuli.* In general, fast pain is elicited by the mechanical and thermal types of stimuli, whereas slow pain can be elicited by all three types.
Some of the chemicals that excite the chemical type of pain are *bradykinin, serotonin, histamine, potassium ions, acids, acetylcholine,* and *proteolytic enzymes.* In addition, *prostaglandins* and *substance P* enhance the sensitivity of pain endings but do not directly excite them. The chemical substances are especially important in stimulating the slow, suffering type of pain that occurs after tissue injury.

No adapting Nature of Pain Receptors

In contrast to most other sensory receptors of the body, pain receptors adapt very little and sometimes not at all. In fact, under some conditions, excitation of pain fibers becomes progressively greater, especially so for slow-aching-nauseous pain, as the pain stimulus continues. This increase in sensitivity of the pain receptors is called *hyperalgesia.* One can readily understand the importance of this failure of pain receptors

to adapt, because it allows the pain to keep the person apprised of a tissue-damaging stimulus as long as it persists.

Rate of Tissue Damage as a Stimulus for Pain
The average person begins to perceive pain when the skin is heated above 45°C, as shown in Figure. This is also the temperature at which the tissues begin to be damaged by heat; indeed, the tissues are eventually destroyed if the temperature remains above this level indefinitely. Therefore, it is immediately apparent that pain resulting from heat is closely correlated with the *rate at which damage to the tissues is occurring* and not with the total damage that has already occurred.

The intensity of pain is also closely correlated with the *rate of tissue damage* from causes other than heat, such as bacterial infection, tissue ischemia, tissue contusion, and so forth.

Special Importance of Chemical Pain Stimuli during Tissue Damage
Extracts from damaged tissue cause intense pain when injected beneath the normal skin. Most of the chemicals listed earlier that excite the chemical pain receptors can be found in these extracts. One chemical that seems to be more painful than others is *bradykinin.* Many researchers have suggested that bradykinin might be the agent most responsible for causing pain following tissue damage. Also, the intensity of the pain felt correlates with the local increase in potassium ion concentration or the increase in proteolytic enzymes that directly attack the nerve endings and excite pain by making the nerve membranes more permeable to ions.

Tissue Ischemia as a Cause of Pain
When blood flow to a tissue is blocked, the tissue often becomes very painful within a few minutes. The greater the rate of metabolism of the tissue, the more rapidly the pain appears. For instance, if a blood pressure cuff is placed around the upper arm and inflated until the arterial blood flow ceases, exercise of the forearm muscles sometimes can cause muscle pain within 15 to 20 seconds. In the absence of muscle exercise, the pain may not appear for 3 to 4 minutes even though the muscle blood flow remains zero.

One of the suggested causes of pain during ischemia is accumulation of large amounts of lactic acid in the tissues, formed as a consequence of anaerobic metabolism (metabolism without oxygen). It is also probable that other chemical agents, such as bradykinin and proteolytic enzymes, are formed in the tissues because of cell damage and that these, in addition to lactic acid, stimulate the pain nerve endings.

Muscle Spasm as a Cause of Pain
Muscle spasm is also a common cause of pain, and it is the basis of many clinical pain syndromes. This pain probably results partially from the direct effect of muscle spasm in stimulating mechano sensitive pain receptors, but it might also result from the indirect effect of muscle spasm to compress the blood vessels and cause ischemia. Also, the spasm increases the rate of metabolism in the muscle tissue, thus making the relative ischemia even greater, creating ideal conditions for the release of chemical pain-inducing substances.

Dual Pathways for Transmission of Pain Signals into the Central Nervous System
Even though all pain receptors are free nerve endings, these endings use two separate pathways for transmitting pain signals into the central nervous system. The two pathways mainly correspond to the two types of pain-a fast-sharp pain pathway and a slow-chronic pain pathway.

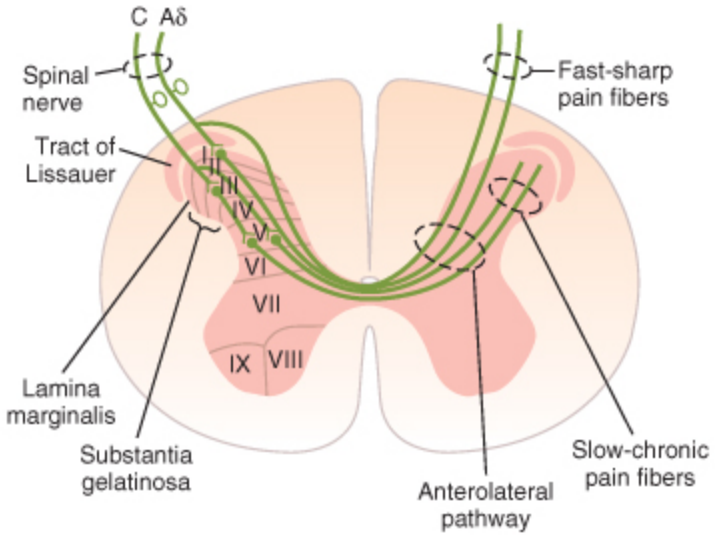
Peripheral Pain Fibers-"Fast" and "Slow" Fibers
The fast-sharp pain signals are elicited by either mechanical or thermal pain stimuli; they are transmitted in the peripheral nerves to the spinal cord by small type Aδ fibers at velocities between 6 and 30 m/sec. Conversely, the slow-chronic type of pain is elicited mostly by chemical types of pain stimuli but sometimes by persisting mechanical or thermal stimuli. This slow-chronic pain is transmitted to the spinal cord by type C fibers at velocities between 0.5 and 2 m/sec.

Because of this double system of pain innervation, a sudden painful stimulus often gives a "double" pain sensation: a fast-sharp pain that is transmitted to the brain by the Aδ fiber pathway, followed a second or so later by a slow pain that is transmitted by the C fiber pathway. The sharp pain apprises the person rapidly of a damaging influence and, therefore, plays an important role in making the person react immediately to remove himself or herself from the stimulus. The slow pain tends to become greater over time. This sensation eventually produces the intolerable suffering of long-continued pain and makes the person keep trying to relieve the cause of the pain.

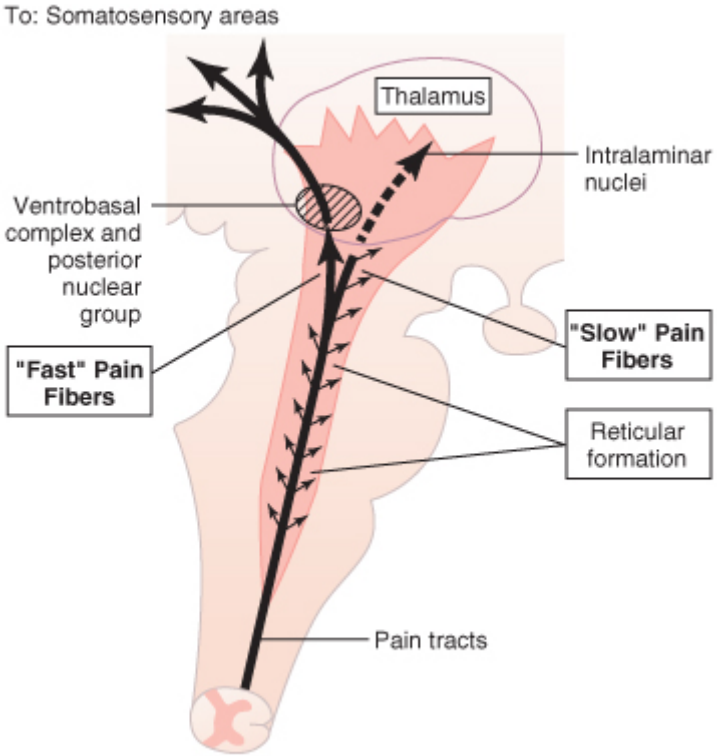
On entering the spinal cord from the dorsal spinal roots, the pain fibers terminate on relay neurons in the dorsal horns. Here again, there are two systems for processing the pain signals on their way to the brain, as shown in Figures.

Dual Pain Pathways in the Cord and Brain Stem-The Neospinothalamic Tract and the Paleospinothalamic Tract
On entering the spinal cord, the pain signals take two pathways to the brain, through (1) the neospinothalamic tract and (2) the paleospinothalamic tract.

Neospinothalamic Tract for Fast Pain
The fast type Aδ pain fibers (30 m / sec) transmit mainly mechanical and acute thermal pain. They terminate mainly in lamina I (lamina marginalis) of the dorsal horns, and there excite second-order neurons of the neospinothalamic tract. These give rise to long fibers that cross (all fibers) immediately to the opposite side of the cord through the anterior commissure and then turn upward, passing to the brain in the anterolateral columns.



Transmission of both "fast-sharp" and "slow-chronic" pain signals into and through the spinal cord on their way to the brain.



Transmission of pain signals into the brain stem, thalamus, and cerebral cortex by way of the fast pricking pain pathway and the slow burning pain pathway.

Termination of the Neospinothalamic Tract in the Brain Stem and Thalamus
A few fibers of the neospinothalamic tract terminate in the reticular areas of the brain stem, but most pass all the way to the thalamus without interruption, terminating in the ventrobasal complex along with the dorsal column-medial lemniscal tract for tactile sensations. A few fibers also terminate in the posterior nuclear group of the thalamus.

From these thalamic areas, the signals are transmitted to other basal areas of the brain as well as to the somatosensory cortex.

Capability of the Nervous System to Localize Fast Pain in the Body
The fast-sharp type of pain can be localized much more exactly in the different parts of the body than can slow-chronic pain. However, when only pain receptors are stimulated, without the simultaneous stimulation of tactile receptors, even fast pain may be poorly localized, often only within 10 centimeters or so of the stimulated area. Yet when tactile receptors that excite the dorsal column-medial lemniscal system are simultaneously stimulated, the localization can be nearly exact.

Glutamate, the Probable Neurotransmitter of the Type Aδ Fast Pain Fibers
It is believed that glutamate is the neurotransmitter substance secreted in the spinal cord at the type Aδ pain nerve fiber endings. This is one of the most widely used excitatory transmitters in the central nervous system, usually having a duration of action lasting for only a few milliseconds.

Paleospinothalamic Pathway for Transmitting Slow-Chronic Pain
The paleospinothalamic pathway is a much older system and transmits pain mainly from the peripheral slow-chronic type C pain fibers (2 m / sec); although it does transmit some signals from type Aδ fibers as well. In this pathway, the peripheral fibers terminate in the spinal cord almost entirely in laminae II and III of the dorsal horns, which together are called the substantia gelatinosa, as shown by the lateral most dorsal root type C fiber. Most of the signals then pass through one or more additional short fiber neurons within the dorsal horns themselves before entering mainly lamina V, also in the dorsal horn. Here the last neurons in the series give rise to long axons that mostly join the fibers from the fast pain pathway, passing first through the anterior commissure to the opposite side of the cord, then upward to the brain in the anterolateral pathway. Most of the fibers cross to the opposite side of the cord, but there are some ipsilateral fibers too.

Tissues from endoderm have only PaleoSpinothalamic tracts (one of the three primitive germ layers of the embryo), ectoderm and endoderm have Neo and PaleoSpinothalamic tracts.

Substance P, the Probable Slow-Chronic Neurotransmitter of Type C Nerve Endings
Research experiments suggest that type C pain fiber terminals entering the spinal cord secrete both glutamate transmitter and substance P transmitter. The glutamate transmitter acts instantaneously and lasts for only a few milliseconds. Substance P is released much more slowly, building up in concentration over a period of seconds or even minutes. In fact, it has been suggested that the "double" pain sensation one feels after a pinprick might result partly from the fact that the glutamate transmitter gives a faster pain sensation, whereas the substance P transmitter gives a more lagging sensation. Regardless of the yet unknown details, it seems clear that glutamate is the neurotransmitter most involved in transmitting fast pain into the central nervous system, and substance P is concerned with slow-chronic pain.

Projection of the Paleospinothalamic Pathway (Slow-Chronic Pain Signals) into the Brain Stem and Thalamus

The slow-chronic paleospinothalamic pathway terminates widely in the brain stem, in the large shaded area shown in Figure. Only one tenth to one fourth of the fibers pass all the way to the thalamus. Instead, most terminate in one of three areas: (1) the reticular nuclei of the medulla, pons, and mesencephalon; (2) the tectal area of the mesencephalon deep to the superior and inferior colliculi; or (3) the periaqueductal gray region surrounding the aqueduct of Sylvius. These lower regions of the brain appear to be important for feeling the suffering types of pain, because animals whose brains have been sectioned above the mesencephalon to block pain signals from reaching the cerebrum still evince undeniable evidence of suffering when any part of the body is traumatized. From the brain stem pain areas, multiple short-fiber neurons relay the pain signals upward into the intralaminar and ventrolateral nuclei of the thalamus and into certain portions of the hypothalamus and other basal regions of the brain.

Very Poor Capability of the Nervous System to Localize Precisely the Source of Pain Transmitted in the Slow-Chronic Pathway

Localization of pain transmitted by way of the paleospinothalamic pathway is poor. For instance, slow-chronic pain can usually be localized only to a major part of the body, such as to one arm or leg but not to a specific point on the arm or leg. This is in keeping with the multisynaptic, diffuse connectivity of this pathway. It explains why patients often have serious difficulty in localizing the source of some chronic types of pain.

Function of the Reticular Formation, Thalamus, and Cerebral Cortex in the Appreciation of Pain

Complete removal of the somatic sensory areas of the cerebral cortex does not destroy an animal's ability to perceive pain. Therefore, it is likely that pain impulses entering the brain stem reticular formation, the thalamus, and other lower brain centers cause conscious perception of pain. This does not mean that the cerebral cortex has nothing to do with normal pain appreciation; electrical stimulation of cortical somatosensory areas does cause a human being to perceive mild pain from about 3 per cent of the points stimulated. However, it is believed that the cortex plays an especially important role in interpreting pain quality, even though pain perception might be principally the function of lower centers.

Special Capability of Pain Signals to Arouse Overall Brain Excitability

Electrical stimulation in the reticular areas of the brain stem and in the intralaminar nuclei of the thalamus, the areas where the slow-suffering type of pain terminates, has a strong arousal effect on nervous activity throughout the entire brain. In fact, these two areas constitute part of the brain's principal "arousal system" This explains why it is almost impossible for a person to sleep when he or she is in severe pain.

Nociception Response

When nociceptors are stimulated they transmit signals through sensory neurons in the spinal cord. These neurons release the excitatory neurotransmitter glutamate at their synapses.

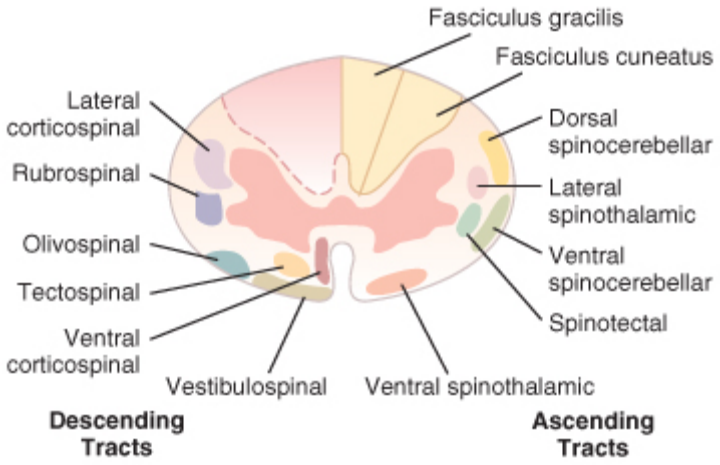
If the signals are sent to the reticular formation and thalamus, the sensation of pain enters consciousness in a dull poorly localized manner. From the thalamus, the signal can travel to the somatosensory cortex in the cerebrum, when the pain is experienced as localized and having more specific qualities.

Nociception can also cause generalized autonomic responses before or without reaching consciousness to cause pallor, diaphoresis, tachycardia, hypertension, lightheadedness, nausea and fainting.

Pain Suppression ("Analgesia") System in the Brain and Spinal Cord
The degree to which a person reacts to pain varies tremendously. This results partly from a capability of the brain itself to suppress input of pain signals to the nervous system by activating a pain control system, called an *analgesia system*.

The analgesia system is shown in Figure. It consists of three major components: (1) The *periaqueductal gray* and *periventricular areas* of the mesencephalon and upper pons surround the aqueduct of Sylvius and portions of the third and fourth ventricles. Neurons from these areas send signals to (2) the *raphe magnus nucleus,* a thin midline nucleus located in the lower pons and upper medulla, and the *nucleus reticularis paragigantocellularis,* located laterally in the medulla. From these nuclei, second-order signals are transmitted down the dorsolateral columns in the spinal cord to (3) a *pain inhibitory complex located in the dorsal horns of the spinal cord.* At this point, the analgesia signals can block the pain before it is relayed to the brain.

Inhibition of Pain Transmission by Simultaneous Tactile Sensory Signals
Another important event in the saga of pain control was the discovery that stimulation of large type Aβ sensory fibers from peripheral tactile receptors can depress transmission of pain signals from the same body area. This presumably results from local lateral inhibition in the spinal cord. It explains why such simple maneuvers as rubbing the skin near painful areas is often effective in relieving pain. And it probably also explains why liniments are often useful for pain relief.



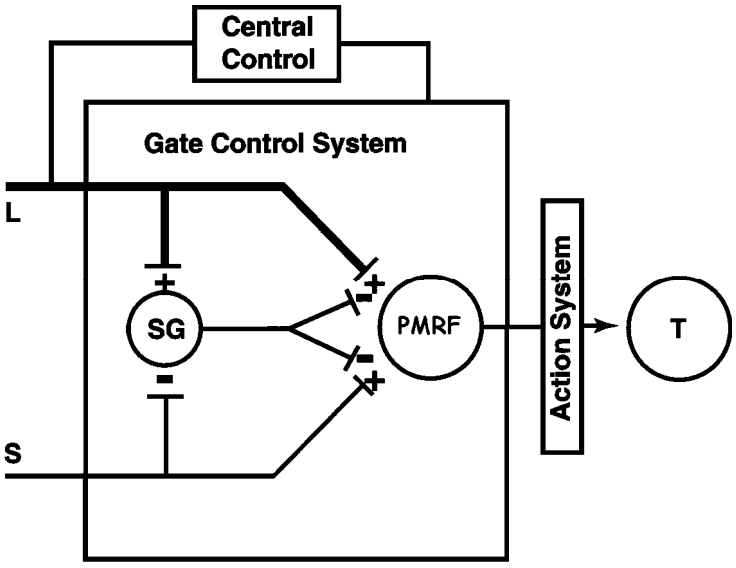
Pontomedullary Reticular Formation (PMRF)
For clinical purposes, I will reduce the PMRF to the greatly oversimplified version. I will focus only on the output. There are four basic output systems from the PMRF. They are;

1. Inhibit nociception at the dorsal horn of the spinal cord ipsilaterally.
2. Activate all ventral horn cells ipsilaterally, by inhibiting inhibition
3. Inhibit the IML ipsilaterally.
4. Inhibit Anterior Compartment muscles above T6 and inhibit Posterior Compartment muscles below
T6, ipsilaterally.

The output travels in reticulospinal pathways and are ipsilateral in nature. The meaning of this is simple, yet profound, in your understanding of human function. For as you can see, it is involved in; the modulation of pain, the tone of postural muscles, the balance of the sympathetic and parasympathetic nervous system, and walking. Think of the output of the PMRF as parasympathetic, and inhibitory to the ipsilateral IML.

NEW: Pain nociceptors dysfunction is a dysfunction of paired fast and slow nociceptors, and their pathways (Neo and Paleo spinothalamic tracts).

The gate control theory of pain describes how the perception of pain is not a direct result of activation of nociceptors, but instead is modulated by interaction between different neurons, both pain-transmitting and non-pain-transmitting. In other words, the theory asserts that activation, at the spine level or even by higher cognitive brain processes, of nerves or neurons that do not transmit pain signals can interfere with signals from pain fibers and inhibit or modulate an individual's experience of pain.



The physiological response to pain is: 1st the fast pain goes through the fast tract (neo spinothalamic) together with the tactile information from tactile propioceptors, and after a few milliseconds it´s blocked by the information that comes from the slow Paleospinothalamic tracts.

The signs to diagnose this condition are related to Flexor Reflex Afferent (FRA) or to Pontomedullary reticular Formation inhibitory response (PMRF). The response is related to the level of the body where the inhibition is trying to block the aberrant or conflicted associated information.

Flexor Reflex Afferent (FRA) is also known as a Nociceptive Withdrawal Reflex (NWR).
If the aberrant information is associated with FRA, the inhibition is blocked in lower medulla or in the spinal cord, and the symptoms are local with only one weak muscle extensor, or regional with two or more extensor muscles weak, or one side of the body with muscles extensors weak. Normally the contra lateral side shows an opposite response, with flexors weak.

If the aberrant information is inhibited in the Pontomedullary Reticular Formation, then the signs showed by the patient are: anterior muscles of Anterior Compartment above T6 and Posterior Compartment muscles below T6 ipsilaterally.

NEW NOCICEPTORS CLASSIFICATION:
Spinothalamic Tracts Nociceptor Dysfunctions

- Neo (over) – Paleo (under)
- Paleo (over) – Neo (under)
- Neo and Paleo over stimulated
- Temperature - Spinothalamic

Basic Pain Nociceptors paired dysfunction

NeoSpinoThalamic over PaleoSpinoThalamic

1. There is a fast nociceptor (Neo spinothalamic tract) paired with a slow nociceptor (Paleo spinothalamic tract).
2. The dysfunction causes no muscle weakness in the clear.
3. The patient shows an extensor weakness in a muscle (s) if the nociceptive dysfunction area is stimulated with pinprick, and a facilitation of the antagonist flexor muscle.
4. Signs to diagnose this condition are related to Flexor Reflex Afferent (FRA).
5. The fast pain area is often located close to the slow pain nociceptor, but it´s not a rule.
6. T.L. alone is negative in the areas where involved nociceptors are located. T.L. plus pinprick stimulation over the fast nociceptor inhibits any strong indicator muscle.
7. T.L. alone is negative in the areas where involved nociceptors are located. Pinprick stimulation over the fast nociceptor inhibits one or more muscles with FRA pattern.
8. Proper Paleo stimulation negates the test.
9. Performing pinprick stimulation over the fast nociceptor area and slapping over the slow nociceptor area plus DTR fixes the involved dysfunction.

PaleoSpinoThalamic over NeoSpinoThalamic

1. There is a slow nociceptor (Paleo spinothalamic tract) paired with a fast nociceptor (Neo spinothalamic tract).
2. The dysfunction causes no muscle weakness in the clear.
3. The patient shows a PontoMedullary Reticular Formation pattern with ipsilateral (and sometimes bilateral) inhibition of the anterior compartment muscles above T6 and inhibition of the Posterior Compartment muscles belowT6 if the nociceptive dysfunction area is stimulated with simultaneous multiple slapping, only if the primary nociceptor is related to the slow Paleo Spinothalamic tract.
4. The slow pain nociceptor is often located close to the fast pain nociceptor, but it´s not a rule.
5. T.L. alone is negative in the areas where involved nociceptors are located. T.L. plus slapping stimulation over the slow nociceptor location inhibits any strong indicator muscle.
6. Performing pinprick stimulation over the fast nociceptor area and slapping over the slow nociceptor area plus DTR fixes the dysfunction.

Multi Pain Nociceptors paired dysfunction

NeoSpinoThalamic over PaleoSpinoThalamic

1. There is one fast nociceptor (Neo spinothalamic tract) paired with two or more slow nociceptors (Paleo spinothalamic tract).
2. The dysfunction causes no muscle weakness in the clear.
3. The patient shows an extensor weakness in two or more muscles, in one region, ipsilateral or bilateral areas if the nociceptive dysfunction area is stimulated with pinprick, and facilitation of the antagonist flexor muscles.
4. T.L. alone is negative in the areas where involved nociceptors are located. Performing pinprick stimulation over the fast nociceptor area inhibits the related muscles (FRA pattern), pinprick plus T.L. inhibits any strong indicator muscle.
5. Performing pinprick stimulation over the fast nociceptor area plus DTR fixes the multi pain nociceptor dysfunction, and then the problem becomes a Basic Pain Nociceptors dysfunction. The basic pain dysfunction is treated as mentioned before.

PaleoSpinoThalamic over NeoSpinoThalamic

1. There is one slow nociceptor (Paleo spinothalamic tract) paired with two or more fast nociceptors (Neo spinothalamic tract).
2. The dysfunction causes no muscle weakness in the clear.
3. The patient shows a PontoMedullary Reticular Formation pattern with ipsilateral (and sometimes bilateral) inhibition of the anterior compartment muscles above T6 and inhibition of the Posterior Compartment muscles below T6 if the nociceptive dysfunction area is stimulated with prolonged multiple slapping, only if the primary nociceptor is related to the slow Paleo Spinothalamic tract.

4. T.L. alone is negative in the areas where involved nociceptors are located. Performing slapping stimulation over the slow nociceptor area inhibits the related muscles, slapping plus T.L. inhibits any strong indicator muscle.
5. Performing slapping stimulation over the slow nociceptor area plus DTR fixes the multi pain nociceptors dysfunction, and then the problem becomes a basic pain nociceptors dysfunction. The basic pain dysfunction is treated as mentioned before.

Hyper Pain propioceptors paired dysfunction

NeoSpinoThalamic over PaleoSpinoThalamic

1. There is one fast nociceptor (Neo spinothalamic tract) paired with one slow nociceptor (Paleo spinothalamic tract).
2. The dysfunction causes an extensor muscle weakness in the clear.
3. The patient shows an extensor weakness in one muscle, and flexor facilitation of the antagonist muscle.
4. T.L. alone is negative in the areas where involved nociceptors are located. Pinprick over-stimulation of the area may cause inhibition of two or more muscles with FRA pattern. T.L. plus pinprick stimulation over the fast nociceptor inhibits any strong indicator muscle.
5. Pressure over the dysfunction area facilitates the related weak extensor muscle.
6. Performing pinprick stimulation over the fast nociceptor area plus DTR fixes the hyper pain nociceptor dysfunction, and then the problem becomes a Basic Pain nociceptors dysfunction. The basic pain dysfunction is treated as mentioned before.

PaleoSpinoThalamic over NeoSpinoThalamic

1. There is one slow nociceptor (Paleo spinothalamic tract) paired with one fast nociceptor (Neo spinothalamic tract).
2. The dysfunction causes one muscle weakness in the clear.
3. The patient shows a PontoMedullary Reticular Formation pattern with ipsilateral (and sometimes bilateral) inhibition of the anterior compartment muscles above T6 and inhibition of the Posterior Compartment muscles below T6 muscles.
4. T.L. alone is negative in the areas where involved nociceptors are located. T.L. plus slapping stimulation over the slow nociceptor inhibits any strong indicator muscle.
5. Pressure over the dysfunction area facilitates the related weak muscle.
6. Performing slapping stimulation over the slow nociceptor area plus DTR fixes the hyper nociceptor dysfunction, then the problem becomes a Basic Paleo nociceptor dysfunction. The basic pain dysfunction is treated as mentioned before.

Hyper multi Pain propioceptors paired dysfunction

NeoSpinoThalamic over PaleoSpinoThalamic

1. There is one fast nociceptor (Neo spinothalamic tract) paired with two or more slow nociceptor (Paleo spinothalamic tract).
2. The dysfunction causes two or more extensor muscles weakness in the clear.
3. The patient shows an extensor weakness in two or more muscles, in one region, ipsilateral or bilateral areas, and facilitation of the antagonist flexor muscles.
4. T.L. alone is negative in the areas where involved nociceptors are located. T.L. plus pinprick stimulation over the fast nociceptor location inhibits any strong indicator muscle and strengthens the previously weak extensor muscle.
5. Pressure over the dysfunction area facilitates the related weak muscle.
6. Performing pinprick stimulation over the fast nociceptor area plus DTR fixes the hyper pain nociceptors dysfunction, but neo stimulation will cause the muscle to weaken again. Then the problem becomes a Basic Pain nociceptor dysfunction. The basic pain dysfunction is treated as mentioned before.

PaleoSpinoThalamic over NeoSpinoThalamic

1. There is one slow nociceptor (Paleo spinothalamic tract) paired with two or more fast nociceptors (Neo spinothalamic tract).
2. The dysfunction causes two or more muscles weakness in the clear.
3. The patient shows a PontoMedullary Reticular Formation pattern with ipsilateral (and sometimes bilateral) inhibition of the anterior compartment muscles above T6 and inhibition of the Posterior Compartment muscles below T6.
4. T.L. alone is negative in the areas where involved nociceptors are located. T.L. plus slapping stimulation over the Paleo nociceptor location inhibits any strong indicator muscle.
5. Pressure over the paleo dysfunction nociceptor area facilitates the related weak muscles.
6. Performing slapping stimulation over the paleo nociceptor area plus DTR fixes the hyper pain nociceptors dysfunction, then the problem becomes a Basic Pain nociceptors dysfunction. The basic pain dysfunction is treated as mentioned before.

Neo & Paleo Nociceptor Dysfunction (Mixed)

Physiology

The patient shows an ipsilateral inhibition weakness and contralateral hemisensory inhibition if the nociceptive dysfunction area is stimulated with pinprick and slapping at the same time for 10 seconds or more.

1. There is two or more fast nociceptor (Neo spinothalamic tract) paired with two or more slow nociceptors (Paleo spinothalamic tract).
2. The dysfunction causes ipsilateral muscle weakness in the clear. If the Ventral Posterior Lateral Nucleus (VPM) is the level of inhibition, the weakness is in the body, If the Ventral PosteroMedial Nucleus is the level of inhibition, the weakness is in the ipsilateral head muscles. If both nucleus are involved, contralateral body and ipsilateral head muscles are inhibited.
3. The patient shows an ipsilateral inhibition weakness and contralateral hemisensory inhibition.
4. Pressure over the dysfunction area facilitates the related weak muscles.
5. Performing pinprick stimulation over the neo nociceptor area and DTR to eliminate the multiples Paleo areas, and then slapping the Paleo Nociceptor area plus DTR to erase the multiples neo areas resolves to a basic nociceptor dysfunction. Treat the basic dysfunction as mentioned before.

Results

More than 200 patients has been diagnosed and treated with this technique, with structural problems and were treated with Pain propioceptors dysfunction (Paleo and Neo spinothalamic pathways and their propioceptors).
In all patients treated, the rules described above worked without exception.

97 to 98% of all patients treated by proprioceptive recalibration responded positively to the treatment, and showed long lasting results, but the treatment required 30 to 45 % less time to complete. 2 to 3% of patients with recurrent problems are attributable to concomitant chemical or emotional sources.

Summary

The initial Neo Spinothalamic response produces an FRA response locally, with more intensity ipsilateral flexors and contralateral extensors are activated. If the stimulus is more extreme all flexors are facilitated and all extensors are inhibited bilaterally. Secondarily Paleospinothalamic response inhibits the Substantia gelatinosa and PMRF pattern responses dominate. The PMRF pattern inhibits anterior muscles above T6 and posterior muscles below T6. This is also scaled according to intensity. Mild-local, Moderate-ipsilateral, Intense-global

The signs to diagnose this condition are related to Flexor Reflex Afferent (FRA), to Pontomedullary reticular Formation inhibitory response (PMRF) or Thalamic VPL or VPM inhibition.
The response is related to the level of the body where the inhibition is trying to block the aberrant or conflicted information associated with it.

Blockade of the Pain

- ❖ Rubbing blocks the pain from mechanoreceptors source
- ❖ Pressure blocks the pain from nociceptors source

Discussion

The preceding procedures will simplify and, at the same time, broaden the scope of treatment of two basic AK testing methods.
These novel testing and treatment procedures find and correct previously described dysfunctions, but at a much deeper level.
This method is fast, safe, and seems to affect the patient in a more profound and long lasting way. Results are quite surprising and we feel that we are only scratching the surface at this point.
Most structural problems seen in the clinical practice can be treated successfully with these new techniques. These procedures, the Kinematic Chain technique and the Event technique are complementary.

References

1. Palomar, Jose. Dynamic Muscle Propioceptors testing, Proceedings ICAK-U.S.A. 2008.

2. Kandel Eric R., Schwartz, James H., Jessell, Thomas M. Principles of neural Science; 4th ed.

3. Arthur C., M.D. Guyton, John E. Hall. Textbook of Medical Physiology, 2006.

4. Walther, David S., Applied Kinesiology Synopsis, 2nd ed., 2009, ICAK-U.S.A.Shawnee Mission, KS 66202.

5. Leaf David, Applied Kinesiology Flowchart Manual, 3rd ed., Plymouth, MA.

6. Silvestre D et Baetcher R., Counterstrain: Technique de Médecine Manuelle . Encycl. Méd. Chir. (Elsevier, Paris, France) , Kinésithérapie – Médecine physique – Réadaptation, 26-075-A-10, 1998, 14 p.

Ligament Stress Reaction and the Anterior Lumbar Adjustment

Greg Shiu, D.C.

Abstract

Objective

This paper shows the necessity for correctly diagnosing anterior lumbar maneuver and ligament stress reaction.

Clinical Features

A 30-year-old fitness trainer that is experiencing lower back pain while seated and walking. Patient works out 4-5 days/week with a circuit training rotation for the last 2 weeks. Blood pressure is 113/ 67 at rest. Pulse is 61/min. Height is 6'0''. I have seen patient 2 visits prior with relief but pain comes back with in approximately one-week post treatment.

Intervention and Outcome

Upon the first visit, the patient had complained of low back problems and discomfort that had presented with in the last week. I had asked him if nothing had changed from his daily routine and his response was nothing out of the norm. Pulse point diagnosis represent an active heart / small intestine meridian as well as the circulation sex / triple warmer. There was no correlation to emotions or biochemical. Adjustments were given to Atlas, C3, C5, Anterior L5 and T6, T8 as well as T2. Patient responded well and walked out with no pain.

Second visit patient returned 10 days later with the same complaint. I asked if there were any accidents or injuries that he might have sustained or any changes to his regular routine but the answer was no again. The pain returned with in 7 days after the initial visit. Using pulse point diagnosis the active meridian was the heart and small intestine meridian. I tried to link the pulse point to emotions and biochemical but there was no correlation. I used Dr. Jeff Spencer's cold Laser protocol to restore normal nerve firing pattern with the Erchonia cold laser using his up and down regulations techniques. I adjusted accordingly and the patient again left with no pain nor achy ness. Anterior lumbar was positive for L5 and an adjustment was performed.

Third visit patient three days later experiencing same low back and ask him again if there was anything that he change in his routine, and finally he revealed that he was doing circuit training. I asked him of his heart rate while he trained and was at 180 beats/min. Psoas muscle were tested and were weak bilaterally and both strengthened to the adrenal alarm point as well as the heart alarm point. Sub scapulars was weak on the right and strengthened to the adrenal alarm point as well as the heart alarm point. I then tested the left middle deltoid muscle and found it strong. Distraction to the joint was performed and

then retested the left middle deltoid, which was now weak. To confirm for ligament stress, the right brachialis was tested in the same manner as the middle deltoid and also showed weakness after distraction was placed upon the elbow joint Adjustments to the patient were C3, C5, T3, T6, and L5 anterior. Adrenal supplementation was given and Ligaplex I. Instructions was giving to maintain a max heart rate of 180 beats/ min – their age as specified by Phil Maffetone. Patient had relief of pain for the next month with no pain while exercising with in the maximum target heart range

Conclusion

This paper suggest the importance the ligament stress test and the importance of supplementation for the adrenals to maintain a lasting adjustment.

Key Indexing Terms

Anterior Lumbar, Adrenals, Kinesiology, Applied

The Internal Frontal Fault with Recurrent Headaches with Physical Exertions

Greg Shiu, D.C.

Abstract

Objective

Determining the efficacy of the Internal frontal fault with Pain behind the eye in a patient with pain for the duration of 30 days with increased pain with physical exertion.

Clinical Features

A 47-year-old female patient claimed that she was working out 30 days prior to visiting my practice and experienced severe headaches and eye pain while exercising that she had never experienced prior. Patient had worked out the same manner for the last 6 years with no prior experiences of headaches or eye pain. She had gone to neurologists for MRI and CT, which were negative and taken painkillers to try to alleviate the pain with no avail. She experience increased pain behind the eye with an increase in physical exertion (weight lifting and cycling). Blood Pressure was taken and was well with in normal ranges –blood pressure110/65, pulse- 58 beats/min and height 5'6''.

Intervention and Outcome

Upon hearing her symptoms, it sounded like classical Internal frontal fault. I challenged the left maxillary bone inferior to the malar surface of the zygomatic arch using pressure from lateral to medial and fond a positive challenge. Visually the patient did not demonstrate a tipped sphenoid. The correction was two part for the cranial. Pressure was applied to the hard palate on the ipsi lateral side of the challenge behind the last molar. Slight pressure was exerted inferior to superior and a slight give on the hard palate was exhibited after 40 seconds. On the contra lateral side superior to inferior pressure was placed onto the ptyergoid pocket for approximately 30 seconds. Adjustments to the body included atlas, C2 and C3 and Category I from SOT was performed. Patient confirmed with me the next day that the pain was reduced by 90%. Another treatment was give to the patient two days later and pain was reported to be negligible after exercise for the last week that previously had given her pain.

Conclusion

This paper suggests the importance of cranial faults and the impact it can have on patients with headaches.

Key Indexing Terms

Cranial Fault, Headache, Internal Frontal Fault, Kinesiology, Applied

Cold and Flu Patterns, A PAK® Perspective

Barton A. Stark, D.C., DIBAK, DIAMA

Abstract

The author discusses four phases of the management of patients suffering with cold or flu infection. A review of the Traditional Chinese Medicine (TCM) and biomedical descriptions of these phases is presented and expanded based on the author's clinical experience. Correlations to typical Professional Applied Kinesiology (PAK) findings and structural factors are clarified. Specific PAK diagnostic and treatment protocols are recommended.

Key Indexing Terms
Professional Applied Kinesiology, Colds, Influenza, Rhinovirus, H1N1, Upper Respiratory Tract Infection, Virus

Introduction

Cold and Flu Facts

Colds and influenza are a leading cause of doctor visits, missed work and school resulting in 114,000 average hospitalizations per year and ~20,000 deaths per year in U.S. Flu season typically runs from October through March with November and February as peak times. September, October, April, and May can also be active periods. Flu vaccine supplies are chronically inadequate, inconsistently effective, and possible triggers for other disease. Adverse reactions are flu-like symptoms usually within first 24 hours after flu shot and are thought to be auto-immune reactions. (1-2)

According to the American Lung Association early use of anti-virals can shorten illness and decrease symptom intensity. The Chinese medicine Materia Medica alone contains many powerful, scientifically and clinically validated antiviral herbal medicines. In fact Traditional Chinese Medicine (TCM) herbology was used during the SARS epidemic in 2003 to effectively treat patients and prevent outbreak among healthcare workers. (2)

One TCM author's perspective on cold and flu infection reflects *"… No other disharmony approaches the dynamism and variety of external pathogens as they move and lodge, transform and linger, intensify and diminish through and within the body's systems of channels, organs, stages, levels, and burners…"* (1)

Discussion

Cold and Flu Definitions

Colds are caused by approximately 100 known cold viruses. Half of cold infections are caused by Rhinovirus and effect mostly the upper respiratory tract (sinuses, ears, throat, adenoids, bronchi). Fevers are less common and usually less than 101.5 F. Headache, fatigue, body aches, loss of appetite, and severe cough are less common and more mild. Conversely, sneezing/stuffy nose and sore throat are common. Cold viruses are very contagious before and after symptoms. Serious complications are rare. Biomedical treatment typically includes OTC medications for symptoms, fluids, rest, avoidance of stress, alcohol, and tobacco. (1-2)

Influenza (flu) infections are caused by Influenza viruses A, B, and C and especially target the lungs. Lungs are vital organs, therefore, flu symptoms are usually more severe than colds. Higher fever (~102-104F), headache, loss of appetite, body aches, fatigue/exhaustion, and cough can all be severe. Possible complications include bronchitis, pneumonia, and death. Biomedical treatment may consist of Rx. anti-virals (5 available) and OTC symptom meds. (1)

Gauging the Patient

In gauging the patient presentation it is helpful to delineate an exuberant **immune reaction** (to the virus in this case) from a **stress reaction**. An adequate to exuberant immune reaction generally results in more intense symptoms. By contrast a stress reaction may involve any number of cold or flu symptoms but, because of fatigued or suppressed immune reaction, the symptoms may not be as pronounced. There is usually a history of pronounced cumulative stress in these patients which tends to suppress the immune reaction. In the case where the immune reaction is strong this author considers immune modulation such as adrenal support, in addition to the usual anti-microbials, lymph support, etc. Balanced adrenal function, for example, works as a modulator that helps prevent immune over-reaction which would otherwise create excessive inflammation, fever, and post-infection debris causing an increased load on an already burdened lymph system.

Traditional Chinese Medicine

TCM theory discusses colds and flu in terms of wind-cold, wind-heat, and “Warm Disease”. Many flu cases are warm disease where the symptoms involve heat and are more intense, especially fever. It is correct to use cooling herbs, for example, during the Initial and Fully Engaged Phases of a warm disease. However, it is usually incorrect to use warming formulae during a warm condition. (1-3)

Viral “heat” causes drying of body fluids and therefore injures the body’s Yin, especially **Lung Yin**, while also consuming the **reserves of available Chi**. This explains the often difficult pattern of fatigue and dry, spastic cough that can linger sometimes for weeks after the infection. As a result it is often helpful to utilize herbal Chi and/or Lung Yin tonics (or other energy related and lung tissue supplements) during the post-infection phase. (1-3)

In warm disease the patient's tongue will have **red, raised points** and/or **red discoloration** (as opposed to pink). A stress reaction, by contrast, may exhibit signs of stress induced exhaustion such as tongue paleness, a purple or blue undertone, and/or red points that are concave, and possibly some red tongue color. (3-4)

Phases of Viral Cold and Influenza Infection

Prevention Phase

The goal of this phase is to strengthen Chi reserves and the immune system to prevent viral invasion of adenoids, lungs (MALT), or digestive system (GALT)._Some effective remedies for the prevention phase are various mushroom formulas, astragalus, ginseng, schisandra, green tea, vitamin C, multi-vitamin/mineral, spleen drainage, and especially Vitamin D. Patient education should address lifestyle factors such as stress management, aerobic exercise, adequate sleep, avoidance of synthetic fats and excessive sugar, and the TCM recommendation to protect the ankles and neck from cold wind. (1-3)

After extensive review of current knowledge regarding immune function this author proposes that much of the human species is overly vulnerable to viral infection due to unhealthy balance of TH1/TH2 lymphocytes. Viral and tumor resistance are especially controlled by the TH1 subsets. TH1 is suppressed by stimulation of TH2. TH2 is stimulated, at least in part, by **fungal overgrowth**. Other factors, such as mercury, will also suppress T-cells. Therefore, it is recommended in the prevention phase to evaluate patients for signs and symptoms of fungal imbalance (particularly fungal dysbiosis) and **heavy metals,** in addition to treating **viruses** as needed.

Initial Exposure Phase

This occurs after viral exposure but before the virus has imbedded into lung or digestive tissues to further replicate. During this phase the virus is sensitive to many natural anti-microbials as it travels from the mouth or nose, down the throat to the lungs and/or digestive tract. When initial exposure is suspected begin strong anti-virals as soon as possible during the first 2-3 days after mild symptoms have begun. Nutrients recommended for initial exposure include andrographis, mushrooms, isatis, sophora, olive leaf, thymus and/or spleen glandulars, green tea (steeped at least ten minutes), zinc lozenges, selenium, and lymphatic drainage support. (1-3, 10)

Fully Engaged Phase

The virus has embedded into the lung membranes and/or digestive system and now must be dug out with dispersing remedies and the constitutional strength of the patient. Typical symptoms: racing pulse, high fever, extreme fatigue, no appetite, cough, and sometimes diarrhea or vomiting. In this phase the body produces thick, colored lung phlegm to isolate the virus in an attempt to force it into latency and eventually expel it. At the gut level purging via diarrhea or vomiting is customary. In addition to the anti-virals from the initial phase some additional remedies to consider for the engaged phase are cats claw, proteolytic enzymes, mucolytic nutrients (ex.: iodine, bromelain, fenugreek), quercitin, and bioflavinoids. Meadowsweet and/or willow can also be used to help suppress fever. Certain TCM herbal formulas are specifically oriented for this

phase and can be used to dislodge the virus from it's attachment in the lung or gut while also continuing the anti-virals. (1-3)

It is important to note that there is also a critical phase where the patient does not respond or continues to worsen in spite of these treatment efforts. Critical phase cases should be referred for medical evaluation. (1-2)

Post Infection Phase

The importance of this phase should not be under-estimated. In the short-term lymph drainage and restoration of Chi and Yin reserves are primary goals. Addressing these factors helps prevent rebound infections, excessive fatigue, and also potential for infection remnants or debri to trigger future, seemingly unrelated, disease. Drainage efforts are mainly focused on the lymphatics, but may also include liver and/or kidneys. Aerobic (low to moderate intensity) exercise is very helpful to stimulate lymphatic drainage as well as PAK evaluation and often lymphatic drainage remedies.

Common PAK lymphatic indicators are the lower and middle trapezius, pectoralis minor, retro- and antero-grade Lymphatic techniques. PAK Liver and Kidney indicators include pectoralis major sternal, rhomboids, and psoas. For restoring energy and Chi reserves evaluate ATP production related nutrients (via Schmitt CO2 challenge), adrenal indicators (sartorius, gracilis), and Pancreas function (Earth element: latissimus and pectoralis major clavicular). Ileo-cecal valve may also require PAK treatment in cases of diarrhea and vomiting. Lung indicators (deltoids, coracobrachialis) may strengthen to water (the source of Yin) and/or herbal Lung Yin tonics. (5, 6, 9)

Educating the Patient

At first sign of cold or flu symptoms begin aggressive natural remedies and PAK evaluation and treatment. Common early symptoms may be neck or upper thoracic stiffness, fatigue, low fever, aches, sensitivity to wind and/or cold (esp. on neck/throat, and ankles).

Note: Patients with fever over 100.5 F, cough, and resting pulse rate >90 BPM should undergo adequate medical evaluation, especially to rule out H1N1 infection, if they do not improve or stabilize within 24 hours. (1-2)

Common AK Considerations in Cold and Flu Cases

- Thymus circuits – infraspinatus
    - Thymus recruits immature stem cells from the bone marrow to specialize them into mature T-cells. Even though thymus function is diminished in adults and especially elderly, it still functions as an endocrine gland to stimulate immune response. (7)
- Spleen circuits -lower trapezius, middle trapezius
    - Spleen synthesizes new anti-bodies and removes from lymph and blood circulation antibody labeled microbes. In many ways spleen functions as a large lymph node and often requires drainage remedies/nutrients to facilitate optimal performance of reticulo-endothelial system. (8) Spleen

indicators such as Liver 13 (tip of 11th rib) may therapy localize in patients with any, seemingly unrelated, problem.

- Lymphatic Techniques -tight/short pectoralis minor usually due to inhibited lower trapezius which is often due to thoraco-lumbar fixation and/or spleen imbalance. (5,9)

H1N1 Flu Virus

H1N1 has shown to be highly sensitive to neuraminidase inhibitor medications. The herbs Sophora root and Isatis root also show strong neuraminidase inhibition effects. (2)

A Word about Vitamin D

"Vitamin D is an amazingly effective antimicrobial agent, producing 200 to 300 different antimicrobial peptides in your body that kill bacteria, viruses and fungi.
In the United States, the late winter average vitamin D level is only about 15-18 ng/ml… considered a very serious deficiency state…over 95 percent of U.S. senior citizens may be deficient, along with 85 percent of the American public … nearly one out of five children …have low blood levels of less than 50 nanomoles per liter, the level recommended by the American Academy of Pediatrics…two out of three children have … below 75 nmol/L--which is still insufficient..."
According to Dr. Mercola the **ideal blood levels of 25-OH vitamin D are 60ng/ml**. (10)

Conclusion

The division of cold or influenza infection into the four phases mentioned is an important clinical consideration in determining the optimal treatment approach. It is common for a patient to utilize remedies throughout a cold or flu that are best for Initial Exposure Phase, for example, but incorrect for Prevention or Fully Engaged Phase. PAK evaluation assists the astute practitioner in the selection of appropriate remedies/treatments.

References

1. Heuertz J. "Treating Colds and Flu with Chinese Herbal Medicines", Herbal Medicine Press

2. Heuertz J. "Chinese Herbal Medicines and the Novel H1N1 Virus", Herbal Medicine Press
3. Monda L. Scott J. Golden Flower Chinese Herbs: Formula Guide, 4th Ed. Jin Hua Press; 2004.

4. Maciocia G. Tongue Diagnosis in Chinese Medicine, Revised Ed. Eastland Press; 1995.

5. Walther D. Applied Kinesiology Synopsis, 2nd ed. ICAK-U.S.A. 2009.

6. Schmitt W. Selected Papers on Metabolic Faults, A.K.S.P., (919) 545-8829

7. Wikipedia.com, “Thymus”

8. Wikipedia.com, “Spleen”

9. Leaf D. Applied Kinesiology Flowchart Manual, 3rd ed., Privately Published, Plymouth, MA, 1995

10. Mercola J. “How to prevent the flu – as easy as 1,2,3…”, Mercola.com

Psoas Strain Counter - Strain Patterns, A PAK® Perspective

Barton A. Stark, D.C., DIBAK, DIAMA

Abstract

The author discusses the management of patients suffering with psoas Strain/Counter-strain (S/CS). A review of the Professional Applied Kinesiology (PAK) diagnosis and treatment of psoas S/CS is presented and expanded based on the author's clinical experience. Correlations to typical Professional Applied Kinesiology (PAK) findings and structural factors are clarified. Specific PAK diagnostic and treatment protocols are recommended.

Key Indexing Terms
Professional Applied Kinesiology™, Psoas, Low Back Pain, Strain/Counter-Strain.

Introduction

The Psoas S/CS, in this author's experience, is the most common cause of acute low back pain. It is also routinely found in hip and lower thoracic problems. Any patient that complains of pain in these areas after bending or twisting should be evaluated for S/CS of the psoas, particularly if the pain occurs **on the way up** from bending or if the twisting involves extension that triggers the pain. Getting out of a car seat, rising from a bed or chair, or even reaching up all are common scenarios where S/CS will trigger pain. Typically, the patient may have over-exerted their low back extensors while gardening or lifting items over a prolonged time period. The initial strain in the extensors is followed by the hypertonicity in the antagonistic psoas. The pain is usually described as somewhere in the low back and lower or even mid-thoracics. Fortunately, most cases can be successfully treated in one or two office visits. This author has observed this problem in countless patients, most of whom had been treated with limited or no success in other ways.

Discussion

Psoas major is a large, thick muscle situated mainly in the abdomen. It is a primary stabilizer for the lumbar spine. Within its substance is the lumbar plexus of nerves, with the branches emerging from its borders and surfaces. (1)

- **Origin**: vertebral bodies and discs from T-12 to L-5 and from the transverse processes of L-1 to L-5. Occasionally it does not insert to L-5.
- **Insertion**: lesser trochanter of femur on posterior medial aspect.

- **Psoas Action and Antagonists**:
    a) Thigh flexion -Gluteus maximus, hamstrings, adductor magnus (posterior portion)
    b) Lumbar flexion -Quadratus lumborum, erector spinae, opposite Psoas, Ilio-lumbar ligament (2-4)

Inhibition of psoas is found on side of the short stride and usually causes over-contraction of the opposite psoas. Internal thigh rotation (checked supine) is decreased on side of shortened psoas. Psoas dysfunction is also associated with diaphragm imbalance, disc instability, ileo-cecal valve, inguinal ligament area pain, meralgia paresthetica, and hip joint problems. (2-4)

Again, the hallmark of psoas S/CS is acute low back pain ***when straightening up*** from sitting or bending over, or sometimes with twisting. The patient may be antalgic in straight lumbar flexion or to one side. The S/CS of psoas compresses lumbars and S-I joint so it can re-aggravate preexisting injuries in those areas.

Psoas S/CS treatment:

1) An intact psoas is inhibited after maximal shortening of the muscle **or**
2) An inhibited psoas becomes less tender at the belly upon maximal passive shortening (4)
3) Test inhibited psoas for need of Glycine or Folic acid
4) Perform Strain/counterstrain correction
    - With patient supine hold firm hand pressure on the psoas belly just medial and superior to the greater trochanter, bend the knee and maximally flex the hip while maneuvering the knee medial or lateral to find which gives maximum relief of psoas tenderness, maintain that position then instruct patient to hold an inspiration as long as possible, after resumption of normal breathing lower the leg to neutral very slowly
    - A palpable click is often felt and usually means there was good correction
5) Adjust **T-12 anteriority** (usually the primary subluxation) and Lovett reactor at C-6
6) Ipsilateral S-I joint is often subluxated or fixated or a lumbar posteriority
    - Drop-piece adjustment is usually adequate once SCS is treated
7) Process mental, chemical, and structural factors related to the contra-lateral psoas inhibition if needed
8) Evaluate lumbar and lower thoracic spinal extensors for inhibition and/or muscle stretch reaction
9) Treat Psoas NL's (2-3)

The S/CS is thought to be a malfunction in the relationship of the intra-fusal and extra-fusal fibers within the muscle. (3)

Additional Nutrient Considerations for PAK Low Back Analysis
In general consider vitamin E for lumbar and pelvic problems. Wheat Germ Oil is an excellent source of complete vitamin E. The author does not find high dose synthetic

vitamin E fractions to be beneficial. Also remember specific lumbar muscle related nutrients. For chronic lumbar/pelvic support test for need of Kidney tonics. (2-3)

Conclusion

Even though the psoas S/CS usually causes intense pain and dysfunction, it has consistently proven to be identifiable and effectively treated through PAK. This is another example of PAK efficacy in treating a very common spinal and extremity problem based on the often neglected, but causal, muscle function factors.

References

1. Wikipedia.com, Psoas major muscle

2. Leaf D. Applied Kinesiology Flowchart Manual, 3rd ed. Privately Published, Plymouth, MA; 1995.

3. Walther D. Applied Kinesiology Synopsis, 2nd Ed. ICAK-U.S.A., 2009.

4. Leaf D. Lower Extremity Conditions Video Tape, Mo-Jo Enterprises.

Case Report - A Case of a Patient with Adrenal Glands Exhaustion Diagnosed and Treated with Applied Kinesiology and Laboratory Analysis

Giovanni Wullschleger, D.C., CCN

Abstract

This is a case report of a patient presenting multiple symptoms associated with adrenal dysfunction. The ability to understand adrenal gland physiology and the interaction with the rest of the body allowed the clinician to correlate the different symptoms presented by the patient with a common denominator: the Adrenal glands. Applied Kinesiology was used in the examination and treatment. The functional approach to this patient presentation was the key to solve a series of condition that the patient has been suffering for many years. The structural, biochemical and emotional complaints were all related to the patient's adrenal glands exhaustion. Treatment by different specialist never solved her problems because they all focused on the specific symptoms. A review of this case allows us to understand all the clinical manifestations of adrenal exhaustion and how they can all be present in a patient. Applied kinesiology testing helped in the diagnostic process and allowed the practitioner to better understand the correlation between the patient's musculoskeletal complaint and the adrenal related symptoms. Laboratory analysis by Adrenocortex stress profile (saliva) test confirmed the applied kinesiology findings.

Key Indexing Words

Adrenal Glands, General Adaptation Syndrome, Circadian Rhythm Of Cortisol, Adrenocortex Stress Profile (Saliva), Gluten Sensitivity

Introduction

Nowadays in our society, it is very common to encounter patients with adrenal related disorders. It is probably the most common functional disorder found today. A poor diet high in refined carbohydrate, hydrogenated fats, caffeine, and smoke, artificial sweeteners, combined with a very stressful lifestyle has created an epidemic of adrenal related disorders.

The adrenal glands help the organism to adapt to any form of stress: physical, chemical, thermal, electromagnetic and emotional. The body responds to stress by modifying its hormonal production allowing the person to overcome a stressful event.
Hans Selye, MD has described this response: **General Adaptation Syndrome**. Three stages of adrenal response occur: the **alarm reaction** is the body's initial response to stress. The adrenal glands go into a hyper functional state to increase cortisol levels to adapt to the demands of stress. The second stage is called the **resistance stage** and occurs if stress is prolonged. During this phase the body increases the cortisol production (Pregnenolone steal phase) at the expenses of the sex hormone precursor DHEA. Some people may remain in this state with elevated cortisol for their entire life, with all the consequences that high cortisol can cause on the organism: anterior pituitary suppression, secondary hypothyroidism, Insulin Insensitivity, syndrome X, hypertension, immune system suppression with thymus atrophy, suppression of secretory IgA and mucosal cell regeneration in the Gastrointestinal system leading to gastric ulcers, dysbiosis, leaky gut syndrome and eventually autoimmune disorders.

The final stage of adaptation is called **exhaustion stage**, where the adrenal glands can no longer adapt to stress and are exhausted. This will lower cortisol production. A state of adrenal gland hypo function is associated to fatigue disorders, anxiety, hypoglycemia, hypotension, the inability to withstand psychological and physiological stress and inflammatory conditions.

Patient presentation:
A 38 years old female patient presents to the office with multiple complaints. She initially came into the office for her low back pain, cervical spine recurring problems and bilateral knee pain. She has been to different practitioners with no lasting effect. Chiropractic and osteopathic manipulative corrections seem not to hold and cortisone injection done by orthopedist for her knee pain only aggravated her symptoms. The patient also complains of fatigue, difficulty starting the day, low blood pressure with episodes of fainting, dizziness, blurred vision, and lightheadedness. She suffers of allergic asthma, difficulty concentrating, mood swings and feels depressed in the fall and winter, suffers of anxiety attacks, difficulty staying asleep, feels under a lot of stress all the time and has severe perspiration at night.

She craves salty foods, sweets in the middle of morning; she feels irritable if meals are delayed, gets lightheaded and has memory problems in midmorning and afternoon. Other complain are premenstrual syndrome, irregular or missed periods, inability to become pregnant. Bloating and gas after the meals, frequent skin eruptions, sensitive to perfumes and sulfites (Found in wine).

Joint pain, stiffness, pain in the neck and stiff shoulders, low back pain, bilateral medial knee pain with no apparent pathology as revealed by a negative MRI.

Physical exam:

Vital signs: Blood Pressure supine: 112/74 standing: 95/67
Indicating a positive **orthostatic hypotension** test as found in catecholamine imbalances (Ragland Effect).

Paradoxyl Pupillary light reflex: a **paradoxyl pupillary reaction** is present, indicating mineral corticoid imbalance.

Postural Analysis: revealed a right convex thoracic scoliosis, bilateral genu valgus, and hypolordotic lumbar curve with a slight left convex scoliosis.

Palpation: revealed a bilateral Sacroiliac subluxation (PIEx) with bilateral weak Sartorius muscle, tenderness on the pes anserinus bilaterally, th9 subluxation and Atlas ASRP subluxation.

Orthopedic and Neurological examination: within normal limits.

Applied Kinesiology examination:
Aspirin, Acetaminophen oral challenge test: positive for essential fatty acid deficiency (Gamma linoleic acid as in Blackcurrant seed oil and Eicosapentaenoic acid as found in fish oil).

Antihistamine oral challenge test: positive for gluten sensitivity, which was removed from her diet (wheat, barley, Oat,).

Aldehide sniff test: positive. Patient was sensitive to many perfumes and sulfites; Molybdenum negated the positive reaction.

Iron was tested, as the patient knew to be anemic, and continued with supplementation.

Systemic structural:
Neurological disorganization was present due to bilateral TMJ dysfunction, which was secondary to immune system weakness (thymus and spleen). Treatment to the Chapman reflex for the thymus and spleen was applied. Cranial faults were found secondary to emotional stress, which was corrected with Emotional Recall technique.

Endocrine:
Bilateral weaknesses of the Sartorius and tibialis posterior muscles were negated by Adrenal gland Chapman reflex. Positive ligament stretch test.
Oral challenge for nutritional supplementation: positive for vitamin B complex (bioglycozyme forte), Adrenal glandular extract (Cytozyme AD) and adaptogenic herbs compound (licorice root, Panax and Siberian Ginseng, Rhodiola, Ashwagandha).

Findings

An *Adrenocortex stress profile test* (saliva) was ordered to confirm the clinical findings. Salivary cortisol and DHEA are measured. An evaluation of how cortisol levels differ throughout the day is possible by collecting four saliva samples at different times. Cortisol levels typically peak shortly after rising in the morning and are at their lowest after the onset of sleep. Cortisol is involved in many important functions of the body, including the metabolism of carbohydrates, proteins and fats, the control of inflammation, regulation of proper glycemic level. Cortisol is also important in blood pressure regulation, normal nerve and brain activity and normal heart and immune function. DHEA also plays a role in the body's metabolism, regulates body weight, blood pressure, and immune function and is the precursor for the hormones testosterone and estradiol.

The patient's profile showed <u>decreased cortisol levels</u> in the morning at rising 7-9 AM, in the afternoon 3-5 PM and late at night 10 PM-12 AM with <u>normal DHEA levels</u>.

Because cortisol levels are typically at their peak shortly after awakening, morning cortisol may be a good indicator of peak adrenal function. Low morning cortisol levels suggest a degree of adrenal fatigue with regard to peak circadian activity.

Afternoon cortisol levels may be a good indication of the adrenal gland 's ability to regulate blood sugar, since they represent a postprandial sample. Low levels reflect a degree of adrenal fatigue, especially in the area of glycemic control.

Late night cortisol levels may be a good indication of baseline adrenal gland function, since they represent the lowest level during the day. The patient cortisol level was low also at this time of the day, confirming an adrenal gland hypo function of the zona fasciculata (the primary source of cortisol).

<u>Results:</u> A degree of adrenal hypo function is suggested, which has been noted in fatigue disorders, physiological and psychological stress, anxiety, hypotension and hypoglycemia.

Treatment

Treatment consisted in the correction of the findings described above:

<u>Diet</u>:
The patient was recommended a gluten-free diet. Elimination of wheat and other cereal with gluten resolved her gastrointestinal symptoms quickly. Avoidance of food allergens is in of primary importance, since gluten sensitivity and food hypersensitivity in general are a contributing factor to adrenal stress.
The patient was recommended to have a good breakfast, which included some proteins and complex carbohydrates, avoid caffeinated beverages, artificial sweeteners, hydrogenated fats, eat snack in the middle of the morning and middle afternoon to have a

better glycemic control (reactive hypoglycemia). A diet rich in fruits and vegetables, complex carbohydrates, lean meat and fish was recommended.

Nutritional supplementation included:
Blackcurrant seed oil (GLA) and Fish oil (EPA-DHA) to improve inflammation and intercellular communication.
Vitamin B complex with minerals as a general support for adrenal hormone synthesis and to help regulate glycemic levels.
Adrenal glandular extracts were recommended in the beginning of care with a combination of adaptogenic herbs (as described above).

Molybdenum was recommended for chemical sensitivity to sulfites and perfumes (aldehides) and iron since the patient was anemic (CBC from previous MD).

Applied Kinesiology and chiropractic treatment:
Injury recall technique with Therapy localization to Small Intestine Chapman reflex and offender in the mouth (wheat) was performed. Allergy desensitization technique as of Dr. S. Walker DC, DIBAK was performed to help desensitize the patient. The patient was then instructed to follow a gluten-free diet.

Chapman reflex for the thymus and spleen were treated to correct neurological disorganization secondary to immune dysfunction. Emotional Recall technique was used to correct emotional issue, which caused secondary cranial faults.
Bilateral Chapman reflexes prolonged stimulation was performed for the adrenal glands, which facilitated adrenal related muscles Sartorius and tibialis posterior originally found inhibited in the clear.
Spinal manipulation was performed on the sacroiliac joints, th9 and C1 with the listings mentioned above.

Aerobic exercise was recommended. The patient is instructed to start walking thirty minutes three to four times a week.

Results

The patient symptoms improved gradually within a short period of time. Her knees started feeling better and was able to climb the stairs right after the first treatment. Her low back improved and she was able to maintain the spinal correction (no recurrence of low back pain). Most symptoms related to hypoglycemia improved with the dietary change we advised (patient used to skip breakfast, eat croissant and coffee for lunch and be under a lot of stress at work). The patient general fatigue improved and so did her symptoms of hypotension (especially orthostatic). Within two months her allergic asthma improved, so that she did not need medication and had no recurrent attack. Sleeping pattern improved, she could sleep all night without waking up, and her mood in general was better (treatment was done in the fall and reevaluation in December; these are the months were subjects who suffer from Seasonal Affective Disorder are more

depressed). And finally after about four weeks after her first visit, the patient was able to become pregnant after trying for many years before (age 37 years old).

Discussion

The value of applied kinesiology in the diagnosis and the treatment of this patient are remarkable. The ability to use muscle testing and its correlation to organs/glands help the practitioner to determine the origin of the patient's functional problem. In this case many symptoms were present and the previous practitioners without success managed them all individually. It is with our holistic approach were the structural, emotional and biochemical component all come together that we can help the patient the most. AK gives us the tools to diagnose functional conditions with remarkable precision as often confirmed by laboratory findings, as in this case.

Conclusion

The life of this patient was changed from the moment that we understood the cause of her multiple complaints. It is by teaching the patient how her body reacted to her life style, and how to improve her health by changing her diet, teaching her how to exercise correctly, by correcting her structural and biochemical faults and helping her overcome old emotional pattern, that we gave her the chance to be in control of her own health. This patient no longer needs the drugs she was taking for her back, knee, shoulder and neck aches, and sleeping and asthma medication. Utilizing the adrenocortex stress profile test to confirm the validity of Applied Kinesiology muscle-organ correlation in adrenal disorder should be the object of future studies.

References

1. J. Alexander Bralley, Ph.D, CCN, Richard S. Lord, Ph.D. Laboratory evaluations in molecular medicine. The Institute for Advances in Molecular Medicine, Norcross, GA 2001. P.302-309.

2. D. Walther, DC, DIBAK. Applied Kinesiology Synopsis, 2 ed., 2009 ICAK-U.S.A., Shawnee Mission, KS 66202.

3. D. Kharrazian, DC, DIBAK, Functional endocrinology, seminar notes. 45

Hyperthyroidism - A Case Report of a Patient Treated with Applied Kinesiology and Nutrition

Giovanni Wullschleger, D.C., CCN

Abstract

This case report reviews the treatment of a patient, who presented to the clinic with a diagnosis of subclinical hyperthyroidism (Basedow = Grave disease). The patient was looking for an alternative to the pharmacological treatment recommended by her physician. Applied kinesiology and nutritional therapy was used in conjunction with laboratory analysis to diagnose, monitor and treat the patient. Treatment was directed to correct thyroid hyper function and restore gastrointestinal integrity through diet modification and specific nutritional supplementation.

Key Indexing Words

Hyperthyroidism, Grave Disease, Basedow Disease, Applied Kinesiology, Vitamin A, Lithium, Thymus Glandular Extract

Introduction

Hyperthyroidism is a condition characterized by over activity of the thyroid gland, therefore excessive hormone is produced. Several causes exist, including immunological reaction (believed to be the cause of Graves` disease), thyroiditis, toxic thyroid nodules (adenoma).

In this case report we describe the treatment of a patient suffering of Hyperthyroidism from autoimmune origin and treated with Applied Kinesiology and Nutritional therapy. High dosage of Vitamin A (Retinoic acid) and Lithium, were used to control thyroid over-activity. Particular attention was dedicated to the restoration of the gastrointestinal system, being a possible site of origin for autoimmune disorders.

Patient Presentation: A comprehensive history reveals a 39-year-old female patient presenting the following symptoms (divided according to the symptom survey form utilized).

Thyroid: nervousness, palpitations, elevated heart beat at rest, tremor, increased appetite, feels irritable and restless, blepharospasm, tiredness, can not tolerate warm temperature, hot flushes, disphagia, morning headaches.

Menstrual cycle: irregular menses, menstrual pain, painful breast before and during menstruation, hot flushes, premenstrual depression.

Gastrointestinal system: difficulties digesting, intestinal bloating and gas right after the meal, diarrhea alternated to constipation and halitosis, intolerance to dairy products.

Other: excessive hair loss. This may be related to a zinc deficiency, often seen in hyperthyroid patients. Oral zinc tolerance test shows that hyperthyroid patients have a basal serum zinc concentration similar to euthyroid individuals, but with greater urinary zinc excretion, which suggests zinc depletion from tissues to the bloodstream, caused by catabolism from the hyperthyroid state.
Hyperthyroidism also causes lower zinc assimilation by tissues after ingestion.
The patient also consumes 4-5 coffees a day and smokes about one pack of cigarettes a day. She has a sedentary lifestyle.

Laboratory findings:

Serum level:
Free T4: 23 pmol/L (10-21)
Free T3: 6.8 pmol/L (3.0-6.4)
TSH: below 0.1 mU/L (0.3-5.0)
Antibodies antitireoperoxidase: 57.1 IU/ml (below 0.2)
Antibodies antitireoglobulin: 17.51 IU/ml (below 0.1)

Ecography thyroid gland:
Hypoecogenicity of the thyroid's parenchyma compatible with autoimmune thyroid pathology. Hyperplasia of the right lobe with global thyroid volume in the norm.

Applied Kinesiology test:
The patient presents with a bilateral weak teres minor. The weakness was negated with oral challenge of Vitamin A, Lithium and thyroid neurolymphatic reflex stimulation. Weaknesses of bilateral Tensor fascia lata, right pectoralis major sternal and right quadratus lumborum were negated by therapy localization to Liver and Large intestine neurolymphatic reflexes. Oral challenge with Ultraclear sustain (support for Gastrointestinal system) negated the weakness.

Food Allergy/Intolerance test:
The test was performed using oral challenge of different foods and manual muscle testing. The patient reacted positive to wheat, caffeine and dairy products. (Food tested: wheat, dairy, chocolate, coffee, corn, soy, grains, peanut, baker and brewer yeast).

Structural evaluation:
Subluxations were identified at C4-C5, D8 and L5.

Treatment

We decided to treat the patient for one month and see if we were able to bring the thyroid under control. In the opposite case the patient would have take the medication as prescribed by the medical endocrinologist
(Neomercazol 5 mg).

Diet:
Eliminate wheat, dairy and caffeine in general. Patient is instructed to eat plenty of fruits, vegetables, fish (salmon, cod…), white meat, gluten free cereals (rice), nuts, and almonds. Soy products, cabbage, pine nuts and millet were increased since they are considered dietary goitrogens, and are able to inhibit thyroid function by interfering with iodine utilization. This food contains isothiocyanates, which have a similar structure and function of propylthiouracil (compound most commonly used to treat hyperthyroidism). Increase water consumption, and avoid all caffeinated beverages.

Nutritional supplementation:
Vitamin A (retinoic acid): Bio Ae-mulsion forte (Biotics research):
First week 100`000 IU/day, (25`000 IU qid).
Next three weeks 50`000 IU (12`500 IU qid).
Lithium: LI-zyme (Biotics research): 2 tablets, tid
Multimineral complex: Multi-mins (Biotics research):
Thymus glandular: Citozyme-THY (Biotics research):

Ultraclear Sustain: (Metagenics):
(Support for gastrointestinal system including many compounds specific for repairing the intestinal mucosa and improve digestive function; L-Glutamine, gamma-oryzanol, probiotics, fructooligosaccarides,).
The process of improving gastrointestinal function, through food allergy elimination and Ultraclear sustain was done for about four weeks prior to the treatment protocol for the thyroid gland.

Chiropractic and Applied Kinesiology treatment:
Spinal manipulation was performed at C4, D8, and L5 level.
Neurolymphatic reflex stimulation of the Thyroid gland, Large Intestine and Liver.
The patient was instructed to walk 30 minutes 3-4 times a week.

Results

The patient is reevaluated after one month of therapy. Improvement was felt by the patient in the intestinal function, with disappearance of gas and abdominal bloating after the meal, the stool consistency returned to normal (she complained of constipation alternated to diarrhea), the menstrual cycle is now regular with less premenstrual pain, she feels less tired, morning headaches resolved, less hot flushes, pulse is regular, dysphagia also improved with no more discomfort (lump in the throat). Manual muscle

testing reveals strong (facilitated) teres minor bilaterally, tensor fascia lata bilaterally and pectoralis major sternal.

Laboratory test:

	Before treatment	After treatment
FreeT4 (10-21)	23 pmol/L H	14.6 pmol/L
FreeT3 (3.0-6.4)	6.8 pmol/L H	3.96 poml/L
TSH (0.3-5.0)	Below 0.1 mU/L L	Below 0.1mU/L L

The patient is feeling better and T3 and T4 level returned within reference range. Treatment is stopped and TSH will be repeated at three and six month distance.
After three months TSH was 2.4, (free T4, 13 pmol/L and freeT3, 4.1pmol/L).
After six month TSH was 2.71 and the patient condition is stable.

Discussion

Dr. George Goodheart has described the effect of Vitamin A and Lithium on thyroid hyper function in the past. Studies conducted mainly in Japan and published on different journals of endocrinology confirm the mechanism by which vitamin A and Lithium influence thyroid metabolism.

By reviewing the mechanism of formation of thyroxin and triiodothyronine we better understand how Vitamin A and Lithium affect T3 and T4 synthesis and release.

Thyroid hormone secretion: Mechanism

The first stage in the formation of thyroid hormones is the transport of iodide from the blood flow into the thyroid glandular cells. The basal membrane of the thyroid cell has the specific ability to pump the iodide into the cell. Iodide concentration inside the cell is about thirty times higher then in the blood. Once in the cell, iodide needs to be oxidized into an oxidized form of iodine in order to combine directly with the amino acid tyrosine. The enzyme responsible for this oxidation process is called peroxidase. The other function of the thyroid cells is the synthesis of a glycoprotein known as thyroglobulin. This protein contains seventy tyrosine amino acids and they are the major substrates that combine with iodine to form the thyroid hormone. T3 and T4 formed from tyrosine remain part of the thyroglobulin protein. When the peroxidase enzyme is blocked the rate of thyroid hormone secretion drops. Vitamin A is responsible to reduce gene expression of thyroid peroxidase and reduce gene expression of thyroglobulin, inhibiting therefore the release of organified iodine necessary for thyroid *hormone* synthesis.

Conclusion

Further studies are recommended to confirm the efficacy of retinoic acid and Lithium in the treatment of autoimmune hyperthyroidism. These two substances can regulate thyroid

activity, without the side effects caused by medication and radiotherapy (irreversible hypothyroidism).

References

1. Arai M, Tsushima T, Isozaki O et al. *Effects of retinoid on iodine metabolism, thyroid peroxidase gene expression, and deoxyribonucleic acid synthesis in porcine thyroid cell in culture.* Endocrinology 1991; 129: 2827-2833.

2. Namby H, Yamashita S, Morita S, et al. *Retinoic acids inhibits human thyroid peroxidase and thyroglobulin gene expression in cultured human thyrocytes.* Depart. Internal medicine, Nagasaki University, school of medicine, J.Endocrinologic. Invest. 1993 Feb., 16(2) 87-93.

3. Tsuchiya Y. Saji M, Isozaki O, Arai H., et al. *Effect of lithium on deoxyribonucleic acid synthesis and iodide uptake in porcine thyroid cellsin culture.* Endocrinology1990 Jan; 126 (1): 460-5

4. Mori M, Tajima K, et al. *Inhibitory effect of lithium on the release of thyroid hormones from thyrotropin-stimulated mouse thyroids in a perifusion system.* Osaka University, Medical school, Endocrinology 1989 Mar., 124 (3) 1365-9.

5. Hokkaido Igaku, Zasshi, *Effect of lithium on iodide uptake and DNA synthesis in porcine thyroid cells in culture.* Department of medicine, Asahikawa Medical College; 1990Mar 65(2) 161-9.

6. Eideneir H., Ph.D., Nutritional protocol manual, DSD International.

7. Goodheart G., Research Tapes.

Division III

Constructive Review

A New Class of Cranial Faults that Accompany the Yaw #2 PRY Pattern

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Abstract

The discoveries of a new series of cranial faults that accompany each of the modular distortion patterns of Pitch-Roll-Yaw that have never been observed before. This paper will deal with the specific of the Yaw # 2 distortion and its associated cranial faults.

Introduction

The modular distortion patterns of Pitch-Roll and Yaw know as (PRT-Technique) was first presented in the 1980, by Dr. George J. Goodheart in the Workshop Procedural Manual.[1] These observation were preceded in by Dr. Goodheart "Ocular-Basic Technique", in the 1979 Workshop Procedural Manual[2] and were derived from information Dr. Goodheart developed from his reading a book titled "Awareness Through Movement".[3] This showed the importance that eyes played in coordinating the musculature of the body and its effect on the neck muscles. This became know as the "visual righting reflexes". The secondary system of reflexes that was connected to the sacrum known as "cloacal reflex", which must be in synchronization meaning that the head and sacrum must be level in order to provide maximum neural communications.

Discussion

After incorporating this technique into my standard AK examination of all new patients in 1980 I became aware the Yaw #2 pattern would return in a short period of time after the initial correction. The other part of the PRY-Technique (Pitch-Roll and Yaw #1) could be corrected during the first treatment and with rare exception would stay corrected for at least six months to a year.

After writing many research papers about this pattern and its connection to neurological disorganization (switching),[4,5,6] dural torque[7,8,9], hiatal hernias and gastroesophageal reflux disorder (GERD),[10] I believed that if the pattern kept returning maybe there was another factor that I was missing. Since PRY-T is not only a modular distortion but also a method to diagnose dural torque I thought there might be a cranial fault that occurs with this pattern. What I found was two specific cranial faults that related specifically to the Yaw # 2 distortion pattern. Currently I have been observing this on every patient that I have treated over the past two years so the sampling is 1,157 at the present time that I have treated it.

Method

I would check each patient the presents of a Yaw #2 modular distortion by using the DeJarnette block in a challenge position. The original testing position described in Walther's book Synopsis describes this test position with the patient prone, with the body modules are rotated by placing the bock under a shoulder and another one under the opposite pelvis. If this pattern were present a strong indicator muscle would weaken.[11]

However, to test this premise of an associated cranial fault the testing would be done supine with the block under one should the other under the pelvis at the PSIS region. The supine position would be the opposite of block position in the prone.

I first examined for all known cranial faults before if present I would then challenge with the above mentioned block position supine and have patient therapy localize (TL) and see if any specific know fault would negate the positive challenge position. What I found was no known AK cranial faults were related to this yaw #2 pattern.

So the next thing I did was check all different patterns what I found was a combination of on thumb of one hand and the index of the other hand would cause a weakness to a strong indicator muscle. If the patient was placed on block, in the challenge position of the Yaw # 2 a thumb and index finger to the palate could negate modular distortion. It did seem to matter which thumb and index were used as long as it was one finger from each hand.

A second fault was found that also associated with Yaw #2 modular distortion pattern. This was TL using the finger tips of Index-Middle-Ring (IMR) placed on the mastoid region and the other hands (IMR) fingers are placed on the parietal bone above the parietal ridge just behind the coronal suture on the same side as the mastoid.

The present of this fault is specific to the side opposite listing of the Yaw #2 pattern. Example if the yaw #2 is left in the supine position this means that pelvis on the right ASIS is up toward the ceiling then this fault will by on the right. This is the most common pattern of pattern of this modular distortion at least 90% or more on the left with the ASIS higher as described on the right side in the supine. If the Yaw # 2 were to be found on the right then the fault would be on the left.
The block challenge for the Yaw #2 supine was negated by using the above mentioned TL hand positions.

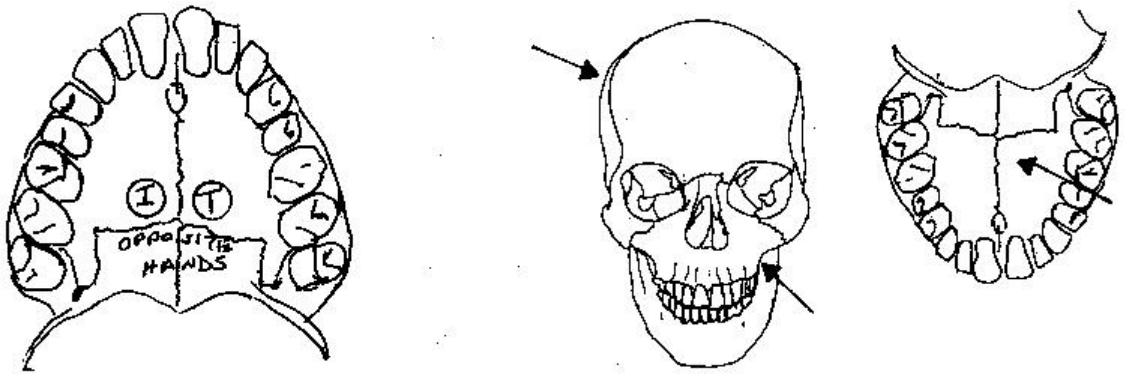
Correction

The structural correction of the Thumb/Index TL-(Yaw #2 Cranial Fault Pattern-1) is done by challenge to the palate with directional force applied as an example on the right side near the cruciate suture vectored towards opposite parietal bone near the coronal suture. As with cranial faults the correction is in the direction that causes the maximum weakness using the phase of respiration that negates the weakness which has always been

to date inspiration. The pressure of correction is similar about 1 to 3 pound of pressure repeated about 4 to 5 times.

YAW #2 CRANIAL FAULT PATTERN-#1

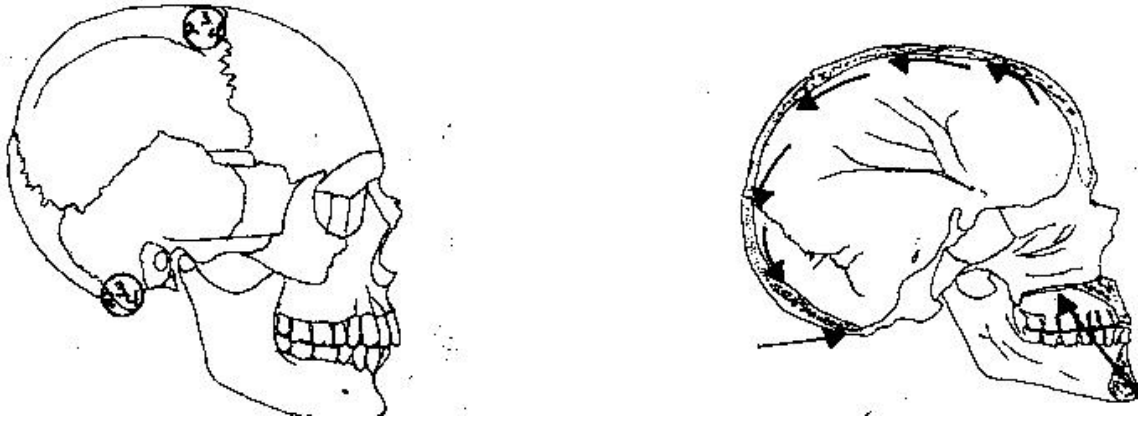
THERAPY LOCALIZATION **CORRECTION**



The (Yaw #2 Cranial Fault Pattern-2) (IMR-Fingers) TL to mastoid and parietal on the same side is corrected in a similar manner to a Sphenobasilar fault. On the side of positive TL pressure is applied to the maxillary part of palate near the cruciate suture on the same side a pressure is applied along the parietal and down into the mastoid in a forward direction. Inspiratory assist is used while the pressure is being applied with about one to three pounds of force about four to five applications. Both faults are rechecked after corrections, which should now be negative.

YAW #2 CRANIAL FAULT PATTERN-#2

THERAPY LOCALIZATION **CORRECTION**



Conclusion

After check and correction these two new cranial faults for the past two years their presence seem to prove that the PRY-Technique is definitely a method of diagnosis and correction dural torque. Since the discovery of this pattern by Dr. Goodheart in 1980 with the methods that have been described in the Workshop Research Manual and in Synopsis, 2nd Edition. The question that needed to be answer was, of what importance would it be to add this to the current body of knowledge?

If what I had discovered is correct this would mean that these two specific cranial faults would be present in every patient that has ever had a Yaw #2 pattern. Since the Yaw #2 modular distortion pattern is almost universally found in 99 percent of any sampling of the general population. It made it easy to get a large sampling in just a two-year period that I have been doing this study.

After I found enough cases presently 1,157, I started testing homeostatic factors of blood pressure (BP), pulse rate (PR) and range of motion (ROM). Each of these factors was measured with the patient being examined in the supine position. Blood pressure was taken on the right arm with a (OMRON Model:HEM-711DLX) automatic blood pressure monitor which also gives pulse rate. The BP and PR were measured before and immediately after the correction. The BP cuff remained on the arm to avoid chance of an error occurring. Significant changes for blood pressure was 5mm of Hg either systolic or diastolic pressure, and the pulse rate changes had to be at least 10 beats up or down.

Range of motion was done on both hip joints with knee locked, with foot externally rotated and moved into abduction as far as the patient would tolerate. The normal ROM for this test should be 90 degrees.

The outcome in BP and PR up or down with significant changes occurred in 70 percent of the cases tested. The ROM study showed an average of 15-degree change to all the studies participants treated. The range of motion change was the greatest with the Yaw #2 Cranial Fault Pattern-#1 comparing against all other known cranial faults. This factor alone seems to warrant this correction being added to the patients treatment plan, because it seem to improve the functioning of the autonomic nervous system.

Reference

1. Goodheart, George, J., D.C., Applied Kinesiology-Workshop Procedure Manual, Private Publication, 16th Edition, 1980, Pages 1-9.

2. Goodheart, George, J., D.C., Applied Kinesiology-Workshop Procedure Manual, Private Publication, 15th Edition, 1979, Pages 23-29.

3. Feldenkrais, Moshe, Awareness Though Movement, Harper and Rowe, 1972.

4. Sprieser, Paul, T. D.C., A New Slant on Switching, Collected Papers of The International College of Applied Kinesiology-U.S.A., Private Publication, 2001, Pages 197-202.

5. Sprieser, Paul,T., D.C., Switching and its Ramifications, Collected Papers of the International College of Applied Kinesiology-U.S.A., Private Publication, 2007 Pages 213-220

6. Sprieser, Paul, T., D.C., The Relationship of Switching to the Yaw #2 of the PRY-T Technique, Collected Papers of the International College of Applied Kinesiology-U.S.A., Private Publication, 2003, Pages 243-247.

7. Sprieser, Paul, T., D.C., The Relationship of Rocker Action to PRY-T Technique As a Method of Diagnosis of Specific Dural Lesion, Collected Papers of the International College of Applied Kinesiology-U.S.A., Private Publication, 2001, Pages 47-48.

8. Sprieser, Paul, T., D.C., Dural Torque and its Association to the Pitch Pattern of (PRY-T) Technique and Improvements in Range of Motion (ROM)- Collected Papers of the International College of Applied Kinesiology-U.S.A., Private Publication 2002, Pages 193-195.

9. Sprieser, Paul, T., D.C., Newly Discovered Causative Factor for Switching and the Yaw #2 Pattern, Collected Papers of the International College of Applied Kinesiology-U.S.A., Private Publication, 2006, Page 91-93.

10. Sprieser, Paul, T., D.C., Gastroesophageal Reflux Disorder and Hiatal Hernia a Universal Problem, Collected Papers of the International College of Applied Kinesiology-U.S.A., Private Publication, 2005, Pages 209-217.

11. Walther, David, S., D.C., Applied Kinesiology-Synopsis 2nd Edition, ICAK- U.S.A., Shawnee Mission, KS 66202, Pages, 213-217.

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