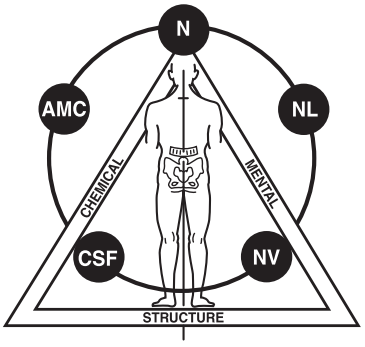
International College of Applied Kinesiology®-U.S.A.

Experimental Observations of Members of the ICAK

Volume I, 2006 – 2007

Proceedings of the Annual Meeting



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

Experimental Observations of Members of the ICAK

Volume I, 2006 – 2007

Proceedings of the Annual Meeting

Presented

June 8 – 11, 2006

Chicago, IL

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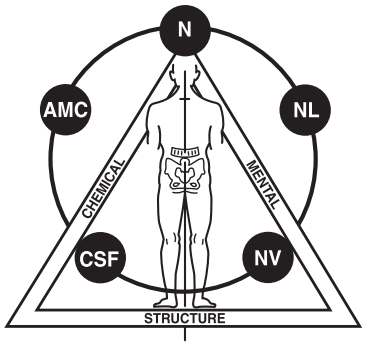

Message From the Chairman

David Leaf, D.C., DIBAK

For 30 years, the members of the International College of Applied Kinesiology®-U.S.A. have shared their insights, outcomes, case histories and research through the papers presented in the *Proceedings*. The ICAK-U.S.A. continues to thrive as an "Arena of Ideas" through which members have the opportunity to present their observations and research results. These published works document the first steps toward the furtherance and development of the authors' hypotheses, concepts and procedural techniques which can culminate in their material becoming part of the accepted body of knowledge we know as applied kinesiology. We invite all members to participate in this endeavor in the future.

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

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

Thank you and congratulations to all of our contributors. And a special thanks to Drs. Rebecca Hartle, David Engel, Jan Calhoon, Alan Zatkin, and Denise Natale for all of their help during the review process, and Dr. Barton Stark, our Research Chair. We look forward to seeing you at the Annual Meeting, June 8–11, 2006 in Chicago, IL.

Introduction

This forty-eighth collection of papers from members of the International College of Applied Kinesiology®-U.S.A. contains 23 papers by 16 authors. The authors welcome comments and further ideas on their findings. You may talk with them at the meeting or write them directly; addresses are given in the Table of Contents.

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

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

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

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

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

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

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

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

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

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

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

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

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

Continuing call for papers includes:

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

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

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

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

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

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

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

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

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

The following editorial policies will apply:

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

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

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

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

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

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

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

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Manuscript preparation

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

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

Approved manuscript style

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

Summary of requirements

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

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

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

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

Detailed preparation procedure

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

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

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

Title page

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

Abstract and key word page

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

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

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

Text pages

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

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

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

Introduction

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

Materials and methods

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

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

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

Results

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

Discussion

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

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

Conclusions

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

Acknowledgments

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

Reference pages

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

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

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

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

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

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

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

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

Journals

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

Books and other monographs

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

Other articles

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

Table pages

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

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

Illustration legend pages

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

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

Illustration preparation

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

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

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

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

Manuscript submission summary

Manuscript components

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

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

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Applied Kinesiology Status Statement

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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Division I

Informative Papers

Manubrium, Meridian Imbalance, and Emotions

Cecilia A. Duffy, DC, DIBAK

Abstract

OBJECTIVE: To compile two sources describing the Manubrium, Meridian Imbalance, and Emotion Technique of Applied Kinesiology.

Key indexing terms: Manubrium, subluxation, acupuncture, meridian imbalance, emotion, applied kinesiology.

Discussion

Goodheart described the Manubrium, Meridian Imbalance, and Emotion Technique in the 1986 Applied Kinesiology Research Manual (1) and the author also heard an updated version of this technique at a seminar given by Goodheart and Schmitt in 1986 (2). Since this technique is not contained in major texts on Applied Kinesiology, a compilation of the technique from the two sources is offered.

Procedure

1. Meridian pulse point testing is negative in the clear, with Eyes Into Distortion, Body Into Distortion, and Breath Cessation. A Temporosphenoidal Line (TS Line) indicator for a muscle involvement reveals no inhibition in the clear or as a 51%er (including therapy localization of the muscle's related acupuncture alarm point).

2. Having the patient therapy localize the acupuncture alarm point related to the muscle that was indicated on the TS Line combined with opening their mouth so as to separate their lips will induce an inhibition of the TS Line related muscle.

3. Challenge the manubrium for subluxation by exerting either a cephal or a caudal pressure just above the Angle of Louis. A positive challenge occurs when one direction inhibits a previously facilitated muscle. Find the phase of respiration (inspiration or expiration) that negates the positive challenge. Adjust the manubrium in the opposite direction as the challenge (as in an extremity subluxation) on the phase of respiration that abolished the positive challenge.

4. Re-evaluate therapy localization of the alarm point with the patient's mouth open utilizing the TS Line muscle (repeating Step 2); if negative, go on to Step 6; if still positive, go on to Step 5.

5. Tap the manubrium.

6. While the patient again therapy localizes the alarm point, have them vocalize "(Name of patient) is (positive emotion associated with the meridian)" (see chart below); this should produce no inhibition of the associated meridian/TS Line muscle. Continue alarm point therapy localization and have patient

vocalize "(Name of patient) is (negative emotion associated with the meridian)"; if this inhibits the meridian/TS Line muscle, re-tap the manubrium.

7. Utilizing a general indicator muscle, have the patient therapy localize the acupuncture associated point on the spine related to the meridian/TS Line muscle; this will be negative. Add the negative vocalization from Step 6 to the associated point therapy localization; if this inhibits the general indicator muscle, examine that vertebral level for an intraosseous subluxation and correct.

Acupuncture Meridian-Emotion Chart[1, 2]

Meridian	Positive Emotions	Negative Emotions
Lung	humble, tolerant, modest	disdain, scorn, contempt, haughtiness, false pride, intolerance, prejudice
Circulation Sex	reject remorse, jealousy, or tension; relaxed, generous	remorseful
Heart	love, forgiveness	angry
Liver	happy, glad, good fortune, cheerful	unhappy, sad
Gall Bladder	love, forgiveness	rage, fury
Spleen	faith in the future, secure	anxiety in the future
Stomach	tranquil, content	disgusted, disappointment, greed, emptiness, deprivation, nausea, hunger
Triple Heater	lightness, buoyancy	heaviness, depression, despair, grief, hopelessness, despondency, loneliness, solitude
Kidney	sexually secure	sexually insecure
Large Intestine	clean, good, worthy of being loved	guilt
Small Intestine	joy	sorrow, sad
Bladder	peace, harmony	restlessness, impatience, frustration

Goodheart describes discovering that a facilitated muscle would inhibit with therapy localization of its alarm point combined with mouth opening, but treatment of acupuncture points related to the specific meridian had no effect on the muscle inhibition. It was ultimately found that tapping the manubrium negated the alarm point therapy localization with mouth open.[1]

Diamond's work influenced the emotional aspect of the technique. Diamond stated that the patient vocalization "I am (related emotion)" is less effective than vocalizing "(Patient name) is (related emotion)." "….the imbalance of the meridians induces the emotions and the body can't tell the spurious emotions from the real thing."[1]

Conclusion

An apparently lesser-known emotional technique in applied kinesiology derived from two sources is described.

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Modifications of the Manubrium, Meridian Imbalance, and Emotion Technique

Cecilia A. Duffy, DC, DIBAK

Abstract

OBJECTIVE: To present modifications of the Manubrium, Meridian Imbalance, and Emotion Technique and briefly describe its usefulness in panic disorder. [1, 2, 3]

Key indexing terms: Manubrium, subluxation, acupuncture, meridian imbalance, emotion, applied kinesiology, temporal tap.

Discussion

Since learning the Manubrium, Meridian Imbalance, and Emotions Technique in 1986, the author has utilized it consistently in practice along with other more well-known applied kinesiology emotional approaches (Psychological Reversal and Emotional Technique).[4] This paper represents the author's clinical experience with some modifications of the technique as originally described by Goodheart.[1, 2, 3]

The Manubrium, Meridian Imbalance, and Emotions procedure was outlined in this volume[3] and is repeated here.

1. Meridian pulse point testing is negative in the clear, with Eyes Into Distortion, Body Into Distortion, and Breath Cessation. A Temporosphenoidal Line (TS Line) indicator for a muscle involvement reveals no inhibition in the clear or as a 51%er (including therapy localization of the muscle's related acupuncture alarm point).

2. Having the patient therapy localize the acupuncture alarm point related to the muscle that was indicated on the TS Line combined with opening their mouth so as to separate their lips will induce an inhibition of the TS Line related muscle.

3. Challenge the manubrium for subluxation by exerting either a cephal or a caudal pressure just above the Angle of Louis. A positive challenge occurs when one direction inhibits a previously facilitated muscle. Find the phase of respiration (inspiration or expiration) that negates the positive challenge. Adjust the manubrium in the opposite direction as the challenge (as in an extremity subluxation) on the phase of respiration that abolished the positive challenge.

4. Re-evaluate therapy localization of the alarm point with the patient's mouth open utilizing the TS Line muscle (repeating Step 2); if negative, go on to Step 6; if still positive, go on to Step 5.

5. Tap the manubrium.

6. While the patient again therapy localizes the alarm point, have them vocalize "(Name of patient) is (positive emotion associated with the meridian)" (see chart below); this should produce no inhibition of the associated meridian/TS Line muscle. Continue alarm point therapy localization and have patient vocalize "(Name of patient) is (negative emotion associated with the meridian)"; if this inhibits the meridian/TS Line muscle, re-tap the manubrium.

7. Utilizing a general indicator muscle, have the patient therapy localize the acupuncture associated point on the spine related to the meridian/TS Line muscle; this will be negative. Add the negative vocalization from Step 6 to the associated point therapy localization; if this inhibits the general indicator muscle, examine that vertebral level for an intraosseous subluxation and correct.

Acupuncture Meridian-Emotion Chart [1, 2]

Meridian	Positive Emotions	Negative Emotions
Lung	humble, tolerant, modest	disdain, scorn, contempt, haughtiness, false pride, intolerance, prejudice
Circulation Sex	reject remorse, jealousy, or tension; relaxed, generous	remorseful
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Liver	happy, glad, good fortune, cheerful	unhappy, sad
Gall Bladder	love, forgiveness	rage, fury
Spleen	faith in the future, secure	anxiety in the future
Stomach	tranquil, content	disgusted, disappointment, greed, emptiness, deprivation, nausea, hunger
Triple Heater	lightness, buoyancy	heaviness, depression, despair, grief, hopelessness, despondency, loneliness, solitude
Kidney	sexually secure	sexually insecure
Large Intestine	clean, good, worthy of being loved	guilt
Small Intestine	joy	sorrow, sad
Bladder	peace, harmony	restlessness, impatience, frustration

The first modification of the technique is at step 7 where the patient therapy localizes the associated point at the spine while vocalizing the negative emotion. Many patients are unable to reach certain areas of the spine to therapy localize. In that case, bypass therapy localization/vocalization combination and directly challenge both sides of the vertebral level for an intraosseous subluxation.

The next modification involves a change in the diagnostic entry point of the technique. In the author's clinic, each examination/treatment room contains a binder with various clinical notes. This technique is contained in the binder with the list of positive and negative emotions for ready reference when utilizing the technique on patients. Have the patient read the positive and negative emotions chart and pinpoint those negative emotions that they feel describe them and/or the positive emotions they wish they could emulate. The patient may choose one or many meridians. The entry point to the technique then changes to step 6 whereby a facilitated muscle related to the emotion the patient chose is tested. Therapy localization of the meridian's alarm point against the meridian related muscle produces no change in muscle strength; add the negative vocalization to the alarm point therapy localization and observe if the muscle inhibits. If this therapy localization with patient negative vocalization is negative (muscle remains facilitated), move on to other emotions/meridians/muscles the patient chose. If an emotion/meridian produces the related muscle inhibition, go back to step 3 and continue through the technique skipping steps 4 and 6.

The final modification is the addition of Temporal Tap Technique[5] instruction. At the author's clinic there are pads available with tear-off sheets containing the Temporal Tap instructions along with pictures to help patients locate the area to be tapped. The right and left sided appropriate beginning phrase is followed by a blank line that is filled in with the negative emotion that originally caused the positive challenge. Patients are instructed to perform the temporal tap on each side of the head three times each, many times throughout the day, for three weeks. If more than one negative emotion/meridian occurred, the patient is instructed to do each emotion independently during the temporal tap.

Temporal Tap Instructions

1. Begin in front of left ear and tap quickly along the temporosphenoidal line while simultaneously speaking out loud, "I get along fine without_____."

   Repeat three times, then immediately do the right side.

2. Begin in front of right ear and tap quickly along the temporosphenoidal line while simultaneously speaking out loud "There is no reason for me to _____."

   Repeat right side three times.

   Repeat this procedure often during the day for a total of three weeks.

The author has found the following effective in panic disorder:

1. Blood glucose regulation: The reader is referred to Duffy's concept of Panic Disorder[6] as a result of a sudden change in blood glucose levels resulting in an adrenal catecholamine response that is designed to raise blood glucose levels but results in other physiological effects such as increased heart rate (pounding heart), visceral vasoconstriction (abdominal distress, "butterflies in the stomach"), and vasoconstriction diminishing blood supply to the skin and diverting it to the muscle (cold, clammy skin). These sensations can frighten a patient unaccustomed to and/or unknowledgeable in their origin. The patient may operantly condition the symptoms to some event or object that can later provoke return of the

catecholamine-induced symptoms resulting in a panic attack. Combine blood glucose regulation via diet and give instructions for managing an acute panic attack per Duffy's suggestions.[6]

2. Diagnosis and application if indicated of Emotional Technique, Psychological Reversal, and Manubrium, Meridian Imbalance, and Emotion Technique.

Conclusion

Modifications to the Manubrium, Meridian Imbalance, and Emotion Technique are presented that are clinically effective in the author's experience.

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1. Goodheart, GJ. 1986 Applied Kinesiology Research Manual. 22nd edition. Geneva, Ohio: AK Printing; 1986, p. 51–54.
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The Missed Relation Between Gait Analysis and Stomatognathic System Causing Eye in Distortion in Golfers.

Robert Jo, D.C.

Abstract

The gait mechanisms are often checked and corrected for lower extremity involvements. In some cases the Doctor may proceed further and check for the involvement in the spine but often misses the foot correlation with the cranial involvement. This paper brings up the importance of the need to identify the gait pattern and foot involvement with its relation to stomatognathic system and eyes into distortion/(EID).

Introduction

Five male golfer subjects with bilateral low back stiffness and right short leg were tested. These subjects have stated their golf strokes had increased lately and felt they were not hitting the ball straight. They were initially treated in the lumbar spine and the foot (twice in one week) for subluxation and muscle imbalance only. All five patients played one round of golf after the second treatment with less stiffness in the lumbar spine but the golf stroke did not return to their average score.

Discussion

The subjects were then re-examined to determine if the adjustments held. Initial AK muscle testing revealed weakness in the quadratus lumborum, gluteus medius and piriformis. Upon re-examination all subjects tested 5/5 and pelvis levels were even. Of the five subjects, three subjects had an increase in their scores.

Subject	Average Score	Present Average Score Before TX	Difference	After Second Treatment
A	83	92	+9	-1
B	81	87	+6	+5
C	94	101	+7	+2
D	90	96	+6	-3
E	90	94	+4	+2

Table 1: Present average is within last one month period from the initial date at same course.

Examination and Therapy

All five of the subjects selected had a right short leg for better comparison. Upon initial testing, all subjects showed at lease one side of quadratus lumborum, piriformis, gluteus medius, and soleus muscles at 4/5 using AK muscle testing.

After the second treatment and a round of golf they were now further evaluated for a dropped navicular with foot shock method. All five subjects showed positive foot shock on the left with left navicular subluxation.

Therapy localization to left TMJ with mouth in open position caused weakness of the indicator muscle. Eye in distortion to the left also caused the weakness of the indicator muscle but not to the right lateral eye distortion. Five treatments within two weeks for the navicular subluxation with arch support, left lateral pterygoid strain counterstrain treatment, left TMJ purcussor, and left sphenobasilar inspiration assist were performed.

After completing the treatment protocol, the subjects reported the result of their golf scores. Four of the five subjects made improvements with two subjects decreasing their score even further below their initial average scores.

Subject	Average Score	Present Average Score Before TX	Score After 5 TX.	Difference
A	83	92	84	+1
B	81	87	78	-3
C	94	101	90	-4
D	90	96	92	+2
E	90	94	91	+1

Table 2

Conclusion

Symptoms in one area of the body are always related to another structurally and biomechanically. Eyes in distortion may be caused by adaptation of oculomotor muscles locked according to the distortion in the body. The body adapts to the distortion which may come from cranial or any other structural abnormality due to tensegrity of the fascia. The dropped navicular due to short leg may influence the muscles of mastication every time the muscle in the jaw or the gait is activated, causing the stomatognatic systems to be one of the causes for eye in distortion. The stomatognatic system and eyes in distortion should be part of routine evaluation in all patients in order for the body to be centrally integrated to maintain its structural balance.

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Advanced Principals of the Neuroendocrine-Immune Stress Response and It's Application to Clinical Practice and Applied Kinesiology

Datis Kharrazian, DC, MS, FAACP, DACBN, DIBAK, CNS, CSCS, CCSP

Abstract

The connections of the neuroendocrine-immune system, and the role it plays with activation of the hypothalamus-pituitary adrenal axis is complex. Cytokines, hormones, neurotransmitters and neural pathways orchestrate responses to acute and chronic stress. This paper reviews the neurophysiological interactions of these systems and proposes models that employ natural medicine and neuronal stimulation to support aberrancies within these physiological systems.

Key Indexing Terms: paraventricular cell of the hypothalamus, nucleus of the solitary tract, hypothalamus-pituitary-adrenal axis, cortisol, intermediolateral cell column, dentorubrothalamocoritocal loop, hemisphericity, locus ceruleus, corticolenticulopallidostraitothalmocortical loop, nucleus raphe magnus, substantia nigra, pontomedullary reticular formation, GABA, glutamate, aspartate, epinephrine, norepinephrine, serotonin, angiotensin, vasopressin, hippocampus, amygdala, dorsal lateral periaqueductal gray, dorsal raphe nucleus, rostral ventrolateral medulla, applied kinesiology

Introduction

Activation of the neuroendocrine-stress response occurs to some degree in almost every chronically ill patient. Stress is the great epidemic of the twenty-first century. As the field of neurophysiology, neuroendocrinology and neuroimmunology expand we learn to appreciate the interconnectedness of human systems. Cell messengers such as hormones, cytokines, and prostaglandins have allowed us to understand how physiological systems integrate. The physiological mechanisms of diseases are being understood at greater depth every year, but its applications to clinical practice are lingering.

Our current medical model of specialists in each human system has already become outdated. It is impossible to look at the function of one system without the influence of other systems. The future education of human physiology may promote an educational model that does not make distinct differentiations of human systems, instead a collective human physiological system may be presented. In order for healthcare professionals to help manage chronic diseases, the impact of stress on physiological systems and understanding of the neuroendocirne-immune system must be appreciated.

Discussion

Neuroendocrine-Immune Interactions

The nervous, endocrine and immune systems are intimately connected by cytokines. These cell messengers allow these physiological systems to work as an orchestrated system. Therefore, it is virtually impossible to stimulate any one of these systems in isolation. For example, any activation of the hypothalamic-adrenal-axis (HPA) will create an inflammatory response, and any inflammatory reaction will activate the HPA axis.

Therefore, we can see that the explanation of many disease processes today can be explained when evaluating the role of cytokines in the activation of the neuroendocrine-immune response.[i]

Up-regulation of the HPA axis has been shown to induce insulin insensitivity, decrease TSH and T3 production,[ii] [iii] increase inactive reverse T3, [iv] decrease phase II glucuronidation and sulfation, suppress pituitary function,[v] increase the potential for gastric and duodenal ulcers, lower intestinal secretory IgA,[vi] [vii] delay intestinal mucosal cell generation,[viii] suppress immunity,[ix] decrease bone density, induce depression,[x] encourage obesity [xi] [xii] [xiii] and increase the risk for cardiovascular and neurodegenerative disorders.[xiv]

Inflammatory responses as a result of antigen and non-antigen based mechanisms promote the release of interleukin-1, interleukin-6 and TNF-alpha by central and peripheral immune cells. These cytokines directly stimulate the synthesis of corticotrophin-releasing hormone and vasopressin at the level of the hypothalamus.[xv]

The release of inflammatory mediators promote the activation of the HPA axis which serves as an immune modulating response to decrease the activity of the inflammatory responses so the immune response does not become overzealous. Glucocorticoids include suppression of the peripheral release of interferon-gamma, granulocyte-monocyte conoly-stimulating factor; interleukin 1, 2, 3, and 6; and 6; TNF alpha; inflammatory mediators such as eicosonaoids, bradykinin, serotonin and histamine; and tissue destroying enzymes, including collagenase and elastase.[xvi]

Therefore, it appears that central activation of the HPA axis initially promotes the release of cortisol and the promotion of inflammatory mediators and peripheral immune responses activate the HPA axis which serves to act as a suppressing response on the self-perpetuating cellular inflammatory response. Dysfunction in the HPA axis modulating response may therefore promote increased inflammatory responses from central responses and a failure to suppress inflammatory immune activation. Inability to suppress the production of these cytokines, which have been shown to promote both TH-1 and TH-2 subset dominance, have been linked to a number of autoimmune and chronic diseases.

The role of the HPA axis in immune responses is not just limited to dampening the inflammatory cytokine response of cellular immunity, but it also serves a role in trafficking immune cells to the site of acute challenge.[xvii] The HPA axis works in concert with the autonomic nervous system to contain acute-phase responses by influencing the redistribution of lymphocytes and macrophages throughout the body.[xviii] [xix] [xx] Hyporesponsivness of the HPA axis increases the inflammatory cytokine response. It is important to note that cortisol secretion is designed to normally counter-regulate the immune response so once the immune response is triggered it will not become overzealous. Therefore, individuals with diminished central responses to stress have susceptibility to inflammatory responses and the promotion of abnormal cytokine synthesis.[xxi]

Chronic stress creates a glucocorticoid-cascade that leads to oxidative damage of the glucocorticoid sensitive hippocampal cells.[xxii] This leads to dysregualtion of the HPA axis and cognitive impairments.[xxiii]

Chronic stress has demonstrated neurodegenerative impacts on the hippocampus through mechanisms involving pyramidal neurons in the CA3 region of the hippocampus through mechanisms involving both glucocorticoids and excitatory neurotransmitters.[xxiv] The damage induced in the hippocampus in acute stress is reversible, but stress lasting many months or years can cause hippocampal neuronal apoptosis.[xxv] These changes in the hippocampus have been illustrated with magnetic resonance imaging.[xxvi]

When the hippocampus is impaired it fails to inhibit the release of excitatory amino acids that modulate the central stress response. This may lead to plasticity of the central neuraxis towards chronic activation of HPA axis despite exogenous sources of stress.[xxvii]

Inflammatory reactions have the ability to suppress the activity of the endocrine system. Inflammatory cytokines released from severe trauma or burns have demonstrated severe suppressive activity on the pituitary-ovarian and pituiraty-gonadal mechanisms leading to anovulation in women and diminished spermatogenesis and depressed testosterone in males.[xxviii] [xxix] A study in which interlukin-1 was injected into the third ventricle demonstrated dramatic suppression on the hypothalamic release of gonadotropin-releasing-hormone (GnRH).[xxx]

Cytokines released by immunocompetent cells have demonstrated the ability to modulate the expression of the endocrine system. For example, the thyroid stimulating hormone (TSH) response to thyrotropin-releasing hormone has been shown to be reduced by TNF-alpha.[xxxi] The pituitary secretion of TSH have also been shown to be inhibited by interleukin-1 and interleukin-6.[xxxii] [xxxiii] Therefore, many of the cellular proteins that have been associated as immune system inflammatory responses appear to have modulating influence on central neurendocrine immune responses.

Summary of the Neuroendocrine-Immune Response to Stress:

- Decreased expression of insulin receptor site signal translocation of GLUT transporters
- Increased expression of interleukin-6 and interleukin 10
- Increased production and sensitivity of IL-6 receptor complexes (sIL-6R)
- Suppressed production of IL-12 and interferon gamma
- Increased expression of tumor necrosis factor-alpha
- Increased hypothalamus-pituitary-adrenal axis activation
- Decreased production of IGF-1 and HGH
- Decreased insulin secretion
- Suppression of LH in men and women
- Suppression of SIgA
- Decreased testosterone in males
- Altered progesterone distribution in females
- Altered expressions of leptin on neuropeptide Y and hypothalamic integration
- Negative nitrogen balance
- Mineral depletion (zinc, cobalt, selenium, copper)
- Suppressed neutrophil function
- Suppressed lymphocyte count and proliferation
- Suppressed natural killer cell count and activity
- Alterations in polymorphonuclear cell priming potentials
- Shift of T helper lymphocytes (TH) to TH2 over TH1 balance which results in the up-regulation of humoral immunity and down-regulation of cell-mediated immunity
- Elevations of urea
- Elevations of C-reactive protein
- Increased production of PGE2 via expression of inducible cyclo-oxygenase 2
- Uncoupling of mitochondria oxidative phosphorylation
- Increased uric acid production
- Decreased iron, hemoglobin, ferritin, TIBC
- Reduced glutamine levels
- Elevated creatine kinase
- Altered catecholamine production and secretion
- Decreased serum tryptophan levels
- Alteration in brain serotonin levels
- Neurotransmitter release and/or membrane sensitivity is altered
- Autonomic shifts of balance into what is called either sympathetic or parasympathetic syndromes
- Alterations in fatty acid metabolism which decreases the chain length of fatty acids which may be attributed to either alterations in synthesis or higher rates of perioxidation.

Hypothalamus-Pituitary-Adrenal Regulation Mechanisms

The parvocellular cells of the paraventricular nucles of the hypothalamus (PVN) is considered the final common pathway of the stress response. The central integrative state of the PVN either toward sodium or potassium equilibrium potentials are mediated by multiple canalized pathways. These pathways include projections for neocortex, paleocortex, mesencephalon and cerebellum. Chemical mediators of these projections include GABA, glutamate, aspartate, epinephrine, norepinephrine, serotonin, angiotensin, insulin, vasopressin and cortisol. The medial parvocelluar cells of the paraventricular nucleus of the hypothalamus is mediated by excitatory and inhibitory interactions with hypophysiotropic corticotrophin-releasing hormone, which represents the final common pathway in the stress response.[xxxiv] In the following paragraphs the influence of the these neurochemicals on the PVN will be reviewed.

Activation of the hypothamolic-pituitary-adrenal axis is centrally mediated by the secretion of corticotropin-releasing factor (CRF) by the paraventricular nucleus (PVN) of the median eminence of the hypothalamus. In addition to the PVN, there are other central areas such as the amygdala which contain CRF-containing neurons and project into the mesolimbic system, the pontine reticular formation and the medial mesencephalon.[xxxv]

There are many interactions between norepinephrine(NE) and CRF in the central response to stress. Both NE and CRF elicit influence on one another. NE can excite the CRF containing cells of the PVN and CRF increases the release of NE in the prefrontal cortex, the PVN and the locus ceruleus.[xxxvi]

It is these interconnected relationships with NE and CRF that may lead to uncontrolled vicious cycle responses of the HPA axis. It is very clear that a positive feedback loop occurs between these two intercellular communicating agents. Noradrenergic neurons stimulate neurons in the PVN to release CRF which activates locus ceruleus NE release. However, these reciprocal activated systems have potentials to lead to escalating activity and sensitization responses that may create an adverse feed-forward cycle. This abnormal mediated feed-forward mechanism has been postulated in exaggerated physiological stress responses, anxiety, panic attacks and depression.[xxxvii]

The hypothalamic paraventricular nuclei receive the majority of andrenergic projections from the nucleus of the solitary tract and a smaller percentage for the locus ceruleus.[xxxviii]

The locus ceruleus is the main adrenergic output center of the neuraxis and its central integrative state is mediated through numerous central structures besides the PVN which include the nucleus paragigantocellularis, nucleus prepositus hypoglossi, Barrington's nucleus, spinal intermediate grey nuclei, parabrachial nuclei, bed nucleus of stria terminalis, periaqueductal gray nuclei, hippocampus, central nucleus of the amygdala, and the Edinger-Westphal nucleus.[xxxix]

Studies of immobilization repeated daily for 6 days decreased basal concentrations of norepinephrine in the PVN and in the central nucleus of the amygdala.[xl] Prolonged stress increased the activity of the norandrenergic system centrally and peripherally. Several studies have demonstrated these types of responses may lead to depleted levels of norepinephrine in the brian.[xli] [xlii]

Brain stem released catacholamines excite CRF neurons in the hypothalamic paraventricular nucleus to activate HPA axis. CRF containing neurons in the PVN and the amygdala project to the locus ceruleus in both direct and indirect pathways.

In addition to catecholamine stimulation of the PVN's central integration, both GABA and glutamate exhibit central modulating influences on PVN activity. Therefore, GABAergic and glutamergic circuits

interact with the parvocellular paraventricular neurons controlling HPA activity. GABAergic neurons in the bed of nucleus of the stria terminalis, preoptic area, and hypothalamus can directly inhibit PVN outflow and thereby reduce ACTH secretion. On the other hand, glutamate excites the PVN projecting cells to the HPA axis. [xliii] Therefore, GABAergic and glutamergic projections express inhibitory and excitatory modulating properties of the central integrative state of the PVN. The GABAergic inhibitory projections to the PVN arise from amygdala and septum. The glutamergic projections are from neurons of the ventral subiculum and prefrontal cortex.[xliv]

Lesion studies of the neurons that project excitatory and inhibitory stimulation to the PVN such as the ventral subiculum have been shown to influence the activity of the HPA axis. Studies in which the ventral subiculum and prefrontal cortex are destroyed demonstrated changes in the central integrative state of the PVN towards potassium equilibrium potentials (inhibitory) to psychogenic stimuli.[xlv] Therefore, areas of the neuraxis that produce GABA and glutamate have influential roles on the activity and responsiveness of the HPA axis. Decreased activity and degeneration of these central structures have been postulated to play a role in disease states of aging and the responsiveness of the HPA axis.

There is strong support for the role the limbic system, or more specifically the hippocampus plays with suppressing exaggerated stress responses. Lesions of the hippocampus and limbic areas have shown to abnormally enhance the HPA response to psychogenic stressors.[xlvi] [xlvii] Researchers have concluded that disease in areas of the neuraxis that project to the PVN underlie HPA axis dysfunction.[xlviii]

Overall, the neurophysiologic data suggests that limbic stress effecter pathways rely through basal forebrain, hypothalamic, and brainstem neurons to modulate HPA axis activation. The hippocampus and prefrontal cortex decrease the excitatory output of the parvocellular cells of the paraventricular nucleus of the hypothalamus and therefore inhibit the HPA axis. In contrast, the pathways from the medial and central amaydaloid nuclei use sequential GABA synapses, indicating that they may excite the parvocellular cells of the paraventricular nucleus of the hypothalamus by disinhibition.

The role of serotonin (5-HT) to attenuate the neuroendocrine stress response is becoming better understood every year. Serotonin has demonstrated abilities to attenuate the stress response, or to act as a modulator on the physiological stress response. Serotonin's role as a modulator is mediated in part by 5-HT release in the dorsolateral periaqueductal gray (DLPAG) and in the rostral ventrolateral medulla (RVLM), an area that contains symphathexcitotory C1 andrenergic neurons. These serotonergic neurons are found in the lateral wings of the dorsal raphe nucleus (DRN). The release of serotonin in either the DLPAG or RVLM inhibits stress induced sympathetic activity.[xlix]

A study conducted demonstrated that discrete injections of 5-HT or 5-HT1A receptor agonists into the RVLM inhibited pre-existing hypertension.[l] Acute administration of serotonin reuptake inhibitors (SSRI) stimulates the hypothalamo-pituitary-adrenal (HPA) axis, whereas continuous treatment desensitizes it.[li] The adrenal 5-HT system regulates the stress-induced adrenomedullary catecholamine synthesis and is also involved with modulation of adrenomedullary Ang II AT1 and AT2 receptors, indicating a close interaction between the serotonin Ang II systems in the adrenal medulla.[lii]

The nucleus of the solitary tract (NTS) contains medullary catecholaminergic neurons which receives inputs from vagal and systemic signaling pathways and relays signals to visceral and central center.[liii] [liv]

It has been documented that peripheral inflammation can signal via systemic and visceral routs within the NTS to topographically organized, functional specific subpopulations of meduallary catacholaminergic and non-catacholaminaergic neurons that would be expected to have differential contributions to neuroendocrine and autonomic responses.[lv]

The central angiotensin system performs modulating control of the HPA axis via its influence on the PVN. It should be made clear that the brain Angiotensin II (Ang II) system, separated from and physiologically integrated with the peripheral, circulating renin-angiotensin system. Ang II is synthesized in the cortex and activates specific AT1 receptors to regulate thirst and fluid metabolism. AT1 receptors located in the subfornical organ, the hypothalamic paraventricualr nucleus, and the medial eminence are involved in the regulation of the stress response.[lvi] When peripheral AT1 antagonists were administered peripherally it demonstrated prevention of the sympathoadrenal and hormonal response to isolation stress, and prevented stress induced gastric ulcers.[lvii] Angiotensin II (Ang II) is a vasoconstrictive peptide that promotes water retention and vasoconstriction. There is a peripheral and central angiotensin response during stress. The peripheral Ang II system will increase blood pressure, heart rate, adrenal and sympathetic nerve catecholamine release during stress. The central Ang II system will express on the PVN medial eminence ANG II AT1 receptor sites and promote the synthesis of CRH and adrenocorticotropin (ACTH) and excite the central andrenergic centers of the neuraxis.[lviii]

Chronic stress appears to induce both direct and indirect effects on central neuromodulation. The well-known canonical feedback model of negative feedback coordination reflects acute stress. With acute stress glucocorticoids are released which then act to inhibit CRF and ACTH secretion. However, with chronic stress, the glucocoriticoid feedback mechanism responds differently. With chronic stress responses, even novel stressors facilitate exaggerated ACTH surges and CRF. This may be a result of loss of dampening responses from the neuraxis on the HPA axis. With chronic stress there also appears to be decreased expression of dopamine-beta-hydorxylase in the locus ceruleus.[lix]

Insulin appears to have profound influences on the glucocorticoid feedback mechanisms. In the presence of insulin and its stimulation by glucocorticoids, the response of the HPA axis may respond differently. In the presence of insulin, elevated glucocorticoids stimulate deposition of mesenteric fat, which inhibits the responsiveness of CRF neurons. When insulin levels are reduced, mesenteric and other fat stores are decreased, and the metabolic feedback loop signal is reduced and the CRF neurons regain responsivness.[lx]

Vasopressin has influences in modulating the central integrative state of the PVN and therefore participates in the neuroendocrine response to stress. The parvocellular neurons of the PVN of the hypothalamus co-express corticotrophin-releasing hormone and arginine vasopressin (AVN) and secrete a mixture of polypeptides into the portal vessels of the stalk-median eminence. The peptides reach the anterior lobe of the pituitary, where CRH and AVP potentiate each other's actions to release ACTH.[lxi]

Nutritional Modulation of Central HPA Activity

Phosphatidylserine (PS) supplementation has profound impact on central HPA axis modulation. It is important to note that the endogenous production of phosphatidylserine is a very difficult and energy consuming process. It requires the combination of L-serine, glycerphosphate and two fatty acids and the aid of methyl donors such as B-12, folic acid, S-adensylmethionine with essential fatty acids. Its arduous chemical synthesis that depends upon commonly deficient nutrients may explain why its exogenous intake has shown such great promise.

Exogenous supplementation of phosphatidylserine has shown the ability to enhance cellular metabolism and communication,[lxii lxiii lxiv] protect cells from oxidative damage,[lxv] decrease anxiety, improve mood, motivation and depression,[lxvi lxvii lxviii lxix] enhance memory and cognition[lxx lxxi lxxii] and decrease cortisol.[lxxiii lxxiv lxxv lxxvi] Perhaps the most clinically significant impact of PS is its ability to lower cortisol. An overactive hypothalamus-pituitary-adrenal axis that induces hypercortisolemia has many adverse impacts on healthy metabolism. The doses of PS required to support the HPA axis have been found to be between 800-2,000 milligrams a day. Therefore, it is unlikely to support this pathway effectively with oral supplementation.

The application of PS in intravenous delivery or liposomal delivery cream (Adrenacalm from Apex Energetics) would be preferred.

Ashwaganda has been shown to modulate central nervous system activities involving cholinergic, dopaminergic and GABAergic pathways.[lxxvii] [lxxviii] Ashwaganda has also demonstrated central modulation of adrenergic and prostaglandin production in physiological stress responses.[lxxix] [lxxx] Ashwaganda apparently has adatogen-like glucocorticoid activity which makes it so helpful in adrenal stress syndromes.[lxxxi] Studies have found that ashwagandha has similar adaptagenic activity to panex ginseng.[lxxxii] It also had the ability to counteract some of the adverse.

Holbasil leaf mechanisms of modulation of the central stress response have been noted to be due to its ability to modulate central dopaminergic influences.[lxxxiii] Holybasil leaf extract is an adaptagen that supports an increased sense of well being. Studies have shown that holybasil prevents the increase of plasma level of cortisol induced exposure to both chronic and acute stress, antagonized histamine, normalizes blood sugar, modulates HPA activity, increases physical endurance, has immunomodulatory activities and enhances gastric mucosal strength.[lxxxiv] [lxxxv] [lxxxvi] [lxxxvii]

Rhodiola appears to act on serotonergic, dopaminergic and adrenergic modulation of the central neuraxis.[lxxxviii] [lxxxix] Rhodiola is an adaptagenic plant that has demonstrated central nervous system enhancement, anti-depressant, anti-carcinogenic and cardioprotective properties. It has shown the ability to increase the swimming time of animals by 135-159 percent. The compounds in Rhodiola have shown the ability to prevent the stress-induced catecholamine activity, reduce adrenaline-induced arrhythmias in animals and prevent stress induced increases in cAMP and decrease cGMP in heart tissues of animals.[xc] [xci] [xcii] [xciii] [xciv] Rhodiola has also been shown to enhance cognitive function and mental fatigue, as well as support immune function.[xcv] [xcvi]

Panax ginseng is also known as Korean ginseng and is probably one of the most recognized stress adaptagens. It appears that ginseng enhances fatty acid oxidation during prolonged exercise by sparing muscle glycogen.[xcvii] The utilization of fatty acid metabolism over glycogen metabolism is an important role pannax ginseng plays in adrenal stress syndrome. If metabolism is shifted into a state that can conserve glycogen levels by mobilizing fatty acids, tremendous stress is taken off the adrenals and blood sugar metabolism. Pannax ginseng apparently influences metabolism so that an adequate supply of oxygen is available for working muscles which will make non-esterfied fatty acids the preferential form of energy over glycogen. Pannax ginseng has the ability to improve stamina, energy and physical performance. Apparently the compounds in Pannax ginseng improve the hypothalamus-pituitary-adrenal (HPA) feed back loop as well as reduce the suppression caused by cortisone on the immune system.[xcviii] [xcix] [c] [ci]

Boerhaavie diffusa has the ability to support both adrenal over and under activation. In stressful conditions it has demonstrated the ability to buffer the elevations of serum cortisol and prevent the suppression of the immune system that takes place with elevated cortisol. On the other hand, Boerhaavia diffusa has also demonstrated the ability to improve cortisol levels with end stage adrenal exhaustion.[cii]

Siberian ginseng also known as eleutherococcus senticosus is an adaptagen. Most of the studies on Siberian ginseng were conducted in the Soviet Union. These studies demonstrated enhanced athletic performance in animals as well as the ability to optimize HPA axis performance under stress.[ciii] [civ] [cv] Studies have also demonstrated that Siberian ginseng has the ability to enhance work output under stressful conditions and to improve mental and physical responses under stress. [cvi]

Neurological Integration of the HPA Axis and Applied Kinesiology

Standard muscle testing in applied kinesiology is based on a system in which different sensory evoked stimuli with temporal and spatial summation is provided to the nervous system. The impacts of sensory evoked potentials are assessed using volitional muscle testing responses. The motor responses of the neuraxis reflect changes in the central integrative state of the anterior horn. If the evoked sensory stimuli modulates the central integrative state of the anterior horn motor neuronal pools closer towards sodium equilibrium potentials then we may observe a conditionally facilitated muscle. If motor neuronal pools are brought closer towards potassium equilibrium potentials, then we may observe a conditionally inhibited muscle. Once again, the underlying premise for a muscle test response is dependent on an integrative system that has the functional capacity to respond predictably through canalized and non-canalized pathways.[cvii cviii]

Canalized pathways of the anterior horn, which is considered the final common pathway, include rubrospinal, vestibulospinal, corticospinal, reticulospinal, and tectospinal pathways. Ipsilateral modulation of the anterior horn also includes neocortex excitation of the pontomedullary reticulospinal tract which results in excitation of the anterior horn cells via inhibition of inhibitory interneurons termed dysinhibition. This dysinhibition pattern will allow for increased sensitivity and decreased gain of ipsilateral muscle spindles which increases the probability of large diameter afferent's proprioceptive excitation of the ipsilateral cerebellum and increased summative states of spinal cord reflexes. The pontomedullary rectiulospinal tract also excites anterior muscles above T6 and posterior muscles below T6. The net result of the integration of these pathways with neocortical and basal ganglionic systems results in the clinical motor responses we term as "strong or weak" or conditionally facilitated or inhibited muscles.[cix cx cxi]

The neurological system is modulated in such a way that one side of the neuraxis increases the function of the other side. The neocortex monsynatpically excites the pontomedullary reticulospinal tract which inhibits inhibition of motor neuronal pools and therefore leads to excitation of these neurons which include the gamma motor apparatus. Excitation of the gamma motor neurons increases the sensitivity of the ipsilateral muscle spindles which then increase the amount of proprioceptive bombardment of the ipsilateral cerebellum. The cerebellum then exicites the contralateral neocortex via the mesencephalon. Now the other neocortex monosynaptically stimulates the ponotmeduallary reticulospinal tract which increases the sensitivity of the ipsilateral muscle spindles, which increase the excitation of the ipsilateral cerebellum, which then excites back the opposite cortex via the mesencephalon again. Therefore, one side of the nervous system promotes the survivability of the other side.[cxii]

Applied kinesiology techniques are heavily centered on assessing pools of neurons of the motor system. However, many neurological and autonomic changes occur that are not clearly exhibited by muscle testing outcomes. In addition, the final common pathway of the anterior horn is influence by multiple canalized pathways and therefore its multisynpatic connections demonstrate less accurate responses to stimulatory input than pathways which represent singularity of projections.

Neurological stimulation always impacts the central integrative state of the entire neuraxis which include not only motoric responses, but autonomic, limbic, tectal, etc.

The vast majority of applied kinesiology techniques emphasize assessment of the patient by change exhibited from pre and post muscle testing challenges. The anterior horn is a multisynaptic site of numerous canalized pathways and therefore it may not be the best window to assess direct influences of the neuraxis, especially with pools of neurons that do not have direct projections into the anterior horn.

However, the goal of most applied kinesiology techniques developed have been to increase the central integrative state of the anterior horn toward a facilitated state due to the need of applied kinesiologist to improve gait, muscle tone and posture. The foundation of AK was based on conditions that caused society to seek chiropractic care such as musculoskeletal disorders.

When assessing the hypothalamus-adrenal-axis, one may be limited with its complexities by limiting assessment to only muscle testing. It is important to assess the entire neuraxis such as pathways that are pre and postsynaptic to the parventricular cells of the hypothalamus (PVN). Just as the anterior horn is referred to as the final common pathway of the motor system, the PVN is considered the final common pathway of the stress response. The central integrative state of the PVN, either towards sodium or potassium equilibrium potentials are mediated by multiple canalized pathways. These pathways include projections for neocortex, paleocortex, mesencephalon and cerebellum. The postsynaptic projections of the PVN may be seen in windows of integration of the mesencephalon, the intermediolateral cell column, the vagal nucleus, and the adrenal glands.

Several neurological integration pathways may influence the central integrative state of the parvocelluar cells of the paraventricular nucleus of the hypothalamus closer towards excitatory postsynaptic potentials that can lead to increased activity of the HPA axis. These include wind-up of the corticolenticulopallidostraitothalamocortical loop as a consequence of lesions (or decreased frequency of firing) of the cerebellar cortex which dampen purkinje output into the mesencephalon and nucleus of the solitary tract, decreased neocortical inhibitory projection into the deep cerebellar nuclei from canalized projections of the ventral tegmental tract, pontine nuclei and the inferior olivary nucleus. The possibility of specific degenerative changes which lead to neuronal apotosis in regions of the mesencephalon such as the substantia nigra and the nucleus raphe magnus also exist. These central wind-up patterns which bring the central integrative state of the mesencephalic neuronal pools towards exaggerated sodium potentials promote increased activity of the contralateral locus cereulus and and intermediolateral cell column which promote increased sympathetic tone. Suspicion of central wind-up of these pathways may demonstrate some characteristic physical examination findings. Mesencephalic integration that is closer towards excitatory potentials may exhibit increased pupil constriction activity that may present as cormeosis or as pupil plasticity which exhibits as quick response of the pupil with accompanying delayed hippus. Increased tectum activity of the mesencephalon may be seen with intolerance to sound and light. With these presentations, the patient may be intolerant to nasal light stimulation or tuning fork stimulation from the contralateral side. There may also be increased flexor tone on the contralateral proximal musculature and increased sympathetic tone.

Ipsilateral hemisphericity may also promote increased ipsilateral sympathetic tone due to diminished projections into the brain stem. Hemisphericity is a term used to indicate decreased frequency of firing of the neocortex. Hemisphericity may increase the activity of PVN from peripheral stimulation of catacholamines which cross the blood-brain barrier and promote activation of the HPA axis. Increased catacholamines generated from hemisphericity are a result of decreased presynaptic firing into the ipsilateral pontomedullary reticular formation which excites inhibitory projections onto the intermediolateral cell column. Increased sympathetic tone may demonstrate clinically as increased blood pressure when tested bilaterally and simultaneously, abnormal pulse amplitudes, skin discoloration, temperature changes, diminished oxygen saturation or tissue perfusion, hyperalgesia, hyperesthesia, and pallanesthesia; all on the ipsilateral side of the hemisphericity.

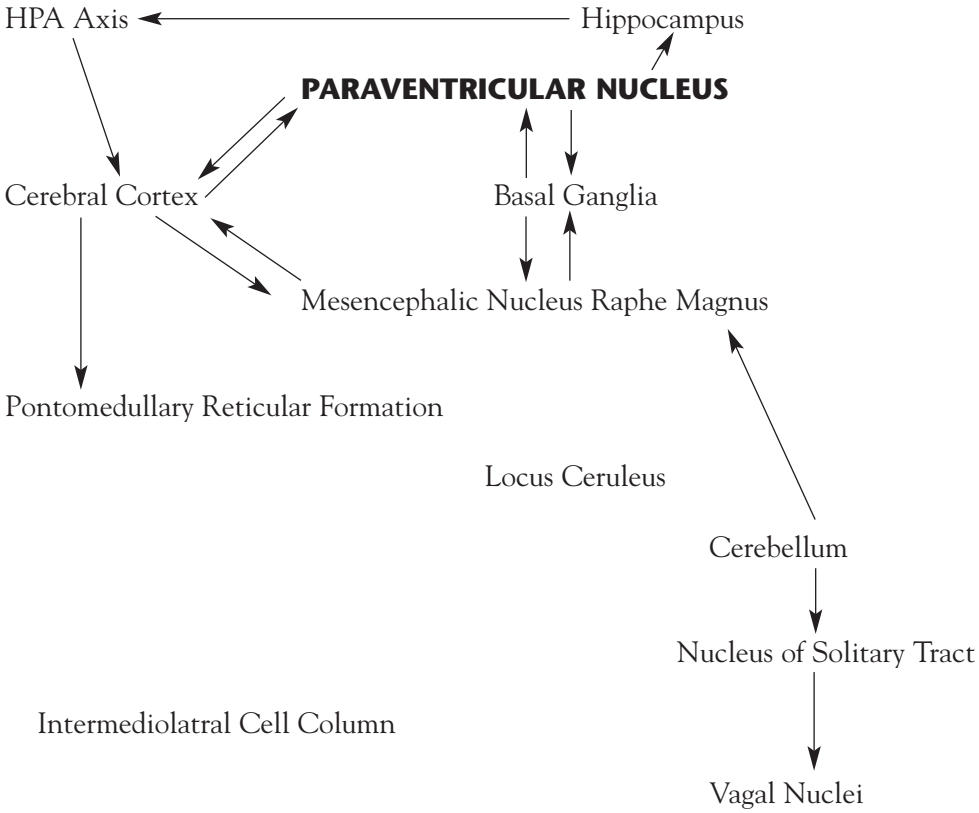
Many different types of applied kinesiology techniques have the potential to stimulate projections into the PVN and modulate the neuroendocrine-immune response to stress. However, the impact of the technique may be best determined by the type of stimulation provided within the basis of the individual's collective central integrative states of neuronal pools. When we look at the nervous system as a complex integrative unit it does not make sense to have so called "adrenal techniques" since any pathway that excites the PVN may have impacts on the HPA axis. Performing simple origin and insertion technique may have a far greater profound influence on adrenal function than taking adrenal supplements or stimulating an adrenal neurolymphatic.

An applied kinesiologist may perform the identical AK technique which demonstrates a change in the facilitatory output of the muscle, but the physiological responses may be different in every individual. The impact of any technique will be based on two criteria. First, which physiological pathways will be evoked with

the technique. Second, the patient's central integrative state of the neuraxis. For example, if you have an individual that has pools of neurons of the PVN that are towards potassium equilibrium potentials as a consequence of decreased cerebellar projections into the dentato-rubro-thalamo-hypothalmic projections they may have greater change in their HPA axis from an origin and insertion technique than from stimulation of an adrenal neurolympahtic. Another individual may have PVN pools of neurons closer towards sodium equilibrium potentials, with increased excitatory potentials and respond better to evoked stimuli of the nucleus of the solitary tract from visceral afferents evoked from massage of their ileocecal valve. Therefore, the impact of an evoked stimuli from any type of technique (neurovascular, emotional neurovascular, cranial, etc.) will depend on the individuals collective state of their neuraxis.

What may be considered a short-coming in standard applied kinesiology techniques are that, the determination of using an evoked stimuli is usually dependent on the influence of pre and post muscle responses. For example, let us say two different people have inhibited muscles which facilitate when they therapy localize a spinal segment. This facilitation may be due to generated proprioceptive projections into the anterior horn of the muscle that is being tested, and only takes into account the impact of such stimuli in pools of neurons related to that specific muscle. Now let us say, in one individual the mesencephalon is firing at a higher rate and manipulation of their spinal segment leads to increased output of the mesencephalon into the IML which leads to increased stress response, hypertension and ischemia. In another individual, the same stimulation increased the activity of their projections of the pontomedullary reticular formation to decrease the activity of the IML and decrease sympathetic tone. The point is, while both individuals responded to pre and post muscle testing challenges by facilitating, the impact on the neuroendocrine-immune axis is substantially different between them because the determination of the technique employed was based solely on one window of integration (anterior horn) instead of multiple windows of integration.

Diagram of Central HPA Axis Integration



Conclusion

In conclusion, the use of simplified protocols and models to support adrenal disorders are self-limiting. The understandings of physiological integration are important for management of chronic stress syndromes. A comprehensive examination should be employed to assess multiple pools of integration. Techniques that promote windows of singular integration should be discouraged. Applied kinesiology techniques must evolve with the advances made in the fields of human science in order to become an efficient and accurate method of diagnosis and treatment.

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Aerobic Muscle Weakness—A Case History

Robert A. Ozello, D.C., DIBAK

Abstract

A case history of aerobic muscle weakness is presented.

Introduction

The relationship between nutrition and aerobic muscle weakness has been long established.[i] Goodheart has written about the need for iron in these cases. I have also found that the patient may need fats to alleviate this condition.[ii]

Discussion

A 43-old woman presented with general muscle aches and fatigue. She had been suffering for several months.

Applied kinesiology examination revealed an aerobic muscle weakness. There were multiple muscle imbalances that were treated with proprioceptor technique and strain and counterstrain technique. Multiple subluxations and fixations were corrected.

Despite the structural corrections the aerobic weakness persisted. She was tested against iron which did not negate the aerobic weakness. Linum B6 from Standard Process Labs negated the aerobic weakness. I find that essential fatty acids more often negate the aerobic weakness. I find that iron rarely affects this condition.

The patient responded slowly. Within a month she stopped responding to the linum B-6. Other EFA supplements such as Cataplex F were tested with no response.

I came across a reference about saturated fats:

Saturated fats are needed for the proper utilization of essential fatty acids.
Elongated omega-3 fatty acids are better retained in the tissues when the diet is rich in saturated fats.[iii]
I had the patient fill out a seven day diet log. Analysis revealed a very low fat diet.

The next time the patient came to the office I was ready with butter and cocoanut butter and lard. All three of these foods negated the aerobic weakness. I then tested her against Cataplex A, G, E, D, chlorophyll, evening primrose oil, sesame seed oil, and fish oil.. There was no response.

I instructed the patient to eat liberal amounts of butter and cocoanut butter. She responded rapidly and within three weeks no longer had an aerobic weakness.

I have also found this nutritional need in several other patients.

Conclusion

Perhaps it is time to look at foods in the American diet that were present years ago and have been eliminated. Saturated fats may be one of these foods.

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Sciatica—A Case History

Robert A. Ozello, D.C., DIBAK

Abstract

A case history of sciatica and excessive nutritional supplementation is presented.

Introduction

It has been recognized that the intake of various foods and nutritional supplements can cause an adverse reaction in a patient. Often when a patient reacts there is a category I pelvic fault along with a temporal bulge cranial fault. Usually there is a piriformis muscle involvement.[i]

Discussion

A forty four year old man presented with low back pain, right buttock pain and severe right posterior leg pain. He was limping and was unable to walk on his right toes.

I had seen this patient several times in the past for other conditions. The last time I treated this patient he presented with right upper quadrant pain. I ascertained that his symptoms were due to an overdose of vitamins A, B, C and E. The patient was tested using applied kinesiology muscle testing techniques to determine food sensitivities. The supplement was placed in the patient's mouth. If a strong muscle weakened, the patient was sensitive to that supplement. Also abdominal tenderness increased markedly when the supplement was in the patient's mouth.

After correcting multiple muscle imbalances with proprioceptor technique and correcting multiple subluxations and fixations and removing the supplements from the patient's diet, he quickly responded. I told him to throw the supplements in the garbage.

At this time for the sciatica his orthopedic surgeon was considering surgery on a bulging disc. The patient had already been on a course of cortisone from which he had a bad reaction.

Applied kinesiology examination revealed category I pelvic fault with a right temporal bulge. I corrected these faults and corrected a right piriformis imbalance with NL technique.

He improved dramatically with some return of the symptoms on a subsequent visit. At that next visit the category I fault had returned. I then palpated his abdomen which was tender to the touch. This was similar to the past condition of abdominal pain I had treated him for.

I asked him if he was still taking the supplements. He said that he was, but only every other day. I informed him that the supplements were causing his problem and to immediately stop taking them. I corrected the category I fault. He stopped taking the supplements and the category I did not return on the next visit He quickly recovered with no return of the symptoms.

Conclusion

This is another example of my being surprised by a patient. He really didn't follow my original recommendation of discontinuing the supplements. Reducing the dosage caused different symptoms to express themselves.

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Division II

Critical Review

Comparison of Applied Kinesiology Neuromuscular Screening and Laboratory Indicators of Adverse Reactions to Foods

Katharine Conable D.C.[1], John Zhang, MD, PhD[2], Terry Hambrick, D.C.[3]

Abstract

Objective: To compare neuromuscular screening for adverse reactions to foods with laboratory markers of immediate and delayed hypersensitivity

Design: Masked comparison

Setting: Chiropractic College

Subjects: Volunteer sample of 30 chiropractic students and spouses with no history of severe allergic reactions to foods.

Methods: Fasting subjects gave blood samples and were tested for baseline state of 10 applied kinesiology (AK) neuromuscular indicators. Each food was placed in the subject's mouth in a masked manner and all AK indicators were retested for change. The mouth was rinsed between trials. Foods tested were egg white, dry milk, corn, soy flour and whole wheat flour.

Main Outcome Measures: IgE RAST and Lymphocyte Response Assay for 5 common foods and AK neuromuscular indicators for adverse reactions to those foods. A value exceeding either laboratory's reference range was considered positive for hypersensitivity. AK was considered positive if any indicator changed from baseline to an abnormal state.

Results: There was no significant correlation between adverse reactions found on AK and those found on laboratory testing. Kappa: egg white .229, soybean -.098, cow's milk -.222, wheat -.118, corn -.065.

Conclusions: AK oral food testing was not shown to identify the same hypersensitivities as the immediate hypersensitivities identified by IgE RAST or the delayed hypersensitivities identified by LRA. AK screening results should be further explored with other comparative methods before making a definitive determination about its application.

Introduction

An oral food challenge is used by applied kinesiology practitioners as a screening test for possible adverse reactions to foods. Versions of this procedure were well established in the AK community by the early 1980s[1, 2]

In an oral food challenge, the patient is asked to taste a small sample of the food in question and then examined for neuromuscular effects such as change of the functional state of a muscle from normal facilitation to inhibition, to an over-facilitated or non-responsive state or the appearance of one of several other physical indicators such as muscle testing response changes with patient digital contact to suspected problem areas ("therapy localizations").[3, 4] Such an adverse reaction is thought to include an immediate hypersensitivity reaction, delayed hypersensitivity reaction, toxicity, enzyme deficiency or other mechanism. According to the Board of Standards of the International College of Applied Kinesiology,[5] any apparent adverse reaction to foods suspected on the basis of an oral food challenge should be confirmed by laboratory testing or an elimination/challenge diet as clinically appropriate.

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Schmitt and Leisman[6] published a pilot study comparing laboratory indicators of allergy with muscle testing screening procedures and found that serum tests for RAST, IgE, IgG and immune complexes confirmed 90.5% of the food allergies suspected on the basis of muscle testing results.

Hermann and Drost[7] compared skin prick, serum IgE, serum IgG, salivary IgA from 5 different laboratories, "kinesiology," Vega testing and Carroll testing (both energetic tests) with an elimination/challenge test on a single subject. They calculated sensitivity, specificity and positive predictive value of each test and concluded that the "kinesiology" testing had a sensitivity of 50%, specificity of 33% and positive predictive value of 60%. The details of the "kinesiology" protocol were not included in the paper. Other weaknesses of the study included considerable time elapsed between the various forms of testing done.

Stegagno[8] presented a study of 364 hypertensive patients evaluated for food sensitivities based on postural deviations due to induced muscle imbalance as evidenced on a computerized quadrilateral weight scale before and after oral challenge with 30 different diluted foods. Seventy percent of the subjects who avoided or were successfully treated for the identified offending foods achieved at least 20/15 mm hg improvement in hypertension, with 30% of the total achieving normalized blood pressure within one year's follow up. Postural distortions and postural reactions to food challenges improved as well.

The intent of this study is to follow up on the Schmitt and Leisman study with a masked evaluation and to compare laboratory indicators of immediate and delayed allergy with applied kinesiology neuromuscular indicators of adverse reactions to foods.

This study compared applied kinesiology neuromuscular indicators of adverse reactions to 5 foods with serum IgE RAST[9] (testing performed by Quest Diagnostics) and lymphocyte response assay[10] (ELISA/ACT Biotechnologies LLC) – a measure of delayed hypersensitivity. Foods tested were wheat, corn, egg white, soy and cow's milk.

The hypothesis is that neuromuscular changes will be evident after oral food challenge when there is a positive laboratory indicator of food hypersensitivity and absent when there is no laboratory indicator of hypersensitivity. It is thought that some neuromuscular reactions occur due to non-immunologically mediated adverse reactions, so it is hypothesized that AK testing would appear to be less specific than the laboratory testing. In other words, A.K. would find most or all of the allergies found by the laboratories but might find some additional adverse reactions not seen on laboratory testing.

Materials and Methods

SUBJECTS

Thirty chiropractic students and student spouses were recruited to participate in this study by classroom announcements and posters. Fifteen males and 15 females ranging from 21 to 46 years old participated. Prior to the study, all subjects completed demographic questionnaires including inclusion/exclusion data. Those subjects who met the inclusion criteria completed an informed consent process and were given a detailed instruction sheet on preparation for the study.

INCLUSION CRITERIA

Subjects over 21 years of age, willing to participate and able to understand and follow preparations for the laboratory tests were considered for inclusion.

EXCLUSION CRITERIA

Subjects taking corticosteroid medications orally or topically or who had had a cortisone injection within 2 weeks of the study date were excluded due to potential interference with the laboratory test. No subjects met this exclusion criterion.

Subjects who medically could not avoid antihistamines or aspirin-containing compounds for 48 hours prior to the study date were excluded. Several subjects voluntarily discontinued antihistamines for 48 hours prior to the study and so were not excluded.

Subjects who were unwilling or unable to fast for 12 hours prior to the test or for whom it was medically inadvisable to fast were excluded. Water was allowed. No potential subjects were unable to or failed to fast. Subjects taking theophylline (aminophylline) were excluded. No subjects met this exclusion criterion. Subjects with known anaphylactic reactions to any food were excluded. No potential subjects met this exclusion criterion. One subject withdrew on the morning of the study.

Subjects were required to fast 12 hours prior to the test, avoid cosmetics and body lotions for 12 hours prior to the test and abstain from medications as listed in the exclusion criteria.

PROCEDURE

The study protocol was reviewed and approved by the Institutional Review Board of Logan College of Chiropractic.

Subjects reported to the study site and inclusion/ exclusion criteria were verified by inquiry. Blood samples meeting the requirements of both laboratories were drawn by an AMT & HEW certified Medical Technologist in the Logan College of Chiropractic Montgomery Clinic laboratory. Subjects then reported to a separate examination area for the A.K. examination.

Examiners were two Diplomates of the International College of Applied Kinesiology, each with over 20 years of clinical experience. Each subject was muscle tested bilaterally for pectoralis major, clavicular division, pectoralis major, sternal division, according to Kendall, McCreary and Provence[11] and Walther[12] and rectus femoris according to Walther.[13] A near-simultaneous style of manual muscle testing was used. Each subject was tested for respiratory change in the state of the first muscle,[14] and for muscle change while the patient touched (therapy localized)[15] the Chapman neurolymphatic reflex[16] for each muscle and while the patient touched acupuncture point Kidney 27[17, 18] bilaterally. If there was a change in the muscle test during respiration, all parameters were tested on both inspiration and expiration and both results noted. This was necessary for only one subject. After baseline neuromuscular status was determined, an assistant gave the subject a sample of powdered food to taste and then the examiner retested the same muscles and indicators. Following the first food, the subject rinsed his/her mouth with water and spit the water out. Each of 5 foods (whole corn flour, soy flour, whole wheat flour, dried milk, egg white powder) was tested in this manner. If any adverse reaction occurred, muscles were tested following rinsing the mouth to ensure that they returned to pre-challenge status before testing the next food. In no case did an indicator fail to return to baseline state. Following the AK testing, the subject was thanked and dismissed. Subjects were provided with copies of their laboratory results several weeks after the completion of data collection.

Food samples were pre-measured powders in numbered envelopes, prepared by an assistant who did not assist during the testing. All samples were obtained from a health food store. The soy, whole wheat and dried milk were labeled as "organic"; the corn and egg white were not. Neither examiner nor subject was informed of which food was which, although the subjects may have been able to identify some of the foods by taste. Subjects were instructed not to tell the examiners or assistants any speculation as to the nature of the foods. Examiners turned away while the food was placed in the subject's mouth to avoid seeing the appearance of the sample.

The majority of testing was conducted on 2 separate days for logistical reasons. Four subjects had to be re-tested due to damage to their blood samples during shipping to the Elisa/ACT lab. Each of these subjects was tested through the entire protocol the second time. Only the complete data from the retest was included in data analysis.

Laboratory tests were graded as positive or negative for hypersensitivity according to each laboratory's criteria for a result suggesting that the food should be avoided.

Applied Kinesiology testing was graded as positive for adverse reaction to the food if any of the following occurred:

- Any of the tested muscles became weak (inhibited) upon oral challenge.
- Any of the tested muscles became non-responsive to autogenic inhibition upon oral challenge. (Failed to weaken after pressure together in the belly of the muscle).
- Any therapy localization which was initially negative became positive upon oral challenge.

Statistical Analysis: SPSS 13.0 (Chicago) was used for Kappa analysis. A Kappa reading of greater than 0.7 was considered showing significant inter-method agreement and a reading below 0.4 was considered low inter-method agreement.

Results

A.K. Testing

Initially, all test parameters were normal (muscles strong and responsive to autogenic inhibition, no respiratory change of strength of muscles, no therapy localizations) for 22 of the 30 subjects. Five displayed 1 or more non-responsive muscles, one had therapy localization to acupuncture point K-27, 1 displayed a respiratory change in muscle tests and one showed both therapy localization to K-27 and a therapy localization to the Chapman reflex for the pectoralis major, clavicular division.

Ten parameters were tested for each food. The number of changes elicited by any single food varied from 0 to 8, with each muscle, right or left counted separately. (*see Table 1*).

Table 1.
AK Result

	Egg White Frequency	Soy Frequency	Wheat Frequency	Milk Frequency	Corn Frequency
no change	20	23	24	21	20
Weak	0	0	1	0	0
Over Facilitated	3	1	2	1	2
K-27 TL	1	0	0	0	2
PMC NL TL	0	0	0	0	0
PMS NL TL	2	0	0	0	0
Rectus Fem NL TL	1	0	2	1	2
normalized	0	3	0	2	0
multiple	3	3	2	5	4
Total	**30**	**30**	**30**	**30**	**30**

A.K. changes upon oral exposure to foods. weak = muscle became inhibited; overfacilitated = muscle became non-responsive to autogenic inhibition, K-27 = muscle test changed upon touching acupuncture point Kidney 27, PMC NL TL = touching the neurolymphatic reflex for the pectoralis major, clavicular division changed the muscle test of that muscle; PMS NL TL = touching the neurolymphatic reflex for the pectoralis major, sternal division changed the muscle test of that muscle; Rectus fem NL TL = touching the neurolymphatic reflex for the rectus femoris changed the muscle test of that muscle; normalized = abnormal indicators became normal; multiple = more than one type of neuromuscular reaction occurred.

Ten subjects showed changes in A.K. indicators upon tasting egg white. Two subjects displayed normalization of one indicator while others became abnormal: Subject 10: A non-responsive muscle became responsive (normalized) while TLs became positive to K-27, PMC NL and PMS NL. Subject 16: Several muscles weakened, one became non-responsive (abnormal) while one muscle which had been non-responsive became responsive (normalizing).

Seven subjects responded to soy, with 4 being counted as adverse reactions and 3 subjects having some indicators which had been abnormal becoming normal. There were no mixed responders to soy.

Six subjects reacted adversely to wheat; no indicators normalized.

Nine subjects reacted to milk with 7 adverse reactions and 2 isolated normalizing reactions. One subject had a mixed response to milk with one indicator normalizing (pectoralis clavicular which was initially non-responsive becoming responsive) and others becoming abnormal (1 muscle became non-responsive and 2 Chapman reflexes became positive).

Ten subjects reacted adversely to corn. One of these subjects had one indicator normalize (K-27 TL became negative) and others become abnormal.

Laboratory Testing

Immediate hypersensitivity testing by IgE RAST demonstrated from 1 to 6 subjects reacting to each food.

Lymphocyte response assay (LRA) testing showed delayed hypersensitivity responses for 0 to 2 subjects for each food. (*See table 2*)

Table 2.

	Egg White		Cow's Milk		Wheat		Corn		Soy	
	IgE	LRA	IgE	LRA	IgE	LRA	IgE	LRA	IgE	LRA
Reactive	3	2	3	2	6	2	1	0	1	0
Non Reactive	27	28	27	28	24	28	29	30	29	30

Laboratory results. IgE = IgE RAST test for immediate hypersensitivity (testing performed by Quest Diagnostics)
LRA = Lymphocyte response assay for delayed hypersensitivity (Elisa/ACT Labs)

Statistical Analysis

There was very little overlap between AK and lab estimates of adverse reactions to foods. For each food, there was a large proportion of subjects who tested negative by all methods. Three subjects tested positive to egg white by both A.K. and one or both of the labs. Wheat was positive on both lab and A.K. for one subject. There was no overlap for milk, soy or corn.

Kappa was non-significant for all comparisons: egg white .229, soybean -.098, cow's milk -.222, wheat -.118, corn -.065. (*See tables 3–7*)

Sensitivity and specificity and positive predictive value were calculated for each food, comparing the A.K. test to a positive result on either or both of the laboratory measures of hypersensitivity. (*See table 8*) Sensitivity ranged from 0 for soybean, cow's milk and corn to .6 for egg white. Specificity ranged from .66 for corn to .86 for soybean. Positive predictive value, which assumes that the prevalence of hypersensitivity seen in the study sample is representative of the population at large, ranged from 0 to .3.

Table 3.

Either Lab Egg White * AK Egg White Crosstabulation

Egg White		AK		
		non reactive	reactive	Total
Either Lab	non reactive	18	7	25
	reactive	2	3	5
Total		20	10	30

Comparison of A.K. result with combined lab result for egg white. "Either lab" = one or both labs identified the food as allergenic. A.K. = one or more A.K. indicators identified the food as reacting adversely.

Table 4.

Either Lab Soybean * AK Soybean Crosstabulation

Soybean		AK		
		non reactive	reactive	Total
Either Lab	non reactive	24	4	28
	reactive	2	0	2
Total		26	4	30

Comparison of A.K. result with combined lab result for soy. "Either lab" = one or both labs identified the food as allergenic. A.K. = one or more A.K. indicators identified the food as reacting adversely.

Table 5.

Either Lab Cow's Milk * AK Cow's Milk Crosstabulation

Cow's Milk		AK		
		non reactive	reactive	Total
Either Lab	non reactive	19	6	25
	reactive	5	0	5
Total		24	6	30

Comparison of A.K. result with combined lab result for cow's milk. "Either lab" = one or both labs identified the food as allergenic. A.K. = one or more A.K. indicators identified the food as reacting adversely.

Table 6.

Either Lab Wheat * AK Wheat Crosstabulation

Wheat		AK		
		non reactive	reactive	Total
Either Lab	non reactive	17	6	23
	reactive	6	1	7
Total		23	7	30

Comparison of A.K. result with combined lab result for cow's milk. "Either lab" = one or both labs identified the food as allergenic. A.K. = one or more A.K. indicators identified the food as reacting adversely.

Table 7.
Either Lab Corn * AK Corn Crosstabulation

Corn		AK		Total
		non reactive	reactive	
Either Lab	non reactive	19	10	29
	reactive	1	0	1
Total		20	10	30

Comparison of A.K. result with combined lab result for wheat. "Either lab" = one or both labs identified the food as allergenic. A.K. = one or more A.K. indicators identified the food as reacting adversely.

Table 8.
AK Test Characteristics Compared to Laboratory Measures

	Sensitivity	Specificity	Positive predictive value
Egg White	.6	.72	.3
SoyBean	0	.86	0
Cow's Milk	0	.76	0
Wheat	.14	.74	.14
Corn	0	.66	0

Sensitivity = proportion of true positives, considering the laboratory as the criterion standard. Specificity = proportion of true negatives. Positive predictive value = the likelihood that a subject with a positive AK test will have a positive laboratory test.

Discussion

While IgE RAST is relatively well accepted as a measure of immediate hypersensitivity reactions, there is no single gold standard laboratory test for delayed hypersensitivity. Elimination/challenge testing, particularly when done double-blind and repeated remains the best choice for verifying suspected adverse reactions to foods.[5]

Compared to these two laboratory measures, AK testing had very poor sensitivity and positive predictive value. In other words, AK testing did not identify many of the subjects who had positive laboratory evidence of either immediate or delayed hypersensitivity. AK testing did have relatively good specificity, or ability to identify patients who did not have laboratory indications of hypersensitivity. In other words, there was good correlation between AK testing and laboratory testing for those who are non-responsive to the foods being tested.

The current study did not achieve the level of correlation between AK indicators and lab values seen in Leisman and Schmitt's study. There are several possible explanations for this:

1. A different lab and somewhat different parameters were used for delayed hypersensitivity testing as the lab used by Schmitt and Leisman is not currently operating. Every effort was made to choose a lab which has stringent internal controls and clinically meaningful testing methods. However, this laboratory may well have been measuring somewhat different laboratory parameters than in the earlier study.

2. AK procedures were more extensive in this study. Schmitt and Leisman identified reactive foods as those which caused a previously intact muscle to become inhibited or test weak. The present study included several

muscles tested both for weakness and for a hyper-facilitated or non-responsive state as well as a number of other indicators commonly used in clinical practice to screen for adverse reactions to foods. This would have been expected to increase the number of adverse reactions identified by AK. Only 11 subjects of a total of 23 who reacted to any foods demonstrated a weakening reaction to a foods, therefore this sample was rather different than the sample in the Schmitt and Leisman study. It is not known whether these different parameters are more or less likely to correlate with IgE and IgG mediated food sensitivities. However, each of the 4 reactions in which AK and either of the labs agreed were "hyper" reactions, or one or more muscles becoming non-responsive to autogenic inhibition. The numbers are so small that this may well be coincidence.

3. The Schmitt and Leisman study did lab testing only on subjects suspected of adverse reactions/ allergies according to AK testing. It did not examine subjects who did not test positive on AK testing. No masking procedure is described in the paper. The present study included a volunteer sample who may or may not have had any clinical symptoms of allergies or food sensitivities. They may have self-selected to some degree toward a more allergic population as the recruiting posters and informed consent process did identify the study as addressing adverse reactions to foods. Nevertheless, the resulting sample had a large proportion of subjects who were identified as non-reactive to most or all of the foods by most of the tests. In the present study, examiners were masked as to which food was being tested at which time, although examiners knew which 5 foods were being tested. Subjects did not know which foods were being tested although they may have been able to identify some by taste.

4. In this study, all subjects were fasting for a minimum of 12 hours prior to testing. Blood was drawn for lab testing prior to exposure to food samples. In the Schmitt and Leisman study there is no mention of whether the subjects were fasting. In the Schmitt and Leisman study, subjects were exposed to food samples in the course of A.K. testing shortly before blood was drawn for lab testing. While it is unlikely that the brief exposure during the AK test would be adequate to elevate immunoglobulin levels, recent meals might have.

The fact remains that there was very little overlap between reactions identified by AK means and those identified by either laboratory. Why this is so is the key question brought up by these results. Various explanations include AK being totally random and invalid, the AK examiners in this study being at fault but the method being valid, the labs being invalid, and methods each measuring different parameters.

AK adverse reaction screening has been used clinically for over 2 decades with many doctors seeing sufficiently useful results to continue the practice. It is certainly possible that they may all be fooling themselves, but that is not the only possible explanation. The neuromuscular manifestations and changes seen on the study were quite apparent to testers, subjects and assistants.

Both testers were Certified Teaching Diplomates of the International College of Applied Kinesiology each with over 20 years experience using these methods in clinical practice. They had drilled the study procedures prior to the study and attempted to use consistent procedure throughout. Based on the published study[19] indicating increased reliability of muscle testing between examiners with greater than 5 years of experience, the examiners in this study should be representative of average or better competence in the A.K. community.

AK may well be picking up on non-immunologically mediated reactions, cell-mediated reactions or allergic reactions earlier in the response before immunoglobulins had developed. AK might also be picking up habitual responses related to taste or associations other than purely physiological adverse responses.

A related, carefully designed study[20] involving AK testing of dental materials for intolerance showed poor test-retest reliability with the same examiner orally testing materials in a double-blinded condition compared with an open test. While not testing foods and not necessarily postulating an immunologically mediated mechanism for any adverse reactions to the dental materials, this study uses similar methodology to the present one and must be taken into consideration when evaluating the utility of this form of testing.

Further examination of the clinical relevance of the neuromuscular changes seen on AK oral food challenge should include a comparison with an elimination/challenge diet with adequate masking. A comparison of symptomatic change in patients who eliminate foods according to an AK test compared to those who eliminate foods according to a lab test would also help to clarify when each of these screening procedures would be most clinically applicable.

Conclusion

AK oral food testing as done in the present study does not appear to identify the same thing as the immediate hypersensitivities identified by IgE RAST or the delayed hypersensitivities picked up by LRA. This confirms the position of the Board of Standards of the ICAK that manual muscle testing screening results need to be further explored with other indicators such as labs and elimination/challenge diet before making a definitive diagnosis. The physiological mechanisms and clinical meaning of the neuromuscular changes seen on oral food challenge are fertile fields for further research.

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Restless Legs Syndrome: A Case Series Report

Scott Cuthbert, D.C.

Abstract

Objective: To present a case series report on the successful treatment of Restless Legs Syndrome (RLS).

Clinical Features: 5–15% of the population has been reported to suffer from RLS. A retrospective analysis of 28 patients is reviewed who presented with numerous complaints and symptoms that also had RLS as part of their clinical picture. The nature of RLS is described, hypothetical causes of the condition reviewed from the literature, and medical treatment for the condition is presented. The nutritional value of riboflavin, niacin, and vitamin B6 are described, and their relevance to RLS proposed.

Intervention and Outcome: Standard AK methods of diagnosis and treatment were employed in all cases. Common to all the cases of RLS successfully treated (n = 23) in this patient cohort was the positive AK oral nutrient testing for a portion of the vitamin B complex (riboflavin, niacin, and B6). With supplementation of these factors, the patients' RLS resolved completely or were noticeably improved. 5 patients who were treated using this protocol did not improve their RLS with this treatment.

Conclusion: Further investigation of these methods of treatment and larger patient cohorts in controlled clinical trials would be of value with concurrent biochemical, EMG, and observational sleep monitoring of the patients treated. RLS is a cause of severe insomnia with costs to both the individual and to society at large.

Key Indexing Terms: *Restless Legs Syndrome; Nocturnal Myoclonus Syndrome; Clinical Trials; Vitamin B Complex; Riboflavin; Niacin; Vitamin B 6; Vasodilator Agents; Diagnosis; Treatment; Chiropractic; Kinesiology, Applied*

The treatment of insomnia, when successful, is one of the most satisfying clinical successes in my practice. "How are you sleeping?" is one of the questions I will often ask new patients. I have found over the years that if my patients cannot get a good night's sleep, they are not going to fully recover their health.

Insomnia is a term used to describe the more than 80 million Americans who routinely have trouble falling asleep or staying asleep. For anyone who has ever experienced a few sleepless nights in a row, a feeling of desperation sets in as you struggle to function during the following day. Close to 40% of the U.S. population over the age of 15 reports they've experienced insomnia at least occasionally.[1]

It is likely that more than 100,000 motor vehicle crashes are caused annually in the United States by driving while drowsy. Major disasters such as Three Mile Island, *Exxon Valdez*, Bhopal and *Challenger* were all officially attributed to sleepiness-related impaired judgment in the workplace.[1]

Restless legs syndrome (RLS) is one of the most common causes of severe insomnia. It is a neurological sensory/movement disorder affecting 5–15% of the general population.[2] A survey conducted in May 2005 of some

1,500 adults reports almost 10 percent of the respondents suffered from restless legs syndrome, and more women than men have it.[3]

RLS is characterized primarily by a vague and difficult-to-describe unpleasant sensation in the legs. This discomfort appears primarily during periods of inactivity, particularly during the transition to sleep in the evening.

Patients often have difficulty in describing the unpleasant sensations; they rarely use conventional terms of discomfort such as 'numbness, tingling or pain,' but rather bizarre terms such as 'pulling, searing, drawing, crawling, shimmering or boring,' suggesting that RLS sensations are unlike any experienced by unaffected individuals. These distressing sensations are typically relieved only by movement or stimulation of the legs.

The Restless Legs Syndrome Foundation puts the condition this way:

> ***"Restless Legs Syndrome is an overwhelming desire to move the legs usually caused by uncomfortable or unpleasant sensations in the legs. The sensation can occur during periods of inactivity and become more severe in the evening and night. RLS may often cause difficulty staying or falling asleep, which leads to tiredness or fatigue. Up to 8% of the US population may have this neurologic condition. Many people have a mild form of the disorder, but RLS severely affects the lives of millions of individuals."*** **(http://www.rls.org)**

Many different techniques have been found by patients for relief: walking about, stomping the feet, rubbing, squeezing or stroking the legs, taking hot showers or baths, or applying ointment, hot packs or wraps to the legs. Although these treatments are effective while they are being performed, the discomfort usually returns as soon as they become inactive or return to bed to try to sleep. The motor restlessness often appears to follow a circadian pattern, peaking between midnight and 04:00.[4] The prevalence of depression and anxiety found associated with RLS is felt to be secondary to the RLS. The relationship between RLS and insomnia generally and psychiatric conditions are bi-directional: depression may cause insomnia, and insomnia may cause depression.[5]

The movement can happen just at night, when the jiggling may drive a spouse or significant other up the wall, and/or it can occur during the day, when it will drive everyone else up the wall with the incessant tapping or twitching. In a small percentage of people, the sensations of restlessness and twitching may also be experienced in the arms. The automatic jerks of the legs can disturb their sleep enough to produce a feeling of fatigue the morning after but not enough to fully awaken them. The sleeper's mate, however, is usually fully aroused by their partner's unintended kicks. Both of these people will be in your office asking about this condition together, and the tired expression on their faces and the circles around their eyes can be a clue for you to investigate this problem in a patient.

Many patients with RLS take tranquilizers, muscle relaxers, and over the counter sleep aides to get them to sleep. But most people who use these medications never go into deep restorative sleep, the deep delta-wave sleep that allows the body to get its physiological rest and restorative repair.

One of the most obvious and immediate effects of RLS and its associated insomnia is the increased risk of accidents. As reported in *Business Week*, "Studies show that someone who has been awake for 24 hours has the same mental acuity as a person with a blood alcohol level of 0.1, which is above the legal limit for driving in most states." But when you consider someone who is a health care worker, pilot, or law enforcement officer, the effects can be catastrophic. Some 39% of health-care workers report that they've had a "near miss or accident" at work due to fatigue in the last year. Further, sleep disorders cost the nation about $45 billion every year in lost productivity, health care and motor vehicle accidents.[6]

Recent studies suggest there may be a susceptibility gene locus in RLS, which would explain why RLS is often familial. [7] RLS is commonly seen in pregnancy, hemodialysis or peritoneal dialysis for renal failure and iron-deficiency anemia. Its relationship with iron metabolism abnormalities has led to studies indicating that RLS is associated with abnormal iron metabolism within the central nervous system.[8]

Studies relating this problem with nutritional deficiencies of riboflavin, niacin, and B6 have not been explored in the literature. A search of Pub Med showed 10,789 papers on riboflavin; 5,591 papers on niacin; 5,968 papers on niacinamide; and 9,070 papers on vitamin B6. A series of searches was performed, using the terms "restless legs syndrome," "nocturnal myoclonus," "riboflavin," "niacin," "niacinamide," and "vitamin B6." None of these papers correlated these vitamins with RLS.

One of the conclusions of this study must be a new nutritional relationship between RLS and these three vitamins.

Recent functional neuroimaging studies have identified thalamic, red nucleus and brainstem involvement in the generation of periodic limb movements in patients with RLS. One PET study found reduced dopamine 2 binding in the caudate and putamen. A subcortical origin of RLS is supported by transcranial magnetic stimulation studies and the successful treatment of the patient cohort in this case series, **i.e. *RLS has physiological causes***.[9]

Demonstration of a continuous hypersensitivity to pinprick, but not to light touch, confirms a sensory component to RLS and may explain the efficacy of AK, nutritional, and opiate medications in RLS.[10] Patient's with RLS will often report that walking, rubbing or massaging the legs, or doing deep knee bends or finger to floor stretches can bring relief, but only briefly. This suggests that there is a structural component to RLS as well.

Medical Treatment of RLS

Medical doctors will employ a variety of medications to treat RLS. Generally, they choose from dopaminergics, benzodiazepines (central nervous system depressants), opioids, and anticonvulsants. Dopaminergic agents, largely used to treat Parkinson's disease (like Sinemet and Levodopa), have been shown to reduce RLS symptoms and are considered the initial treatment of choice. Good short-term results by treatment with levodopa plus carbidopa have been reported, although patients usually develop augmentation, meaning that symptoms are reduced at night but begin to develop earlier in the day than usual. Dopamine agonists such as pergolide mesylate, pramipexole, and ropinirole hydrochloride may be effective in some patients and are less likely to cause augmentation. The dopaminergic anti-Parkinsonian medications are a particularly common treatment medication now.[7]

Benzodiazepines (such as clonazepam and diazepam) may be prescribed for patients who have mild or intermittent symptoms. These drugs help patients obtain a more restful sleep but they do not fully alleviate RLS symptoms and can cause daytime sleepiness. Because these depressants also may induce or aggravate sleep apnea, they should not be used in people with this condition.

For more severe symptoms, opioids such as codeine, propoxyphene, or oxycodone are prescribed for their ability to induce relaxation and diminish pain. Side effects include dizziness, nausea, vomiting, and the risk of addiction.[11]

Anticonvulsants such as carbamazepine and **gabapentin (Neurontin)** are also considered useful for some patients, as they decrease the sensory disturbances (creeping and crawling sensations). Dizziness, fatigue, and sleepiness are among the possible side effects.

The medical and drug company literature shows that no one drug is effective with RLS. What may be helpful to one individual may actually worsen symptoms for another. In addition, medications taken regularly may lose their effect, making it necessary to change medications periodically.

For one of the patients in this study (age 68 F, see *Table 1*), Sinemet produced urinary incompetence and systemic muscle weakness as well as blurred vision, light-headedness and depression. These kinds of side effects from the medications our patients are taking are not uncommon in my experience. 6 of the patients treated successfully with the applied kinesiology protocols were taking Sinemet and Levodopa (for Parkinson's disease), or muscle relaxers, sedatives, or painkillers for control of this condition. The use of Sinemet and Levodopa was short-lived for them however.

[I was amazed anew when I realized that several of the patients in this case series reported that they had been given drugs designed for the treatment of other diseases (Parkinson's disease and seizures) for the treatment of their RLS. These are examples of a worrisome tactic of the Pharmaceutical Companies (Big Pharma) called "Off-Label Marketing," that will be discussed in another paper in this volume.]

[See following paper.]

On May 5, 2005, the FDA approved the first ever drug for treatment of restless legs syndrome: ropinirole (Requip).

The side effects of this "great advancement in medical therapy for RLS" are announced:

Possible side effects

SIDE EFFECTS that may occur while taking this medicine include feeling of warmth, dry mouth, sweating, weakness, fatigue, dizziness, drowsiness, lightheadedness, stomach pain, heartburn, gas, nausea, or vomiting. If they continue or are bothersome, check with your doctor. CHECK WITH YOUR DOCTOR AS SOON AS POSSIBLE if you experience swelling of ankles, feet, or hands; unusual fatigue or tiredness; unusual pain; unusual muscle movement; loss of appetite; fast or irregular heartbeat; falling asleep during daily activities; mental or mood changes; impotence; trouble breathing; sore throat; or vision changes. CHECK WITH YOUR DOCTOR IMMEDIATELY IF YOU EXPERIENCE fainting, or chest pain. An allergic reaction to this medicine is unlikely, but seek immediate medical attention if it occurs. Symptoms of an allergic reaction include rash, itching, swelling, severe dizziness, or trouble breathing. If you notice other effects not listed above, contact your doctor, nurse, or pharmacist.

Drug interactions

Drug interactions can result in unwanted side effects or prevent a medicine from doing its job. Use our drug interaction checker to find out if your medicines interact with each other. ADDITIONAL MONITORING OF YOUR DOSE OR CONDITION may be needed if you are taking ciprofloxacin, medicines for anxiety (such as diazepam), medicines for mental or mood problems (such as risperidone), medicines for depression (such as fluoxetine), digoxin, theophylline, levodopa, estrogens, phenothiazines (such as chlorpromazine), butyrophenones (such as haloperidol), thioxanthenes (such as thiothixene), or metoclopramide."

The cost of Requip, the great advancement in medical therapy for RLS is $219.96 for 100 tablets (approximately 2 months supply).

AK treatment for RLS

A better, safer course of action would be to treat the true causes of the problem, meaning the abnormal neuro-physiology going on in the patient with RLS.

There is no laboratory test that can identify RLS, and the condition cannot be diagnosed by a medical physician other than by symptoms reported by the patient. For us this should strongly suggest a functional problem (one of the 5-factors of the IVF) is the root cause of the problem.

In each of the cases successfully treated here, pelvic subluxations and weakness of pelvic and leg muscles on MMT were found. Frequently, these patients were hypoadrenic, and several were obese with an anterior sacral base subluxation that increased the lumbo-sacral lordosis. Each of these cases responded to chiropractic adjustments and Cataplex G ™ [12] vitamin supplementation, and this element of treatment was the common factor among all the cases successfully treated (n = 23) for RLS.

Table 1.

Restless Legs Syndrome Patient List (n=23)

Patient Age and Sex	Problems with Insomnia? (I) Adrenal Stress Disorder? (ASD) Low Back or Leg Pain? (LBLP)	Intensity of RLS: • Severe • Distracting • Moderate Duration of RLS pre-treatment?	Treatment time until resolution of RLS (days)	Did RLS return? (# Months): Maintenance dose of Cataplex G needed? (Yes: No)
80; M	I, LBLP	D: 7 years	12	12: N
50; F	I, ASD	D: (lifetime)	7	17: Y
63;M	LBLP	S: (lifetime)	15	N: N
61; F	I, ASD	M: (3 years)	13	N: Y
77; F	ASD, LBLP	D: (10 years)	4	Every 6 months: Y
69; F	ASD, LBLP	S: (10 years)	8	N; N
68; F	I, LBLP	S: (lifetime)	11	Every 4 months: Y
49; F	ASD	S: (10 years)	11	Every 2 months: Y
64; F	I, ASD	D: 5	6	N: Y
62; F	ASD, LBLP	M: (12 years)	8	N: N
68; F	I	D: (6 years)	7	N: N
47; F	ASD	D: (lifetime)	10	N: N
57; M	LBLP	M: (20 years)	40	N: N
44; F	ASD	S: (10 years)	9	N: N
80; M	LBLP	S: (2 years)	12 (This patient resolved his RLS with Cataplex E2)	N:Y
73; M	LBLP	D: (1 year)	5	Every 4 months: Y
61; M	ASD, LBLP	D: (3 years)	2	Every 4 months: Y
70; F	ASD	S: (2 years);	4	N: Y
54; M	LBLP	also in arms D: (5 years)	24	N: N
66; F	I, ASD, LBLP	S: (10 years)	15	N: N
63; F	LBLP	D: (lifetime)	4	Every 2 months: Y
80; M	I	S: (10 years)	28	N: Y
93; M	LBLP	S: (6 months)	3	N: N

Since 2001, the author has treated 28 patients with RLS who presented for chiropractic treatment for a wide variety of other problems. Of the 23 patients successfully treated for RLS, only 2 came to the clinic specifically for treatment of their RLS.

As part of a thorough whole-body examination, most of the muscles in the body were tested, and the relationship of muscular inhibition on MMT to the patients' primary complaints was explained. Because AK examinations and treatments are interactive, the patient is educated about the relationships between the different areas of the body and how one dysfunction may be creating problems elsewhere. During the course of treatment of patients who presented with more conventional chiropractic problems, the irritation of the RLS was revealed.

The commonality among all the patients with RLS was the finding of facilitation on MMT of inhibited muscle(s) in the legs with oral nutrient testing of Cataplex G (SP). Since discovering the consistent success of AK evaluation and treatment and Cataplex G supplementation for RLS after so many years, I decided to present this case series report.

I have been very impressed by the relationship between Cataplex G supplementation and the speedy resolution of RLS in patients. It appears that Cataplex G is almost a "specific" for RLS in the patients I have treated.

Another important part of the successful treatment of RLS in these patients was the reduction of pain in the legs that was present in 14 of the patients listed above (listed as LBLP in *Table 1*). Treatment of this factor by standard AK methods to the 5-factors of the IVF, including cranial, pelvic category, vertebral, and muscular corrections to the structures found disturbed in the patient proved successful in reducing the low back and leg pain in these cases. In terms of the structural factors, there was no one recurring "specific" I found in my review of these cases with RLS. The heterogeneity of the chiropractic presentation – each patient showing unique and peculiar features – was the rule here.

8 of the patients who had problems with insomnia (sometimes a major or minor factor in their lives before the onset of RLS), were treated using standard AK evaluation procedures. The patient would be tested in a closed room with the lights off, and the factor that corrected the global inhibitions on MMT with the lights out would be treated.

Additionally, patients who only had trouble falling into sleep often responded to supplementation with Calcium Lactate(SP); patients who had trouble staying asleep and awoke frequently without cause often responded to Cataplex B(SP); patients with depression, irritable bowel syndrome, and insomnia often improved with 5-HTP(NW) supplementation.[13] (5-HTP should be taken with 4 ounces of fruit juice, as insulin is required to carry the 5-HTP past the blood brain barrier.) These have been consistent findings in my practice, and were a part of the improvement in the overall picture of some of these patients who had the RLS also.

12 of the patients in this case series report showed signs and symptoms of adrenal stress disorder (postural hypotension, paradoxical pupillary reaction, Rogoff's sign, weakness of the sartorius, gracilis, or tibialis posterior muscles that showed strengthening upon TL to the relevant reflex, the ligament stress reaction, etc.). In my own practice, Drenamin(SP) is frequently used for support of the hypoadrenic patient, and interestingly Drenamin is a combination of adrenal glandular tissue, vitamin C and vitamin G. It is possible that those patients with adrenal stress disorder and RLS were receiving the Cataplex G supplement in two forms.

The vitamin "G" complex includes riboflavin, niacin, B6, folic acid, PABA, choline, inositol, biotin, and betaine.

According to Dr. Royal Lee in *Vitamin News* from 1934 and 1952; Dr. George Goodheart in his articles on vitamin B deficiencies; Dr. Wally Schmitt in *Compiled Notes on Clinical Nutritional Products* (1990); and Dr. Philip Maffetone in *Complimentary Sports Medicine* (1999), the signs of a vitamin "G" deficiency include:

1. Cardiovascular — tachycardia, extra ventricular beats, increased 1st heart sound with a long silence after 1st sound (increased time of diastolic ventricular filling), angina pectoris, and pre-myocardial infarction
2. Psychological — Excessive worry, apprehension, moodiness, depression, suspicion
3. Digestive — Insufficient stomach acid production and excess alkalinity, spastic gall bladder
4. Liver — Cirrhosis of the liver and loss of fat metabolism activity, deficient formation of Yakitron, a physiologic anti-histamine
5. ***Neurological*** — Insufficient acetylcholine activity and cholinesterase activity (for breaking down acetylcholine and for recycling choline), **restless, jumpy, or shaky legs, body or limb jerks upon falling asleep**, can hear heartbeat on pillow
6. Skin and mucous membranes — Cheilosis (cracking at corners of mouth), friable skin, especially on face and neck (when shaving), bright red tongue tip, strawberry tongue (purple), loss of upper lip (thin upper lip), irritated mucous membranes of the rectum, vagina, and conjunctiva (frequent tears), excessive oil on face and nose, roughness, cracking and exfoliation of the soles of the feet, and psoriasis
7. Visual — Burning or itching of eyes, photophobia, blepharospasm, blood shot eyes due to capillary engorgement, seeing only parts of printed words (circumcorneal injection), pallor of the temporal half of optic disc, transient ischemia of retina – like looking through a fish bowl
8. Endocrine — excess estrogen and menstruation, cystic mastitis or gynecomastia, premenstrual tension, and excessive adrenal function.

Dr. Royal Lee, one of the great chroniclers of deficiency signs and symptoms in relation to vitamins and minerals, listed the following deficiency signs for the B vitamins that make up the Cataplex G formula.[12] These might help explain why Cataplex G was of value in the cases of RLS reported upon here:

Signs and symptoms for vitamin B2 (riboflavin) deficiency:
- a. Myelin degeneration
- b. Incoordination
- c. Loss of strength in arms or legs
- d. Central neuritis, symptoms resembling degeneration of spinal cord

Signs and symptoms for vitamin B3 (niacin or nicotinic acid) deficiency:
- a. Myelin degeneration (motor and sensory)
- b. Headaches, dizziness, insomnia, depression and impairment of memory
- c. Burning hands and feet
- d. Pain in calves
- e. Numbness and weakness of extremities
- f. Difficulty in walking
- g. Absent knee jerk

Signs and symptoms for vitamin B6 (pyridoxine) deficiency:
- a. Severe sensory neuritis
  - i. Numbness and tingling in hands and feet
  - ii. Hyperesthesia

In general, Cataplex G has the physiological effect of relaxing nerves and thereby acts as a vasodilator upon the vascular system, helping some individuals with their hypertension and smooth muscle spasm.

In the early investigations of the vitamin B/G complex, the investigators called vitamin B/G an anti-neuritic vitamin. Many neurological syndromes and specific nerve dysfunctions have been related to inadequacies of each of the B/G-vitamins.

All nerves require B/G vitamins for normal function. Pyridoxine (B6), riboflavin (B3), and folate are especially important. Supplementation should also include B12, biotin and pantothenic acid.

As an example of this fact: inositol has been used in Europe for the past 30 years to lower cholesterol and improve nerve function in diabetics.[14, 15] Because of its role in cell-membrane function, it has also shown beneficial effects for depression and general neurological function.

Pyridoxal phosphate (vitamin B6) has been implicated as critical in lipid metabolism because its deficiency causes myelin degeneration in man.[16] Impairment of temperature perception is present in a high percentage of RLS patients, and the sensory deficits are at least in part caused by small nerve fiber neuropathy.[17]

Practically all of the neurotransmitters in the brain are metabolized with the aid of vitamin B6, including dopamine, norepinephrine, serotonin, GABA, histamine, acetylcholine, insulin, growth hormone, follicle-stimulating hormone, luteinizing hormone, aldosterone, glucagon, cortisol, estradiol, testosterone, and epinephrine.

Vitamin B6 is also required for the conversion of tryptophan to niacin. Serotonin, a critical factor for normal sleep patterns, is derived from pyridoxal-5 phosphate and 5-hydroxytryptophan, and so another possible improvement in nighttime RLS may have an explanation.

Since dopamine is synthesized with the aid of vitamin B6, and dopaminergic drugs like levodopa and dopamine agonists are the first line of medical treatment choice in idiopathic RLS, the effect of Cataplex G may have another corroboration. However with the use of dopaminergic drugs, augmentation and rebound phenomena are consequences of this type of medical treatment, and must be carefully monitored in long-term treatment.

Niacin has long been used for cardiovascular conditions, especially those involving lipid metabolism. It may also help Raynaud's disease (excessive blood vessel constriction due to cold and symptoms of intermittent claudication) caused by insufficient blood supply to the calf muscles while walking.

According to Carlton Fredericks, Ph.D, "Niacin restores the electrical charge to red blood cells so they don't aggregate and thereby are able to pass through small blood vessels in single file." Deficiency of niacin has been reported to cause weakness, dry skin, lethargy, headache, irritability, loss of memory, depression, insomnia, delirium, and disorientation. Unless the nerve damage or degeneration has gone too far in cases of RLS, the administration of these nutritional factors for healing of the nerve provided quick relief.

Most of the B vitamins, particularly thiamine and riboflavin, are involved in energy production. Folic acid, B12, B6 and niacinamide all contribute to DNA repair and synthesis.

The B vitamins also play an important role in immunity. This is because with immune activation during an infectious invasion, trillions of white blood cells and other immune cells must be mobilized. All of these immune cells have a very high metabolic rate that greatly increases their requirement for the water-soluble B vitamins. Once again, these include riboflavin (B2), pantothenic acid (B5), pyridoxine (B6), folate and cobalamin (B12). We also know that during major infections these vitamins are rapidly depleted.

They are also depleted during intense or chronic stress (and that RLS is a chronic and often intense stressor will not be denied by anyone who has the condition). The importance of this factor is demonstrated by the B vitamin formulation (primarily Cataplex G) in the adrenal gland supplement Drenamin.(SP)

Numerous studies have shown that the elderly (18 of the patients in this study were members of the 'senior set') are usually deficient in the nutrients contained in Cataplex G, as well as others.[18]

Further study of the relationship between dopamine, central nervous system and vitamin B metabolism will be of great interest and value to neurophysiologists, neurochemists, neuropharmacologists and, of course, our patients with RLS.

The scientific literature supporting nutritional supplements is extensive and goes back over 100 years. Throughout my years practicing and studying the art and science of chiropractic and applied kinesiology, I have expanded my understanding of the importance of nutritional treatment in conjunction with conventional treatment modalities. Pain reduction is one aspect of our responsibility to our patients; helping them prevent disease, recover from disease, and achieve their optimum health is another. Frequently, nutritional supplementation is required. In the 23 patients with RLS successfully treated in this report, nutritional supplementation was a critical factor.

I hope that this paper will encourage you to discover whether or not your patients with sleep problems have RLS, and if they do you will try this method of treatment with them.

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Balancing Acupuncture Meridians to Treat the Mental Side of the Health Triangle Using Injury Recall Technique

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Abstract

This paper introduces the use of injury recall technique[1] to balance acupuncture meridians to treat various emotional components of the mental side of the health triangle which affect the chemical and structural sides and interfere with the healing process(s).

Introduction

Applied Kinesiology was discovered and developed in chiropractic. It was the first technique to use a practical treatment of the mental side of the health triangle which would affect itself and the chemical and structural sides of the triangle. Beginning with holding the neurovascular emotional points to numerous other techniques developed over the years pioneered by John Diamond, MD[2] and Roger Callahan, Ph.D[3], meridian therapy has now emerged to a number of various therapies called Energy Psychology. Injury Recall Technique discovered and developed by Walter H. Schmitt Jr., DC, adds a new, faster method of eliminating emotional problems.

Discussion

In 1981 Roger Callahan, Ph.D,[4] , developed what he called, *The Five Minute Phobia Cure*. He presented a paper at the winter meeting of the ICAK at in Key West, FL demonstrating that tapping on a specific acupuncture beginning or end point would eliminate most phobias in five to fifteen minutes. At that time he was using only the Stomach and or Spleen meridians. He subsequently wrote a book by the same name published in 1985 by Enterprise Publishing Co, which this author read and successfully applied the technique in an office situation. It soon became apparent that using additional meridians would help patients more specifically with other things in addition to phobias such as Issues and Negative life beliefs. I wrote a book, *Freedom From Fear Forever* which included the twelve organ meridians published by Van Ness Publishing in 1995. Simultaneously, Scott Walker, DC developed Neuro Emotional Technique[5] At first he used the twelve organ meridians and latter added the governing and conception meridian with their respective emotions. The treatment method was using TBM adjusting sequences for the various organs that were associated with the emotion that the patient had.

After treating a patient in my office for a physical problem which the injury recall technique was used and explained, the patient asked to be treated to help her stop smoking. I used the regular tapping method having her think of smoking which inhibited a strong muscle and TLing the beginning or end points of each meridian until I found one which facilitated the muscle and began to have her tap on the points bilaterally. She then asked me that if the use of the foot (talus) flick which removed the memory of the injury, could be used to treat the smoking addiction? I accepted her suggestion and used it.

Method

This was done by having her TL the same meridian points bilaterally TL'd which were earlier determined when thinking of smoking. Instead of tapping on these points she was instructed to hold the points while the talus was adjusted in the injury recall technique. After the procedure was done she released her desire to smoke.

Conclusion

While this only gave here a temporary period of not smoking did as tapping does, (I have found that tapping procedure takes a lot of repetition for any kind of addiction) I started applying the same procedure to other emotional components of physical problems as indicated using NET procedures, eliminating phobias anxieties, negative life beliefs and life issues. So far the talus adjustment (IRT) while holding the acupuncture meridian beginning or end point has been just as successful as the tapping procedure and takes less time.

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Additional Therapy Localization Procedures to Uncover the Emotional Side of the Triad of Health

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Abstract

The emotional side of health is extremely important in balancing a patient. Various methodologies have been developed in applied kinesiology and elsewhere to help find and correct this particular cause of a patients symptomatology. Three additional procedures are presented to help uncover the emotional causes of body dysfunction.

Introduction

The introduction of therapy localization in 1974 has been of tremendous contribution in both understanding and diagnosis of body dysfunction. The Bennett reflexes for the stomach/bladder are also known as the emotional neurovascular reflexes (ENV). Either direct therapy localization to these points or using a two handed therapy localization; that is one hand to another reflex, vertebrae, and or joint, and cross therapy localization to the emotional neurovascular points will allow entrance into the emotional side if involved. Treatment may consist of holding the ENV's until a synchronous pulse is felt. Other methods may include encoded memory technique, Calahan technique, N.E.T., Homeopothy and various other protocols.

Additional therapy localization procedures have been discovered by this author that uncover covert emotional causes when standard therapy localization may fail to show involvement. These include a two handed therapy localization to the emotional neurovasculars, a cross therapy localization to the emotional neurovasculars (right hand on left neurovascular and left hand on right neurovascular), and a prayer position therapy localization to the metopic suture (similar to sacral wobble therapy localization).

Discussion

The Bennett reflexes for the stomach/bladder have long been utilized as the window into the emotional side of the triad of health. These reflexes were first discovered in the 1930's by Terrance Bennett, D.C. When these points therapy localize it is assumed that an emotional component is involved. They are contacted and held until a bilateral synchronous pulse is palpated.

These reflexes may be two pointed (one hand on another reflex, vertebrae, joint/muscle, etc. and the other hand on the ENV's) to determine if there is an emotional component to the problem. One may become more specific by having the patient think about finance, romance, him/herself in some particular role in life for more specificity.

However there have been times when the ENV's would not therapy localize either in the clear or by two pointing or by thinking a stressful thought and yet it seemed apparent there was an emotional involvement. Further research uncovered the following three patterns.

The first pattern has negative therapy localization in the clear either palm down or palm up with either hand. However if the patient places one hand on top of the other very often this displays a positive therapy localization. Structural corrections may include occiput, atlas, the first thoracic, and/or Category I which may not show in the clear until the two handed therapy localization to the ENV's. Extremity subluxation correlations may include the radial head, jammed carpals, fibular head and talus. Find the associated feeling involved (finance, romance, role playing) and correct the subluxation via encoded memory technique.

The second pattern also has negative therapy localization to the ENV's either palm up or palm down. However if the patient places the left hand on the right ENV and the right hand on the left ENV a positive therapy localization occurs. Common spinal correlations include T 10/11, L 5, and Category I. Extremity findings may include the radial head, jammed carpals, femur heads, tibia/fibula/patella, and talus subluxations. Again use the encoded memory technique. Biochemically a balanced mineral product may be useful.

The third pattern involves a particular type of therapy localization utilized in the sacral wobble fault. The hands are placed in the prayer position on the metopic suture. If a positive therapy localization is obtained in this manner then look for an occiput, sacral/pelvic, and/or upper thoracic involvement. Extremity subluxations may include a talus. Again utilize an encoded memory technique type of correction. Biochemically similar to the previous pattern, a balanced mineral product may be helpful.

Conclusion

These new therapy localization patterns to the ENV's allow access to the emotional side of the triad of health previously unobtainable by other procedures. These include the two handed therapy localization with one hand on top of the other; a cross therapy localization with the left hand to the right ENV and the right hand to the left ENV; and finally a prayer position of the hands to the metopic suture area. Correction involves utilizing the encoded memory technique to the appropriate axial/appendicular skeletal correlations. These corrections enable the body to function in a more balanced manner.

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The Upper Thoracic Subluxation/ Fixation Complex as a Common Cause of a Category II Pelvic Fault

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Abstract

A category II pelvic fault is an osseus subluxation of the ilium in relation to the sacrum. There are various structural, chemical, and emotional causes for this lesion. A common structural cause is an upper thoracic/rib head subluxation/fixation complex.

Introduction

The category II pelvic fault is a common structural finding with patients in a clinical setting. Many varied symptoms may be associated with this subluxation. Utilizing a two handed therapy localization (T.L.) to the sacroiliac joint involved and then cross therapy localizing to the upper thoracic/rib head area often will negate the positive category II findings. Osseous correction of the upper thoracic/rib head subluxation manually will then correct the category II.

Discussion

The category II pelvic fault was first described by Dr. DeJarnette (founder of sacro-occipital technique). It was later incorporated into applied kinesiology by Dr. George Goodheart. This is one of three pelvic category lesions first described by Dr. DeJarnette.

Category II is an osseous misalignment of the ilium to the sacrum. There are two basic types; a flexion malposition (PI-Gonstead terminology) and an extension malposition (AS-Gonstead terminology). The two subtypes are external (ex) and internal (in).

A flexion malposition involves disassociation of the upper portion of the sacroillic joint (the amphiarthrotic section) with possible superficial edema. The upper sacral surface is concave while the corresponding illic portion is convex. When the upper portion dissociates, the normal biomechanical listing is PIEX (Postero Inferior-External) following normal joint biomechanics. However if the joint is traumatized, then a PIIN (postero inferior and internal) is possible; going against normal sacro iliac biomechanics. Three dysfunctional muscles are commonly associated with a PI ilium; sartorius, gracilis, and the rectus femoris on the ipsilateral side. With a PI ilium flexion of the hip joint is easy while extension is difficult. This subluxation of the ilium corresponds to an ipsilateral short leg. Symptoms may be found anywhere in the body, in a structural, chemical, and emotional basis.

An extension malposition involves disassociation of the lower S/I joint (the diarthrotic section) with possible deep edema. Rarely pain may radiate into the ovaries or testicles. The lower portion of the sacral side is convex while the iliac portion is concave. Therefore when this portion is involved the ilia moves internal making the normal biomechanical listing ASIN (Gonstead terminology). If the S/I joint is traumatized then

the listing would be ASEX indicating abnormal joint biomechanics. Two common dyskinetic muscles are the gluteus maximus and/or the hamstrings ipsilaterally. With an AS ilium extension of the hip is easy while flextion is more difficult. An AS ilium corresponds to a long leg ipsilaterally. Symptoms may be found anywhere in the body on a structural, chemical, and or an emotional basis.

The two subtypes of a category II are ex and in. Ex is an external disassociation of the ilium in relation to the sacrum. The muscle association is an ipsilateral gluteus medius. IN is an internal disassociation with the ilium in relation to the sacrum and the muscles involved are the obliques.

Therapy localization involves a one handed contact to the S/I joint in question being careful to touch the entire joint. Traditionally the patient is in the supine position. Therapy localization may require the palm up or down position. However many times a category II is not revealed in this manner but is still present. Flexion of the cervical spine with or without rotation may often uncover a covert category II. Likewise, a weight bearing posture such as sitting or standing may be employed. Therapy localization to the S/I joint in a forward/backward gate position is also necessary sometimes. (As well as lateral flexion or rotation of the spine).

Once a category II has been identified, the S/I joint may be challenged on a rebound basis to determine the subluxation listing. The arm-fossa test (S.O.T.) may be utilized as well as medial or lateral knee tenderness. These procedures help to determine the osseous correction required.

Correction of the category II may be performed with a high velocity thrust with the patient in a side posture or prone utilizing a drop cable (Thompson). A non-high velocity protocol (S.O.T.) may be done with the patient supine laying on wedge shaped blocks. Proper correction may be verified if the therapy localization, challenge, leg length, knee tenderness, associated muscles and arm-fossa test are all negative post therapeutic effort.

As with any correction in the body, a question often asked by the practitioner and patient alike is; how long will that last? If after a category II correction have the patient run in place, run up and down stairs, chew, swallow, think a stressful thought, and or ingest a food substance and then recheck the category II findings, many times stressing the body will cause a recidivism of the pelvic fault.

Utilizing a two handed cross therapy localization, one hand on the sacroiliac joint, and one hand on the upper thoracic area often times this will negate positive therapy localization of the category II. This may involve a subluxation/fixation of the upper thoracic/rib head complex. One may also utilize a temporal tap procedure to determine if indeed this is the primary structural cause of the category II. It is this author's observation that well over half of the category II faults are in actuality due to this upper thoracic/rib head subluxation/fixation complex. After manual osseous correction of the subluxation/fixation area, recheck the category II and if negative then restress the patient structurally, chemically, and emotionally to determine if indeed the therapeutic attempt holds. In a vast majority of cases (assuming the osseous correction was performed properly) it does, demonstrating that this is where the cause originated.

Conclusion

A category II pelvic fault may be compensatory to other factors involved in the body on a structural, chemical, and emotional basis. A common structural cause is an upper thoracic/rib head subluxation/fixation complex which is easily demonstrated via a two handed therapy localization and temporal tap procedure. Proper manual osseous correction of this subluxation/fixation complex will ultimately correct the category II pelvic fault if this is the cause.

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The Role of Low Level Laser Therapy in Modulation of Neuromuscular Spindle Function With Emphasis on Temporomandibular Joint Applications

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Abstract

Temporomandibular Joint (TMJ) dysfunction is a common finding in clinical practice. TMJ dysfunction may cause secondary imbalance in cranial, cervical, pectoral and other spinal structures. Additional neurological consequences, especially via cranial and upper cervical nerve effects add to the complexity of presenting patient symptomology.[1] The science of Applied Kinesiology includes many effective interventions to improve TMJ function, including treatment of the neuromuscular spindles (NMS) of muscles of mastication. Treatment, especially of the internal and external pterygoid muscles, may at times prove difficult for the clinician and painful for the patient. This paper discusses the effect of low level laser therapy (LLLT) on neuromuscular spindle function and presents an application to TMJ NMS therapy that reduces difficulty and virtually eliminates any pain associated with the procedure.

Key Words: TMJ, temporomandibular joint, low level laser therapy, LLLT, neuromuscular spindle, NMS

Introduction

Identification and treatment of temporomandibular joint dysfunction is a common and important clinical procedure in most applied kinesiology practices. The powerful effect of masticatory muscles on cranial structures via attachments to sphenoid, zygomatic, occipital and temporal bones make TMJ balance essential for any lasting correction of cranial problems and the myriad of musculoskeletal and autonomic effects of poor cranial function. Adverse effects of TMJ imbalance conveyed via the trigeminal nerve add to the overall importance of TMJ diagnostic and treatment protocols.[1]

There are many techniques for improving TMJ function. This paper will focus on one of the most common approaches, treating hypertonicity of TMJ muscles via attention to the neuromuscular spindle (NMS). During this paper the author will use the following conventions in reference to the results of manual muscle testing: A conditionally inhibited muscle will be referred to as "weak" and a conditionally facilitated muscle will be referred to as "strong."

Discussion

Standard applied kinesiology (AK) tests[1] for TMJ NMS function involve having the patient touch or therapy localize (TL) the TMJ just below the zygomatic arch near the mandibular head. The doctor then requests the patient to go through a sequence of masticatory functions while the doctor tests a previously strong muscle (PSM) during or after each movement. Weakening of a PSM while or after the patient lightly or forcefully occludes the teeth suggests a need for therapy to the masseter, buccinator, temporalis, or internal pterygoid muscles. Weakening with the mouth half open suggests need for therapy to the external pterygoid

or anterior digastric muscles. Weakening with lateralization of the mandible and ipsolateral TL suggests therapy to the temporalis on the side of TL. Weakening with lateralization of the mandible and contralateral TL suggests therapy to the internal pterygoid on the side of TL.

Therapy is usually accomplished by pressing together over the belly of the muscles indicated to "turn down" the over-firing NMS. This is fairly easy to do with the masseter, buccinator and temporalis muscles but more difficult with the internal and especially with the external pterygoid muscles. NMS Therapy to the pterygoid muscles is usually accomplished by simply rubbing with one finger over the muscle belly.

While the above therapy is effective it is sometimes difficult and painful for the patient, especially pterygoid muscle treatment. Intra-oral treatment may result in pain for the doctor as well if younger patients decide to retaliate while the doctor's finger is still in their mouth! The search for improved patient (and doctor) outcomes led me to investigate low level laser therapy (LLLT).

Low level laser (aka "cold") therapy as used in this paper refers to lasers with an aperture output of less than one watt. This includes class I, II, IIIa and IIIb lasers as regulated by the FDA. Low level and "cold" lasers have an output low enough that they do not cause heating of the tissues which differentiates them from the higher wattage "hot" lasers used in laser surgery.

LLLT has gained popularity over the last several years for a variety of applications. A search of the National Library of Medicine, using "LLLT" as a keyword, turns up over 700 articles on a wide range of metabolic topics. A previous article by this author relates the value of LLLT in nerve regeneration as well as symtomology associated with vagus related digestive impairment.[2] One of the areas of metabolic application referenced in several articles relates to myospasm.[3, 4, 5] Since the purpose of NMS therapy for TMJ dysfunction is to correct hypertonicity of the muscles of mastication, it seemed appropriate to investigate LLLT applications.

Materials and Methods

There is a wide array of laser equipment currently available. A perusal of almost any chiropractic publication that accepts advertising will turn up many laser advertisements. Most of the advertised equipment runs several thousand dollars. I have a unit I'm quite fond of that cost $6500. Although my more expensive equipment allows a wider range of applications, for my TMJ investigation I decided to try a laser that would be affordable by any doctor. The unit I chose is a laser pointer. The best one I have found to date is available from www.laserpointer.net . It has the most precise 635 nanometers (nm) wavelength rating I have found. It is also the brightest laser pointer I have tried to date.

I found that application of laser energy to the belly of the masticatory muscles yielded results similar to more traditional manual therapy. In addition to providing an additional treatment option, I discovered several advantages that have made laser therapy my treatment of choice for TMJ muscle NMS therapy.

I'm sure most of us have encountered the pain, sometimes extreme, that patients experience during manual NMS therapy to the TMJ muscles. This is especially true of the pterygoid muscles. LLLT to the TMJ muscles is completely pain free. Some patients, especially children, are uncomfortable with intra-oral therapy. I have a personal policy to respect the right of children to say "no" to invasive procedures. LLLT offers two alternatives to manual intra-oral therapy. If the patient is willing to open their mouth, laser therapy may be applied by shining the laser directly on the belly of the internal pterygoid or into the pterygoid pocket for the external pterygoid. Even if the (usually young) patient is unwilling to open their mouth, therapy is still possible by taking advantage of the ability of laser energy to travel through soft tissue. By angling the laser slightly posterior from just anterior to the masseter, the internal pterygoid can be treated externally. The external pterygoid muscle can be treated from the exterior by angling the laser sharply posterior from just anterior and inferior to the mandibular head.

Laser TMJ Analysis and Treatment to the NMS

I. Analysis
   A. Find a previously strong muscle (PSM) to use in testing, preferably a lower extremity muscle such as gluteus medius or tensor fascia lata.
   B. Have the patient TL to the TMJ, just below the zygomatic arch
      1. Right hand to right TMJ
      2. Left hand to left TMJ
   C. Ask patient to activate various TMJ muscles
      1. Clench teeth, test PSM
         a. Weakening indicates involvement of masseter, buccinator, temporalis, internal pterygoid
      2. Open mouth, test PSM
         a. Weakening indicates involvement of the external pterygoid, anterior digastric muscles
      3. Lateralize mandible and test PSM
         a. Weakening indicates involvement of temporalis on side of positive TL or internal pterygoid on side opposite TL
      4. Protrude mandible and test PSM
         a. Weakening indicates involvement of internal pterygoid, anterior temporalis
      5. Retrude mandible and test PSM
         a. Weakening indicates involvement of posterior temporalis
   D. Treatment
      1. Use upper extremity PSM such as pectoralis clavicular for further testing
      2. For positive test above, discover which hand yields positive TL = side to treat
      3. As patient maintains positive TL, shine laser on suspect muscle
         a. Cancellation of positive TL (restoration of strength to muscle weakened by TL) indicates proper treatment location
            (1) masseter or buccinator belly
            (2) temporalis belly
               (a) anterior, middle and posterior sections
            (3) anterior digastric belly
            (4) internal pterygoid belly
               (a) internal treatment through open mouth
               (b) external treatment directed 45° posterior from just anterior to masseter
            (5) external pterygoid belly
               (a) internal treatment through open mouth to pterygoid pocket
               (b) external treatment directed 70° to posterior from just anterior and inferior to mandibular head
      4. Maintain laser output at treatment location for 20 seconds
         a. Laser may be applied directly to skin for external treatment
   E. Repeat positive test as above to verify correction

Conclusion

Low level laser therapy has proven to be a useful addition to or replacement for standard manual treatment to the neuromuscular spindles in TMJ therapy. Advantages include elimination of patient pain and discomfort that may be associated with manual therapy as well as the ability to treat pterygoid muscles externally, avoiding intra-oral treatment if desired. LLLT has become my treatment of choice for TMJ NMS therapy. The elimination of manual treatment to the pterygoid muscles is also appreciated by my patients. In my clinical experience, the effectiveness and longevity of LLLT treatment equals or exceeds manual therapy.

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Diagnostic Test To Find Adhesions Between Muscles

David W. Leaf, D.C.

Abstract

The treatment of adhesions between structures in the body has been a part of standard treatment since at least the 1930's. Since then many treatments for this condition have been created. The problem is knowing when to apply the various treatments. This paper presents a simple diagnostic sign that indicates the presence of adhesions between muscles or their tendons.

For the past five years, George Goodheart, D.C. has been using a procedure known as pincer palpation to find structural problems of muscles. Basically, a muscle is pinched and a strong muscle on that side of the body is tested for weakening. He developed this procedure from the work of Travell. She originally described pincer palpation in her volume I of her text.

Goodheart added in the muscle test to make it more objective than subjective.

In conversations with Michael Leahy, D.C., founder of ART (Active Release Technique), I pointed out that there was no diagnostic procedure for the use of his procedures. In ART, therapy is applied to stretch and release adhesions or cross-links between muscles, articular capsules, tendons and ligaments. I discussed with him the possible use of muscle testing as a diagnostic procedure. I showed him that placing the median nerve under torque by rotation of the arm, pronation of the forearm and extension of the wrist would uncover entrapments that would normally be missed. The movie file here shows the positioning done to put the arm in a position to stress the nerve.

This is accomplished by first contracting the scalenes, bring the arm into extension to aggravate a thoracic outlet syndrome, the stretching the biceps, fully pronating the forearm and hand and then extending the wrist. The second picture shows the end position that the arm should be in to stress the median nerve.

The problem was there where were still entrapments. Leahy had a lengthy list of possible places where the median nerve could be entrapped.

Using the pincer palpation of Goodheart did not uncover all of the problems that Leahy demonstrated. It was found that by applying pressure against a muscle, or muscle section, at 90 degrees, (perpendicular) to the line of the muscle fibers would at times cause weakness of the muscle being tested.

Linear (perpendicular) pressure applied to a muscle as it contracts should not cause weakening of the muscle, Muscles must be able to adapt to external forces that do not confine their expansion during contraction.

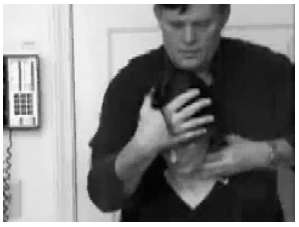
Using this diagonal stress to the muscle as a testing procedure quickly identified those muscles that were restricted in their normal motion by cross-links. The accompanying video picture shows testing the extensor carpi radialis while diagonal pressure is applied causing weakness of a strong muscle.

Using the example of the median nerve, the arm is placed in a position to stress the nerve and the opponens pollicis is tested. The various muscle areas that can be involved are then tested with linear pressure against the muscle being held by the examiner or by the patient and the muscle is retested. Therapy is then applied to those muscles that are found to weaken under linear pressure.

Example

In a case of retro-patella pain, it was obvious that there were problems in the firing of different sections of the quadriceps. The tests to isolate the sections of the quadriceps were used but showed no abnormal findings. The muscle sections were then retested with linear pressure being applied at a 90-degree angle to the line of the muscle fibers. This elicited a weakening effect in different sections of the muscle. These sections were then palpated for the presence of cross-links or adhesions. Therapy was then directed at releasing these adhesions and the muscle then retested.

The patient was then asked to bend and twist and reported that the knee was now more free.



This same technique has been used for shoulders, forearms and especially in cervical problems.

A common finding is restriction in motion between the three scalene muscles. This can quickly be determined by applying linear pressure against one of the scalenes while performing the regular muscle test. The video movie file here shows the testing of a section of the scalene while directional pressure is applied. This is done to a muscle that has been tested and found strong and you suspect either a decreased range of motion or entrapment of a nerve or blood vessel.

This linear pressure does not cause global weakness or one-sided weakness. The pressure to the muscle causes only weakness of the muscle being tested.

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Use Of One Of The Latest Laser Therapy Devices In Treating Complications Of Chronic Injuries

David W. Leaf, D.C.

Abstract

Four cases will be described using a cold laser in improving the function of patients with chronic problems from a severe Pot's fracture, a patient who had unsuccessful ACL reconstruction, a case of RSD of the lower extremity and a patient with severe hip socket degeneration.

The following cases were treated with a cold laser with the following specifications:

"Laser parameters: 910 nm pulsed laser producing a peak power of up to 40 W and an average power of up to 250 mW. Pulse repetition rates up to 50,000 Hz. ….thus delivering laser photons deep into the tissue with a high precision probe. Treats specific tissues and areas at 4"+ depth."

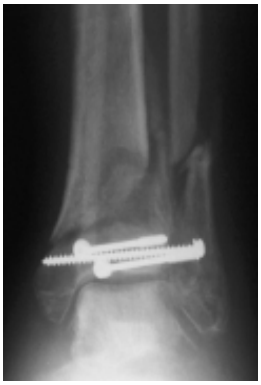
Discussion

Case I

The patient, at age 53, fell suffering a compound fracture of the left leg. There were eleven fracture sites. The tibia was protruding 2 inches through the leg with the foot at an 80-degree angle after the fracture.

The fracture was treated using an external fixator for 12 weeks. Following removal of the fixator, therapy was begun slowly.

In the beginning, severe atrophy of the leg had occurred to a point where it was impossible for the patient to raise his weight using the gastrocnemius and the soleus muscles.



Initial therapy included ultrasound, interferential current, percussion, and laser therapy with a dual diode unit and pool therapy. After one-month exercise using a BowFlex was added.

The strength in the leg improved over the first 8 months with the patient being able to support his weight on the leg at that time.

However, the patient was unable to twist and turn on the ankle or to run more than three steps without severe pain.

This condition continued and stabilized at this level for five years.

This past September, the leg was again evaluated. There was residual swelling of the lower leg that worsens as the day progresses. The patient was still unable to run or twist on the leg as in dancing.

Therapy was begun using a different laser. According to the manufacturer, this laser is able to deliver the energy into the deeper structures of the body. Therapy sessions were 10 minutes in duration with a total of 42 joules of energy. The probe was moved around the leg at 20-second intervals. During the treatment, the patient was involved in active PNF motions while the laser therapy was administered.

The results of this change in therapy were dramatic. The patient was able to dance for 90 minutes after two treatments and after 8 treatments began to run on a treadmill.

The improvement in the ability to run has increased to 20 minutes with no adverse reactions.

Case II

This 45-year-old female was an active tennis player who injured her knee skiing. The injury included a complete tearing of the ACL on the right. She underwent surgery to repair the ligament.

The patient was seen three months after surgery and completion of the standard physical therapy. The major complaint was a failure of the knee to bend past 30 degrees and a limp when she walked. The knee stayed in slight flexion at the mid-stance of the gait.

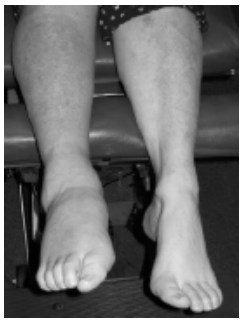
Adhesions were found in the quadriceps, patella tendon along with shortening of the gastrocnemius and the hamstring muscles. These were treated with a combination of massage, percussion, and reactive muscle – spindle cell corrections. This increased her range of motion to 50 degrees. However the limp in her gait was not markedly improved.

Laser therapy was then employed to the knee for 10 minutes with a total of 40 joules of energy. The probe was aimed at the popliteal space and from the anterior both medial and lateral to the lower pole of the patella. While the laser was being used, the patient performed resisted PNF. After the first treatment the knee improved to 80 degrees. By the third treatment, the knee was bending to 120 degrees. After two treatments, she was able to ambulate normally with short strides. By the fifth treatment, she was able to begin jogging normally for short distances.

She was then able to begin a strengthening program and is now again playing tennis.

These two cases demonstrate the use of laser therapy when applied with AK procedures being an effective addition in the treatment of chronic injuries.

Case III



This 58-year-old female has RSD of the right lower leg. This condition has existed for 5 years. The shoe size was 9 on the left foot and 11 1/2 on the right. The foot and lower leg were extremely discolored from light pink to dark pink on the foot. Palpation of the lower leg was extremely rigid almost the density of wood. On the first treatment, she was unable to lace her sneaker on the right foot due to the swelling.

Laser therapy was then employed to the lower leg and foot for 15 minutes with a total of 48 joules of energy. The probe was moved every 20 seconds starting proximally and moving distally. After 5 minutes, the patient was asked to perform resisted PNF motions of dorsiflexion and plantarflexion for 1-minute intervals every other minute while the laser was used. After the first treatment, the swelling had reduced so that the patient could lace the shoe.

After 5 treatments, the skin color had returned to normal, the foot had decreased in size to only 1/2 size larger than the left and the tissue was more like a minor pitting edema except for a small area under the metatarsals.

Case IV

This 48 year old, physically active female has severe hip degeneration. When seen, she was scheduled for a hip replacement.

Important findings: She had leg abduction of 10 degrees, internal and external rotation of the femur was restricted at 5 degrees. She walked like Walter Brennan. She lifted her pelvis to clear the leg when she walked. She had been and wished to return to horse training.

Before using the laser, we had used a combination of strain counterstrain, fascial technique, reactive muscle corrections and percussion while performing PNF motions. The leg abduction and increased to 30 degrees and internal and external rotation to 15 degrees. She was able to walk with a very short stride properly but not with a long stride. She could not ride at this point so was considering the surgery as the orthopedist saw signs of inflammation in the joint. Laser therapy, 10 minutes with a total of 42 joules of energy, was added to the mix of therapies being done. The laser probe was aimed at the acetabulum and the GIGO muscles while the percussion and PNF motions were being performed. After two treatments, abduction had increased to 60 degrees and internal and external rotation to 40 and 30 degrees. She was seen by her orthopedist after the fifth treatment. She was told that the operation was being indefinitely postponed due to her improvement. She was now able to walk with long strides with no visible limp and had returned to her horse training. Currently she is stabilized at one treatment per month.

In chronic cases, the use of laser therapy, especially when added after and during our normal therapy options, is excellent at increasing our outcomes.

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Visceral Parietal Pain (VPP)

Jose Palomar Lever, M.D., DIBAK, Orthopedic Surgeon

Abstract

Organs have different ways to perceive pain. There are also different pathways to transmit pain. The first is the true pain conducted by autonomic nerves, and which refer pain to the VRP areas. The second, coming from parietal surfaces, are conducted directly into the local spinal nerves from the parietal peritoneum, pleura, or pericardium, and these sensations are usually localized directly over the painful area (VPPs').

Introduction

"Referred Pain"

Often a person feels pain in a part of his or her body that is considerably remote from the tissue causing the pain. This pain is called referred pain. The pain usually is initiated in one of the visceral organs and referred to an area on the body surface. The pain may be referred to an area of the body not exactly coincident with the location of the viscus producing the pain. A knowledge of the different types of referred pain is important in clinical diagnosis because many visceral ailments cause no other clinical signs except referred pain.

Mechanism of Referred Pain

The following is the probable mechanism by which most pain is referred. Branches of visceral pain fibers synapse in the spinal cord on the same second-order neurons (1 and 2) that receive pain signals from the skin. When the visceral pain fibers are stimulated, pain signals from the viscera are then conducted through at least some of the same neurons that conduct pain signals from the skin, and the person has the feeling that the sensations originate in the skin itself.

Visceral Pain

In clinical diagnosis, pain from the different viscera of the abdomen and chest is one of the few criteria that can be used for diagnosing visceral inflammation, visceral infectious disease, and other visceral ailments. In general, the viscera have sensory receptors for no other modalities of sensation besides pain. Also, visceral pain differs from surface pain in several important aspects.

One of the most important differences between surface pain and visceral pain is that highly localized types of damage to the viscera seldom cause severe pain. For instance, a surgeon can cut the gut entirely in two in a patient who is awake without causing significant pain. Conversely, any stimulus that causes diffuse stimulation of pain nerve endings throughout a viscus causes pain that can be severe. For instance, ischemia caused by occluding the blood supply to a large area of the gut stimulates many diffuse pain fibers at the same time and this can result in extreme pain.

Causes of True Visceral Pain

Any stimulus that excites pain nerve endings in diffuse areas of the viscera causes visceral pain. Such stimuli include ischemia of visceral tissue, chemical damage to the surfaces of the viscera, spasm of the smooth muscle of a hollow viscus, excess distention of a hollow viscus, and stretching of the ligaments.

Essentially all the true visceral pain that originates in the thoracic and abdominal cavities is transmitted through pain nerve fibers that run in the autonomic nerves, mainly along the same pathways as the sympathetic nerves. These fibers are small type C fibers and, therefore, can transmit only the chronic aching-suffering type of pain.

Ischemia
Ischemia causes visceral pain in the same way that it does in other tissues, presumably because of the formation of acidic metabolic end products or tissue-degenerative products such as bradykinin, proteolytic enzymes, or others that stimulate the pain nerve endings.

Chemical Stimuli
On occasion, damaging substances leak from the gastrointestinal tract into the peritoneal cavity. For instance, proteolytic acidic gastric juice often leaks through a ruptured gastric or duodenal ulcer. This juice causes widespread digestion of the visceral peritoneum, thus stimulating broad areas of pain fibers. The pain is usually excruciatingly severe.

Spasm of a Hollow Viscus
Spasm of a portion of the gut, the gallbladder, a bile duct, a ureter, or any other hollow viscus can cause pain, possibly by mechanical stimulation of the pain nerve endings. Or the spasm might cause diminished blood flow to the muscle, combined with the muscle's increased metabolic need for nutrients. Thus, ischemia could develop, which can cause severe pain.

Often pain from a spastic viscus occurs in the form of cramps, with the pain increasing to a high degree of severity and then subsiding. This process continues intermittently, once every few minutes. The intermittent cycles result from rhythmical contraction of smooth muscle. For instance, each time a peristaltic wave travels along an overly excitable spastic gut, a cramp occurs. The cramping type of pain frequently occurs in gastroenteritis, constipation, menstruation, parturition, gallbladder disease, or ureteral obstruction.

Overdistention of a Hollow Viscus
Extreme overfilling of a hollow viscus also can result in pain, presumably because of overstretching of the tissues themselves. Overdistention can also collapse the blood vessels that encircle the viscus or that pass into its wall, thus perhaps promoting ischemic pain.

Insensitive Viscera
A few visceral areas are almost completely insensitive to pain of any type. These include the parenchyma of the liver and the alveoli of the lungs. Yet the liver capsule is extremely sensitive to both direct trauma and stretch, and the bile ducts are also sensitive to pain. In the lungs, even though the alveoli are insensitive, both the bronchi and the parietal pleura are very sensitive to pain.

Parietal Pain Caused by Visceral Damage
When a disease affects a viscus, the disease process often spreads to the parietal peritoneum, pleura, or pericardium. These parietal surfaces, like the skin, are supplied with extensive pain innervation from the peripheral spinal nerves. Therefore, pain from the parietal wall overlying a viscus is frequently sharp. To emphasize the difference between this pain and true visceral pain: a knife incision through the parietal peritoneum is very painful, even though a similar cut through the visceral peritoneum or through a gut wall is not very painful, if painful at all.

Localization of Visceral Pain - The "Visceral" and the "Parietal" Pain Transmission Pathways
Pain from the different viscera is frequently difficult to localize for a number of reasons. First, the brain does not know from firsthand experience that the different internal organs exist; therefore, any pain that originates

internally can be localized only generally. Second, sensations from the abdomen and thorax are transmitted through two pathways to the central nervous system-the true visceral pathway and the parietal pathway. True visceral pain is transmitted via sensory fibers in the autonomic nerves (both sympathetic and parasympathetic), and the sensations are referred to surface areas of the body, often far from the painful organ.

Conversely, parietal sensations are conducted directly into the local spinal nerves from the parietal peritoneum, pleura, or pericardium, and these sensations are usually localized directly over the painful area.

Localization of Referred Pain Transmitted via the Visceral Pathways

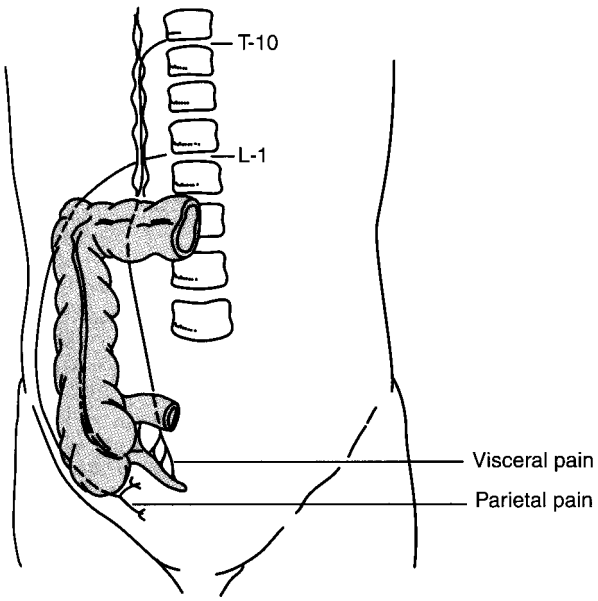
When visceral pain is referred to the surface of the body, the person generally localizes it in the dermatomal segment from which the visceral organ originated in the embryo, not necessarily where the visceral organ now lies. For instance, the heart originated in the neck and upper thorax, so that the heart's visceral pain fibers pass upward along the sympathetic nerves and enter the spinal cord between segments C-3 and T-5. Therefore, pain from the heart is referred to the side of the neck, over the shoulder, over the pectoral muscles, down the arm, and into the substernal area of the upper chest. These are the areas of the body surface that send their own somatosensory nerve fibers into the C-3 to T-5 cord segments. Most frequently, the pain is on the left side rather than on the right because the left side of the heart is much more frequently involved in coronary disease than the right.

The stomach originated approximately from the seventh to the ninth thoracic segments of the embryo. Therefore, stomach pain is referred to the anterior epigastrium above the umbilicus, which is the surface area of the body subserved by the seventh through ninth thoracic segments. And several other surface areas to which visceral pain is referred from other organs, representing in general the areas in the embryo from which the respective organs originated.

"Direct Pain"

Parietal Pathway for Transmission of Abdominal and Thoracic Pain.

Pain from the viscera is frequently localized to two surface areas of the body at the same time because of the dual transmission of pain through the referred visceral pathway and the direct parietal pathway. Thus, the figure shows dual transmission from an inflamed appendix.



Pain impulses pass from the appendix through sympathetic visceral pain fibers into the sympathetic nerves and then into the spinal cord at about T- 10 or T- 11: this pain is referred to an area around the umbilicus and is of the aching, cramping type. Pain impulses also often originate in the parietal peritoneum where the inflamed appendix touches or is adherent to the abdominal wall. These cause pain of the sharp type directly over the irritated peritoneum in the right lower quadrant of the abdomen.

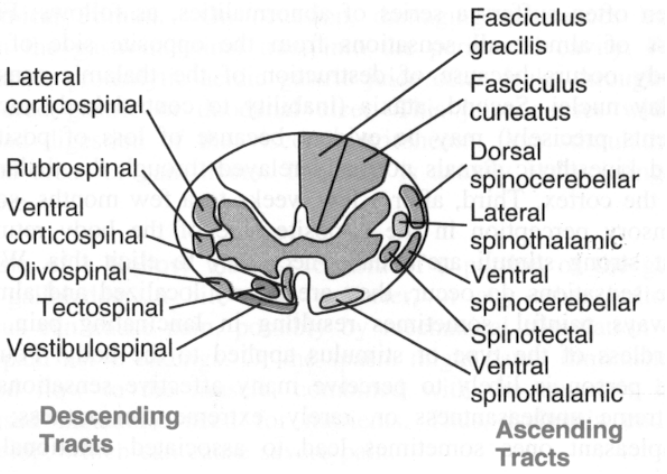
Some Clinical Abnormalities of Pain and Other Somatic Sensations

Hyperalgesia

A pain nervous pathway sometimes becomes excessively excitable; this gives rise to hyperalgesia, which means hypersensitivity to pain. The basic causes of hyperalgesia are (1) excessive sensitivity of the

pain receptors themselves, which is called primary hyperalgesia, and (2) facilitation of sensory transmission, which is called secondary hyperalgesia.

An example of primary hyperalgesia is the extreme sensitivity of sunburned skin, which results from sensitization of the pain endings by local tissue products from the burn-perhaps histamine, perhaps prostaglandins, perhaps others. Secondary hyperalgesia frequently results from lesions in the spinal cord or the thalamus.



Spinal cord ascending track on right, descending track on left

Procedure

Method

I studied 150 random patients, 40% of which were males and 60% were females, who sought consultation for various reasons, and who showed weak muscles, not responding to the 5 IVF standard treatments. Sometimes they would become strong after rubbing the VRP related area, but it's not a rule. Sometimes the muscles are strong but become weak after a small nociceptive stimulus over the VRP related area (Pinching). The standard treatment for VRP area problems, using treatment to increase PS (Parasympathic) and SYM (Symphatic) didn't work, or the correction was lost after a few seconds or by the next visit.

1. Initially I ruled out the possibility of switching and /or ligament stretch reaction.
2. Look for a positive VPP challenge. (the VPP challenge is when the doctor applies a direct pressure over the organ to be tested and then suddenly releases the pressure (**rebound challenge**). Find the vectors causing the greatest amount of strengthening of a weak related muscle. If the previously weak muscle becomes strong after the challenge, I consider it a VPP positive test.
3. If a strong muscle related to the organ becomes weak while the doctor applies direct constant pressure over the related organ I consider it a VPP positive direct test (**Not rebound test**). Find the vectors causing the greatest amount of weakness of a strong related muscle
4. Treat for the VPP, using:
   a. Visceral Parietal Challenge Technique (VPP). Treat the related organ with IRT (Injury recall technique from Dr. W. Schmitt) with the same challenge (direct pressure over the related organ) at the same time. Use the same vector of force previously found.

b. NSB Technique (Nociception Blockade Technique) with direct pressure to the organ

c. Set Point Technique with simple hand contact over the organ.

d. IRT (Injury Recall Technique) to the NL of the organ.

There is no particular breathing pattern except in the case of the lung. In organs placed close to the diaphragm, there is not a specific breathing pattern associated, but inspiration appears to improve the correction.

Results

94 % of all weak muscles unsuccessful treated by the 5 IVF and IRT, and VRP techniques, with positive Visceral parietal Challenge, became strong with long lasting results.

Conclusion

These procedures help to determine the presence of organ involvement. The viscera has two different pathways to send nociception info to the brain.

Positive Visceral parietal pain (VPP) challenge cause a strong indicator muscle to become weak, but the best muscle test is against an extensor muscle on the same side.

Referred pain reflex (VRP) is difficult to elicit precisely, although stimulation will weaken an extensor muscle on the side of referred pain. Pinwheel stimulation and (sometimes) vibration will be positive over the referred pain area, weakness will be in the extensor muscles of the opposite side of the body.

ENS is the second largest structure composed of nervous tissue, thus it is one of the most powerful organs to balance in all the body.

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Neurogait

Jose Palomar Lever, M.D., Orthopedic Surgeon & DIBAK

Introduction

It is now known that the feet send a great quantity of information to the brain, with which one can know the condition of the terrain, and adapt to the same, it is also known that the gait can be altered by foot subluxations. It is not unknown that should the feet contribute aberrant information, this may cause neurological disorganization, and also by means of the fascia the feet may cause multiple problems.

This study has sought to add more information around the physiology of gait and also elucidate more about the information originating in the feet. This research is about the neurophysiology of the foot and the gait. At first I will comment a little of that which is now established and published, and then add new material.

Background

Sensory Input From Moving Limbs Regulates Stepping Patterns

Although normal walking is automatic, it is not necessarily stereotyped. Outside the laboratory mammals constantly use sensory input to adjust their stepping patterns to variations in the terrain and to unexpected events. Three important types of sensory information are used to regulate stepping: somatosensory input from the receptors of muscle, ligaments and skin, input from the vestibular apparatus (for controlling balance), and visual input.

In considering the role of somatosensory input in stepping, Charles Sherrington made the distinction between proprioceptors and exteroceptors. Proprioceptors are located in muscles and joints and are excited by body movements; input from proprioceptors is involved in the automatic regulation of stepping. Exteroceptors are located in the skin; their main function is to adjust stepping to external stimuli. This distinction is still considered valid, although it is now recognized that skin afferents can provide important feedback about body movements.

Proprioception Regulates the Timing and Amplitude of the Stepping Patterns

One of the clearest indications that somatosensory afferents from the limbs regulate the step cycle is that the rate of stepping in spinal and decerebrate cats matches the speed of the motorized treadmill belt on which they stepping. Specifically, afferent input regulates the duration of the stance phase. As the stepping rate increases, stance duration decreases, while the duration of swing phase remains relatively constant. This observation suggests that some form of sensory input signals the end of stance and thus leads to the initiation of swing.

Sherrington was the first to propose that proprioceptors in muscles acting at the hip were primarily responsible. He noticed that rapid extension at the hip joint but not at the knee and ankle joints, led to contractions in the flexor muscles of chronic spinal cats and dogs. More recent studies have shown that preventing hip extension in a limb suppresses stepping in that limb, whereas rhythmically moving the hip can entrain locomotor rhythm. During entrainment, a burst activity in flexor motor neurons is initiated in synchrony with hip extension. The afferents responsible for signaling hip angle for swing initiation arise from the muscle spindles in hip flexor muscles. Stretching hip flexor muscles in decerebrate animals to mimic the lengthening that

occurs at the end of the stance phase inhibits the extensor half-center and thus facilitates the initiation of burst activity in flexor motor neurons during walking.

Other important signals for regulating the step cycle arise from the Golgi tendon organs and muscle spindles of extensor muscles. Electrical stimulation of the afferents from these receptors prolongs the stance phase, often delaying the onset of swing until the stimulus has terminated. Both groups of afferents are active during stance, with the Golgi tendon organs providing a measure of the load carried by the leg. The excitatory action of the Golgi tendon organs on extensor motor neurons during walking is opposite to their inhibitory action when locomotor activity is not being generated. The functional consequence of this reflex reversal is that the swing phase will not be initiated until the leg is unloaded and the forces exerted by extensor muscles are low (signaled by a decrease in activity from Golgi tendon organs). Limb unloading normally occurs near the end of leg extension, when the animal's weight is being borne by the other legs and the extensor muscles are shortened and thus unable to produce optimal forces.

In addition to regulating the transition from stance to swing, proprioceptive feedback from muscle spindles and Golgi tendon organs contributes significantly to the generation of burst activity in extensor motor neurons. Reducing feedback from these afferents in cats reduces the level of extensor activity by more than 50%, while in humans up to 30% of the excitatory input to the ankle extensor motor neurons arises from Ia afferents of extensor muscles.

At least three excitatory pathways transmit information from extensor sensory fibers to extensor motor neurons: (1) a monosynaptic pathway from Ia fibers, (2) a disynaptic pathway from Ia and Ib fibers, and (3) a polysynaptic pathway from Ia and Ib fibers. The polysynaptic pathway includes the extensor half-center of the central rhythm generator, so in addition to regulating the level of extensor activity this pathway also controls the stance duration. The disynaptic excitatory pathway is active only during the extension (possibly by excitatory input to the interneurons in this pathway from the extensor half-center). The continuous regulation of the level of extensor activity by proprioceptive feedback presumably allows automatic adjustment of force and length in extensor muscles to unexpected unloading and loading of the leg.

Sensory Input From the Skin Allows Stepping to Adjust to Unexpected Obstacles

Exteroceptors in the skin have a powerful influence on the central pattern generator for walking. One important function for these receptors is to detect external obstacles and adjust the stepping movements to avoid them. A well-studied example is the stumbling corrective reaction in cats. A mild mechanical stimulus applied to the dorsal part of the paw during the swing phase produces excitation of flexor motor neurons and inhibition of extensor motor neurons, leading to rapid flexion of the paw away from the stimulus and elevation of the leg in an attempt to step over the object. Because this corrective response is readily observed in spinal cats, it must be produced to a large extent by circuits entirely contained within the spinal cord.

One of the interesting features of the stumbling-corrective reaction is that corrective flexion movements are produced only when the paw is stimulated during the swing phase. An identical stimulus applied during the stance phase produces an opposite response: excitation of extensor muscles that reinforces the ongoing extensor activity. This extensor action is appropriate; if a flexion reflex were produced, the animal might collapse because its weight is being supported by the limb. This is an example of a phase-dependent reflex reversal: the same stimulus will excite one group of motor neurons during one phase of locomotion and excite the antagonist motor neurons during another phase.

Descending Pathways Are Necessary for Initiation and Adaptive Control of Walking

Although the basic motor pattern for stepping is generated in the spinal cord, fine control of stepping movements involves numerous regions of the brain, including the motor cortex, cerebellum, and various sites within the brain stem. Recordings from neurons in all these regions have shown that many are rhythmically

active during locomotor activity and hence involved in some way with the production of the normal motor pattern. Each region, however, appears to play a different role in the regulation of locomotor function.

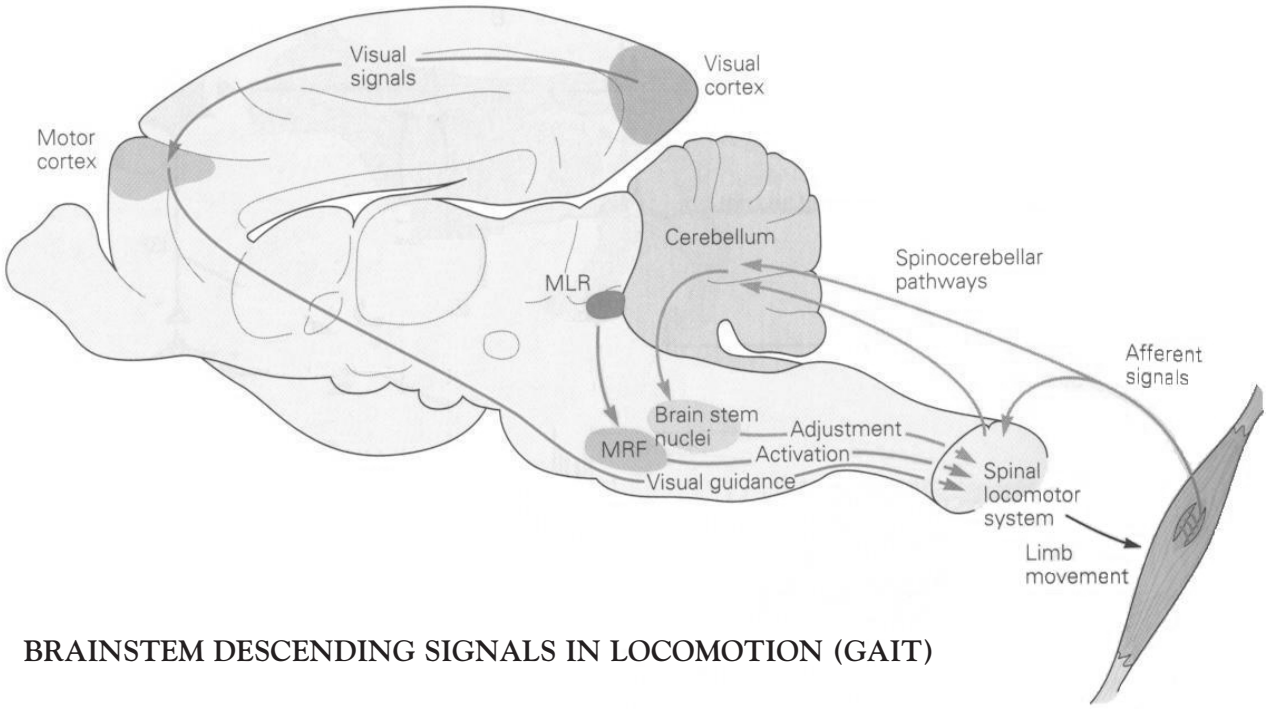
Supraspinal regulation of stepping can, in broad terms, be divided into three functional systems. One activates the spinal locomotor system and controls the overall speed of locomotion, another refines the motor pattern in response to feedback from the limbs, and the third guides limb movement in response to visual input.

Descending Pathways From the Brain Stem Walking and Control Its Speed

In their seminal studies of decerebrate cats,: Mark Shik, Fidor Severin, and Grigori Orlovsky found that tonic electrical stimulation of the mesencephalic locomotor region initiates stepping when animals are placed on a freely moving treadmill. The rhythm of the locomotor pattern is not related to the pattern of electrical stimulation but depends only on its intensity. Weak stimulation produces a walking gait that increases in speed as the intensity increases; progressively stronger stimulation produces trotting and finally galloping. Thus, a relatively simple control signal from the brain stem, modulated only in intensity, not only initiates locomotion but also controls the overall speed of walking.

It is especially interesting to note that the changes in gait are associated with changes in the coordination between legs: an out-of-phase relationship between left and right legs in walking changes to an in-phase relationship in galloping. These shifts in interlimb coordination are most likely implemented by local circuits in the spinal cord, since they are also observed in spinal cats on a motorized treadmill as the treadmill speed is increased.

In addition to the mesencephalic locomotor region several other motor regions of the brain, including a subthalamic motor region and a pontine locomotor region, can produce locomotion when stimulated experimentally. How these different brain stem regions interact in normal control of locomotion is not yet known. Of course we know that the cerebellum fine tunes the locomotor pattern by regulating the timing and intensity of descending signals.



BRAINSTEM DESCENDING SIGNALS IN LOCOMOTION (GAIT)

Abstract

The feet send a myriad of gait information to the brain; this gait research shows a neurological cycle during gait.

This paper is about neuro physiology of the gait, and the information sent by the ligaments of the joints. The most important part of the cycle is when the foot is stimulated by the contact with the floor. This is during the stance phase. I could not find any paper related to specific links between muscles and the foot's ligaments.

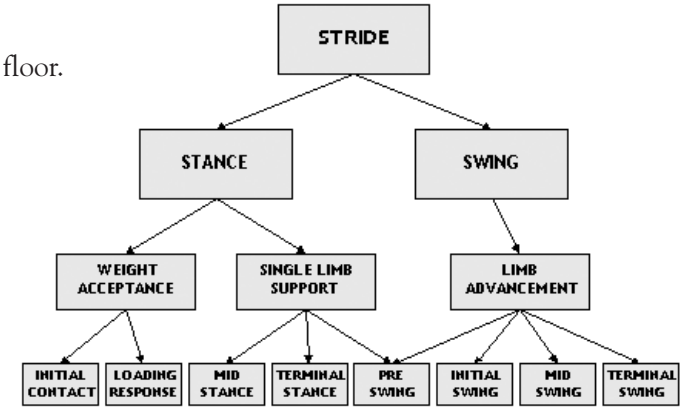
Introduction

Normally, the stride of the gait is divided in two different phases: A.- Stance and B.- Swing.

Also, the stance and swing divides into different phases.

Stance is subdivided into:

1. Initial contact of the calcaneus with the floor.
2. Supination of the middle foot, and contact of the external border of the foot.
3. Load on the metatarsal heads (starting with the 5th, then 4th, 3rd, 2nd, and finally the 1st.
4. Load on the big toe and launch.



It's a good description, but it's only a mechanical description. I suggest a more complete Neuro biomechanical description.

Procedure

I test more than 200 (two hundred) normal or at least asymptomatic people.

I test the ligaments of many different joint of the foot. The test consist in spread apart the ligament and test 40 different muscles, and verify if they inhibits or facilitates.

Method

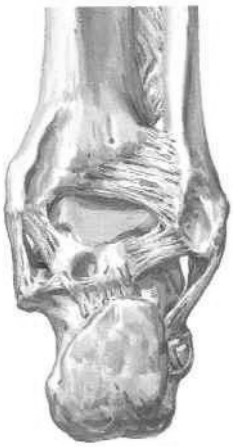
I test the facilitation with two different tests:

1. Placing the tested people in gait position (supine position), that causes the tested muscle become weak, and then stress the tested ligament and verify if it negates the position.
2. Asking to the tested people to look at the "II" parallel lines (looking at the parallel lines causes all muscles become weak) and verify if the stress of the tested ligament causes the muscles becomes strong.

Results

I suggest this more complete gait description:

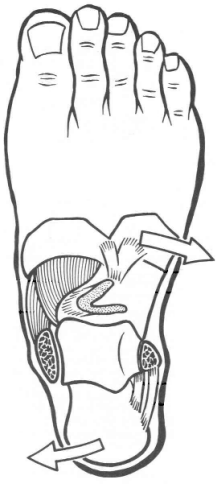
1. When the calcaneal bone strikes the floor, but there is not valgus or varus of the foot (Lisfrank, Chopart neither subtalar joints).
2. But then the calcaneus moves in valgus and the Chopart joint moves into supination.
3. When the external border of the foot starts to load the body weight, the first bone to load is the external border of the calcaneus, then the cuboids, then the 4th and 5th metatarsals.
4. After that the load of the metatarsal heads (starting with the 5th, then 4th, 3rd, 2nd, and 1st at the end. But when the metatarsal heads load the body weight, the toes rotate externally while loading some weight in the toe tips.
5. The medial border of the foot moves into pronation (normally between 4 to 5 degrees). Loading the weight in the metatarsal-cuneal joint, then in the Cuneo - navicular joint, and lastly in the talo – navicular joint.
6. The big toe then loads the body weight first in the metatarsal head.
7. Then in the interphalangeal joint of the first toe.
8. End of the ipsilateral stance and start of the contralateral one.



Step 1.
When the calcaneus strikes the floor, there is no valgus or varus of the retro foot (neither Lisfrank, nor Chopart, nor the subtalar joints).

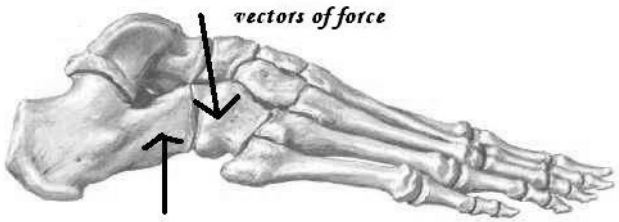
The dorsiflexión of the foot stresses the posterior Tibio – talar and posterior Talo - calcaneal joint (ligaments) and facilitates the quadriceps.

Then the ankle moves into plantar flexion.



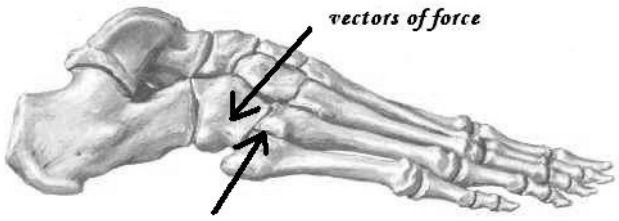
Step 2.
After the calcaneus strikes the floor, it moves in valgus, at the same time the mid foot moves into supination by means of the Chopart joint.

Valgus of the calcaneus stresses the tibionavicular joint and the deltoid ligament, and inhibits the quadriceps.



Step 2.

Supination of the mid foot, by means of the Chopart joint, stresses the calcaneocuboid joint and its ligament, this inhibits the Ipsilateral TFL and the Peroneus tertius, longus & brevis muscles

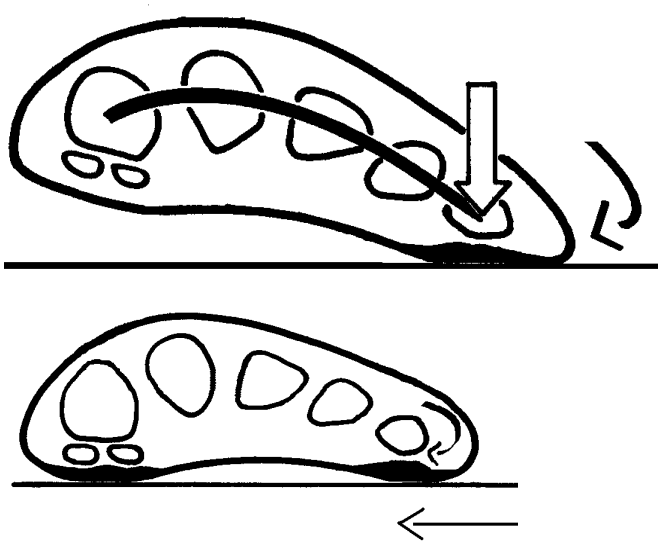


Step 3.

The mid foot supination continues.

The next ligaments to be stressed are in the joint between the cuboids and the 5th metatarsal.

This stress facilitates Ipsilateral TFL and the Peroneus tertius, longus & brevis muscles.

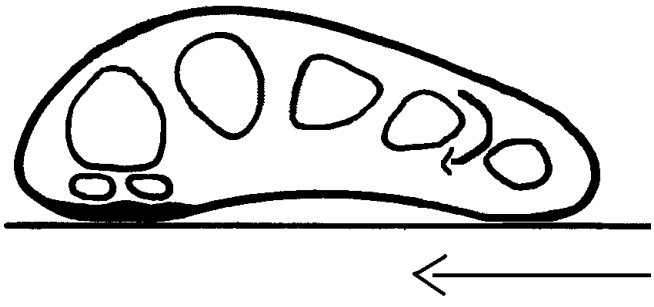


Step 4.

The anterior foot Supinates and loads the 5th metatarsal head.

Then the 5th metatarso phalangeal joint with the 5th toe twists and moves into adduction (the toe action must include metatarso-phalangeal, interphalangeal proximal and distal joints and ligaments).

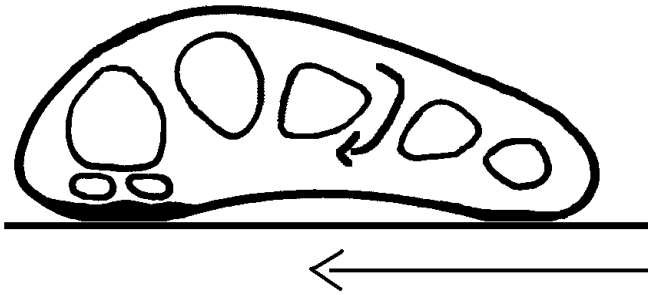
The stress on the ligaments between 5th and 4th metatarsals, and the metatarsophalangeal joints together inhibit ipsilateral adductor muscles of the leg, and contralateral adductor muscles of the arm.

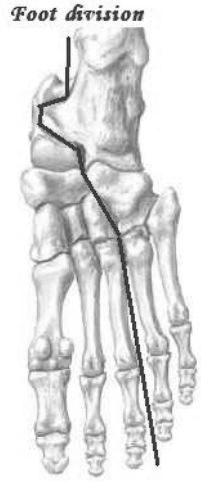


Step 5.

The 4th metatarso phalangeal joint with the 4th toe twist and moves into adduction (the toe action must include metatarso-phalangeal, interphalangeal proximal and distal joints and ligaments).

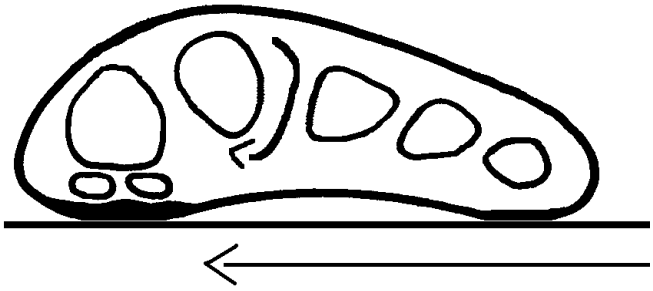
The stress on the ligaments between 4th and 5th metatarsals, and metatarsophalangeal joints together inhibit ipsilateral gluteus medius and contralateral abdominal oblique.





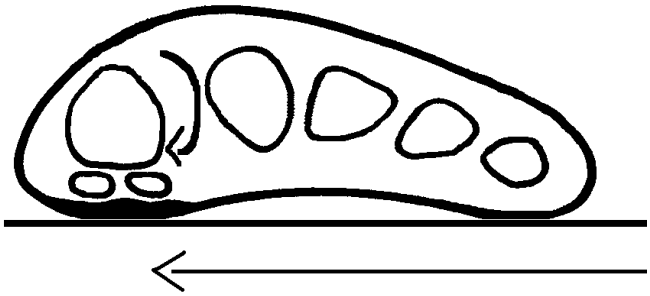
Step 6.
The 3rd metatarso phalangeal joint with the 3rd toe twists and moves into adduction (the toe action must include metatarso-phalangeal, interphalangeal proximal and distal joints and ligaments).

The stress on the ligaments between 3rd and 4th metatarsals, and metatarsophalangeal joints together inhibit the ipsilateral abductor's muscles of the leg and contralateral abductor muscles of the arm.



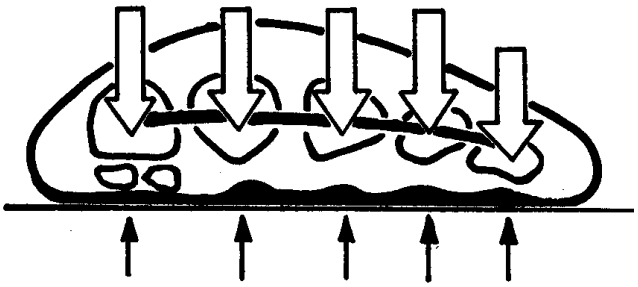
Step 7.
The 2nd metatarso phalangeal joint with the 2nd toe twist and move into adduction (the toe action must include metatarso-phalangeal, interphalangeal proximal and distal joints and ligaments).

The stress on the ligaments between 2nd and 1st metatarsals, and 2nd metatarsophalangeal joints together inhibit ipsilateral flexors muscles of the leg and contralateral flexors muscles of the arm.



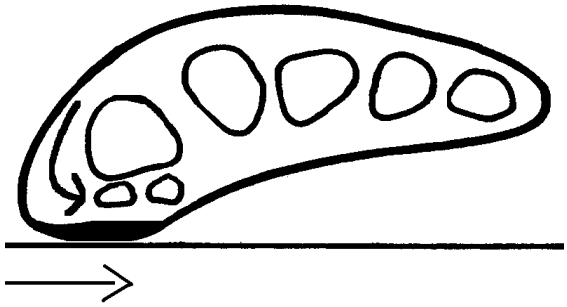
Step 8.
The 1st metatarso phalangeal joint with the 1st toe twist and move in adduction (the toe action must include metatarso-phalangeal, interphalangeal proximal and distal joints and ligaments).

The stress on the ligaments between 1st and 2nd metatarsals, and 1st metatarsophalangeal joints together inhibit ipsilateral extensors muscles of the leg and contralateral extensors muscles of the arm.



Step 9.
In this step all metatarsal are in neutral position and are raised to form a virtual arch.

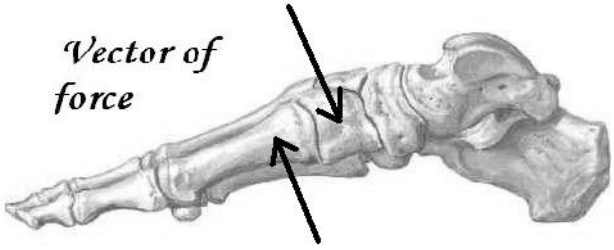
The stress on the ligaments between the 2nd, 3rd and 4th metatarsals inhibits the ipsilateral Psoas and contralateral Pectoralis Sternal muscles.



Step 10.
The 1st metatarso phalangeal joint with the 1st toe twists and moves into abduction (the toe action must include metatarso-phalangeal, interphalangeal proximal and distal joints and ligaments).

This step stresses the ligaments between 1st and 2nd metatarsals, and 1st metatarsophalangeal joints.

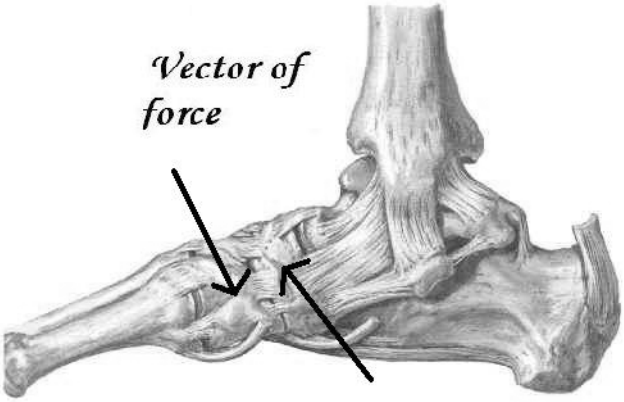
This is the movement previous to the launch of the foot.



Step 11.
Before the body weight translates to the first toe, the mid-foot rotates with 4º degrees of pronation.

This pronation stresses the ligaments of the Cuneo metatarsal joint.

This is the movement previous to the launch of the foot.

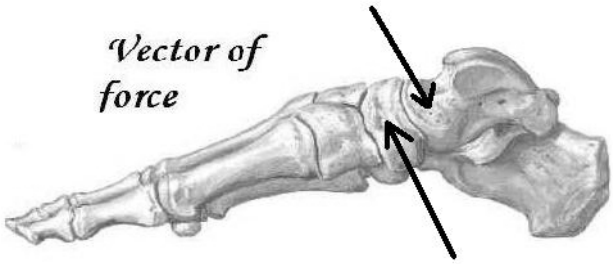


Step 12.
This movement stresses the ligaments of the Cuneo Navicular joint during pronation. These ligaments inhibit:

Ipsilateral anterior deltoid, peroneus longus & brevis and tertius.

Also the contralateral upper Trapezius and Pectoral major Clavicular.

Facilitates: ipsilateral posterior deltoids.



Step 13.
This movement stresses the ligaments of the Talo Navicular joint and anterior deltoid ligament (tibionavicular).

This ligament inhibits:
Piriformis, SCM, Anterior Tibialis , Posterior Tibialis, Psoas, Latissimus Dorsi, and Abdominal Obliques (all muscles are ipsilateral).

Neuro Gait

(Joint ligaments / involved muscles)

Talo-navicular joint ligaments
Ipsilateral Piriformis (Inhibition)
Ipsilateral SCM (Inhibition)
Ipsilateral Anterior Tibialis (Inhibition)
Ipsilateral Posterior Tibialis (Inhibition)
Ipsilateral Psoas (Inhibition)
Ipsilateral Latissimus Dorsii (Inhibition)
Ipsilateral Abdominal Oblique (Inhibition)

Cuneo Navicular joint ligaments
Ipsilateral Deltoides Anterior (Inhibition)
Ipsilateral Deltoides Posterior (Inhibition)
Ipsilateral Peroneus Brevis, Long and Tertius (Inhibition)
Contralateral Upper Trapezius (Inhibition)
Contralateral Pectoral Major Clavicular (Inhibition)

Cuboid calcaneal joint ligaments
Ipsilateral TFL (Inhibition)
Ipsilateral Peroneus longus & brevis (Inhibition)

Subtalar Joint ligaments
CALCANEUS VARUS
Ipsilateral Gastrocnemius y Soleus (Inhibition)
Ipsilateral Hamstrings (Inhibition)
Ipsilateral Gluteus maximus (Inhibition)

PRONATION O VALGUS OF THE CALCANEUS (retro foot)
Ipsilateral Quadriceps (Inhibition)
Ipsilateral Recto anterior (Inhibition)

Interphalangeal joint of the 1st toe ligaments
Ipsilateral Quadriceps (Inhibition)

Metatarso phalangeal joint of the 1st toe ligaments
Ipsilateral Quadriceps (Facilitation)
Contralateral Quadriceps (Inhibition)
Ipsilateral Popliteus (Inhibition)
Popliteus (Contralateral - Do not alter facilitation)

Tibio Talar Joint ligaments (Dorsal flexion: normal range of motion)
Ipsilateral Quadriceps (Facilitation)
Ipsilateral Popliteus (Inhibition)
Contralateral Quadriceps (Inhibition)
Popliteus (Contralateral - Do not alter facilitation)

Tibio Talar joint ligaments (forced dorsal flexion)
Ipsilateral Quadriceps (Inhibition)
Ipsilateral Popliteus (Inhibition)
Contralateral Quadriceps (Facilitation)
Contralateral Popliteus (Do not alter inhibition)

5th metatarso phalangeal toe ligaments (twist (plantar surface toward medial side and adduction)
Ipsilateral adductors muscles of leg (Inhibition)
Contralateral adductor muscles of the arm (Inhibition)

4th metatarso phalangeal toe ligaments (twist (plantar surface toward medial side and adduction)
Ipsilateral Gluteus medius (Inhibition)
Contralateral Abdominal oblique (Inhibition)

3rd metatarso phalangeal toe ligaments (twist (plantar surface toward medial side and adduction)
Ipsilateral abductors muscles of leg (Inhibition)
Contralateral abductor muscles of the arm (Inhibition)

2nd metatarso phalangeal toe ligaments (twist (plantar surface toward medial side and adduction)
Ipsilateral anterior muscles of leg (Inhibition)
Contralateral anterior muscles of the arm (Inhibition)

1st metatarso phalangeal toe ligaments (twist (plantar surface toward medial side and adduction)
Ipsilateral posterior muscles of leg (Inhibition)
Contralateral posterior muscles of the arm (Inhibition)

1st metatarso phalangeal toe ligaments (twist (plantar surface toward lateral side and abduction)
Ipsilateral Quadriceps (Inhibition)
Contralateral Piriformis (Inhibition)
Contralateral Psoas (Inhibition)

Raise of the metatarsal heads (Ki1) (ligaments)
Ipsilateral Psoas (Inhibition)
Contralateral Pectoralis major (Inhibition)

Anterior Tibio talar Deltoid ligament
Ipsilateral Upper Trapezius (Inhibition)

Deltoid ligament (Tibio navicular)
Ipsilateral Quadriceps (Inhibition)

Posterior Tibio talar Deltoid ligament
Ipsilateral Subscapularis (Inhibition)

Anterior TaloFibular ligament
Ipsilateral Neck flexors and SCM (Inhibition)

Middle CalcaneoFibular ligament
Ipsilateral Hamstrings (Inhibition)

Posterior TaloFibular ligament
Ipsilateral Neck extensors (Inhibition)

Discussion

Curiously, calcaneal ligaments affect pelvic and lower limb muscles, while talar ligaments are more involved with neck, upper thoracic and shoulder muscles.

Also worthy of attention is the fact that the hyoids, which sends such a large quantity of information to the brain, should have so many muscular insertions but none ligamentous, while the talus has no muscular insertions but is ligament rich, and yet is another key information center for the brain.

Foot lesions can cause neurological as well as structural problems and should be evaluated for complete correction of any health problem.

The ligaments may be affected by foot subluxations in any of the many foot articulations, but they are more frequently injured by traction or compression trauma, traction injury being more common.

Treatment for these lesions consists in IRT followed by manual manipulation of the ligaments so as to approximate the ends towards the middle in the case of distraction injuries or tension on the ends to separate the ends from the middle in the case of compression injuries. The duration of treatment may be from 30 seconds up to 3 minutes. In some cases NSB technique may be needed to treat nociceptors.

It is possible that the current success of treatment to the foot gait receptors (PALO_A points) is more due to stimulation of ligaments and other structures than to electromagnetic considerations.

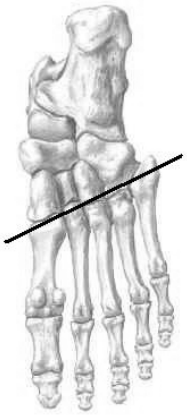
Exteroceptors in the skin have a powerful influence on the central pattern generator for walking, they influence the cerebellum, and adapt to the uneven surface of the floor or changes in direction. Ligaments and joints send afferent information to the basal ganglia during the normal gait and to the cerebellum when the patient starts or finishes the gait. We can inhibit different muscles just pulling the skin of the sole of the foot

in different directions. For example: pulling the skin with caudal vector we can inhibit the quadriceps muscles, and with cephalad vector, we can inhibit the hamstrings muscles, with lateral vector we can inhibit Peroneus muscles and with medial vector we can inhibit Posterior Tibialis.

Conclusion

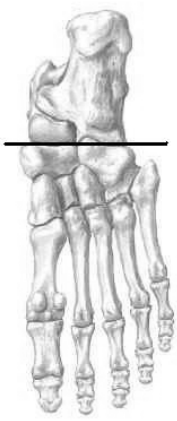
In Light of the quantity and importance of foot proprioception, and realizing their causal relation in so many problems from neurological disorganization to gait, facial disturbances, and inhibition of any muscle, we should reevaluate our physical exploration and pay more attention to the feet, especially in light of their vulnerability and frequency of injury and also the insult to them in daily life. Difficult to resolve or recurrent cases are often helped with more attention to the feet, often there lies the answer. We need more investigation to continue advancing the knowledge of human functional neurology.

P.S.: To unclear not commonly known terms, I'm describing them.



Lisfrank joint: (tarsometatarsal), Junction between the midfoot and forefoot.

Chopart (transverse tarsal joint): Junction between the hindfoot and midfoot.



Hindfoot: Division of the foot comprised of the calcaneus and talus.

Midfoot: the portion of the foot located between the hindfoot and forefoot; includes the navicular, cuneiform, and cuboid bones

Forefoot: the most distal part of the foot, comprised of the metatarsals and phalanges.

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Newly Discovered Causative Factor for Switching and the Yaw #2 Pattern

Paul T. Sprieser, D.C., DIBAK

Abstract

A new factor that causes the immediate reoccurrence of the Yaw #2 modular distortion and the K27 switching after it has been corrected. The significance of this factor in stabilization and correction of patients with gastroesophageal reflux disorder.

Discussion

On June 7, 2005 a psychotherapist came into my office saying that she tells her patients not to put their glasses on the top of their head because it causes a polarity change. She uses Thought Field Therapy in her practice so she is familiar with muscle testing and neurological disorganization (switching).

I asked her what exactly she meant by polarity she said that she would muscle test her patients and have them place their hand over the vertex of the skull of GV-21.[1] She referred to this are as the crown chakra and said that the placement of glasses over this point caused a disturbance in the patient polarity. I explained to her that what she was referring to was probably better classified as stitching or neurological disorganization rather then polarity such as north or south pole as in magnetic fields.[2]

Since I always start my AK examination when the patient is supine by checking K27 switching and evaluating for the yaw #2 pattern. I check her and she showed a positive K27 pattern and correct the yaw #2 to the left, meaning the pelvis at the ASIS was forward on the right. I made my usual correct of the yaw #2 and rechecked K27 it was now negative.[2]

I then took the therapist's reading glasses and placed them over GV-21 point. I immediately noticed the pelvis raised off the table on the right and caused the reoccurrence of the Yaw #2 pattern and K27 switching.

I now wondered if this was a consistent occurrence and would this happen on all patients, and if so, it might explain the persistence of the Yaw #2. Despite adequate correction of this modular distortion it would seem to return in a short period of time. It is a rather common practice of patient when not using the glasses to put them on the top of the head.

Besides the use of earphones, headsets for phone, and even hair-bands that commonly cover this area. This new factor could give another explanation for the persistence of this pattern despite proper corrective techniques.

Method

I started testing every patient that showed the Yaw #2 pattern with (S.O.T.) blocks in the supine position. One block is placed under the crest of the ileum on the right and under the shoulder on the left.[3] This would cause a strong indicator muscle to weaken I would then ask the patient to contact K27 points and note if it negated the weakness proving the causative factor for switching is the Yaw #2.[4]

At the time that I am writing this paper the total number patients seen with this pattern is 717 with the causal connection of Yaw #2 to switching has been 711 and Pitch pattern caused the remaining 6 cases.

Placing anything that crossed GV-21 and down either side of the head would cause the immediate return of Yaw #2 and switching if it caused by this pattern. A strong indictor muscle would weaken with anything over this region when such as glasses or a head-band if they were moved forward the weakness would stop but the modular distortion and switching would need to be corrected again. This pattern would be reproduced as many times if you had that region covered.

As I started collection data on this pattern this reoccurrence could be reproduced on every patient. It happened for the first 17 patients in a row, then on the 18th patient it did not cause the return of the pattern. As Dr. Goodheart would say that the exception proved the rule. I asked this patient what she was doing. She explained to me that a book that I had recommended called Energy Tapping, by Fred Gallo, Ph.D.,[5] suggested that you should tap the GV-21, and a point referred to as the third eye twice a day. I thought this might be the method for correcting this pattern. I checked the acupuncture charts and found that there was no point over the third eye. The two closest points to this region were GV-24 at the hairline and GV-24.5 between the eyebrows above the bridge of the nose.[6]

I tried tapping each point individually by this did not prevent the reoccurrence but when all three points were tapped simultaneously (GV-21, GV-24, and GV-24.5) for thirty second, this would prevent the glasses to the top of the head from causing the reoccurrence.

What I originally believed was that the tapping of these points prevents the contact over the top of the head from causing the return of the Yaw #2 pattern and stitching. When I realized what the truth was this patient, who was the only exception, did not have the Yaw #2 distortion to begin with, and therefore putting glasses on the top of the head did cause the Yaw #2 pattern, but if it were present it would allow for the reoccurrence after correction.

I continued to investigate what points that caused this pattern and found that the B6 point that lies just lateral of GV-21 about 1 inch rather then the GV-21.

Next I started testing other homeostasis factors on all the patients I used range of motion at the hip and blood pressure and pulse rates. I found that the tapping of the three GV points made a change of 15 degrees as and average. It could cause either a rise or fall in blood pressure I only excepted change of 5 mm or greater to be significant. Pulse rate changes of 10 points or greater were considered significant.

I found that tapping of these three points GV-21, GV-24 and GV-24.5 for thirty seconds would also change the muscle weakness that cause the Yaw #2 pattern to the left. The muscle weakness in this pattern is a weak left psoas and weak right latissimus dorsi. In 75% of patients test it would improve the rib cage mobility which is found restricted in the case of Fulford's and Dr. Goodheart observation of the restriction of torso rotation found in seven out of ten patient showing limitation to the right but not the left in the seated position.[7, 8] This is the observation that is seen in all patients suffering with GERD. So the importance of keeping this corrected in order to eliminate this problem is essential.[9]

Conclusion

When the patient maintained the structural correction of the Yaw #2 pattern I found that there was still value of the tapping of the GV-21, GV-24 and GV-24.5 points for thirty seconds. It seems to improve the working of the autonomic nervous system. It shows a consistent change of ROM measured at the hip of an average of 15 degrees on every patient tested. This range of motion improvement is due to a change of tone being set in the hamstring and abductor muscle by the spindle cell feed back.

I will also tell the patient with GERD to tap these points periodically during the days because it seem to maintain the correction of the Yaw #2 an eliminate the Fulford pattern and there by stabilization of the gastroesophageal reflux patient.

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The Association of the Learning Disability Cranial Fault to Brain Chemistry Disorders and Depression

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Abstract

In recent months a connection between what I had called the Learning Disability (LD) Cranial Fault and the brain neurotransmitters of serotonin, dopamine, epinephrine and norepinephrine has been found. The learning disabilities of attention deficit disorder and hyperactivity have a commonality of dopamine, epinephrine and norepinephrine. Conditions of depression, general anxiety disorders, social anxiety disorders, and obsessive-compulsive disorders, are effected by serotonin, epinephrine and norepinephrine. The common link, within these two classes of disorders is the epinephrine and norepinephrine.

Introduction

I have been gathering data over the last two years on the connection of the LD fault to depression and other neurologic disorders.

Depression is a common problem. In any given year approximately at least 9.5 percent of the general population (18.8 million) American adults have depressive illness. The cost to the American economy is enormous in addition to the suffering that this disorder causes the patient, their families and friends.[1]

Depressive disorders effect body, mood and thoughts; it effects the way a person eats and sleeps. It effects the way one feels about oneself. Depressive disorders are not the same as feeling blue, which is usually a passing pattern. People with a depressive illness often will not seek treatment even though they can be helped. These illnesses can be helped today with medications (selective serotonin reuptake inhibitors or SSRI–drugs called tricyclics, and monoamine oxidase inhibitors (MAOs) for severe depression and with herbal treatment of St. John Wart (Hypericum perforatum). Other alternative treatments include be SAMe (S-Adenosyl-Methionine) and fish oil. However, in the case of severe depression that could lead to suicide, drug therapy will be necessary. Research has shown that the most effective method to treat depression is a combination of "talk therapy" such as cognitive/behavioral and drug therapy.

Other research using EEG (electroencephalogram) and fMRI (functional magnetic resonance imagery) has shown the same regions of the brain to be involved in learning disabilities and depressive disorders. The regions seem to be the prefrontal and some of the temporal brain areas. Depression occurs in women about twice as often as in men and learning disabilities occur about twice as often in men as in women. These differences are probably due to hormonal factors. For those with depression, the rate of suicide for men is four times that of women, however more women attempt to commit suicide.[2]

Discussion

Depressive disorders come in a variety of different forms much like heart problems. The first form is know as **major depression** which has a combination of symptoms that can affect the ability to sleep, work, eat, and other activities of daily living. The next type is less severe. It is known as **dysthymia.** These are long standing and chronic symptoms which are not disabling. The patient can usually function fairly well, but dysthymia can lead to major depression over a lifetime. The third major type is known as **bipolar disorder**, many times is referred to as manic–depressive illness. This type is not as prevalent as other forms of depressive illness. It is often characterized by cyclic mood changes from severe highs referred to as (mania) to the severe lows (depression); these mood cycles can be dramatic and rapid.[3]

To characterize the symptoms for this disorder I will break them down into depression and mania.

Depression: Persistent anxious, empty or sad mood. Feelings of hopelessness, worthlessness, and guilt. Loss of interest in pleasure such as hobbies and activities that, were once enjoyed including sex. Other symptoms that are common include decreased energy, fatigue, difficulty in concentrating, making decisions, and remembering. Feelings of restlessness and irritability are possible. Changes in appetite, which may manifest in abnormal weight loss or gain. The most serious symptoms are thoughts of death or suicide.

Mania shows the following symptom patterns-Excessive elation, unusual irritability, decreased need for sleep. Other important signs include grandiose notions, increased talkativeness, racing thoughts, unusual increase in sexual desire with marked increased energy. Poor judgment and inappropriate social behavior can also be seen in mania.[4]

If we look at some of the characteristics of the ADD and AD/HD individual we will see the parallel between these and the different types of depression. In a book by Daniel G. Amen, M.D. Healing ADD, he has divided this condition in six categories the following are the descriptions. Compare them with above description of depression and you will see the connection.[5]

Type 1 Classic ADD-Inattentive, distractible, disorganized, hyperactive, restless and impulsive.

Type 2 Inattentive ADD-Easily distracted with a low attention span, but not hyperactive. Instead, often appears sluggish or apathetic.

Type 3 Overfocused ADD-Excessive worrying, argumentative and compulsive; often get locked in a spiral of negative thoughts.

Type 4 Temporal Lobe ADD-Quick temper and rage, periods of panic and fear, mildly paranoid.

Type 5 Limbic ADD-Moodiness, low energy. Socially isolated, chronic low-grade depression. Frequent feeling of hopelessness.

Type 6 Ring of Fire ADD-Angry, aggressive, sensitive to noise, light, clothes and touch; often inflexible, experiencing period of mean, unpredictable behavior, and grandiose thinking.

Dr. Amen has a novel approach for treatment of ADD/ADHD that fits the chiropractic model. He also has a medical model that is different from conventional treatment protocols. His approach uses not just stimulants but anticonvulsants and anti-psychotics.

	Nutritional Approach	Drug Approach
Type 1 & 2	L-Tyrosine 500 to1500 mg daily, 1/2 for children	Stimulants such as Adderall, Concerta
Type 3	St. John's Wart 300-900 mg, Tyrosine 00-1500 mg.	(SSRI) Prozac, Paxil
Type 4	Gama-aminobutyeric acid (GABA) 100-500 mg. Adults, 1/2 children	
	Ginkgo biloba 60-120 mg, Vitamin E	Anti convulsants, Depakote,
	Vitamin E 400-600 I.U.	Carbatol, Neurontin
Type 5	DL Phenylalanin (DLPD), Tyrosine SAMe (S-Adenosyl-Methionine)	Stimulating & Antidepressant Norpramin, Tofranil, and Wellbuyrin
Type 6	GABA, Omega-3-Fatty Acids	Anti psychotic & AntiSeizure Risperdal or Zyprexa

The connections are in the neurotransmitters and also the cranial fault which I will describe later in this paper.

Depression and or ADD/ADHD, run in families which might suggest biological factors such as heredity as one possible causative factor. Other possible factors are related to stress (because of the connection to the neurotransmitter of epinephrine and norepinephrine). Stresses of work, home or school may begin the onset of symptoms.

Difficult relationships, serious loss of family or friends and financial problems can be a precipitating factors. The depressed individual may have low self-esteem, and will view the world or themselves in a pessimistic way leading to easily being overwhelmed by stress, which then can lead to depression.

It also should be mentioned that women and men might manifest symptoms of depression pregnancy, menopause and postpartum depression. In addition to other factors such as work, home responsibilities and caring for children. The female may display this as a feeling of the "blues" or crying and not being able to accomplish anything. The male may show depression as irritability, anger and being discouraged. In men alcohol or drugs often cover these symptoms, or by more socially acceptable methods such as over work what we term a (the "workaholic" personality).[6]

The bipolar form of depression deserves special consideration because it has a number of subcategories. The depressive and manic states have been mentioned, but there may be a state classified as "mixed state." This has psychosis and suicidal thinking. It may be accompanied by hallucinations, which involve seeing hearing, or other sensory stimuli that are not actually there.

There is a form of bipolar disorder, which is termed "rapid cycling" which is defined as four or more occurrences within a 12-month period. This form is more resistant to treatment than the non-rapid cycling types.

Combinations of this disorder vary among bipolar patients. The symptoms can go from being completely "out of control," with major impairment, to a milder form know, as hypomanic episodes that may cause little impairment in function. In its most severe forms the patient suffers with incapacitating depression that may prevent them from working, going to school, or even being able to interact with family or friends. This form often requires hospitalization.[7]

The other classifications are **bipolar I disorder** (when a person has experienced at least one episode of severe mania), **bipolar II disorder** (when a person has at least one hypomanic episode but does not meet the full criteria of a manic episode). **Cyclothymic disorder** is a milder form with longer duration of experiences over at least a two-year period (In children and adolescents over a one-year period), with hypomanic symptoms and numerous periods of depression. The final classification is **schizoaffective disorder** when persons who meet criteria for bipolar or unipolar depression who also experience chronic psychotic symptoms, persisting after the mood symptoms have cleared.[8]

Another classification set which was recently published in the science section of The New York Times 3/22/05, the following article titled "Hypomanic? Absolutely. But Oh So Productive!"[9]

The article describes a less serve form of bipolar disorder that they call hypomania that is characterized by great mental energy and concentration that is less reckless then full-blow manic frenzies and unspoiled in many cases by subsequent gloom. The article also two additional classification bipolar III and bipolar IV. The following chart appeared in this article.

When Moods Swing

Although bipolar disorder is widely recognized, scientists do not agree about its milder forms. Some people who rate high on psychological measures of exuberance are on the bipolar spectrum, and some are not. Estimated percentage of population belied to have:

MORE SEVERE				**LESS SEVERE**
Bipolar I disorder:1%	Bipolar II disorder: 0.5%	Cyclothymic disorder: 0.4–1%	Unspecified bipolar: 1–5%	Exuberant temperament: 6–10%
Typified by manic episodes followed by depressive episodes.	Typified by mild manic episodes followed by depressive episodes.	Rapid cycling between moderate depressive and manic symptoms.	Includes hypo-mania without depression; mood swings less extreme than bipolar.	Disinhibited, highly expressive in childhood highly excitable, energetic as adults.

Discoveries

During the past two years I realized there was a relationship between what I had termed the learning disability cranial fault and depression. This has made me to believe that (LD) cranial fault should be renamed or referred to as the neurotransmitter cranial fault, or (NTCF).

All of these discoveries stem from the research on switching that I presented in the Collected Papers of ICAK 2001, in a paper titled A New Slant on Switching.[10] These finding were preceded by the 1984 paper on Learning Disabilities.[11] The accumulated information is more than twenty years of collecting data. This represent more than 20,000 cases. Other papers that stem from this research and are connected to depression and this current paper are: The Relationship of Switching to the Yaw #2 of the PRT-T Technique,[12] Supraspinatus Muscle as an Indicator of Brain Serotonin Levels,[13] The New Alarm Points For The Governing and Conception Vessels,[14] and The Relationship of The Governing and Conception Vessels to Switching and Cross Therapy Localization.[15]

Switching or neurological disorganization had one main cause and that was the modular distortion pattern that we refer to as PRY-T Technique. The main causative factor is the effect on the dura and its effect on the brain. This switching pattern is the one that effect K27.

The other less common pattern is Governing Vessel GV27 and Conception Vessel CV24 which is specifically related to the LD fault and cross therapy localization (switching). Both the K27 and GV27-CV24 are both connected to the secondary meridians of the Governing and Conception vessel.

This pattern is consistently connected to all form of learning disabilities as well as all types of depression.

Method

Patients are examined by manual muscle testing using either the gluteus medius or the tensor fascia lata muscles. If the muscle tests strong, the patient is asked to touch just under the nose above the upper lip (GV27). This should not cause any weakness. The patient is then asked to touch just below the lower lip, which is CV24 using the same indicator muscle. This also should not cause any muscle weakness. Next, the patient is asked to touch with the index fingers both GV27 and CV24 simultaneously. This causes the strong indicator muscle to weaken.

When this is found, there will also be a weakness of the supraspinatus muscle (usually one side only). There will also be a specific cranial fault that is therapy localized (TL) with both index fingers in the center of the palate at the cruciate suture. This fault is unique because both fingers must be in contact with the palate to produce the muscle weakness. If only one finger is in contact with the palate, there will be no weakness. The other factor is that inspiration will not strengthen the positive TL, but is required to make the correction.

The other consistent feature is the presence of cross therapy localization most often at the region of the ileocecal valve region (at McBurney's point) if the condition is present. The normal TL pattern of each finger tips over this point with the right on the right and left one the left will be negative. If you have the patient place one hand over the other one way will show a weakness. If the right hand is on the bottom, then the left supraspinatus will be weak and if the left hand is on the bottom then, the right supraspinatus will be weak.

Findings

The study over the past tow years has consisted of a total of 604 patients, 152 males and 452 females. Anytime that a positive GV27/CV24 therapy localization or a cross TL pattern is found there is also the LD cranial fault that accompanies this pattern. When I found this situation I would ask the patient if they were having any memory problems or depression. Almost all had some problem usually minor with memory or said that they could not think clearly or concentrate properly. When inquiring about medication I would be able to get positive response about the use of the SSRI drugs, which usually surprised the patient that I would know this from performing this test.

Note

The above mentioned method of therapy localization I described was presented in research paper titled "Ileocecal Valve Syndrome a Universal Problem" published in 1989. I had noticed that many patients with this problem were missed by the standard method of TL. What I originally thought was this was double therapy localization away of increasing the neural signal. However, I have since discovered that it is a method of cross therapy localization. In other word if you place the right hand fingers to the left side and the left hand fingers to the right side of McBurney's point it will give you a positive response causing a strong muscle to weaken.

This pattern of TL I have been observing for the past 17 years on tens of thousands of patients, but did not realize its significance to cross therapy localization (CTL) until about three years ago. I also did not see it connection to LD fault and depression until about two years ago.

Nutritional and drug testing has been done against the supraspinatus weakness, against the positive TL to GV27 and CV24, and also to the LD cranial fault. All are negated by the amino acids, L-Phenylalanine, L-Tyrosine and L-Tryptophan, precursors for the neurotransmitter dopamine, serotonin, norepinephrine and epinephrine. These factors can also be tested against drug therapy such as SSRI and MAO inhibitors and they will negate to the correct drug.

Conclusion

This cranial fault is definitely a constant in neurotransmitters problems of serotonin, dopamine, norepinephrine, and epinephrine within the brain. It can be used to judge the correct treatment for depression based upon the response to muscle testing. This is based on the fact that proper prescribed drug or nutrient will work 50% of the time if given on a general symptomatic basis. A more objective method should be used this was pointed out by a study by Ian a. Cook, M.D., done at UCLA Medical School in Los Angles, CA. Dr. Cook using EEG on unipolar depression using two SSRI fluoxetine or venlafaxine vs. placebo. An electrical change could be measured 48 hours after the drugs were taken in the pre-frontal and temporal regions of the brain in the group that should clinical response, but not in the group taken the placebo. The most important fact this study made is that the batting average is 50% in random prescription and 38% improvement with a placebo. Since it takes sometime 4 to 6 week to get a clinical response a lot of time and money is being wasted if the patient does not improve with treatment.[16]

Since most doctors in general practice or for that matter most psychiatrists do not have an EEG in their office a simpler method of using a more objective means to get the right medication. Use muscle testing while TL to the cranial fault (LD), or to GV27 & CV24 simultaneously to see what medication neutralize the test muscle weakness. Or you can see which medication negates the weakness of the supraspinatus muscle, which is always weak in the depressed patient.

I will finish with a short case history of the effectiveness of using this method. In December of 2004 a long-term (15 years) patient came in for reoccurring headaches. She is 42 years old and generally in good health. On examination an open ileocecal valve syndrome was diagnosed, (this has headaches as one of its many symptom patterns).

What I found was a positive the cross therapy localization to McBurney's point, which indicated the presence of the three described patterns and the LD fault. I knew she had no learning disabilities or ADD because she had an MBA for the Wharton School of Business. I questioned her asking if she was experiencing any memory problems and she said that this has been going on for the past few weeks. I also told her that I had found a relationship of this to SSRI drugs. She said she was taking Lexpro for PMS symptoms and was surprised that I knew this from my examination.

She also told me that her doctor had changed her medication for no apparent reason to Effexor, which she said was not working, in fact she stopped it because she was having suicidal thoughts. I asked her to bring in the medication so I could test her on them. What I found was the Effexor caused a universal muscle weakness to any test muscle, which meant that it was not compatible with her body. On the other hand the Lexpro negated all positive findings, which indicated it was the correct medication. This problem could have been avoided if her physician was using muscle testing to check the prescription instead of using the empirical method to treat symptoms. This can avoid iatrogenic problems for the patient and the legal ramifications of the doctor.

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Ankle Sprain and Related Injuries, Part I: An Applied Kinesiology Perspective

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Abstract

The author's clinical experience treating patients with acute, traumatic ankle inversion sprain and relevant literature are reviewed. Specific Applied Kinesiology (AK) diagnostic and treatment protocols are recommended. Correlations to other lower extremity injuries are made.

Key Indexing Terms
Ankle sprain, ankle injuries, talus, foot injuries, muscles, ankle joint, lower extremity, ligaments, joint instability, bone malalignment, Applied Kinesiology, tibia, fibula, tarsal bones, ankle, foot.

Introduction

The foot and ankle are an architectural and biomechanical masterpiece. Clinically, they deserve thorough evaluation and relentless attention when needed. This paper differentiates the joint and soft-tissue injuries following ankle inversion injury from the more common eversion injuries and subluxations that mainly result from extended pronation. With an understanding of the inversion related injury one can explore and direct manipulative and other therapies more specifically. In the author's experience this dramatically decreases acute and sub-acute healing time and allows for improved results following the repair and remodeling phases of healing. Related injuries are also considered for the purpose of supplementing the practitioner's clinical discernment regarding case management.

Discussion

The foot is made up of twenty-six bones which form the ankle, top and bottom of the foot, and toes. These bones are articularly specialized, allowing a wide range of flexibility, while being able to withstand the incredible amounts of stress placed upon them. It is estimated that each stride of an adult places approximately 900 pounds per square inch on the bottom of the foot.[1] Seven of these bones form the compact arrangement of the ankle, or tarsus, and the heel. Ligaments connect the bones of the foot together and allow the muscles of the calf to remotely influence these bones.

Ankle Inversion Sprain Definition: A traumatic injury usually consisting of extreme ankle inversion and plantar flexion with the resultant stretching, tearing, or rupture of the **lateral** ankle ligaments.[2] The patient presents with ankle swelling, tenderness, and loss of function and may have heard an audible "pop" during the injury. Most common is injury to the anterior talofibular ligament which can demonstrate lack of integrity during anterior drawer test of the ankle. In about twenty percent of cases the calcaneofibular ligament is also injured, exhibiting abnormal movement during the talar tilt or inversion stress test. Rarely, the posterior talocalcaneal ligament is involved. Inversion, or "lateral" sprains represent **80–85% of ankle sprains.**[3]

Ankle inversion sprain grading:[2]

A. First degree lateral ankle sprain

1. Mild pain and swelling (possible to walk)
2. No joint instability
3. Anterior talofibular ligament stretched
   - Localized tenderness on palpation

B. Second degree lateral ankle sprain

1. Moderate pain and swelling with bruising
2. Pain with walking
3. Moderate lateral ankle instability
4. Partial tear of anterior talofibular ligament
5. Snapping or tearing sensation felt during injury

C. Third degree lateral ankle sprain

1. Severe bruising and swelling (>4 cm at fibula)
2. Unable to bear weight
3. Severe lateral ankle instability
4. Total disruption of lateral ligaments
   a. Anterior talofibular ligament
   b. Calcaneofibular ligament
5. Audible "pop" with immediate pain and swelling

Note: *The mechanism of injury involved in the lateral sprain can also cause a* ***fracture of the distal fibula****. Any sprain with severe swelling, pain, and/or local crepitus should be X-rayed to rule out fracture. Delayed healing with persistent symptoms beyond six weeks, including crepitus or locking or catching sensation, may also require evaluation for the less common fractures of the talus, navicular, calcaneus, or fifth metatarsal.*[2, 3]

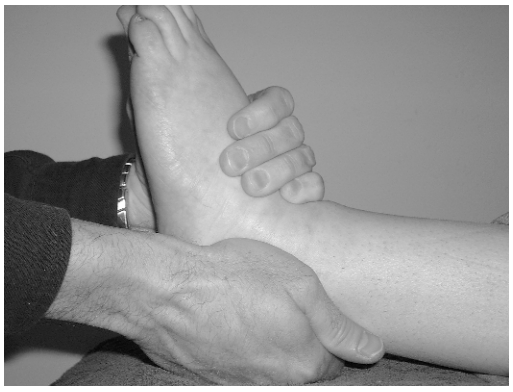
Inversion sprain can also rupture the peroneal ligament allowing the ***peroneal tendon*** *to escape from its bony groove. The condition can become chronic and surgical repair is often required. Characteristic symptoms include pain over the peroneal retinaculum, located on the lateral calcaneus inferior to the subtalar joint.*[2] *Inversion, eversion, and manual muscle testing of the peroneal muscles, especially the tertius, may elicit acute pain or subluxation of the peroneal tendon.*

Chiropractic and AK treatment considerations:

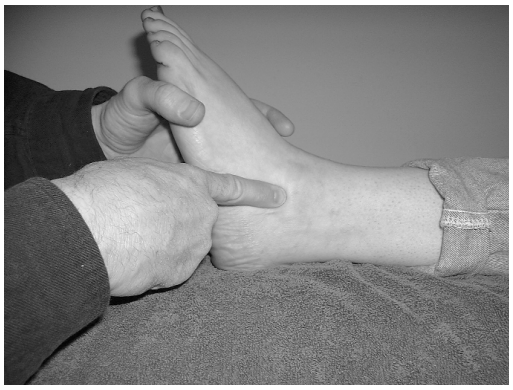
Chiropractically, the lateral talus subluxation is the primary bony consideration for the lateral sprain.[4] This is in contrast to the medial talus commonly observed in the patient with extended pronation tendencies.[5] The severity of inversion involved in this traumatic injury creates a tremendous lateral and

somewhat anterior force against the talus. The talus bone has no muscles attached to it, only ligaments. The lateral ligamentous structures alone are not strong enough to prevent significant lateral subluxation of the talus, as well as soft-tissue injury.

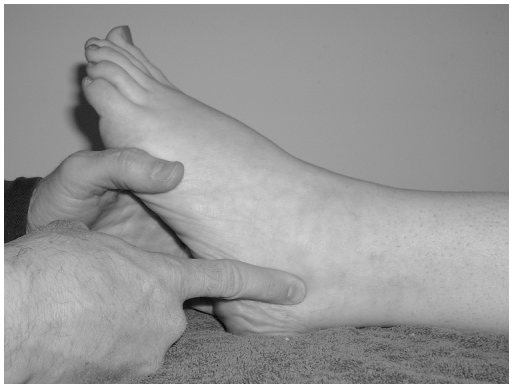
1. Adjusting the **lateral talus** subluxation
   a. Patient supine, doctor standing at patients feet contacting inferior to the lateral maleolus with thenar eminence and ipsilateral fingers wrapping around posterior calcaneus; doctors other hand is over dorsum of foot with fifth finger just under distal-anterior tibia
   b. With the patient's foot in slight dorsiflexion or neutral the doctor slowly tractions the ankle joint inferiorly then delivers a traction impulse straight inferior. *The corrective line of drive is provided by the constant lateral-to-medial pressure of the doctor's thenar eminence against the talus*.[4] (see picture #1)



1. Lateral Talus Adjustment



2. Anterior Talo-fibular ligament



3. Calcaneo-fibular ligament

   c. This adjustment can be performed (even during the acute injury phase) as soon as the possibility of fracture has been ruled out

2. Treat any muscle dysfunction
   a. Peroneal muscles are typically strained and potentially require any of the IVF 5 Factors (primarily the neuro-lymphatic reflex), golgi-tendon organ, muscle spindle cell, origin-insertion, and/or myofascial release techniques to correct
      - muscle stretch reaction initially is too painful to test and may not be positive until two-four weeks after injury when the scar tissue and myofascial adhesions in the lateral compartment are significant enough to create shortening
   b. Medial head of gastrocnemius often needs strain/counterstrain procedure once patient is able to invert and dorsiflex the ankle without pain[6]
      - this may be performed without inversion initially in order to obtain at least partial improvement

3. Rest, Ice, Compression, and Elevation (R.I.C.E.), high-dose proteolytic enzymes and antioxidants are recommended during acute phase (or until all swelling and bruising are eliminated)

4. Treat tender lateral ankle ligaments
   a. The following ligaments may be treated directly with low level laser therapy (low pulse setting initially for anti-inflammatory effect), topical homeopathic arnica cream, ice therapy, and/or ligament interlink technique. Treatment should continue as long as there is tenderness on palpation and/or positive therapy localization to the ligament
      - Palpate the **anterior talofibular ligament** immediately anterior to the distal fibula (see picture #2)

- The **calcaneofibular ligament** is found posterior and inferior to the lateral maleolus (see picture #3)

5. AIRCAST, range of motion activities (especially dorsiflexion), and walking after approximately three days[2, 3]
   a. Weight-bearing activities may be limited for two to three weeks due to discomfort
   b. Slowly progress to normal activities only after walking is painless

6. Proprioceptive exercises such as wobble board, and stretching (especially dorsiflexion) will help prevent re-injury in the long term[3]

7. For the long term consider soft-flexible orthotics casted while weight-bearing, possibly with the addition of a lateral posting wedge under the second through the fourth metatarsal heads to increase lateral ankle stability[7]

Patients who experience repeated inversion sprains will usually display signs of peroneus longus, brevis, and/or tertius inhibition:

1. Signs of peroneal muscle weakness[5, 8]
   a. Foot turned in during gait
   b. Lateral ankle instability when weight is shifting to front of foot during beginning of gait
      - Observe for lateral wiggle of ankle here
   c. Excessive foot supination during swing phase of gait

2. Considerations[5, 8]
   a. Taping or other non-elastic support just below the head of the fibula to approximate tibia and fibula often strengthens the peroneal muscles
   b. Tertius, then brevis, then longus contract sequentially as the heel is lifted from the ground during gait
   c. Tertius gives lateral stability at heel strike
   d. Tertius weakness is often related to lateral cuboid subluxation

Conclusion

Severe lateral ankle sprains may require months for full recovery. First and second degree lateral sprains progress much quicker. In the authors experience the above diagnostic and treatment recommendations afford above average results in all categories of ankle sprains, often eliminating long-term dysfunction. For all patients, especially the elite athlete, Applied Kinesiology methods, thoroughly performed, are more comprehensive and offer better and faster results than conventional protocols alone.

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Ankle Sprain and Related Injuries, Part II: An Applied Kinesiology Perspective

Barton A. Stark, D.C., DIBAK, DIAMA

Abstract

A review of the literature regarding acute, traumatic medial ankle sprain is presented and expanded based on the author's clinical experience. Correlation to the high ankle sprain is clarified. Specific Applied Kinesiology (AK) diagnostic and treatment protocols are recommended and differentiated from those for the lateral ankle sprain.

Key Indexing Terms
Ankle sprain, ankle injuries, talus, foot injuries, muscles, ankle joint, lower extremity, ligaments, joint instability, bone malalignment, Applied Kinesiology, tibia, fibula, tarsal bones, ankle, foot.

Introduction

With approximately 23,000 per day in the United States, ankle sprains are considered the most common acute injury for all levels of athletics.[1] This paper differentiates the medial ankle sprain and high ankle sprain from the ankle inversion injuries and subluxations considered in **Ankle Sprains…Part I**, by the same author (in press). These ankle injuries generally require a much longer healing process than the more common inversion injuries and if not treated properly can even lead to arthritic changes in the ankle.[1] With an understanding of the eversion related injury one can explore and direct manipulative and other therapies more specifically. In the author's experience AK methods decrease acute and sub-acute healing time and allow for dramatically improved results following the repair and remodeling phases of healing.

Discussion

Medial Ankle Sprain Definition: A traumatic injury consisting of ankle eversion with the resultant stretching, tearing, or rupture of the **medial** ankle ligaments and other soft-tissue structures.[1]

The patient presents with ankle swelling, tenderness, and loss of function and may have heard an audible "pop" during the injury. Most common is injury to the **anterior talofibular ligament**, the **interosseus ligament**, and/or the **deltoid ligament**. Major trauma is usually required to produce the medial (eversion) sprain, thus their occurance is rare.[2]

Ankle syndesmotic sprain, or **"high ankle sprain,"** accounts for ten percent of ankle sprains. It is an injury to the anterior–inferior tibio-fibular ligament and the interosseus membrane. This type of sprain should not be confused with medial or lateral sprains. The mechanism of injury here is eversion, rotation, and hyper-dorsiflexion, exactly opposite that of the inversion sprain. Signs and symptoms may include **medial** ankle pain, difficulty bearing weight, minimal swelling, pain and disability out of proportion with the injury, spongy feeling in the ankle, tenderness over the anterior and proximal ankle, and pain at the distal tibiofibular joint.[2]

X-Ray: The high ankle sprain involves tibiofibular clear space widening >6 mm on anterior to posterior X-ray.[2]

Orthopedic tests: positive Squeeze test and/or External rotation test indicate high ankle sprain. The Squeeze test is performed by pressing the distal tibia and fibula together with the ankle in slight dorsiflexion. It is considered positive if this elicits pain between the tibia and fibula. The external rotation test involves stabilizing the distal tibia and fibula with one hand while externally rotating the foot with the doctor's other hand. Pain during this maneuver indicates sprain of the syndesmosis of the distal tibia and fibula.[2]

Chiropractic and AK treatment considerations:

For the high and medial ankle sprains thorough AK evaluation should focus on the major medial ankle stabilizer: **tibialis posterior**. In the high sprain, for example, the pinnate fibers of tibialis posterior help to prevent separation of the tibia and fibula.[3]

Signs of tibialis posterior weakness include: extended pronation, flexor hallucis brevis weakness with weight-bearing, foot externally rotated at end of swing phase of gait, and tenderness on palpation at lateral calcaneus, medial knee, greater trochanter, lumbar paraspinal muscles, rhomboids, anterior scalenes, and/or pterygoid muscles which improves with adequate support under navicular. The posterior tibialis is the most common lower leg muscle weakness.[4, 5]

The medial talus subluxation is common in simple cases of extended pronation.[4, 5] The medial ankle sprain, however, may produce a lateral talus subluxation, especially in the presence of the Pott's fracture.[2] Challenge the talus from various directions to determine the corrective vector. ***Before correcting a talus subluxation after medial sprain the practitioner must rule out the Pott's fracture of the distal fibula***.

In the previous paper **Ankle Sprains…Part I,** by this author (in press), AK case management recommendations were made for lateral ankle sprains. These protocols are amended for the medial and high ankle sprains in the following summary:

1. Adjust the **talus** subluxation
2. Treat any muscle dysfunction, especially tibialis posterior
3. Natural anti-inflammatory treatments until all swelling and bruising are eliminated
4. Treat tender ankle ligaments: **anterior talofibular, interosseus,** and/or **deltoid ligaments**
5. AIRCAST and range of motion activities throughout healing phases
   - For high sprain: disregard early ROM activities
6. Proprioceptive exercises, conservative stretching, and soft-flexible orthotics for long term support and prevention
7. Taping or other non-elastic support just below the head of the fibula to approximate tibia and fibula may help prevent further disruption of the tibiofibular syndesmosis. At least three layers of cotton athletic tape can be applied with light to moderate tension based on patient comfort

Conclusion

The medial and high ankle sprains may require months for full recovery. In the authors experience the above diagnostic and treatment recommendations afford above average results, often eliminating long-term dysfunction. Conventional treatment modalities are often limited and may offer a bleak prognosis for these injuries. The Applied Kinesiology methods discussed in this paper represent the spirit of AK which applies clinical and academic competence to unleash the bodys' innate ability to heal itself.

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Nose Adjustment Technique

Sue Tusiri, D.C.

Abstract

The purpose of this paper is to demonstrate an effective technique used to adjust a dislocated nose which may have been caused by trauma. Sinus congestion, breathing difficulty, and allergies have been improved after this technique.

Introduction

After over 10 years of treating patients with Applied Kinesiology, I had become competent at adjusting almost every part of the body… except the nose.

A 42 year old female had complained of chronic sinus congestion, difficulty breathing and allergies. She had sustained several physical traumas, including a dislocated nose after an accident 20 years ago. Since then she could only breath through one of her nostrils. I had adjusted virtually every joint of her body except her crooked nose. I decided to challenge her nose with muscle testing and eventually performed a respiratory adjustment on her nose.

Immediately following treatment, she told me that she could breath through both of her nostrils. After a few nose adjustments her sinus congestion, breathing difficulty, and allergies have gone. Also her nose had appeared more visually straight. After that, I began checking the rest of my patients, even those who had no obvious physical deviation of their nose. I found this to be a very common subluxation.

Methods and Summary of Procedures

1. Begin with a muscle which is neurophysiologically intact (strong indicator muscle).
2. Perform a static challenge on the osseous portion of the patient's nose with a thumb and index finger (pinch) contact.
3. The challenge is performed in both a clockwise and counterclockwise direction.
4. While maintaining the static challenge in the direction that created a conditional inhibition of the strong indicator muscle, determine the phase of respiration that negates the challenge.
5. Correction is performed in the direction of strength on the phase of respiration that negated the challenge. The correction should be performed 3–5 times.
6. Recheck the patient by statically challenging the nose in any of the four primary vectors and correct as detailed in #5 above.
7. After the correction has been performed the strong indicator muscle should maintain strength in any vector of challenge. If not, repeat this procedure until the static challenge creates no further weakness of the strong indicator muscle.

Discussion

The Nose Adjustment Technique has been shown to restore proper physiologic function to the nasal passage. Potential future pursuits as to the far reaching effects of this Nose Adjusting Technique could include pre and post treatment allergy studies as well as pre and post treatment air flow studies.

Conclusion

Physical trauma has the potential to cause nose misalignment which can produce many respiratory symptoms such as: sinus congestion, breathing difficulty, asthma, allergies, and even migraine headaches. This Nose Adjusting Technique is a simple technique that can help restore normal physiologic function in a very time efficient manor with sustained results. Therefore, the Nose Adjusting Technique can get rid of all the above symptoms if they were caused by a nose misalignment.

Tendon Adjustment Technique

Sue Tusiri, D.C.

Abstract

The purpose of this paper is to demonstrate a technique used to adjust dysfunctional tendons of any joint. Applying this technique has been shown to relocate aberrant joint mechanics, thus reducing pain and associated crepitus. It also vastly improves ranges of motion of the joints.

Introduction

Din Lim is a legendary bone setter in Thailand. He is a 90 year old man whom people travel from all over the world to be treated by. He is also a special instructor for several medical schools in Thailand. After hearing of the profound results he was creating, I decided to travel to Thailand to investigate his techniques. I was intrigued by a unique approach to manipulating tendons that seemed to create immediate, profound results. He described his maneuvers in getting rid of pain in any joint of the body by realigning the tendons and pulling them (which "got pushed deep down") up.

After being treated by Din Lim I was amazed in the way that all my ankle, knee, hip, shoulder, neck, and back pain had gone after his treatment. I had received several more treatments while learning his approach as it was very different from traditional Thai massage. Upon returning to the states I correlated what I had derived from his technique with what I had understood from 10 years in Applied Kinesiology.

The result is a new technique "**Tendon Adjustment Technique**" After using it in literally hundreds of patient visits for dysfunctions in shoulders, elbows, wrists, hips, knees, ankles, sacroiliac joints, and the spines and the results have been immediate and inspiring.

Methods and Summary of Procedures

1. Begin with a "strong indicator muscle."
2. Confirm the need for the Tendon Adjusting Technique by therapy localization to the suspected joint. When dysfunction is present a "strong indicator muscle" will become conditionally inhibited (weak) during contact with the dysfunctional joint.
3. After determining where therapy needs to be administered the appropriate vector needs to be delineated. Place the joint in a relaxed position as to not have the associated muscles in stretch during the diagnostic phase. The treatment is then administered in the direction that maintains strength during a static challenge.
4. Treatment is done by using the fingers to push the affected tendons in the direction that maintained strength while gently flexing the joint and moving it through its pain-free range of motion. This activity is repeated 3–5 times while the practitioners palpating hand is feeling for the joint to relax and typically spontaneously correct (adjust).
5. Evaluate the joint opposite the treated joint as it will often also need correction.
6. Other painful joints may be treated during the same office visit if appropriate.

Discussion

The Tendon Adjustment Technique helps restore normal joint mechanics by balancing aberrant muscle and tendon tensions in that joint. Since muscles and tendons move bones, dysfunctional muscles and tendons move joints in dysfunction. Therefore, after utilizing this technique, the painful/dislocated joint that was held in dysfunction by those tendons virtually self correct.

Conclusion

The Tendon Adjusting Technique is a very powerful, effective, and painless procedure. It has been successfully used to treat all joints in the body (shoulder, elbow, wrist, hip, knees, ankle, sacroiliac, and the spine). This technique is very safe and gentle. Therefore, it works well for patients who have chronic joint problems and or patients who may not well tolerate a forceful adjustment. Patients often feel immediate relief of pain and crepitus as well as improved range of motion right after the treatment. For patients with more chronic problems, more treatments will be needed.

Division III

Constructive Review

Off-Label Marketing and Prescribing: How Chiropractic Patients with Restless Legs Syndrome End Up Taking Anti-Parkinsonian, Anti-Epilepsy, Muscle Relaxers, or Depressant Drugs for RLS

Scott Cuthbert, D.C.

"The hand that stocks the drug stores rules the world." – Bokonon
— (From *Cat's Cradle*, By Kurt Vonnegut)

While writing the paper *Restless Legs Syndrome: A Case Series Report* for this year's ICAK Collected Papers, I was amazed when I realized that 6 of the patients from that case series reported that they had been given drugs designed for the treatment of other diseases (Parkinson's disease, painkillers, and seizures) for the treatment of their RLS.

These were examples of a worrisome tactic of the Pharmaceutical Companies (Big Pharma) and medical doctors called "Off-Label Marketing and Prescribing." I have subsequently explored this issue and want to discuss it here.

From the Food and Drug Administration (FDA):

> "Off-label marketing and prescribing is the recommendation of a medication in a different dose, for a longer duration of time, ***or for a different medical indication than recommended in the prescribing information.*** Although the Food and Drug Administration approves a drug for a specific indication, the physician has discretion to prescribe it as indicated above, or in combination with other medications in the treatment of patients.
>
> ***In 1998, the agency proposed an amendment to the*** **FDA Modernization Act** ***that allows dissemination of information by manufacturers about unapproved uses of drugs and medical devices***. The act allows a firm to disseminate peer-reviewed journal articles about an off-label indication of its product, provided the company files a supplemental application based on appropriate research to establish the safety and effectiveness of the unapproved use."[1]

"Off label promotion" is the term used for a pharmaceutical company's advocacy of a prescription drug for a use that the FDA has not specifically approved. This type of advertising often involves the distribution of medical journal articles that discuss such uses – Sinemet or Levodopa for RLS, or Paxil for shyness, for example – with the intent of subtly encouraging physicians to, let's just say it, experiment.

Off-Label Marketing and Direct To Consumer (DTC) advertising really shifted into high gear in 1999, when a district court judge finally ruled on the original off-label marketing case, to the favor of the Washington Legal Fund (and, by monetary daisy chain, Wiley, and the American Association of Advertising Agencies, or AAAA). The key to the win was the "antipaternalism argument" that, in the U.S. Supreme Court, Justices Scalia and Thomas disliked so much. Paternalistic governmental policies, according to the district court judge "cannot justify a restriction of truthful, non-misleading speech on the paternalistic assumption that such restriction is necessary to protect the listener from ignorantly or inadvertently misusing the information," the court wrote.[2]

Of course, that "non-misleading" part – that could be tricky.

Should a drug company be able to distribute, via highly trained medical affairs people, studies showing that a drug for one approved purpose also "seemed" to help kids with, say, stage-four brain cancer, an unapproved use? The average reader would likely say yes.

But should a drug company also be allowed, as has been the case with antidepressants, to dispatch tens of thousands of young, barely trained sales reps, most just out of college, to hand out to general practitioners, with no experience in psychiatry, studies that "suggest" that adult antidepressants "might" help kids with depression? The murk blossoms anew.

The FDAs traditional response before 2000 was to come down hard on the latter cases.

After the case that sanctioned Off-Label Marketing in 1999 came George W. Bush who (like Justices Scalia and Thomas) brought with him no small contempt for corporate regulation, restrictions, and, of course, plaintiffs' lawyers.

One good indicator of Big Pharma's success in toppling the FDAs restrictions on Off-Label Marketing is Neurontin. I have personally been keeping track of a good number of patients who were put on Neurontin for extraordinarily diverse conditions among my new patients in the past year. They were put on the drug for conditions that applied kinesiology chiropractic therapy was able to cure. Neurontin is an older drug indicated for epilepsy. Neurontin is now one of Big Pharma's biggest billion-dollar babies. The reason: almost 80% of its sales come from off-label uses, everything from bipolar disorder to chronic pain to RLS attention deficit disorder.[3]

Neurontin had been a reliable but modest performer in Parke-Davis' anti-epilepsy cabinet. This was because Neurontin was approved only for adjuvant therapy, for people suffering from seizures that were not well controlled by conventional seizure medications. That made its market small. It was approved only in relatively low doses, further shrinking its potential. The drug – its chemical name was gabapentin – was also intriguing to a number of people in the sales department at Parke-Davis. For years they had been hearing a small but steady number of comments from some doctors who had experimented with the drug off label, using it to treat neuropathic pain, bipolar disorder, attention deficit disorder, as monotherapy for seizures (instead of as an adjuvant) – even for migraine, ***restless legs syndrome***, and alcohol and drug withdrawal.[3]

(See the paper on RLS in this volume in the section "**Medical Treatment of RLS**")

Before the 1998 court ruling, the question was: How could Parke-Davis increase its sales without overtly promoting its off-label use, which was illegal? "Change the law" has been Big Pharma's method from the beginning and, as in any marketing-driven enterprise, once the gray areas are banished, color emerges, first at the edges, then at the red-hot center.

At a recorded meeting of marketing managers at Parke-Davis, executive John Ford stated the company's expectations in the new world of unregulated Big Pharma DTC advertising:

> "I want you out there every day selling Neurontin. (Read off-label drugs for insomnia and RLS here.) Look, this isn't just me, it's come down from Morris Plains [Parke-Davis headquarters] that Neurontin is more profitable than Accupril so we need to focus on Neurontin...We all know that Neurontin's not growing for adjunctive therapy [its approved indication]. Besides that's not where the money is. Pain management, now that's money. Monotherapy, that's money.

> "We don't want to share these patients with everybody, we want them on Neurontin only. We want their whole drug budget, not a quarter, not a half, the whole thing…we can't wait for them to ask, we need to get out there and tell them out front. Dinner programs, CME (continuing medical education) programs, consultantships, all work great, but don't forget the one-on-one. That's where we need to be, holding their hand and whispering in their ear, Neurontin for pain, Neurontin for monotherapy, Neurontin for bipolar, Neurontin for everything…I don't want to see a single patient coming off Neurontin before they've been up to at least 4,800 milligrams a day.
>
> "I don't want to hear that safety crap either – every one of you should take one just to see there is nothing. It's a great drug."[3]

"Gloom and doom," one might safely say, are things of the past for Big Pharma today. Increasingly, Big Pharma has converted government regulatory agencies into subsidiaries of the companies. The Food and Drug Administration routinely allows researchers with ties to the industry to sit on drug approval advisory committees. In many cases, half the panelists on such committees have a financial stake in the outcome, through links to the drug manufacturer or to a competitor.

The "consumer's right to know" – Ralph Nader's great triumph of the 1970s – has become the corporation's right to "subtly encourage." Put another way, the reigning governmental, legal, and political ethos about prescription drug promotion has morphed from "patient trust thy doctor" into "buyer beware."

Off-Label Marketing and DTC advertising are no longer just about giving patients useful information. Have you seen the commercials in the past 10 years offering Paxil for treatment of social anxiety disorder, promising to "help you become yourself again"? Talent agencies have begun packaging celebrities with pills. The tennis star Monica Seles, apparently employing a long-hidden expertise in brain chemistry, became the spokeswoman for prescription migraine pills. Lynda Carter, once known for portraying Wonder Woman, now can deploy her superpowers to talk up drugs for irritable bowel syndrome. Ads for Viagra show fat, balding men partying in the street. Celebrex spots show arthritics dancing in the street. There are ads for GERD and ads for toenail fungus relief. There are ads for Claritin that have no information at all – just pretty people running through pretty fields of flowers. *Happy Days*' Tom Bosley offers us homely pharmaceutical advise on mental health issues.

Skating star Dorothy Hamill was drafted for a campaign promoting Vioxx for osteoarthritis. "This is my favorite time to skate," Ms. Hamill said in a commercial. "I guess it's from all those years of five A.M. practices. But it's also the time when the pain and stiffness of osteoarthritis can be at their worst." The result was that a new, expensive drug once intended for a fairly small population became the expensive default prescription for most forms of adult arthritis pain, while many less expensive and equally effective drugs were submerged. All until October 2004 when one more daily drug was consigned to the national apothecary of harm. The rest of Vioxx's history you know.

As part of the new American corporate pattern, in June 2002, the *New England Journal of Medicine* made a startling announcement. The editors declared that they were dropping their policy stipulating that authors of review articles of medical studies could not have financial ties to drug companies whose medicines were being analyzed.

The reason? The journal could no longer find enough independent experts! Drug company gifts and "consulting fees" are so pervasive that in any given field, you cannot find an expert who has not been paid off in some way by the industry. So the journal settled for a new standard: Their reviewers can have received no more than $10,000 from companies whose work they judge. Isn't that comforting?

Big Pharma's growing "teaching" function can now play large at major medical association conferences. The industry provided 57 percent of the revenue for the nation's 686 accredited continuing medical education (CME) providers, a whopping $720 million in 2002 alone. That same year saw another important shift as well: drug companies spent more money on advertisements in newspapers and magazines than they did in medical journals.[4]

The FDA and organized medicine are not the only culprits in Off-Label Marketing and DTC advertising.

The White House (which appoints the officials who are supposed to oversee the process), and the Congress (which funds the FDA and holds subpoena power for all Big Pharma records), are supposed to regulate what the FDA does. The federal government is part of what passes for this nation's pharmaceutical condom. In election after election, and in legislative session after legislative session, Big Pharma and brand name companies have pumped huge amounts of cash into the Grand Old Party.

Interestingly, Alexander Strategy Group, based in Georgetown, a giant among the Washington lobbying corporations for Big Pharma, was shut down in January 2006 because of its close ties to lobbyist Jack Abramoff and former House Republican majority leader Tom DeLay. Alexander Strategy Group was the "Washington arm" of the Pharmaceutical Research and Manufacturers of America (PhRMA).

Former Tom DeLay aide Edwin A. Buckham, who manages the Alexander Strategy Group, told the *Washington Post*: "the company was fatally damaged by publicity about the ongoing federal investigation into the affairs of Abramoff, who pleaded guilty last week to fraud and conspiracy charges." (August 11, 2005) Buckham said that his former boss, Tom DeLay, is one of several lawmakers under scrutiny in the Abramoff case.

Another interesting angle on all of this is provided by Dr. Joseph Mercola on his well known website www.Mercola.com. Type in the search "Donald Rumsfeld" there and you will find many interesting and on-going connections between the present Secretary of Defense and Big Pharma. The present Secretary of Defense is a past CEO of G.D. Searle, Inc., makers of a wide variety of drugs and chemicals, including the anti-inflammatory we all know, Celebrex.

If this isn't enough, go on line to: www.citizen.org/documents/Medicare_Drug_War%20_Report_2004.pdf. This is the most thorough "exposure" of the penetration of the United States government by Big Pharma that I have yet seen, from the public interest group *Public Citizen*. I have used Public Citizen's book *Worst Pills, Best Pills* for years to demonstrate for patients the dramatic, worrisome effects of the medications they are taking. That book has been very helpful for me as I move patients from a pharmacological to a natural and physiological point of view.

In 2000, the CEO of Bristol-Myers Squibb sent messages to all of his company's top managers to "donate the maximum" to the Bush campaign, $1,000 individually and $1,000 in their spouse's name; the company itself ponied up $2 million.[5] By 2002 there were six Big Pharma lobbyists for every sitting U.S. senator, a kind of political day care for potentially errant legislators. Among the 600 full-time lobbyists, 24 of them were former members of Congress.

Our view into the extent of Big Pharma's influence within the U.S. government and its regulatory agencies came into sharp focus with the *Medicare Prescription Drug Act* (enacted December 8, 2003), which the industry was able to rewrite to forbid the federal government from negotiating for lower drug prices – something citizens of every other industrialized democracy (and many undeveloped ones as well) take for granted. That the congressman who shepherded the bill is now the $2-million-a-year president of Pharmaceutical Research and Manufacturers of America (PhRMA), the industry's big lobbying group, takes no one by surprise.

The American people buy drugs advertised for them on television (DTC and Off-Label Marketing strategies usually) at record levels now. Consider that the amount spent to advertise prescription drugs directly to consumers in 1980 was $2 million. In 2004, it was $4.35 billion and soaring. The number of Americans, annually, who request and receive a prescription drug after seeing a specific commercial for it is…8.5 million.

The average number of prescriptions per person, annually, in 1993 was seven. The average number of prescriptions per person, annually, in 2000 was eleven. In 2004, it was twelve. The total number of annual prescriptions in the United States now stands at about 3 billion. The cost per year? About $414 billion in 2001. There's another number to consider here: In 2004, almost half of all Americans used at least one prescription drug on a *daily* basis; amazingly, about one in six take three or more a day.[6]

Pretty soon, as the saying goes, and we are talking real money.

All of this connotes unseemliness at best and dereliction of duty or economic criminality at its worst. But is anyone really hurt by Off-Label Marketing and prescribing?

Certainly the answer is yes. Today the FDA says that at least 8,000 people a year become seriously ill because of it. Since physicians report only about 1 in 10 such instances to the agency, the figure is in reality more like 80,000.

My interest in discerning the implications of off-label prescribing for my own patients drove me to investigate a recent, comprehensive medical textbook, called *Drug-Induced Liver Disease*. A chapter by Dr. Neil Kaplowitz, coeditor of the book, has even more fun news for us. "In the United States drug-induced liver disease is the most common cause of acute liver failure, a more frequent cause than viral hepatitis and any other single cause." Elsewhere he says, "The frequency and economic impact is a major problem for the pharmaceuticals industry and the regulatory bodies, especially since the toxic potential of some drugs is not evident in the clinical setting." "To identify acute liver failure (in a new drug) with 95 percent confidence would require 30,000 study patients." He notes that today most new drugs get by, thanks to the reforms discussed previously, with about 3,000.[7]

The list of liver damage alone from the pharmaceuticals our patients are taking is staggering. There are painkillers that cause acute hepatitis, blood pressure meds that block up bile ducts, antidepressants that cause hepatocyte damage, COX-2 drugs that run up liver enzymes and cause artery damage, and diabetes medications that inflame the liver so badly that people die. Several specific drugs that we have all seen our patients using – that are wildly popular – have substantial hepatotoxicity, but their merits have been judged to outweigh these deficits. COX-2 inhibitors like Celebrex (still on the post-Vioxx market) and Statins (among them Lipitor and Zocor) are two of the biggest-selling classes of chronic disease drugs, and liver testing is always recommended for both types. Many have been removed or "voluntarily" withdrawn from the market for causing liver damage, with some fanfare but rarely any criminal prosecution. Others are on the cusp of such an action.

As the Food and Drug Administration (FDA), the American Medical Association (AMA), and every leading medical institution in this country and abroad has noted — liver damage, once rare, is now the leading reason for withdrawing a drug, usually a new drug, from the market.[8]

Here is a list of eleven medications withdrawn from the market since 1997 because of serious, often lethal side effects:[9]

- **Vioxx.** Merck did a huge amount of direct and indirect advertising showing that Vioxx caused fewer gastrointestinal hemorrhages, hospitalizations, and deaths than the other anti-inflammatory drugs on the market. Merck had minimized the cardiovascular events in the clinical trial it had used to win FDA approval, a trial known as Vioxx Gastrointestinal Outcomes Research, or VIGOR. Vioxx may have caused upwards of 140,000 cases of heart disease and between 56,000 and 100,000 deaths. The case against Merck has just begun. Withdrawn September 30, 2004.
- **Rezulin.** Given fast-track approval by the FDA, the FDA later linked it to 63 confirmed deaths and possibly hundreds more. "We have real trouble," an FDA physician wrote in 1997, just a few months after Rezulin's approval. The drug wasn't withdrawn until 2000.
- **Lotronex.** Withdrawn after nine-months. The FDA had received reports of 93 hospitalizations, multiple emergency bowel surgeries, and 5 deaths.
- **Propulsid.** Linked to hundreds of cases of heart arrhythmias and 100 deaths.
- **Redux.** Taken by millions for weight loss after its approval in 1996, Redux was soon linked to heart valve damage and a disabling, often lethal pulmonary disorder.
- **Pondimin.** A component of Fen-Phen, the diet fad drug. Approved in 1973, Pondimin's link to heart valve damage and a lethal pulmonary disorder wasn't recognized until shortly before its withdrawal in 1997.
- **Duract.** A painkiller linked to severe, sometimes fatal liver failure.
- **Seldane.** America's top selling antihistamine for a decade, it took the FDA five years to recognize Seldane was causing cardiac arrhythmias, blackouts, hospitalizations, and deaths – and another 8 years to withdraw it.
- **Hismanal.** Approved in 1988 and soon known to cause cardiac arrhythmias, the drug was finally withdrawn in 1999.
- **Posicor.** Linked to more than 100 deaths due to life threatening drug interactions. An expert on the advisory committee said, "Posicor should not have been approved."
- **Raxar.** Linked to cardiac toxicities and deaths.

One drug side effect that every applied kinesiologist should be alerted to is with the Statin drugs, like Baycol. In 2000 Baycol, made by Bayer, was removed from the market for causing 31 deaths from rhabdomyolysis. All Statins carry some risk of "rhabdo," a sudden and devastating weakness of muscle tissue; as it turns out, the same Statin action that inhibits coenzyme A also suppresses other vital cell maintenance enzymes, particularly one that maintains muscle and heart tissue. Celebrex is also associated (as was Vioxx) with the same kidney damage, liver injury, nerve injury, depression, swelling, anxiety, hot flashes, ringing in the ears, increased sweating, elevated cholesterol, and other side effects of the older anti-inflammatories that they replaced like Motrin, Voltaren, Relafen, Orudis, and others. Today about 12 million Americans, as a result of DTC advertising, now take one of six approved Statins.[9, 10]

For a class of drugs that require daily, lifetime usage, there are surprisingly few studies of long-term use. Our early twenty-first century pharmo-capitalism doesn't invest in such questions: there is no money for seemingly obscure drug-safety issues. As Buckminster Fuller pointed out, the problem isn't production, but distribution. Enough new pharmaceuticals *are* being produced, but seeing that only the people who truly *need* them, and that the drug does not slowly kill the people who take them, is a problem capitalism hasn't solved.

Add to this one other cheery number: There are more than 106,000 deaths a year from serious drug reactions – from drugs that have been *properly prescribed* and taken. The JAMA study that reported this also tallied 2,216,000 severe medication reactions in U.S. hospitals annually. Considering that Vioxx alone may well have caused more than 100,000 deaths during its 6-year run, the figure may be low. Even then, such reactions are now among the top 10 causes of death in the United States. And that does not include deaths from overdose, drug abuse, or noncompliance.[11]

A large proportion of our patients are medical misfits who have already been to many other doctors and are therefore on a variety of prescription medications, desperate to find the royal road out of their physiological nightmare. Many of the medications they are taking are more harmful than helpful. I have come to the conclusion that prescription medication doesn't offer much hope for these people. I have learned from my immersion in applied kinesiology theory, art and practice that correcting the person's structural irritations to the nervous system and to their biochemistry is a key to restoring their inborn healing mechanisms. Prescription medications can, in some cases, be helpful, but they can never be better than the patient's own self-regulating, innate healing mechanisms.

The longer we study disease and its treatment from the medical paradigm, and health and its promotion from the chiropractic paradigm, the clearer our duty as chiropractic physicians in the world becomes.

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