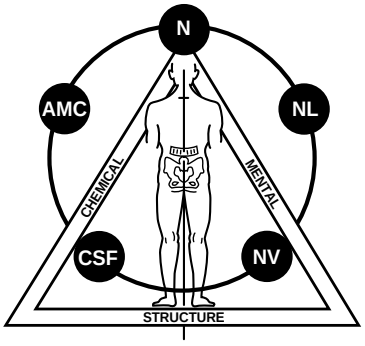
International College of Applied Kinesiology®-U.S.A.

Experimental Observations of Members of the ICAK

Volume I, 2003 – 2004

Proceedings of the Annual Meeting



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Volume I, 2003 – 2004

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Presented

June 26 – 29, 2003

Chicago, IL

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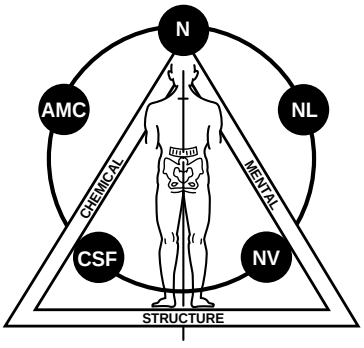

Message From the Chairman

David Leaf, D.C., DIBAK

The members of the International College of Applied Kinesiology®-U.S.A. are fortunate to share their insights, concepts and research through the papers presented in this issue of 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 "another piece of the puzzle," as Dr. Goodheart often says.

Thank you and congratulations to all of our contributors. And a special thanks to Drs. Rebecca Hartle, David Engel, Jan Calhoon and Alan Zatkin for all of their help during the review process. We look forward to seeing you at the Annual Meeting, June 26-29, 2003 in Chicago, IL.

Introduction

This forty-fifth collection of papers from members of the International College of Applied Kinesiology®-U.S.A. contains 45 papers (including 11 case histories) by 23 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).

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.

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.

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

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

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

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

Electronic illustration submission

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

Manuscript submission summary

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

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

Mailing instructions

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

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

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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 AK 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.

AK 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. AK 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 AK approach are specific joint manipulation or mobilization, various myofascial therapies, cranial techniques, meridian therapy, clinical nutrition, dietary management, and various reflex procedures. With expanding investigation there has been continued amplification and modification of the treatment procedures. Although many treatment techniques incorporated into applied kinesiology were pre-existing, many new methods have been developed within the discipline itself.

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

- Myofascial dysfunction (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 AK procedures does not necessarily equate with the practice of applied kinesiology as defined by the ICAK-U.S.A.

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

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

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* Does not conform to the ICAK Status Statement.

Division I

Informative Papers

Endometriosis; A Case Study

Glen P. Alis, D.C. and Supna Alis, D.C.

Abstract

The purpose of this paper is to demonstrate the effectiveness of applied kinesiology in the evaluation and treatment of a patient diagnosed with endometriosis. Modalities used were chiropractic adjustments, supplementation, correction of the ileocecal valve, balancing of the acupuncture meridian system, and stimulation of various neurolymphatic points.

Key Words: Endometriosis, Applied Kinesiology, Hormone Balancing, Manual Muscle Testing, and Nutrition.

Introduction

Endometriosis is a benign disease in which functioning endometrial tissue is present in sites outside the uterine cavity, attaching to the ovaries, the bladder wall, the intestinal walls, and membranes in the abdomen. The endometrial tissue responds to the monthly surges of estrogen by becoming blood filled, and at menstruation, when the uterine endometrium is shed, the endometrial islets also "shed" blood. The blood in the tissues creates local inflammation. Symptomatology includes pelvic pain, pelvic mass, alterations of menses, and infertility. The treatment protocol for this condition is to get pregnant, to have surgery to remove the scar tissue, or to start birth control pills to stop the menses which in theory prevents further scar tissue formation.

Hormonal imbalances found with endometriosis are consistent with Dr. Lee's findings about estrogen dominance. Estrogen dominant women are more likely to experience pain with menses, breast cancer, and ovarian cysts. To alleviate this condition, a patient needs to administer progesterone cream to decrease the effects of excess estrogen.

Discussion

A comprehensive history reveals a 25 year old female patient with severe abdominal pain and cramping with menstruation, especially in the lower right quadrant. She also experiences pain upon intercourse. She has experienced this pain during menses for two months. Moist heat and Advil provided minor relief, along with lying down and lumbar flexion. Standing up straight exacerbates the pain. The pain is rated 10 on a pain scale of one to ten (10 being the worst).

The patient first sought medical care and was diagnosed with endometriosis. Within two months, she chose to have a laproscopy surgery, as recommended by her gynecologist, to remove the endometrial and scar tissue. Six months following the surgery, the symptoms returned with the same severity. It was at this point that she turned to chiropractic care.

Menstrual cramps began since the patient started her cycle at the age of 12, but never this severe. At the time of examination, the patient's diet consists of 1-2 cups of coffee and 1-2 cans of diet cokes daily. She has a high carbohydrate and very low protein diet. She has moderate stress levels and does not exercise on a regular basis.

Examination reveals an open ileocecal valve, right piriformis weakness, subluxations of L3 and L5, and a sacral misalignment. Bilateral pectoralis clavicular and right sartorius weaknesses were also found. It was revealed by the patient that she experiences digestive disturbances and is extremely sensitive to light. Blood pressure taken from the lying to standing positions showed a drop by 8mmHg in systolic pressure, revealing adrenal hypofunction.

Her history, signs and symptoms are classic for someone who is estrogen dominant and progesterone deficient, as Dr. Lee describes in his book, What Your Doctor May *Not* Tell You About Premenopause. Treatment included daily use of natural progesterone cream for one year. After that, she was supplemented with Chaste Tree for eight months (dosage: 1 every morning). During this course, it was recommended that she take digestive enzymes, Symplex M, essential fatty acids, and adrenal support.

Diet modifications were implemented, as well. These included following the blood type A diet, eliminating white flour and sugar from the diet, and cutting out coffee and sodas. She was also prescribed an exercise regimen, which included 20-30 minutes of cardiovascular exercise 3-4 times a week. The goal of the above recommendations was to balance her hormones.

Chiropractic adjustments were made 1-2 times per week, specifically at the levels listed above. Applied kinesiology procedures to alleviate the open ICV were also implemented. Neurolymphatic therapy was applied to the adrenal, stomach, small intestine, and large intestines reflex points. Neuro Emotional Technique was used to address the emotional component of her symptoms. Tonification points for the large intestine and triple heater meridians were stimulated using a vibratory device. The gall bladder B&E point was stimulated for pain management.

Results

Within one month of treatment, the patient's symptoms decreased significantly. She did not have to take time off from work due to the pain. Within three months, the symptoms subsided. She now feels minor bloating and discomfort with menses but does not need to resort to medication for relief. She continues to maintain her lifestyle changes and receives monthly treatments to maintain her health.

Conclusion

By using applied kinesiology, we are able to locate and reverse different imbalances within the structural, nutritional, emotional, and acupuncture systems in order for the body to heal. By looking at these four factors the body is able to withstand any disease state, such as endometriosis.

In conclusion, the patient is now able to live relatively pain free and has no symptoms that would indicate the return of her endometriosis.

References

1. Walther, DS. Applied Kinesiology: Synopsis, 2nd edition. Systems DC. Pueblo, CO. 2000.
2. Maffetone, P. Complementary Sports Medicine. Human Kinetics. Champaign, IL. 1999.
3. Lee, JR. Hanley, J. Hopkins, V. What Your Doctor May Not Tell You About Premenopause: Balance Your Hormones And Your Life From Thirty To Fifty. Warner Books, Inc. New York, NY. 1999.

Medial Epicondylitis; A Case Study

Glen P. Alis, D.C.

Abstract

The purpose of this paper is to demonstrate the effectiveness of applied kinesiology in the evaluation and treatment of medial epicondylitis. The specific techniques used were gait point stimulation, ligament interlink, muscle stretch reaction, and stimulation of various neurolymphatic points.

Key words: Medial Epicondylitis, Applied Kinesiology, Tennis Elbow, Manual Muscle Testing.

Introduction

A 48 year old male presents with medial right elbow pain of six months duration. The pain was rated a seven out of a scale of ten (10 being the worst). There is also significant tenderness and swelling over the medial epicondyle. The patient is an avid tennis player, playing about four to five times per week. The patient also reports significant tenderness upon the first few strokes of playing which then goes away. The pain was definitely worse with his forehand and serves. After about an hour of play, the sharp pain returns to the point where he is unable to continue playing. He received two cortisone shots, which did not alleviate the problem. The patient was also taking four ibuprofens per day in order to continue playing.

By using applied kinesiology techniques, there has been significant improvement of the patient's symptomatology in seven treatments spanning about six weeks.

Discussion

General examination

Blood pressure:148/89 supine, 160/95 standing
Temperature: 97.3 F
Pulse: 73

Orthopedic examination

Elbow active range of motion: within normal limits
(+) Cozen's test
(+) elbow medial ligament stress test

On the initial visit, a thorough history revealed the patient experienced a left patella dislocation in his early teens. His diet consisted of a high carbohydrate, low protein diet, which would aggravate the adrenal glands that provide the integrity for the ligaments and an increase in inflammation. He ate toast and cereal for breakfast, sandwich for lunch, and frozen dinners for supper. He drank two cups of water daily and four to six cans of diet soda. His occupation consisted of eight hours of sitting in front of a computer. Manual muscle testing revealed weaknesses in the following muscles: right bicep, right supraspinatus, right pronator quadratus, left popliteus, left tibialis posterior, and left rectus femoris. Subluxation was present at T8 and the left navicular bone. I (the author) recommended the patient drink at least eight glasses of water, moderate the intake of white flour products, and eliminate the frozen dinners and the diet sodas. I also recommended supplementation of Ligaplex I from Standard Process to be taken two capsules, three times per day.

On the second visit, using manual muscle testing, the weaknesses of the muscles found on the first visit returned, however, there was a slight improvement of the symptomatology. On this visit, supine gait testing was performed. The left oblique and anterior gait points were found active and were stimulated using a vibratory device. After the treatment, the tenderness in the left elbow had significantly improved. A stretch reaction in the right pectoralis major clavicular brought about the original pain in the elbow. Myofascial flush of the right pectoralis major clavicular relieved the elbow pain.

On the third visit, the patient was able to play tennis longer than an hour and ibuprofen was no longer needed. The patient had been icing the area after his tennis matches everyday. Exam noted swelling in the elbow was gone. The left gait points found on the second visit were stimulated. Ligament interlink between the right medial elbow and the left medial knee was performed. There was still a lot of weakness in the right bicep. He was unable to pick up a jar of food. Muscle spindle stimulation of the right bicep muscle belly was performed. Bicep curls with his tennis racket were recommended because that was all he could pick up.

Results

The patient received four more treatments and was 80-85% improved. The patient decided to discontinue care at this time. He is now able to play for two hours with only slight discomfort. He is now rehabbing his elbow using bicep strengthening exercises with a ten pound weight, stretching the right pectoralis major clavicular muscle, and icing after matches to keep the pain and inflammation down without the use of ibuprofen.

Conclusion

The use of applied kinesiology enables me to look at the body as a whole and not only at the suspect joint in question. I was able to locate and reverse different imbalances in the patient's opposite limb which could have caused overcompensation, thus suffering pain in his elbow, which I suspect came from an old injury in the left leg.

It is my opinion that the healing process was accelerated through the dietary changes and supplementation. Now the patient can return to an activity that he enjoys.

References

1. Walther, DS. Applied Kinesiology: Synopsis, 2nd edition. Systems DC. Pueblo, CO. 2000.
2. Hoppenfeld, S. Physical Examination of the Spine and Extremities. Connecticut: Appleton-Century-Crofts. 1976.
3. Maffetone, P. Complementary Sports Medicine. Human Kinetics. Champaign, IL. 1999.

Functional Tests and Treatments for Male Menopause and Penile Dysfunction

Eugene Charles, D.C., DIBAK

Abstract

Solomon is attributed with saying, "There is nothing new under the sun." Respectfully, this paper will attempt to show that what is new is old and what is old is new and the key is to combine new findings with time-proven clinical procedures and strategies. Sun Tzu's, *The Art of War* is considered to be perhaps the most prestigious and influential book of strategy in the world today. In the introduction the sage is reported to say, "The healing arts and martial arts are parallel in several senses; in recognizing that less is better; that both involve strategy in dealing with disharmony; and in the sense that in both knowledge of the problem is key to the solution." It further emphasizes that keeping healthy is like foiling an enemies' plot and is the best strategy; while surgery, which can likened to besieging their cities, is the worst scenario and usually results from poor planning.[31]

That's what is old, now what's new? Prostate cancer now ranks second behind lung cancer in mortality among men and according to Lue, penile dysfunction affects between 20 and 30 million men in the United States alone.[18] Presented here will be a review of the nutritional needs of men and functional tests that can be performed in the office to help evaluate these nutritional needs as well as possible endocrine functional imbalances. Nutrients and herbs to help correct these symptoms along with exercises and physical modalities to counteract the effects of gravity and aging on the prostate will be taught.

Screening methods such as the prostate-specific antigen (PSA) blood test and digital rectal examination (DRE) to diagnose benign prostatic hypertrophy (BPH) and prostate cancer are well documented and will not be further elucidated here. In the same light as Sun Tzu's timeless classic, this paper will deal with more subtle examination procedures and less invasive treatment protocols to potentially avert the risk of these more severe health problems.

Introduction

There is a growing body of evidence to suggest that men experience menopause, a decrease in function of the sex organs, just like their female counterparts and suffer with the same myriad of health-related problems. In the men studied by Smith, McGovern et al., "Decreased production of gonadal hormones definitely contributed to bone loss."[27]

While the fact of male menopause, sometimes referred to in the literature as "andropause," is just coming to public light, it has been quietly mentioned in the medical literature for years. In 1976, Schirren wrote an article entitled, "The male climacteric" in an issue of *Nippon Funin Gakkai Zasshi*.[26] More recently, no less than 129 studies have concluded that testicular weight, number of Leydig cells, and bioavailable testosterone levels (free and albumin bound) decrease with age. Concurrently, testosterone bound to sex hormone binding globulin (SHBG) increases, presumably due to increase synthesis by the liver. Excess of this globulin appears to affect the level of available testosterone.

The good news is that something can be done about this. Recent clinical findings reveal that the prostate gland requires a rich supply of zinc and essential fatty acids, substantiating folklore regarding certain herbs long believed to alleviate masculine sexual dysfunction. Let's look at some of the new research regarding the role of nutrients and herbs on male endocrine function.

Discussion

The prostate gland has a unique characteristic of containing high levels of zinc. In fact, healthy prostate tissue contains a higher concentration of zinc than all other tissue in the human body. For this reason it can be said that, "A man needs zinc in the same manner that a woman needs iron." What does a recent review of the literature say about this?

- Researchers studied 109 patients with BPH (50 cases) and prostate cancer (109 cases). They showed that the zinc concentration in prostate tissue was significantly lower than the concentration found in healthy prostate tissue.[35]
- An interesting study found a correlation between citrate metabolism in the prostrate, zinc levels, and prostate cancer. They discovered that as levels of prostate zinc dropped, the threat of citrate oxidation increased. Perhaps the most interesting discovery of these researchers was that prolactin and testosterone regulate the concentration of zinc in the prostatic epithelial cells that produce citrate.[6]
- A study in Greece[17] also confirmed the correlation between zinc levels and BPH and cancer.
- Alarmingly, a study at Tufts University found zinc to be the #1 deficiency among children in the United States.[25]

Essential Fatty Acids

The body can synthesize certain fats, but there is also a group of fats that the body is incapable of manufacturing on its own. These are known as "essential fatty acids" or EFAs They include the omega 3 (linolenic) and omega 6 (linoleic) fatty acids. The fats one eats influences the cholesterol-based hormones, certain inflammatory processes, and the fluidity of every cell in the body since they all have lipids contained in their membrane.

Essential fatty acids help regulate inflammatory processes via the production of prostaglandins. It was unknown how aspirin worked for seventy years until the discovery of these prostaglandins. Interestingly, the word prostaglandin comes from the fact that these products of fatty acid metabolism were originally found in the prostate gland (prosta - gland - in).

1. A recent study by medical researchers in Korea established a connection between BPH, prostate cancer, and essential fatty acids. It has been called a landmark study in that it makes a direct correlation between omega 3 fatty acid deficiencies and prostate disease.[34]
2. Researchers at the Veterans Affairs Medical Center in San Francisco concluded that arachidonic acid stimulated, while the omega 3 fatty acid Eicosapentaenoic inhibited growth of human prostate cancer PC-3 cells.[15]
3. Attiga, Fernandez, et al. found that inhibitors of prostaglandin synthesis, particularly prostaglandin 2 (Pg2) from arachidonic acid, inhibit prostate tumor cell invasiveness and reduce the release of matrix metalloproteinases. They concluded that inhibiting Pg2 inhibition might be very useful in the prevention and therapy of prostate cancer invasion.[4]

To fully understand the above, I have included the following chart illustrating prostaglandin synthesis.

Essential Fatty Acids and Their Relation to Prostate Disease

Let's look at what scientific research is revealing about herbs that historically have been used to help with sexual function, prostate health, and overall vitality.[23]

Serenoa Repens (Saw Palmetto)
Saw palmetto is a native plant of North America. It is a member of the fan palm family and has characteristic sharp edges that literally "saw" through clothing, hence its common name, saw palmetto. Saw palmetto is known to contain the following medicinal constituents:

- free fatty acids together with triglycerides, diglycerides, and monoglycerides, as well as phytosterols (particularly beta-sitosterol)
- flavonoids and polysaccharides.

Kerry Bone, the Principal of the Australian College of Phytotherapy, reports that "Saw palmetto has been used for the treatment of BPH for hundreds of years. The modern clinical evidence supporting the efficacy of its liposterol extract for this disorder is compelling. While the evidence is not yet incontrovertible, it is sufficient to justify the use of this plant for the treatment of mild to moderate BPH in circumstances where conventional therapy is either not wanted or inadvisable. The safety profile of this herbal preparation is very good."[3]

Blumenthal concludes that saw palmetto is indicated for use in mild-to-moderate prostatic hyperplasia.[29] Additionally, Marks finds that saw palmetto "appears to be a safe, highly desirable option for men with moderately symptomatic BPH."[20]

- An overview of the medical literature points to the following clinical effects of saw palmetto (Serenoa repens):
- Decreases urinary urgency
- Decreases perineal pain
- Decreases nocturnal frequency
- Increases urinary flow rate
- Prevents infections due to residual urine
- Reduces residual urine
- Improves quality of life for patients with BPH

Stinging Nettle Root (Urtica Dioica)
Nettle root is a weed that has a particular affinity for nitrate-rich soil and is found in most temperate regions. Nettle root has a strong affinity for SHBG (sex hormone binding globulin) thereby limiting the amount of testosterone and estrogen that can bind to it and influence cell proliferation.

- Stinging nettle root is known to contain the following medicinal constituents:
- Lignans, which have an affinity for human sex hormone binding globulin (SHBG)
- Sterola and steryl glycosides (including sitosterols)
- Scopoletin (a coumarin)

The popular use of stinging nettle root extract in Germany encouraged the onset of numerous clinical trials. Collectively, these trials demonstrated the benefits of using nettle root for the treatment of BPH. There is only one recent placebo-controlled trial that researchers have undertaken. A French study successfully demonstrated that nettle root extract was superior to placebo in terms of the International Prostate Symptom Score (IPSS).[11]

In an older placebo-controlled trial, 79 patients with BPH were given either 600 mg of nettle root extract or placebo for as long as two months. Nettle root proved superior in all parameters that were measured including urinary flow, residual urine, and urinary volume.[9]

Clinically, stinging nettle root (Urtica dioica):

- Decreases urinary urgency
- Encourages hormonal balance
- Decreases nocturnal frequency
- Increases urinary flow rate
- Prevents infections due to residual urine
- Reduces residual urine
- Improves quality of life for patients with BPH

Adrenal Glands

Physical and mental stress can also adversely affect the endocrine system and the ability of the penis to achieve or maintain erection and properly orgasm. Hans Selye — the man who discovered the General Adaptation Syndrome and the man responsible for making the word "stress" a household term — observed that animals under prolonged stress developed sexual derangements. Selye, who also coined the terminology mineralcorticoids and glucocorticoids, found that the ovaries and testes shrank and became less active in proportion to the enlargement and increased activities of the adrenal glands.[13]

Smith, McGovern, et al. report in the *New England Journal of Medicine* that adrenal hormones (glucocorticoids) alter gonadal function by acting on the pituitary, gonads and adrenal glands. These anti-inflammatory agents inhibit the response of lutenizing hormone in the pituitary; inhibit adrenal hormone production by suppressing the secretion of corticotropin and the production of androstenedione, a substrate for testosterone; and may directly inhibit gonad function.[25]

Along with their role in adaptation, the adrenal cortex produces approximately 15% of the sex hormones. The highly respected Walther states, "Under normal conditions, the adrenal cortex is expected to mature in its estrogen-androgen production at approximately the same time in life that gonad function decreases. Normally the adrenal cortex takes over gonadal activity, including protein anabolism, without undue symptoms or stress to the individual."[32]

Clinically, 15% is a small amount but at a time when the testes are diminishing their production it would be logical that if we could support the patient's adrenal glands this would be helpful in limiting menopausal symptoms. Licorice and ginseng have long been reputed to help the adrenal glands as well as overall virility and vitality.

Licorice (Glycyrrhiza Glabra)

In the late 1940s a Dutch doctor named Revers noticed that patients with gastric ulcers were being cured by high doses of Licorice extract dispensed by a local pharmacist. This investigation led to the development of carbenoxolone, the major anti-ulcer drug of the 1960s.

Licorice inhibits the breakdown of cortisol, potentiates the action of hydrocortisone, inhibits prostaglandin synthesis, and is a demulcent in the gastrointestinal tract. The immunostimulating, antiviral, anti-tumor, anti-inflammatory, hepatoprotective and choleretic activity of licorice could possibly be interpreted as correlated to the induction of the glutathione-dependent adenosyl methionine transferase and resultant methylation processes.

Medicinal constituents of licorice:

- Triterpenoid saponins, predominately glycyrrhizin
- Glycyrrhetinic acid
- Flavonoids
- Sterols

Short-term use of licorice may be helpful in alleviating symptoms of stress as proposed by Selye in his General Adaptation Syndrome where he described the effects of prolonged stress on the adrenal gland, thymus, and digestive tract. The successful treatment of chronic fatigue syndrome has been reported in a case study by Baschetti.[2]

- Licorice can be used for:
- Gastric, duodenal, and esophageal ulcers or inflammation
- Asthma
- Corticosteroid dependency and lowered adrenal function
- Depression

Contraindications include cholestatic liver disorders, liver cirrhosis, hypertension, hypokalemia, severe kidney insufficiency, pregnancy, edema and congestive heart failure.[30]

Ginseng

Ginseng (panax ginseng or eleutherococcus) assists the body to counteract and adapt to stress of many origins. It is classified as an adaptogen, immunomodulator, and a tonic. Since the 1960s it has been the subject of numerous scientific investigation, probably prompted by the work of Russian scientists who demonstrated its potential as an adaptogen in the 1950s. It was provided to their Olympic athletes starting in the late 1970s.

More than 500 studies have been published on the pharmacological activity of ginseng. Ginseng appears to act mainly on the hypothalamus and has a sparing effect on the adrenal cortex. It has been found to have a sparing effect on glucocorticoids

Recent studies suggest that the antioxidant and organ-protective actions of ginseng are linked to enhanced nitric oxide synthesis in the endothelium of the lung, heart, kidney, and in the corpus cavernosum. (Nitric oxide will be reviewed later.) The recommended regime is generally considered to be a course of six weeks followed by a two-week break.

The following medicinal uses can be attributed to ginseng:[22]

- Short-term use (weeks to months) to cope with increased tiredness or stress
- To assist recovery from disease or surgery and to minimize the side effects of radiation or chemotherapy
- Long-term use for the elderly and infirm as a tonic to improve well being
- Impotence and poor sperm count or function
- Symptoms of menopause
- Long-term use to prevent cancer
- To improve resistance to infection

Along with general overall health, the adrenal glands are critical to proper penile function directly. Lue writes, “The maintenance of the intracorporeal smooth muscle in a semi-contracted state results from three factors: intrinsic myogenic activity, adrenergic neurotransmission, and endothelium-derived contracting factors such as prostaglandin F2 and endothelins.”[18] The same article states that “nitric oxide released during nonadrenergic, noncholinergic neurotransmission and from the endothelium is probably the principal neurotransmitter mediating penile erection.” This correlation with nitric oxide is discussed by Burnett, Lowenstein, et al.[5] and Snyder and Bredt. These were the first discussions of the biological role of nitric oxide as the “first of a new class of neurotransmitters.”[28]

Nitric oxide is essential for a myriad of bodily functions in addition to mediating penile erection. Nitric oxide is responsible for maintaining the endothelial-relaxing factor that stabilizes blood pressure. The nitric oxide is released from the intimal lining of the blood vessel resulting in relaxation of the walls of the vessel and a lowering of the blood pressure.

Phagocytes produce nitric oxide in their battle against viruses, bacteria and parasites. Two immune system stimuli — gamma interferon and lipopolysaccharide — cause production of nitric oxide synthase. This enzyme converts arginine to nitric oxide. Nitric oxide is an end product of the arginine cycle. This is a pertinent physiological fact that we will be able to influence biochemically. (This will be discussed in the clinical application section.)

Let's go from a specific case of the amino acid arginine to a general discussion of protein. While the general consensus is that men in civilized societies eat an abundance of protein, it is this author's opinion that quantity does not equate to quality and that there is an endemic deficiency in both men and women of usable protein. How does this effect the hormone status and prostate function in men?

Protein
Independent studies in 1958,[12] 1962,[8] 1978,[7] and two in 1990[19, 33] determined that amino acid supplementation with alanine, glutamine, and glycine offers favorable results in patients suffering with prostate conditions such as BPH, prostatitis, prostatic adenoma, and urinary disturbances.

What if the patient's complaints are those of an overall decrease in vitality including infertility, impotence, decreased libido, and menopause? Recent attention has focused on the Bulgarian research of a standardized extract of Tribulus leaf used as a male tonic for the treatment of infertility and menopause. The results of these open clinical trials indicated that Tribulus had a stimulating effect on sexual function. Improvements were noted in sperm motility and volume, increased libido, sexual activity, and fertility.[29]

Tribulus (Tribulus Terrestris)
The active constituents of Tribulus include:

- Steroidal saponins, mainly furostanol glycosides
- Phytosterols
- Harmala alkaloids (tryptophan-derived B-carbolines)

Summary

Let's recapitulate. The prostate gland is a walnut-sized gland that sits at the base of the bladder, astride the urethra. It secretes fluid that helps to make up semen and functions to energize and alkalize sperm. A close review of the medical research verifies the safety and efficacy of nutritional and botanical agents such as Serenoa repens (saw palmetto), Urtica dioica (stinging nettle root), essential fatty acids, and zinc to promote and maintain prostate health in addition to alleviating painful urological symptoms.

The adrenal glands are located above the kidneys and are comprised of two parts, the medulla and the cortex. The medulla produces epinephrine and norepinephrine, which are collectively called the catecholamines. The cortex is responsible for producing the corticosteroids — the mineralcorticoids, glucocorticoids, and small amounts of sex hormones. Physiologically, the adrenal production of sex hormones is needed to pick up the slack from the gonads around the years currently known as menopause. Vitamins B and C, and herbs such as licorice and ginseng have been found to be useful in supporting proper adrenal function.

All this new information is promising but unless we can use it clinically it just makes us interesting conversationalists. How can this be used in helping patients? It is prudent to recommend to men these valuable supplements when a deficiency is suspected. How can we minimize the randomness of our recommendation and

minimize overloading the patient with a barrage of tablets, capsules, and tinctures? The following are excellent clinical signs and symptoms and some potentially promising in-office and inexpensive functional tests to help determine which supplements may be warranted to aid in preventing and alleviating male menopause and penile dysfunction.

Clinical Application

Now we have an expansive armamentarium from which to choose. However, it is only truly useful if we can most effectively decide when to give what to whom. I am not from the "pop and pray" school where the physician gives the patient a bunch of pills to *pop* then he or she *prays* that the right one or combination was given. How are we to ascertain when a patient is most likely to benefit from supplementing with any or all of the nutrients and herbs reviewed here?

I have often said at my lectures that I am more of a searcher than a researcher. In scouring the literature I have come across many different signs, symptoms, and tests for measuring human function. Some are obscure, some are well known. However, just like the saying, "There is beauty and tragedy in the world, you will find whichever you search for," I have found some of the most useful signs, symptoms, and functional tests to aid the physician in establishing what natural course of treatment may be most efficacious for the patient.

1. Symptoms of a zinc deficiency can reveal itself as:

- white spots on the fingernails
- premature graying of the hair
- A simple taste test was developed and reported in the *Lancet* where the ability to taste a 0.1 percent solution of zinc sulfate in a base of distilled water could be readily assessed in the office for the determination of who may benefit from zinc supplementation.[4]
- The zinc taste test may be a valuable and cost-effective tool to evaluate zinc levels before the effects of a gross deficiency occur.

2. Symptoms of an essential fatty acid deficiency include:

- dry skin
- stiff joints
- sensation of feeling cold with an inability to warm up
- inflammation (any –it is, such as arthritis, bursitis
- memory loss
- decreased bone density
- mood disorders, fatigue, and depression

Harold Hawkins DDS, Professor of Bacteriology and Preventative Dentistry at the University of Southern California wrote what some consider to be the definitive book on nutrition. In *Applied Nutrition*, he writes that oral pH should be approximately 7.6 in adults and 7.8 in children. An oral pH below that level may indicate an essential fatty acid deficiency.[14] He arrived at these levels by evaluating thousands of children and adults and found that those who were free of dental caries, periodontal disease, and in overall excellent health displayed an oral pH of 7.8 and 7.6 respectively. He concurrently measured the blood levels for calcium, potassium, phosphorous, and chloride and found them to be at the optimal ranges for these individuals. As an indicator of dietary needs he found that an oral pH below that level might indicate an essential fatty acid deficiency. In summary, a patient whose oral pH is too acidic and whose urinary pH is too alkaline will probably benefit by increasing the levels of natural fats and oils.

Oral pH may be a valuable and cost effective tool to evaluate essential fatty acid levels before the effects of a gross deficiency occur. The patient should not eat for a minimum of one hour before testing to ensure an accurate reading.

3. Indications that there may be an imbalance in nitric oxide production include:

- Hypertension
- Immune problems indicative of decreased phagocytic activity
- Hypercholesterolemia
- Impotency
- Nitric oxide is an end product of the arginine cycle. When a patient presents with impotency and one or more of the listed signs or symptoms (or additional signs of problems with arginine metabolism such as elevated uric acid levels or gout), he may be a likely candidate for supplementation with the enzyme arginase. Though empirical findings hold great promise, further controlled clinical studies are still needed.

4. Indications that the patient may benefit from adrenal support include:

- pain
- fatigue
- lightheadedness upon arising
- increased sensitivity to light
- need for corticosteroids
- endocrine dysfunction

Two arcane tests that may be helpful include Idiopathic Orthostatic Hypotension and Arroyos Sign. Orthostatic hypotension, a condition whereby the systolic blood pressure drops upon arising, has been found to be affiliated with, among other things, a marked depletion of norepinephrine secondary to a decrease in normal sympathetic response. A normal sympathetic response is an increase in systolic pressure by 4-10 mmHg due to the vasoadaptation of the sympathetic nervous system. This is mentioned by Ziegler, Lake, and Kopin[36] and by Kontos, Richardson, and Norvell.[16]

Arroyo's sign is listed by Mazion as failure of the pupils to maintain constriction for 40 seconds to the sustained stimulation of a bright light due a relative functional hypoadrenia syndrome.21 While the exact mechanism for this response is unknown, the theory is that when there is functional hypoadrenia, the elevated intracellular potassium causes inadequate response of the cholinergic nerves that are responsible for the functioning of the sphincter muscles of the eye.

Blood pressure readings taken from a seated position and then from a standing position (Orthostatic Hypotension) and shining a penlight into a patient's eyes (Arroyo's sign) are simple and time-tested neurological tests that can be performed in the office and may be early indicators of relative adrenal dysfunction. Early intervention in supporting the adrenal glands and their ability to modulate the stress response can help maintain the harmony of the patient's overall endocrine function.

5. Indications that the patient may benefit from prostate or male endocrine support include:

- lack of sex drive or vitality
- sexual dysfunction
- urinary problems (retention, flow, frequency)
- perineal pain
- bone loss

• inflammation, hyperplasia, or cancer of the prostate gland

Unfortunately, no in-office functional test is available. The one very important functional assessment is best measured outside of a clinical setting and with the patient reporting results to you.

Protocols and Indicated Therapies:

1. Supply zinc if the patient fails the zinc taste test, has white spots on the fingernails and/or is experiencing premature graying. (Chezyn, Allorganic Trace Minerals - Standard Process Labs)

2. Supply essential fatty acids if the patient has an oral pH below 7.0, complains of dry skin, herpes lesions and/or any localized inflammatory condition such as gastritis, arthritis, prostatitis, etc. (Black Currant Seed Oil, Linum B6, Cataplex F Perles or Tablets – Standard Process Labs)

3. If the patient is sympathetic dominant and has trouble achieving or maintaining an erection, supply cholinergic supporting nutrients such as choline, calcium, potassium, and the vasodilator portion of the B complex – riboflavin, niacin (Organic Minerals, Choline, Cataplex G – Standard Process Labs)

4. If the patient is parasympathetic dominant and has trouble ejaculating, supply the adrenergic supporting nutrients such as licorice, adrenal extracts, Vitamin C (Licorice, Drenamin, Cataplex C – Standard Process Labs)

5. General prostate support for prevention or if history of problems such as urinary flow disturbances or sexual dysfunction: Saw Palmetto, Stinging Nettle Root, Lycopenes (Palmettoplex, Serenoa – Standard Process Labs)

6. If low sex drive, general sexual dysfunction, low testosterone, or low sperm count: Tribulus, zinc, ginseng (Korean), and Vitamin E have been shown to improve overall vitality. (Tribulus, Cataplex E, Ginseng – Standard Process Labs)

7. If indications are that there may be an imbalance in nitric oxide production or arginine metabolism such as hypertension, immune problems indicative of decreased phagocytic activity, elevated cholesterol and/or uric acid, and impotency supply a source of the enzyme arginase. (Arginex – Standard Process Labs makes the enzyme from a culture grown on beet pulp and rice bran)

Preventative

a) Support the adrenal glands to prevent adrenal exhaustion and the depletion of cortical hormone, which include 15% of the body's supply of sex hormones. Supply appropriate herbs for the adrenal glands, an often-overlooked player in hormonal balance. The adrenals are often in the exhaustion phase of the General Adaptation Syndrome by the time of male and female menopause. Helpful supplements: Licorice, Vitamin C, Adrenal extracts

b) Support the overall body with nutrients believed to improve vitality and have been proven essential for proper endocrine function.

Helpful supplements: Zinc, Ginseng, Tribulus, Essential Fatty Acids (Olive, Black currant seed, Flaxseed, Eicosapentaenoic)

c) Specifically support the prostate with herbs found to be beneficial in clinical studies.

Helpful supplements: Saw Palmetto, Stinging Nettle Root, Amino Acids, Lycopenes.

Patient Responsibilities

A man was asked, "Do you smoke after sex?" He replied, "I don't know, I never looked." Seriously, smoking destroys vitamin C, decreases circulation and vitality, and disrupts essential fatty acid metabolism, which is essential for optimal sexual function. It is number one on a patient's "do's and don'ts" list.

1. Stop smoking.
2. Take recommended supplements.
3. Learn to relax and to communicate thoughts and feelings.
4. Snack on raw pumpkin seeds instead of pizza. (Exception for Monday Night Football)
5. Perform exercises to counteract the effects of gravity on the prostate. The goal is to strengthen the lower abdomen, the pelvic diaphragm, and the pubococcygeal muscles to alleviate any possible mechanical pressures on the prostate or bladder.

The pelvic diaphragm can be thought of as a hammock that prevents ptosis of the pelvic structures. Toned abdominal muscles do the same for the contents of the abdomen. The Kegel exercises commonly given to postpartum women help to relieve the ongoing stress of gravity upon the pelvic structures. These exercises are also useful in alleviating hemorrhoids, varicosities, and urinary frequency of unknown origin.

Male Version of Kegel Exercises

1. Practice pulling the umbilicus in towards the spine and up towards the back of the ribcage while expiring through the mouth strongly. Imagine you are lifting the pelvic structures with your abdominals. Hold contracted in and up for five seconds.
1. Then breathe in and let the abdominal relax, accentuate the low back curve and draw the diaphragm down.
2. Repeat for ten repetitions. Perform three times daily, before meals is best.
3. Contract and draw up the area between the scrotum and the anus. It should almost feel like you are stopping or holding in a bowel movement. Do this on expiration and hold for five seconds. Repeat for ten repetitions. Perform three times daily.
4. When coming out of the shower and drying off, gently push your hands into the lower stomach to the outer sides of the bladder just above the groin and lift up with your fingers as you expire. Lift up as if you were lifting your lower intestines up off your prostate. Do five gentle repetitions as you hold your breath out for five seconds. Perform once daily. This is also helpful for people suffering with inguinal hernias.

Note: When you contract the abdominals and pubic muscles, be sure to squeeze or draw in the muscle. Do not make the mistake of pushing out.

Conclusion

The nutritive and physical modalities presented here may be very helpful in detecting, preventing, and alleviating the symptoms of male menopause and penile dysfunction.

Even a cursory review of any standard text of biochemistry will substantiate the necessity of the nutrients mentioned here for optimal physiological function. This presents us with intelligent alternatives to harsh drugs and invasive surgery — alternatives that people seem to be looking for. In fact, a study in *The Journal of Family Practice*[25] discovered that 21% of family practice patients also use "alternative" care. It seems that the newest clinical research is validating to even the most cautious of doctors what patients seem to already suspect, namely that health is restored and maintained by using nature to replenish what the body needs whenever possible.

The functional tests offered here are left to your scrutiny for clinical application. They are not to be used to diagnose disease but rather to give a general measure of bodily functions. This has been an attempt to take the newest research and incorporate it into time tested and usable protocols. The most classic protocol is the philosophy of treating the person who has the condition and not treating the condition that has the person. Specifically illustrated here is not how to treat male menopause and penile dysfunction, but how to treat the person experiencing male menopause and penile dysfunctions.

Author's Remarks

This paper was presented at a symposium in March 2002 organized by The Institute of Functional Medicine. I was requested to write a standard scientific paper and to lecture to physicians who had no expertise in applied kinesiology. The challenge was to teach the medical doctors about functional tests and treatments using medical vernacular and without relying on the valuable information that manual muscle testing provides. It is my hope that this paper serves as an introduction to functional testing and treatments to those doctors who do not yet utilize applied kinesiology in their armamentarium to treat the person and not the disease.

References

1. Attiga FA, Fernandez PM, Weeraratna AT, Manyak MJ, Patiern SR. Inhibitors of prostaglandin synthesis inhibit human prostate tumor cell invasiveness and reduce the release of matrix metulloproteinases. *Cancer Research*. 2000 Aug;60(6):4305-4165.
2. Baschetti R. Liquorice and chronic fatigue syndrome. *N Z Med* J 1995 Jun 28;108(1002):259.
3. Bone K, Mills S. Principles & Practice of Phycology. Harcourt College Publishers, Orlando, FL. 1999.
4. Bryce-Smith D, Simpson RI. Case of anorexia nervosa responding to zinc sulphate. *Lancet* 1984 Aug 11; 2(8398):350.
5. Burnett AL, Lowenstein CJ, Bredt DS, Chang TSK, Snyder RH. Nitric oxide: A physiologic mediator of penile erection. *Science* 1992 259:401-403.
6. Costello LC, Franklin RB. Novel role of zinc in the regulation of prostate citrate metabolism and its implications in prostate cancer. *Prostate* 1998 Jun 1:35(4):285-96.
7. Cuervo Blanco E, Francia Bengoechea A, Fraile Gomez B. Clinical study of a phytosterol extract of Prunus arborea and 3 amino acids: glycine, alanine and glutamic acid. *Arch Esp Urol* 1978 Jan-Feb:31(1):97-8. Spanish.
8. Damrau F. Benign prostatic hypertrophy: Amino acid therapy for symptomatic relief. *J Amer Ger Soc* 1962 10(5):426-30.
9. Dathe G, Shnid H. Phytotherapy for benign prostatic hyperplasia. Double blind study with Urtica dioica extract. *Urology* 1987 27:233-236.
10. Dawson-Hughes B. Bone Loss Accompanying Medical Therapies. *NEJM* 2001;345(13):989-91.
11. Engelman U, et al. Therapy for benign prostatic hyperplasia with nettle liquid. *Urology* 1996;36:287-291. Translated from French.
12. Feinblatt HM and Gant JC. Palliative treatment of benign prostatic hypertrophy; value of glycine-alanina-glutamic acid combination. *J Maine M.A.* 1958;49:99.
13. Ganong WF. Review of Medical Physiology. 18 ed. Appleton & Lange, Stamford, CT. 1997.
14. Hawkins, H. Applied Nutrition. 2 ed. Mohave Books, 1977.

15. Hughes-Fulford M, Chen Y, Tjandrawinata RR. Fatty acid regulates gene expression and growth of human prostate cancer PC-3 cells. *Carcinogenesis*. 2001 May;22(5):701-7.

16. Kontos HA, Richardson DW, Norvell JE. Norepinephrine depletion in idiopathic orthostatic hypotension. *Ann Intern Med.* 1975 Mar;82(3):336-41.

17. Lagiou P, Wuu J, Trichopoulou A, Hsieh C-C, Adami, H-D, Trichopoulos, D. Diet and Benign Prostatic Hyperplasia: A study in Greece. *Urology* 1999 54(2):284-90.

18. Lue TF. Erectile Dysfunction, *NEJM* 2000;342(24):1802-1813.

19. Maekawa M, Kishimoto T, Yasumoto R, Wada S, Harada T, Ohara T, Okajima E, Hirao Y, Ohzono S, Shimada K, et al. Clinical evaluation of Cernilton on benign prostatic hypertrophy – a multiple center double-blind study with Paraprost. *Hinyokika Kiyo* 1990 Apr;36(4):495-516. Article in Japanese.

20. Marks LS, Partin AW, Epstein JI, Tyler VF, et al. Effects of a saw palmetto herbal bland in men with symptomatic benign prostatic hyperplasia. *The Journal of Urology* 2000 May;5(163):1451-1456.

21. Mazion JM. Illustrated Manual of Neurological Reflexes/Signs/Tests Orthopedic Signs/Tests/Maneuvers For Office Procedure. Imperial Litho/Graphics, Phoenix, AZ. 1980.

22. Natural Therapies for the Prostate Gland: Scientific Review of Nutrient and Herbal Combinations that Promote Prostate Health. MediHerb Professional Literature. Queensland, Australia. 1998.

23. Nick GL. Natural therapies for the prostate gland: Scientific review of nutrient and herbal combinations that promote prostate health. MediHerb/Standard Process, Palmyra, WI. 2000.

24. Palinkas LA, Kabongo ML. The use of complementary and alternative medicine by primary care patients. *J Fam Pract.* 2000 Dec;49(12):1121-30.

25. Roberts SB, Heyman MB. Micronutrient shortfalls in young children's diets: common, and owing to inadequate intakes both at home and at child care centers. *Nutr Rev.* 2000 Jan;58(1):27-9. Review.

26. Schirren C. The male climacteric. *Nippon Funin Gakkai Zasshi* 1976 Apr;21(2):207-13.

27. Smith MR, McGovern FJ, Zietman AL, et al. Pamidronate to prevent bone loss during androgen-deprivation therapy for prostate cancer. *NEJM* 2001 345:948-55.

28. Snyder SH, Bredt DS. Biological roles of nitric oxide. *Sci Am* 1992 May;265(5):68-71, 74-7.

29. Stanislavov R, Nikolova V. *Dokl Bolg Akad Nauk* 2000 53:107.

30. The Complete German Commission E Monographs: Therapeutic Guide to Herbal Medicines. Blumenthal M, Hall T, Rister R, Riggins C (eds.) Rister R (trans.) Integrative Medicine Communications, Newton, MA. 1999.

31. Tzu S, The Art of War, Cleary T (trans.) Oxford University Press, 1983.

32. Walther, DS. Applied Kinesiology: Synopsis. 2 ed. Systems, DC. 2000.

33. Yamaguchi O, Shiraiwa Y, Kobayashi M, Yokata T, Ohinata M, Aoki H, Tsuzuki T, Ohori M. Clinical evaluation of effects of prazosin in patients with benign prostatic obstruction. A double-blind, multi-institutional Paraprost-controlled study. *Urol Int* 1990:45 Suppl 1:40-6.

34. Yang YJ, Lee SH, Hong SJ, Chung BC. Comparison of fatty acid profiles in the serum of patients with prostate cancer and benign prostatic hyperplasia. *Clin Biochem* 1999 Aug;32(6):404-9.

35. Zaucgucj V, Sviridova TV, Zaichick SV. Zinc in the human prostate gland: normal, hyperplastic and cancerous. Int Urol Nephrol 1997;29(5):565-74.

36. Ziegler MG, Lake CR, Kopin IJ. The sympathetic-nervous-system defect in primary orthostatic hypotension.N Engl J Med. 1977 Feb 10;296(6):293-7.

Objectivization of Manual Muscle Testing Through Analysis of the Dynamic Force Spectrum

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Abstract

Background: The purpose is to develop and study a new objectivization method of manual muscle testing (MMT) which is the main diagnostic tool in the applied kinesiology.

Method: It is offered to measure dynamic effort made by the patient and record the amplitude of low-frequency component (lower than 2 Hz) in the dynamic force (DF) spectrum (MacLab-4E, ADInstruments) as a criterion of muscle strength/weakness. A portable DF transducer, containing piezoelectric ceramic cylinder, has been developed and made.

Materials: Eight volunteers were recruited for carrying out a pilot blind experiment. Every patient had been previously MMT examined along the therapeutic localizations (TLs) to reveal positive (weak muscle) and negative (strong muscle) provocations with musculus deltoideus as indicator. The examiner's assistant recorded 6 positive and 6 negative TLs in the arbitrary protocol. Every patient was twice examined objectively according to the protocol. In the first series the patient pressed the DF transducer with his thumb and middle fingers of the same hand, examiner not taking part in it. In the second series the examiner pressed the patient's arm through DF transducer. The examiner's assistant pressed upon TLs during both series.

Results: Developed method and apparatus show moderate agreement with the results of subjective diagnostic MMT evaluation (kappa = 0.47). There is less agreement when the patient's dynamic force spectrum is measured without the examiner's participation (kappa = 0.31). To refine on the effectiveness of the offered method it is necessary to perform enlarged clinical testing in multicenter double blind trial.

Key Words: Manual Muscle Testing, Objectivization, Dynamic Force, Spectrum, Agreement.

Introduction

The problem of objectivization of manual muscle testing (MMT), which is the main diagnostic tool in applied kinesiology (AK), is quite actual because of the procedure subjectiveness and indefiniteness of the phenomenon biophysical nature. Both specialists using AK and their opponents recognize it.

Certain tentatives of using different mechanical parameters have been made to objectivize MMT.

Measuring the force of random isometric muscle contraction seemed the most natural. However, a number of researchers (Rybeck et al., 1980;[1] Grossi, 1981;[2] Kenny et al., 1988[3]) have marked the alienation of this parameter and the MMT data.

Electronic dynamometry turned out to be more adequate. It means that the force transducer should be placed between the examiner's hand and the patient's limb. Thus, Marino et al., 1982,[4] Bohannon, 1986[5] have

shown that there is statistically reliable correlation between the MMT results and those of electronic dynamometry. Hsieh et al., 1990[6] have demonstrated high repeatability of the electronic dynamometry results. Caruso and Leisman, 1999 [quoted from[7] proposed to measure the force, displacement and compute the muscle stiffness coefficient. The stiffness coefficient of the muscles tested by the experienced examiners as strong differed from the stiffness coefficient of the muscles classified as weak. But only examiners having practical experience not less than 5 years prove to correctly identify strong and weak muscles. In their next research Carruso and Leisman, 2000[8] offered to register leading edge of the force pulses. They managed to show that a significantly large slope is indicative of weak muscles (as perceived by the clinician), and a small slope is indicative of strong muscles. They worked out threshold criterion coinciding with the subjective evaluation of the examiners having more than 5 years' practical experience in 98% of cases but if the examiners are less experienced it coincides only in 64% of cases.

To objectivize MMT certain variants of using electrophysiological parameters have been studied.

Thus Perot et al., 1991[9] compared MMT with electromyography (EMG) of the examiner's triceps and discovered significant difference in electrical activity of his muscle when strength or weakness were subjectively evaluated. Leisman et al., 1995[10] revealed essential agreement between the patients' electromyograms and the MMT data.

The authors of the article have used the EMG method[11] to objectivize MMT. The distinct feature of it is threshold. Processing the ratio of amplitude to the duration of application electromyograms of the patient's muscle being tested. Comparison of electromyographic readings of homeopathic nosodes influence on patients (the key set of Metabolics Ltd.) and subjective evaluations independently given by qualified examiner according to the results of 152 diagnostic and 100 reference tests (double blind procedure) revealed coincidence in 87.5% of cases.[12] Homeopathic nosodes were used as provocative challenges. They were very suitable to perform double blind procedure. You can find details in authors' translation of this paper www.manus.vladivostok.ru/ep_scien.htm. In the article Vasilyeva et al., 2001[13] the authors showed that the application EMG of the tested muscle allowed to distinguish 3 phases of long-term resistance efforts assumed to be related to different regulation mechanisms. Evaluation of the amplitude of muscle electric activity proved to be the closest to the experienced examiner's subjective perception of the muscle strength/weakness in the 3rd phase of contraction (81.3% agreement).

However, in spite of intensive researches, there is no MMT objectivization method which could extend beyond the walls of research laboratories. The purpose of this article is to develop and study a new MMT objectivization method.

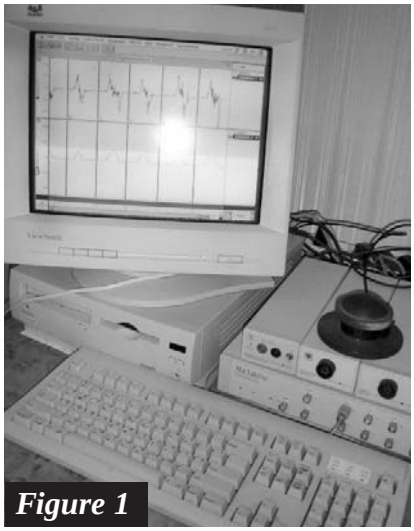


Figure 1

Experimental apparatus: (a) – measuring set; (b) – dynamic force transducer.

Materials and Methods

Analysing the results of the experiments,[13] the authors noticed that the EMG spectrogram pattern in the low frequency range (0–2 Hz) changes greatly in cases of subjectively evaluated muscle strength and weakness. In the latter case it steadily correlates with the examiner's feeling of the patient's muscle tremor. To simplify the equipment, the authors decided to record this effect when measuring not EMG but an "effort mechanic parameter." We offer to measure dynamic effort made by the patient and record the low-frequency component (lower than 2 Hz) of the dynamic effort spectrum as a criterion of strength/weakness.

A portable dynamic force transducer (Fig.1) for measuring equipment has been developed and made. It contains piezoelectric ceramic cylinder. The cylinder faces are carried by ebonite flanges. Electric signal from the electrodes of the piezoelectric ceramic cylinder compressed by the flanges is transmitted to the input of measuring apparatus "MacLab-4E"

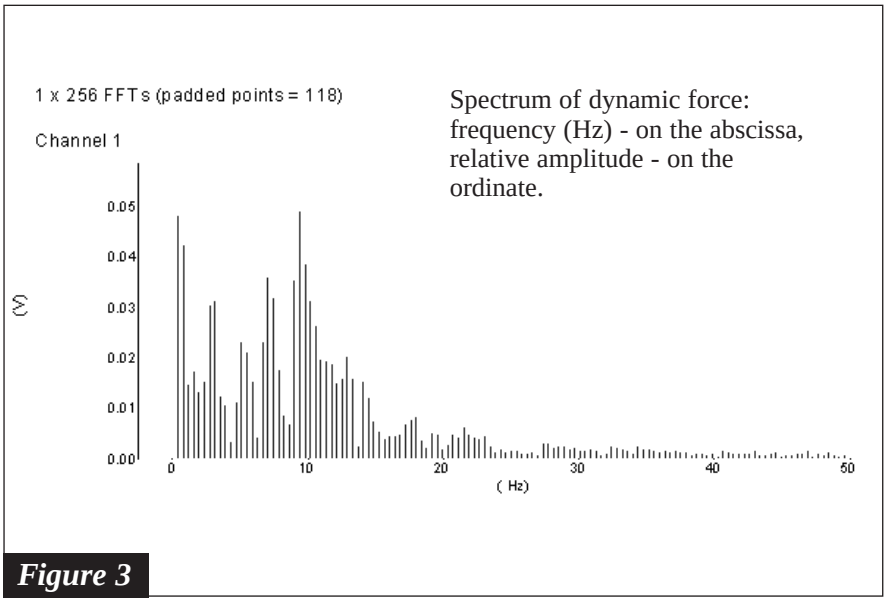
(ADInstruments). Signal recording is performed on PC "Macintosh Performa 6360" by means of software "Chart-3.6.1." Spectrum analysis is performed on PC IBM Pentium II by means of software "Chart-4.1 for Windows."

Eight volunteers (informed consent had been obtained from each subject) were recruited for carrying out a pilot experiment. Every patient had been previously examined by the same experienced examiner (T.N.Ch.) The purpose of this subjective MMT examination along with the therapeutic localizations (TL) was to reveal positive (weak muscle) and negative (strong muscle) provocations with musculus deltoideus as indicator. Reflex points were used. Points of thalamus, heart, kidneys, lungs, stomach, spleen, etc. were used as TL. The patients were not informed of the results. The examiner's assistant recorded 6 positive and 6 negative TLs. Neither the examiner nor patients knew about the TL sequence in the protocol. The operator of the measuring apparatus did not know it either. Then every patient was twice examined objectively according to the TL sequence in the protocol. In the first series the patient pressed the flanges of the dynamic force transducer with his thumb and middle fingers of the same hand, examiner not taking part in it. In the second series the examiner pressed the patient's arm. The upper flange of the dynamic force transducer was held by the examiner's hand and the lower one was put on the bend of the patient's arm. The examiner's assistant pressed upon TLs during both series. Pressure on the transducer was initiated according to the operator's command and lasted for 2 or 3 seconds. Both measuring series consisting of 12 attempts were preceded by 3–4 background attempts without pressing upon TL.

Figure 2 Time diagram of dynamic force: time (sec.) - on the abscissa, relative amplitude - on the ordinate.

The signal was registered as a time diagram (Fig. 2) during every MMT attempt. The signal spectrum processing was performed (Fig.3). When being recorded the signal discretisation frequency was 100 Hz. FFT procedure was used while performing spectrum analysis of the middle part of the dynamic effort time diagram. It comprised subsamples 512 counts long, 50% overlap, Hamming's window, amplitude spectrum, zero-frequency component removed. Amplitude of low-frequency peak of spectrum (Fig. 3) in frequency range from 0.39 to 1.17 Hz was measured by cursor. Numerical data resulting from the experiment were processed by means of software "Microsoft Excel–97," "Statistica 5.0 - StatSoft Inc." Protocols of measuring sequences were opened only after the numerical data of the measured parameters had been fixed in the electronic tables.

Value of threshold amplitude of low-frequency peak of spectrum separating positive result (weak muscle) from negative one (strong muscle) was determined individually for every patient according to the results of background experiments, excluding patients E. and F. whose threshold levels were chosen according to the basic experiments. Moreover we noticed paradoxical dependence of these patients' results: increase of the amplitude of the low-frequency peak when the muscle was evaluated subjectively as weak and decrease of the amplitude of the low-frequency peak when the muscle was evaluated subjectively as strong. Thus, the authors treated the



Spectrum of dynamic force: frequency (Hz) - on the abscissa, relative amplitude - on the ordinate.

Figure 3

events connected with the rise over the threshold (for patients E. and F. – drop below the threshold) as muscle weakness evaluated objectively. Other cases were treated as objective evaluated muscle strength. Then we calculated the total percentage of agreement with the results of subjective MMT evaluation concerning every patient individually and the whole group, computed index kappa.[14] To determine correlation of subjective MMT evaluation, results of the deltoid muscle and fingers measuring we calculated the Kendall tau index.

Table 1.
Number of coincidences of diagnoses according to objective measuring sequences and subjective MMT evaluation.

Patient	1st sequence	Amplitude threshold	2nd sequence	Amplitude threshold
A.	11 of 12	0.00405	12 of 12	0.06
B.	7 of 12	0.00571	9 of 12	0.03
C.	7 of 12	0.0059	8 of 12	0.03
D.	7 of 12	0.0105	9 of 12	0.0145
E.	8* of 12	0.00291	6* of 12	0.022
F.	7* of 12	0.00635	8* of 12	0.0175
G.	8 of 12	0.0053	9 of 12	0.03
H.	-	-	9 of 11	0.048

Note. Figures marked with * show paradoxical relation (dependence).

Discussion

Information of data coincidence of objective measuring and subjective MMT evaluation for every patient is given in the table 1. It should be mentioned that for some technical reason patient H. was not tested according to the first measuring sequence, and only 11 attempts were done according to the second measuring sequence.

Data given in table 1 show quite high interindividual variation of the coincidence number of objective and subjective conclusions. Group average agreement numbers are 73.7% for the second sequence and 65.5% for the first one. Evaluating agreement of the results of two independent examinations it is recommended[14] to calculate index kappa (K). K2 = 0.47 for the second measuring sequence and K1 = 0.31 for the first one.

Index kappa calculated for the second measuring sequence (K2) characterises, according to,[15] moderate agreement of conclusions. K1 value shows only little agreement of conclusions. This inference is confirmed (table 2) by the analysis of correlation (Kendall tau) between subjective MMT conclusions and objective results of the first and second measuring sequences (yes/no). We can see that 5 of 8 examined patients have

statistically reliable correlation (from moderate to strong) between diagnostic MMT conclusions and the second measuring sequence. Whereas only one patient has statistically reliable correlation of diagnostic conclusions between MMT and the first measuring sequence as well as between the first and second sequences.

Thus objectifying MMT directly (the second measuring sequence) we receive good agreement with the results of subjective diagnostic evaluation of MMT result (73.7%, K2 = 0.47), which is just acceptable for practice. While trying to objectify the patient's dynamic effort agreement with the results of subjective diagnostic evaluation of MMT result turns out to be essentially lower (65.5%, K1 = 0.31). This fact can be explained both by the examiner's influence on the patient when the measuring sequence was being performed and by the examiner's partial memorizing the data of the previous MMT examination (the experiment was not absolutely blind in the part of the second measuring sequence in distinction from the first one). It should be also mentioned that the studied effect correlated with behaviour described earlier. It means absence of agreement with MMT when measuring the force of voluntary isometric muscle contraction[1-3] and quite good agreement with MMT when measuring the force between the examiner's limbs and the patient's ones.[4, 5] The result is likely to be affected by the following fact. In the first measuring sequence several muscles were involved as indicators whereas in the second measuring sequence only deltoid muscle took part. In any case to refine on the effectiveness of the method and study influence of examiner or indicator muscle on this effectiveness it is necessary to perform additional clinical testing in which several examiners would take part and the procedure should be absolutely blind.

Table 2.
Cross-correlation of diagnostic conclusions (Kendall tau).

Patient	MMT and 2nd sequence	MMT and 1st sequence	1st and 2nd sequences
A.	1.0 $p < 10\text{-}5$	0.845 $p = 0.00013$	0.845
B.	0.507 $p = 0.022$	s/u	s/u
C.	s/u	s/u	s/u
D.	0.577 $p = 0.009$	s/u	s/u
E.	s/u	s/u	s/u
F.	s/u	s/u	s/u
G	0.577 $p = 0.009$	s/u	s/u
H.	0.633 $p = 0.0067$	-	-

Note: s/u – statistically unreliable ($p > 0.05$).

Conclusion

1. Method and apparatus for MMT objectivization have been developed. According to the pilot experiment they show moderate agreement with the results of subjective diagnostic MMT evaluation.

2. According to the pilot experiment there is bigger agreement with the subjective diagnostic MMT evaluation when measuring the dynamic force spectrum between the examiner's limbs and the patient's ones than when the patient's dynamic force spectrum is measured without the examiner's participation.

3. To refine on the effectiveness of the offered method it is necessary to perform enlarged clinical testing in multi-center research and the procedure should be double blind.

References

1. Rybec CH, Swenson R. The effect of oral administration of refined sugar on muscle strength. J Manipulative Physiol Ther 1980;3(3):155-61.
2. Grossi JA. Effects of applied kinesiology technique on quadriceps femoris muscle isometric strenth. Phys Ther 1981;61(7):1011-6.
3. Kenney JJ, Clemens R, Forsythe KD. Applied kinesiology unreliable for assessing nutrient status. J Am Diet Assoc 1988;88(6):698-704.
4. Marino M, Nicholas JA, Gleim GW, Rosenthal P, Nicholas SJ. The efficacy of manual assessment of muscle strength using a new device. Am J Sports Med 1982;10(6):360-4.
5. Bohannon RW. Manual muscle test scores and dynamometer test scores of knee extension strength. Arch Phys Med Rehabil 1986;67(6):390-2.
6. Hsieh CY, Phillips RB. Reliability of manual muscle testing with a computerized dynamometer. J Manipulative Physiol Ther 1990;13(2):72-82.
7. Motyka TM, Yanuck SF. Expanding the neurological examination usingfunctional neurologic assesment: part I methodological considerations. AK Review 1999;8(1):21-32.
8. Caruso W, Leisman G. A force/displacement analysis of muscle testing. Percept Mot Skills 2000;91(2):683-92.
9. Perot C., Meldener R., Goubel F. Objective measurement of proprioceptive technique consequences on muscular maximal voluntary contraction during manual muscle testing. Agressologie 1991;32(10):471-4.
10. Leisman G, Zenhausern R, Ferentz A, Tefera T, Zemcov A. Electromiographic effects of fatigue and task repetition on the validity of estimates of strong and weak muscles in applied kinesiology muscle-testing procedures. Percept Mot Skills 1995;80:963-77.
11. Chernysheva TN, Korenbaum VI, Apukhtina TP. .Patent of Russia 2171623. Method of muscle testing objectivization. Filled 10.08.1998. Published 10.08.2001.
12. Apukhtina TP, Korenbaum VI, Chernysheva TN. Objectivization of the muscle testing method used in the applied kinesiology. Vestnik novykh meditsinskikh tekhnologuiy 1998;5(3-4):100-2.
13. Vasilyeva LF, Chernysheva TN, Korenbaum VI, Apukhtina TP. About peculiarities of the effect of muscle functional weakness. ICAK Proceedings. Atlanta 2001. p.63-6.
14. Vlasov VV. Effectiveness of diagnostic research. Moscow: Meditsina; 1988. p.256.
15. Lawson A, Calderon L. Interexaminer agreement for applied kinesiology manual muscle testing. Percept Mot Skills 1997;84(2):539-46.

Case Study: Eczematous Dermatitis and the Deep Tendon Reflex Examination

Robert Ciprian, D.C.

Abstract

A patient presents with right ankle, right knee and low back pain. The patient also has a history of eczematous dermatitis. Applied kinesiology diagnosis for structural treatment provided the patient with more than he expected.

Key Words: Lordosis, Eczematous Dermatitis.

Introduction

A 28-year old Latino male presents with chronic right ankle, knee, and low back pain. The history showed that the injuries were traumatic and were caused by playing basketball. On examination the lumbar spine showed decreased lordosis. He had right knee pain over the popliteus muscle. The right foot was excessively pronated and there was decreased dorsiflexion upon gait analysis. Past medical history showed chronic eczematous dermatitis for the past year and a half. The patient was using a prescription cream from his dermatologist that gave no relief from his condition.

Discussion

The treatment consisted of basic applied kinesiology protocol. Important points for this case study are the corrections of pelvic category III, category II, right lateral rotated tibia, right lateral talus, right inferior navicular, right superior 1st cuneiform and right lateral cuboid. There was still some residual low back pain. So I performed the deep tendon reflex examination[1] as per Richard Belli, D.C., DACNB. The right quadriceps reflex was struck. The test showed more than one inhibition of the ipsilateral hamstrings tested as a group and contralateral quadriceps as a group. This was a positive test of the deep tendon reflex examination. Therapy localization to L3 negated the positive test. L3 PL was adjusted in side posture and the patients' low back, knee and foot pain were improved.

By the third visit the patient's musculoskeletal complaints had subsided. The patient continued to tell me that he was very surprised that his dermatitis had also disappeared. Despite the topical prescription ointment he had been using, this was the first relief that he had from the dermatitis.

Conclusion

The deep tendon reflex examination is a very powerful tool. When the proper structural corrections are delivered and neurology is normalized amazing things can happen. Structure effects chemistry. This is just an example how a very simple treatment can have profound effects.

References

1. Walther, DS. Applied Kinsiology: Synopsis, 2nd Edition. Systems DC. Pueblo, CO. 2000.

Case Study: Multiple Sclerosis

Robert Ciprian, D.C.

Abstract

Multiple Sclerosis is a dreaded disease that most of the public believes to be incurable. This is a case study of a patient who was determined to change her life.

Key Words: Multiple Sclerosis, L4 Reflex, Ileocecal Valve.

Introduction

A 28-year-old Mexican female presented with symptoms of multiple sclerosis and had been wheel chair bound for 2 years. She had had some relief from pain in the past from chiropractic care. Her goal at the time was to relieve her low back pain. She was driven in general to overcome her handicap and I let her know that we would work together to accomplish as much as possible.

Discussion

The patient was an energetically vibrant mother of two. Her husband had recently died of causes unknown to medical doctors about the same time that the patient started displaying multiple sclerosis symptoms. As she had been wheel chair bound for 2 years, she had considerable atrophy to her lower extremities. The patient had a decreased L4 reflex. Her toenails were discolored, brittle and flaking. She had a major fungal infection. On questioning she said that the fungal infection began just before she started having the multiple sclerosis symptoms. She also confirmed a history of digestive problems.

Applied kinesiology examination showed a positive closed Ileocecal valve therapy localization and a category III pelvic fault.[1]

Treatment

Treatment consisted of a basic applied kinesiology protocol. Important to note for this case study is the correction of a category III pelvic fault and Ileocecal valve reflex treatment with spinal adjustments to the spinal correlations. She tested positive for the following nutrients: Calcium Lactate, Spanish black radish, and Zymex II, as well as SF 722 (10-undecylenic acid from castor bean oil). The patient was also instructed to remove sugar, wheat, corn, dairy, soy and fermented foods from her diet and to eat whole foods.

Results

After 5 months of treatment with standard applied kinesiology protocol the patient's re-exam showed a 60% increase in lower extremity strength, 90% decrease in low back pain, normal L4 reflex, improvement of digestion and a healthier appearance of toe nails. The patient also stated that she was now able to go to the bathroom by herself and stand up at the kitchen sink to wash the dishes. I informed her that it was time to seek physical therapy to help with the atrophy of the lower extremities.

Conclusion

I treated this patient very early in my career using what I had learned from my mentors, which was to treat the person and not the problem. There is so much information available about conditions such as multiple sclerosis that trying to find where to start can be daunting. A person can easily be led down the wrong path following recipes and cookbook solutions. Applied kinesiology diagnosis and treatment allows for the opportunity to ask the patient to ask the patient for the solution to any health challenge.

References

1. Walther, DS. Applied Kinsiology: Synopsis, 2nd Edition. Systems DC. Pueblo, CO. 2000.

Applied Kinesiology Management of Nocturnal Enuresis; A Case Study

Cecilia A. Duffy, D.C., DIBAK

Abstract

A four-year, five-month-old male patient with a chief complaint of nocturnal enuresis was examined and treated successfully utilizing applied kinesiology methods.

Introduction

Enuresis is the involuntary passage of urine with nocturnal enuresis being involuntary passage of urine while sleeping. It can be divided into Primary Nocturnal Enuresis and Secondary Nocturnal Enuresis.

Primary Nocturnal Enuresis comprises approximately 85% of cases of enuresis and is defined as the child never having a dry bed. Secondary Nocturnal Enuresis is defined as nighttime bladder control for a minimum of three months followed by wetting.

Both Primary and Secondary Nocturnal Enuresis can be caused by maturational, organic, or psychosocial problems.

Maturational causes include small bladder capacity and immature sleep arousal pattern.

Organic causes include urinary tract infection or structural abnormality, bladder innervation disorder, myelomeningocele, diabetes mellitus, diabetes insipidus, hyposthenuria, sickle cell anemia/trait, or sensitivity to foods. Organic problems are a rare cause of nocturnal enuresis.

Emotional causes include temporary stress, regressive behavior due to birth of sibling, or severe emotional disturbance.

Males are affected two times more than females and 75% have a positive family history.

Medical treatment options for non-organic nocturnal enuresis includes: counseling, bladder exercises, elimination diet, hypnosis, medications, or buzzer-alarm conditioning.[1]

Discussion

A four-year, five-month-old male patient presented with the chief complaint of enuresis. He had never experienced a dry night and while napping during the day he would lose bladder control as well. He was otherwise toilet trained during waking hours.

Examination revealed the following: weight 47 pounds; height 44.5 inches; axillary temperature 98.6°; salivary pH 6.8; blood pressure seated 90/60, standing 94/60, supine 88/58; pulse seated 80, standing 88; Lingual Ascorbic Acid Time test 5 seconds bilaterally; right dominance of hand, eye, ear, and foot; urinalysis was within normal limits; Sulkawich Test for urinary calcium was Grade 1; and Koensburg Test for urinary sodium was 30 plus.[3]

Postural examination revealed an elevated left occiput, low left shoulder, low left pelvis, and bilateral foot pronation.[3]

Manual muscle testing revealed a conditionally inhibited left upper trapezius which became conditionally facilitated upon oral insalivation of Cataplex B.[2] [3]

Challenge mechanism revealed subluxations at L5 (anterior), Category II (right posterior ischium), T4 (anterior), and the sphenobasilar suture (inspiration fault on the right).[3]

Correction of subluxations was accomplished, the patient was given Cataplex B at a dose of one tablet, three times per day, they were instructed to reschedule in two weeks, and the mother was to obtain an accurate count of the volume of liquids the patient was consuming.

The patient returned in two weeks and the mother reported that the patient had five dry nights in the two-week period. The patient had never experienced a dry night up to this point. The mother reported the patient's intake of water at three eight-ounce glasses per hour with approximately two urinations per waking hour. Based on this, I examined and treated him utilizing applied kinesiology methods and rescheduled the patient in two weeks for treatment and venipuncture to measure antidiuretic hormone levels to rule out diabetes insipidus. Antidiuretic hormone was reported as 1.3 pg/ml with 0.0-6.9 pg/ml being the reference range. Osmolality was reported as 295 mOsm/kg with the reference range being 275-295 mOsm/kg. At that time the mother reported that daytime wetting during naps had stopped and the patient had three dry nights in two weeks. The patient was examined and treated utilizing applied kinesiology methods. He was given Drenamin[2] at a dose of 1 tablet, three time a day and the mother was instructed to restrict the patient's water intake to eight ounces per hour.

Two weeks later the mother reported that the daytime wetting was still gone, but only two dry nights. It was noted during this examination and treatment that the left upper trapezius posture (elevated left occiput and low left shoulder) had resolved, the pelvis remained low on the left, and the feet remained pronated. The mother was instructed to restrict water intake to four ounces per hour.

Two weeks later the mother reported that the patient did not comply with the four ounces per hour restriction. He had five dry nights. He was examined and treated again and restriction of water to four ounces per hour was reinforced, plus no water past 6:00 PM.

Three weeks later the report was thirteen dry nights and seven wet nights. He was examined and treated and the mother instructed to continue water restriction and to have the patient keep his shoes on during the day to support the bilateral foot pronation.

Three weeks later she reported sixteen dry nights and five wet nights, with two of the wet nights occurring when the patient fell asleep early and did not urinate before going to bed. He was examined and treated and instructed to discontinue the Cataplex B and Drenamin once his current prescription was finished.

He was seen twice more at one month intervals. Reports of night wetting occurred only when the patient consumed water past 6:00 PM. On the last visit, the patient's posture was level at the head, shoulders, and pelvis. The bilateral foot pronation remained. The patient was placed on a self-schedule basis at that time and encouraged to maintain the four ounces of water per hour restriction, no water past 6:00 PM, and to wear shoes during the day.

Conclusion

Management of a case of enuresis in a four-year-old male utilizing the three sides of the triad of health through applied kinesiology is presented.[3] Structural treatment was applied after diagnosing via applied kinesiology methods; chemical support was diagnosed via applied kinesiology methods and included supplementation with Cataplex B and Drenamin; and mental treatment was given in the form of behavior modification of water intake. It should be noted that some dry nights occurred and resolution of daytime wetting during naps was accomplished with structural and chemical treatment prior to water restriction.

The causes of nocturnal enuresis should be re-evaluated and research directed towards expanding the theory of the cause of enuresis to include subluxations within any or all of the three sides of the triad of health.

References

1. Schwartz, M. W., et al, Pediatric Primary Care, A Problem Oriented Approach, Second Edition, Chicago, Year Book Publishers, Inc., 1990.
2. Standard Process Laboratories, Palmyra, WI 53156.
3. Walther, David S., Applied Kinesiology Synopsis, 2nd Edition, Pueblo, CO, Systems DC, 2000.

The Intraosseous Subluxation, Associated Points of Acupuncture, and Redox Problems

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

Abstract

The spinal intraosseous subluxation has been found to invariably coexist with signs of acupuncture meridian imbalance indicated by positive therapy localization of an acupuncture alarm point. The lesion is invariably found at the associated point of the meridian indicated by the alarm point. Correction of the lesion seems to especially benefit the chronically ill patient thought to be "stuck" at one end or the other of the redox curve.

Goodheart's discovery of the intraosseous lesion solved the problem of the presumptive nature of the diagnosis of subluxations involved in the "bent spinous".

Key Words: Intraosseous Lesion, Subluxation, Applied Kinesiology, Acupuncture Meridian Points, Positive Therapy Localization, Challenge Technique, Redox Curve, Over-oxidation, Over-reduction.

Introduction

This article discusses the author's clinical experience of a consistent finding of an intraosseous subluxation found at acupuncture alarm point locations on the spine whenever there is an indication that the acupuncture system is imbalanced via diagnosis of meridian alarm points.

Discussion

The intraosseous lesion provided the final solution to the question of the "bent spinous."[1] Prior to the discovery of applied kinesiology, every chiropractic adjustment was based upon a presumptive diagnosis. The therapy localization and challenge techniques of applied kinesiology eliminated the reliance upon presumptive diagnosis. 100% of chiropractic adjusting can now be based upon confirmed diagnoses. The Journal of the American Medical Association 1999 reports that 85% of medical treatment continues to be based upon presumptive diagnosis using information taken from the patient's history. (Archives, www.mercola.com).

A functional relationship exists between the anatomy and the acupuncture meridian system. The factors involved include: the associated points of acupuncture, located at the spinal vertebra; the Governing Vessel, located on the midline of the spine at the spinous processes; and the Bladder meridian, located in close proximity to the vertebral transverse processes and rib heads.

The Bent Spinous Effect: The intraosseous lesion is presumptively diagnosed via therapy localization of the spinous and transverse process of the same vertebra simultaneously. This presumptive diagnosis is confirmed by the challenge technique via muscle testing, whereby a separation challenge or approximation challenge of the spinous process and transverse process produces a weakening of a previously intact muscle. Challenge technique includes the respiratory factor, either held inspiration or expiration will negate the positive challenge. In most cases the challenge is negated by inspiration. Involvement of the respiratory factor adds to the

evidence suggesting involvement of a meridian-based/energy-flow type of subluxation/dysponesis. Proof of correction is elimination of therapy localization and positive challenge. In considering such a lesion one must consider the dysponetic signaling arising from the aberrant movement in response to the cranial respiratory movement which occurs at ten to twelve cycles per minute and the respiratory movement occurring at an average of sixteen to eighteen plus times per minute. One would suspect such a lesion to play a huge part in each of the following:

1. Seemingly multifactorial dysfunction associated with redox problems.
2. Widespread reflex effects (holographic effect on Penfield's homunculus[2]).
3. Influence on split-brain activity due to universality of polarization effects.
4. Influence on paired (adrenal) vs. singular (thymus) and bi-lobed (thyroid) glands/organs.
5. Autonomic nervous system effects.
6. Differential responses to lingual magnesium and choline factors. Magnesium has inhibitory effects on the sympathetic nervous system at the cord by shutting off sympathetic activity at the spinal ganglion. Choline and cholinesterase factors apply to both sympathetic and parasympathetic function and especially the parasympathetic-related upper cervical and sacral lesions.
7. Direct musculoskeletal responses related to cord level of involvement.

The most common pattern for correction of the intraosseous lesion described herein is separation of the spinous and transverse process on only one side of the vertebra during inspiration, often, but not always, on the side of positive alarm point involvement. Pressing the spinous and transverse process apart usually weakens the indicator muscle, inspiration coincidental with the challenge usually negates the weakness, indicating that the spinous/transverse processes must be separated during an inhalation respiratory effort. The correction is performed by having the patient breathe in and hold their breath while the doctor makes several thrusts with the thumbs in a direction to separate the spinous and transverse processes.

The adjustive thrust on the proper phase of respiration immediately eliminates:

1. Positive simultaneous therapy localization of the spinous and transverse processes of the involved vertebrae at the acupuncture associated point on the spine.
2. Positive therapy localization of the associated alarm point.

This author's experience with correction of the intraosseous lesion also results in the following measurable changes: increased range of motion of formerly restricted body parts, meridian over-activity, normalization of pulse rate and blood pressure, increase in respiratory rate/volume/vital capacity, elimination of orthostatic hypotension, increased range of motion, increased visual acuity (tested before and after correction), increased auditory function, increased muscle strength and immediate improvement in salivary pH and lingual ascorbic acid time.

The intraosseous correction is often found necessary at T5 (Circulation Sex meridian relating to the adrenals) and/or L1 (Triple Heater meridian related to the thyroid) and seems to be especially beneficial in the chronic patient suspected of suffering from redox-based imbalances. Their histories include lengthy and varied treatment approaches involving, and often limited to, heavy nutritional intervention. Such patients are frequently encountered in the non-crisis care alternative medicine clinics where their symptoms are frequently attributed to allergy based, autoimmune problems.

Autoimmunity and allergy are the latest clinical buzzwords replacing hypoglycemia, parasites and "stress" as the end-all-cure-all answers to the questions of chronicity and recidivism. Such patients appear to be "stuck" at one end or other of the redox curve of cell metabolism in an over-oxidized or over-reduced metabolic state with symptoms usually attributed to: allergies, manic-depressive states, digestive tract dysfunctions,

endocrine/skeletal system based disorders, viral infections, etc., many of which defy all manner of diagnosis and therapy over long periods of time. The patients and many of their doctors are convinced that the cause of the conditions is "Candida," "hyper/hypothyroidism," "schizophrenia," "hyper/hypoadrenia," "hyper/hypochlorhydria," "high sugar," "low sugar," "fibromyalgia;" (another recent favorite, sharing the spotlight with hypoglycemia and Candida). Textbook labels, along with symptom based conventional medicine therapies, often add to, rather than subtract from, the patient's problems. The effect of the intraosseous lesion alleged herein adds to the already incommensurable nature of applied kinesiology on physiological function.[5]

Imbalances of the interspinal muscles cause a structural distortion between the transverse and spinous processes. The distortion causes a discrepancy in the representational picture between the brain and the vertebral segments. The result is an intraosseous subluxation. The much derided and oft ridiculed subluxation hypothesis of D.D. Palmer has been replaced by the medically acceptable "dysponesis."[6] Both terms describe the entity first suggested by Palmer as "interference with nerve energy flow."

The levator costales muscles that link the vertebra and rib head are also closely linked to the intraosseous lesion adding to the connection between spine and rib cage. The positioning of the Bladder and Governor Vessel meridians at the mid and lateral points of the vertebra suggest the importance of proper positioning in accordance with their expected holographic image representation in brain tissue. This further suggests that bony changes brought about by long continued muscle imbalances would eventually distort the holographic image transmitted to the brain that could result in the improper neurological signaling to body parts as suggested herein. Considering that nerves convey trophic factors to their respective end organs, one can immediately appreciate the widespread effect of an intraosseous lesion that affects a nerve to a gland.

The intraosseous lesion also directly effects the pumping of cerebrospinal fluid, the supply of which is vital to all nerve function and which is directly associated with the functioning of the respiratory factor, adding further evidence of an involvement of the acupuncture meridian system, alleged to be dependent upon a "pumping" of energy flow around the body.

"Pumping" of cerebrospinal fluid is equated with pumping of the flow of energy in the acupuncture meridians. The flow of an energy "wave" does not exist apart from the flow of a "substance", for example, the conduction of a nerve impulse is associated with movement of minerals back and forth across cell membranes. All waves, as all function, arise from movement of structure and all movement in the body requires a pumping system with a supply of energy in the form of food, air and water. Nothing moves without a pump. No wave of energy is unaccompanied by a solid, measurable substance. Proof of the existence of substance in the acupuncture meridians is demonstrated by the effect of sweeping a meridian with the hand and achieving a demonstrable effect of a related muscle function.[7] The sweep of the hand, magnetically, therefore momentarily affecting the Bonghan corpuscles in the body fascia conducting its load to the consumer end points. Ribonucleic acid (RNA) and the memory molecule of the body was postulated by Goodheart in the early 1970's. This is more evidence of the Nobel Prize quality of his work which continues to be not only ignored but blasphemed by conventional medicine. The relationships discussed herein were first reported by Goodheart in 1966, they are now 47 years old. Two generations have passed since these effects were incorporated into the body of knowledge known as applied kinesiology. During the 1960's Goodheart stated:

> "The RNA unlocks the cell door and lets the thyroid hormone enter."[3]

> "The concept of RNA…[as a] concept of cellular memory is a relatively new idea."[4]

Goodheart reminds the reader that this pertains to cellular memory rather than memory associated with old age and senility.

Each spinal vertebra is a pump. The pumping action of a vertebra has three sources. In order of their importance I would list them as:

1. The cranial respiratory motion of Sutherland.[8]

2. The motion imparted via thoracic respiration.

3. Normal physical movement of the body.

In this regard we consider two basic facts of life, that is, tendencies tend to cause accumulations and rhythm adds to that effect in exponential rather than linear fashion.

The spinous process of a vertebra moves inferiorly on inspiration and superiorly on expiration. The constant inferior to superior movement of the spinous with respiration pumps cerebrospinal fluid in the spinal column and thence down the nerves. Dysfunction involving the intraosseous lesion interferes with the pumping action. This in turn interferes with split brain activity (left/right, up/down, fore/aft problems), nerve energy flow, respiration, lymph flow, vascular flow, etc.

Thyroid and adrenal function acutely influences oxidation/reduction and the redox curve. The under oxidized, low thyroid state often fails to respond to the best conventional medicine therapy due to the failure of T3 at the cell level which is linked to (among other factors) the reduced pumping action of its physiological pump, the teres minor. The muscle gland relationship was reported in the mid 1960s by Goodheart.[9]

Failure of the teres minor over a long period of time can lead to decreased thyroid function which results in decreased liver function, indigestion, lack of absorption, protein deficiency due to decreased gastric hydrochloric acid, and eventually, a malnourished state. The end result, the bottom line, is a lack of RNA (the key to the cell door) resulting in the sequence of events from thyroid function to cellular memory.

The beneficial, often immediately observable, results of a correction of the intraosseous lesion at T5 and/or L1 is thought to be related to the widespread effects of thyroid and adrenal function in their control of metabolic rate (thyroid) and intensity (adrenal). Such widespread effects can only be attributed to a central cause.

Goodheart further explained that thyroidal control over bone marrow blood cell production (via temperature control) and liver function (via metabolic rate control) can result in misleading widespread effects and symptoms: lowered resistance, increased infections, dry rough skin, hair loss, brittle flaking fingernails, indigestion, frequent headaches, mild deafness, poor memory, big flat tongue, gonadal dysfunction in both male and female and progesterone levels particularly in menstrual difficulties.[3] [4] These patients show dysfunction of the teres minor muscle and extreme tenderness of the costal cartilages or posterior rib cage that responds to tasting nutritional grade iodine or rubbing pharmaceutical iodine on the skin. The costal cartilage soreness is partly the result of lymphatic reflex point involvement and is similar to the “ouch point” of Acupuncture. The patients are under oxidized and over reduced as the slightest stress brings into play the adrenal gland until it too fatigues. The result is poor oxidation/reduction overall.

The coincidental location of the Bladder and Governor Vessel meridians on the spine give rise to the opportunity to diagnose specific cause-effect relationships between structure and endocrine function. Penfield’s original representational system of the brain[2] was described by Goodheart in a clinically useful manner as representing a “holographic picture” of body structure, and because a holograph requires three elements, it brings in, once again, the three-fold nature of man.

In retrospect this article serves as both a reminder and a warning concerning the powerful tool made available by Goodheart’s momentous discovery of the non-invasive, comprehensive, diagnostic value of muscle testing that has been placed in the hands of mankind. The discipline is explicable, demonstrable and reproducible, fulfilling all the requirements of a scientific discipline.

The healing professions, much less society, have not yet begun to appreciate AK. Because of popular books on the subject, there are probably more laypersons than doctors in touch with the discipline. One could spend a lifetime simply investigating and demonstrating scientifically the remarkable relationship between applied kinesiology and the ancient acupuncture meridian system, a dovetailing that finally brings together the endocrine, organ, and neuromusculoskeletal systems, under one easily accessed "musculo-visceral" roof.[10]

Goodheart's brilliant combining of the acupuncture meridian system with the musculoskeletal system has illuminated human biological function in a manner that is so mind boggling in its simplicity and ease of access and effectiveness as a therapeutic tool that one would have to be brain dead not to be thrilled by it!

History offers a caveat with the following three examples: About 5,000 years passed before the acupuncture meridian system was brought to light, it took 1,500 years for scientists to reject the notion of a flat, earth-centered universe and to accept the heliocentric universe, and it took 50 years to implement the prevention and cure of scurvy *after* its efficacy was demonstrated at a time when the British fleet was losing over ten thousand sailors a year to the disease.

Given the fact that no less than a former physician to a president of the United States publicly recognized applied kinesiology as "a new method of diagnosis" a quarter century ago, (remark uttered by Janet Travell, MD, President Kennedy's physician, during her lecture and demonstration at the 25th Annual Rowe Smith Dental Conference, San Antonio, Texas, March 1978), future historians will shake their heads in disbelief when noting the stifling effect on the propagation of AK that politically motivated "keepers of tradition" have exerted. Vested interests in the status quo continue to prevail in all quarters of society.

New ideas and methods are rejected with a force that operates in direct proportion to the effect the idea has on society. This writer continues to encounter physicians who continue to deny the reality of the meridian system! Given this fact one can only conclude that it might be a century or two before the full potential of applied kinesiology is accepted and popularized as the "new method of diagnosis" identified over a quarter century ago by Dr. Travell.

No therapeutic technique or claim escapes evaluation by the all-encompassing umbrella of AK.

Conclusion

The intraosseous lesion is related to the associated points of acupuncture and specific acupuncture meridians. The lesion is diagnosable via non-invasive applied kinesiology technique and has been found to have wide ranging physiological effects. Search for an intraosseous lesion at the associated point of the involved meridian is recommended whenever acupuncture meridian imbalance is diagnosed.

References

1. Goodheart, George J., Jr., "The Interosseous Vertebral Holographic Subluxation, Part I", Chiropractic Economics, Vol. 29, No. 2, Sept/Oct 1986 and "The Interosseous Vertebral Holographic Subluxation Part II," Chiropractic Economics, Vol. 29, No. 3, Nov/Dec 1986.
2. Penfield, W., Rasmussen, T., The Cerebral Cortex of Man, Macmillan, New York, 1950.
3. Goodheart, George J., Jr., The Thyroid-An Overlooked Problem, The Digest of Chiropractic Economics, 1969.
4. Goodheart, George J., Jr., The RNA Meridian Relationship To Applied Kinesiology, The Digest of Chiropractic Economics, Mar/Apr 1973.
5. Duffy, Daniel H., Sr., Subluxation, The Incommensurable Paradigm of Chiropractic, AK Printing, Geneva, Ohio, 1991.

6. Dorland's Illustrated Medical Dictionary, W.B. Saunders Co., Philadelphia, 1974.
7. Walther, David S., Applied Kinesiology, Synopsis, Second Edition, Systems DC, 2000, page 238.
8. Ibid, page 376.
9. Ibid, page 14.
10. Duffy, Daniel H., Sr., Musculovisceral, A Recommendation for the Use of a New Term in Chiropractic, Proceedings of the International College of Applied Kinesiology, 1990.

The TMJ, Opener or Closer? Clarification of a Principle

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

Abstract

Because of the relative use of temporomandibular joint (TMJ) closer vs. opener muscles, clinical experience indicates a need for more treatment to closer than opener muscles and dysfunction thought to be due to the TMJ opener muscles are quite often caused by the closers. The most common pair requiring muscle spindle treatment to inhibit function is the right masseter and left temporalis muscles.

Key Words: Temporomandibular Joint (TMJ), Opener, Closer, Applied Kinesiology.

Introduction

In the 1980's, during one of my monthly visits to George J. Goodheart, Jr., DC, DIBAK in Detroit, I was asked to make a tape on the TMJ. The tape was made within an hour of the request without any time for preparation or organization of the material. It was an off the cuff, unrehearsed, completely extemporaneous delivery that was later published in Goodheart's annual Research Manual of Applied Kinesiology, 1988 issue.[1]

Discussion

A recent teaching experience brought about a discussion of opener vs. closer TMJ problems as I demonstrated TMJ technique. The patient showed positive therapy localization on jaw opening on the right only on opening with lateralization to the right. The initial postural exam had revealed that her jaw deviated to the right on closing. While therapy localization to the TMJ was negative in the clear, therapy localization to the belly of her right masseter was positive in the clear. The positive therapy localization of the right TMJ with right lateralization on opening was negated by simultaneous therapy localization to the belly of the right masseter. The left TMJ failed to therapy localize in any position, however the posterior belly of the left temporalis (over the squamous portion of the temporal bone just above the left ear lobe) did therapy localize in the clear.

The point of this discussion is that the jaw only showed on opening but related to two closers muscles, an ipsilateral masseter and contralateral temporalis. One point of my original discussion in the 1988 Research Manual was that I was never able to achieve satisfactory results in opener TMJ problems by treating the external pterygoid via the usual recommended muscle spindle/Golgi tendon organ techniques. Not until the discovery of the strain counterstrain treatment was this author able to observe reliable changes in jaw opener problems by treating the opener muscle.

Since my treatment to the patient mentioned herein was only to the muscle belly, a student questioned my prior comments (in the Goodheart manual) stating that I usually ended up "pulling the muscles apart" to get the results. He mentioned that my former comment did not stack up with what I had just performed and questioned what he thought to be a historical progression in technique. I had to remind him that the tape was performed on the spur of the moment in unrehearsed fashion and thus was incomplete. The statement, "pulling apart" was meant to confer the meaning of weakening or turning down, or detuning or sedating a muscle which was most often accomplished by pushing together on the muscle belly with little or no attention required to the origin insertion.

I demonstrated on the case at hand that no "pulling apart" at the Golgi tendon insertions was necessary. A technique for therapy localizing the temporalis was discussed, that is to say, therapy localization of the ends of the temporalis muscle on the side of the skull with the fingers and therapy localization with the thumb at the other end at the TMJ.[2] I demonstrated that this technique did not show positive therapy localization while therapy localization of the posterior belly as mentioned above did show positive therapy localization. Some confusion arises in teaching the sections of the temporalis muscle since the posterior belly visually give more of an appearance of being inferior rather than posterior. The nomenclature often misguides the student in the initial learning of proper hand placement for therapy localization of the different sections of the temporalis muscle.

This author recommends the AK Flowchart[2] for in office use by clinicians with Synopsis, Second Edition[3] for home study to get the in depth information. Both are outstanding contributions to the body of knowledge known as applied kinesiology.

The suggestion for the use of AK is to ask questions rather than make statements. Reading body language accurately begs a questioning attitude. The focused clinician simply searching for dysfunction and observing in accordance with the Goodheart workshop procedures is, in a body language way, asking questions rather than making statements. As a result the clinician so acting, receives answers, the questions need not be verbal but rather are better expressed by behavior. Knock and the door shall be opened. It has been said that one needs know only a small amount, but to learn that small amount a very large amount must first be known. The method of knocking is a small thing to learn but opens up a world of things to do. The method of knocking here is that knack of simply looking and observing and allowing nature to take its course. This is preferable to the habit of the arrogant entrepreneur seeking to make statements rather than asking questions in the proper manner.

Conclusion

TMJ problems found on jaw opening often require treatment to closer muscles. Students are taught to think of the masseter as the quadriceps of the jaw muscles. Grinding and chewing exerts much more exercise on the masseter muscle than opening the jaw requires of the external pterygoid muscle. This is similar to the relatively excessive use of the quadriceps muscle in the thigh in comparison to the abdominal muscles that creates a situation allowing the quadriceps to overwhelm, hence neurologically inhibit, the less exercised abdominals.

References

1. Goodheart, George J., Jr., 1988 Applied Kinesiology Workshop Procedure Manual, privately published, 1988.
2. Leaf, David, Applied Kinesiology Flowchart Manual, 2nd Edition, privately published.
3. Walther, David S., Applied Kinesiology, Synopsis, Second Edition, Systems DC, 2000.

Transverse Flicking Technique

G. Kingman Fung, D.C.

Abstract

This paper contributes to the existing body of soft tissue therapies accumulated in applied kinesiology. It can be used with chronic tendonitis, reoccurring origin insertion problems, muscle adhesions and especially in chronic cases where patients have very low metabolic rates.

Introduction

Applied kinesiology has introduced some original therapies and adapted others in an effort to have wide range of treatments for soft tissue injuries. Applying Transverse Flicking Technique can further free up scar tissue and adhesions in muscle spindles and tendons. It is also very effective in origin insertion problems that reoccur.

Methods and Summary of Procedures

To determine a positive challenge, take a strong indicator and with some pressure cross the area in question three times in one direction. If the indicator weakens, you've found the direction of correction. If there is no change in the indicator in either direction, there is no need for the technique.

The technique can be applied in one of two directions, either away from the doctor or toward the doctor. The thumb is utilized in the away direction. At the place of a positive challenge, use a slight tissue pull towards yourself, as your thumb drops off the muscle or tendon, then "flick" or push in a smooth motion away from yourself, across the muscle or tendon. The same technique is utilized with two or three fingers, but in the opposite direction. For example, the "flicking" motion is performed by pulling towards you.

In large areas, like the back, a doctor would use both of his thumbs and two to three fingers of each hand, with the patient prone. On the extremities, one or two hands can be used, as needed, sometimes with one hand stabilizing while the other hand flicks.

1. Isolate the tendon or muscle.
2. Find a strong indicator.
3. Challenge across the tendon or muscle three times in one direction.
4. Apply the technique in the direction that weakened the indicator.
5. Repeat the flicking technique five to seven times across the muscle or tendon.
6. Re-challenge the area three times. If there is no change in the indicator, the technique is successful. If the indicator re-weakens, repeat the flicking technique and re-challenge.

Discussion

Tendonitis occurs as a result of muscle imbalances in repetition and overuse injuries. The load exceeds the muscle's capability to endure the load, and the load falls to the tendon, which then becomes inflamed. Scar tissue and adhesions eventually build, decreasing that area's ability to fire its 1A afferents signals to the brain.

The Transverse Flicking Technique frees up tendons and spindles from adhesions and re-modulates 1A afferent signals to the brain. 1A afferents are the largest diameter afferents and are primarily responsible for most of cortical afferentation. That is to say that the summation of 1A afferents from muscle spindles and mechanoreceptors dictate the frequencies of firing of the entire neuraxis as postsynaptic effects spread through the homologous pools of neurons at every level of the neuraxis. This also happens to be the same pathway that is fired as a result of an adjustment of a joint.

One of the positives of the Transverse Flicking Technique is that it has a low metabolic effect on the body because of its light nature and can be used very successfully on chronic or "weak" patients without exceeding their metabolic rate as deep tissue therapies might.

The same type of patients that may feel worse or suffer from headaches or autonomic concomitances after a treatment with low amplitude, high speed osseous adjustments to the cervicals, side postures or deep tissue work will usually have a positive response to respiratory adjustments, blocking and the Transverse Flicking Technique.

Conclusion

Transverse Flicking Technique provides an additional tool that can help in various soft tissue needs and especially where soft, but effective stimulus would not exceed the patient's metabolic rate.

References

1. Walther, David, *Applied Kinesiology Synopsis, 2nd Edition,* Pueblo Colorado Systems D.C. 2000.
2. Carrick, Frederick Robert, *Post Doctoral Program of Study in Chiropractic Neurology,* San Francisco, CA, 1995–1998.
3. Dobkin, Gene, *Neurostructural Integration Technique,* Seminar, Seattle, WA, 2000.

Acid-Base Metabolism: A Study to Evaluate Different Measurement Methods

(Including Summary of 5 Case Histories)

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Abstract

In this study measurements of the acid-base metabolism were performed; blood gas analysis according to Astrup, urine acid titration according to Sander, lactate measurement and sensory provocation as by applied kinesiology. The obtained parameters were related and compared. The result was that no single measurement method could furnish an evident diagnosis regarding the acid base situation of the patient. Instead the parameters which were obtained with various methods have to be assembled to an overall picture. The therapy must address metabolic, mechanical and emotional stressors. Therapy concepts for metabolic stresses are proposed as well as several practical examples given.

Key words: Latent Acidosis, Latent Alkalosis, Actate, Diagnosis of Acid Base Metabolism, Astrup, Urine Titration, Applied Kinesiology, Therapy of Acid Base Imbalances.

Introduction

In all chronic health disorders, imbalances of the acid base metabolism have to be considered and evaluated. Both latent acidosis and latent alkalosis have negative impact on the immune system (immune suppression as well as induction of allergy) and are accompanied by an increased potential of inflammation and pain mediators. Chronic acidosis seems to induce an increased production of kinines and a decreased activity of the kininase.

According to Wiley (1987) acid base imbalances are accompanied by psycho-emotional disorders. This illustrates that practically every health disorder can be caused by acid base imbalances (among others). It has to be avoided, however, to focus exclusively on the so-called latent acidosis. The frequently used urine titration according to Sander offers, as will be demonstrated, no conclusive evidence for metabolic acidosis even if there is a typical acidosis picture.

There are principally two slightly diverging theories regarding acid base imbalances: one has its origin in German speaking natural health care with Sander (1985) and Worlitschek (1994). The former developed the urine titration method, the latter bases his theory mostly on the acid titration method of the blood according to Jörgensen (1985). Both authors do not even consider alkalotic situations or: “all evil lies in acidity.”

The second opinion is from Wiley (1987) and was introduced into applied kinesiology literature by Goodheart, Leaf and Shafer. As a principle, Wiley describes three types of individuals: the first type reacts to stress with a shift to alkalosis. The second towards acidosis and the third is an intermediate type who has a general tendency towards acid shift. He bases his doctrine on approximately 200 pH-measurements in venous blood.

The discussion about the different theories is certainly not completed. In this study a correlation between base excess and pH from blood gas analysis according to Astrup, urine acid titration, lactate in venous blood, as well as outcomes of diagnostic provocation with applied kinesiology, is presented. The different measurement methods of acid base metabolism and their potential errors are discussed.

Definitions and Physiological Basics

Alkalosis
Decompensated state with plasma pH values above 7.45 (arterial blood) with exhaustion of buffering mechanisms (Schmidt-Thews, 1990).

Latent (Functional) Acidosis
Compensated state, where the pH value of the blood is maintained normal (7.37 to 7.45 in arterial blood) by means of the buffer capacity of the organism. Decrease of base reserve caused by increased utilization of the buffer capacity.

Acidosis
Decompensated state with plasma pH values below 7.37 (arterial) with exhaustion of the buffer mechanisms.

Latent (Functional) Alkalosis
Compensated state with increase of base reserve.

pH Value
The pH value according to the following equation is a measure for H+-ion concentration in a solution: pH = -log [H+]. The normal values for arterial blood (plasma) according to Schmidt-Thews (1990). are between 7.37 and 7.45 at 37º C. For venous blood they are between 7.32 and 7.43. The erythrocyte pH seems to measure between 7.2 and 7.3 (Schmidt-Thews, 1990) but can be measured only with difficulty.

Buffering Capacity
The common chemical definition is as follows: (Δ=deviation, BB=buffering bases).

$$\text{Buffering capacity} = \frac{\Delta\ BB}{\Delta\ pH}$$

The buffering capacity therefore is the relation of the added amount of acid to the resulting change in pH (for instance by one unit). For the buffering capacity, apart from the concentration of the buffering system, the proximity of the actual pH to the pK-value of the system is crucial. Reduced hemoglobin has a buffering capacity which is 0.3meq/10gHb (0.48meq/meqHb) higher than the one of oxygenated hemoglobin (Jörgensen, 1985). This fact has to be taken in consideration when comparing arterial and venous blood samples.

Total Buffering Bases
Their concentration in arterial blood is 48meq/l (Schmidt-Thews 1990). They are strong bases: HCO_3^- (22-28meq/l), hemoglobin (7.3-9.6meq/l), HPO_4^{2-} and plasma proteins with their ionisable side groups (17-18meq/l), which, with the corresponding weak acids, form the three buffering systems. The concentration of the buffering bases is little influenced by CO2 partial pressure and therefore serves well to describe an increase or decrease of non-volatile acids in the blood (Schmidt-Thews 1990). The buffer systems of the blood can be described in the following simplified Van Slyke equation:

$$pH = pK + \lg = \frac{HCO_3^- + HPO_4^{2-} + \text{Hemoglobinaic} + \text{Proteinate}}{H_2CO_3 + \text{Lactate} + \text{Acetoalctate} + \text{ß - Hydroxybutyrate}} = 6.1 + \lg \frac{48\ \text{mmol/l}}{2.4\ \text{mmol/l}} = 6.1 + 1.3 = 7.4$$

pK is the dissociation constant, that is the pH - value at which acid and base in a buffering solution are in equilibrium. For blood, pK = 6.1.

H_2CO_3 contributes only 50% of the total acidity of the blood as CO_2 concentration is calculated from pCO_2

as follows: 40 x 0.03 = 1.2 meq/l. This means that the other 50% acidity (the resting 1.2 meq/l in the denominator of the equation) are non-volatile acids which are derived from intermediate metabolism, lactate, keto acids and others.

Bicarbonate Buffering System
The bicarbonate buffering system is described by the Henderson-Hasselbalch equation.

$$pH = pK + \lg \frac{HCO_3^-}{H_2CO_3} \quad (pK = 6.1)$$

By means of this equation blood gas machines calculate carbonate from pCO2 and pH. The pK of the bicarbonate buffer is 6.1. It therefore is relatively distant from the pH value of the plasma (7.4), and therefore the buffering effect is only great because of the high concentration of the buffer.

pCO_2 (CO_2 Partial Pressure)
Increase and decrease of CO_2 partial pressure indicates that there is a primary respiratory acid base disorder: decreased pCO_2 is associated with primary respiratory alkalosis. A metabolic acidosis within a short delay is compensated by hyperventilation generating decreased pCO_2, a metabolic alkalosis by hypoventilation causing increased pCO_2.

Base Excess (BE)
In general terms: BE = BB measured -BB normal.

The value of the normal buffering bases is 48meq/l (Schmidt-Thews 1990). The base deviation (positive or negative base excess) therefore is a calculated value derived from pH and pCO_2. The value is dependent on Hb concentration. A positive BE with normal pCO_2 indicates that there is a primary metabolic alkalosis, that is a decrease of nonvolatile acids. Negative BE (actually base deficiency) indicates that there is a primary metabolic acidosis, that is an increase of nonvolatile acids.

These values are essential, for changes in pH only occur relatively late as the buffering mechanisms of the body (see above) prevent this. The independence of BE from HB and pCO_2- is postulated in Schmidt-Thews (1990). In Thomas (1998). In opposition to this, it is not considered so and therefore the utilization of BE is considered inaccurate. When the BE is calculated, CO_2 is regarded to cause formation of equivalent amounts of H+ and HCO_3- while the H+ is balanced by proteinate.

Base Deviation of the Extracellular Fluid (BDecf)
This value is defined as base concentration in extracellular fluid, measured by titration with strong acid or base towards pH 7.4 at pCO_2 of 40mm Hg. The term ‘extracellular fluid’ comprises the interstitial fluid, the plasma and the cellular components of the blood (Thomas, 1998). The value unlike BE is practically independent of pCO_2 and Hb. Normal is between -2 and +3.

Anion Gap (AG)
The anion gap consists of the unmeasured ions in plasma (proteins, phosphate, sulphate, organic ions).
It is calculated according to the equation AG (meq/l) = cNa^+-cCl^- + $cHCO_3^-$.

An increase of the anion gap is a sign of increased concentration of acids like lactate, ketone acids and various acid radicals (Thomas, 1998). Low HCO_3^- values and a high AG value normally coincide, which is the basis for the practice to deduct the total buffer base situation from the HCO_3^- concentration and the BE, which both are calculated from pH and pCO_2. Increased AG values also can be accompanied by normal $cHCO_3^-$ values (Harrison, 1998), which can be relevant for the interpretation of blood analyses.

Titratable Acid
It is defined as the eliminated acid which is neutralized by buffering. The amount of the eliminated acid in the urine is measured by titration with a base (see also acid titration according to Sander).

The $H_2PO_4^{2-}$/ $H_2PO_4^-$ buffer is responsible for 80%, uric acid for 20% of the buffering. The titratable acidity only comprises one third to one half of the total acid eliminated daily.

Total Acidity
This is defined as the sum of titratable acidity and the acid which is eliminated by the ammonia (NH_4^+) mechanism. NH_4^+ eliminates 50-65% of the total acidity.

Metabolic Regulation of Acid Base Metabolism
For internal medicine, orthopedic, natural health care and pain clinic the respiratory acid base disturbances are less important than the conditions with increased production or decreased elimination of nonvolatile acids or a lack of the latter. Respiratory disturbances on the other hand are a common problem in intensive care and pneumology. Compensatory changes of pCO_2 occur when the metabolic regulatory mechanisms to eliminate the nonvolatile acids are exhausted.

The kidney plays the crucial role in acid elimination: if acid were eliminated only as H^+, with a minimal urine pH of 4.4 the amount of 1,250 litres of urine would be necessary to eliminate 50meq of acid (Devlin, 1997). The daily production of acid is as much as 60-100meq.

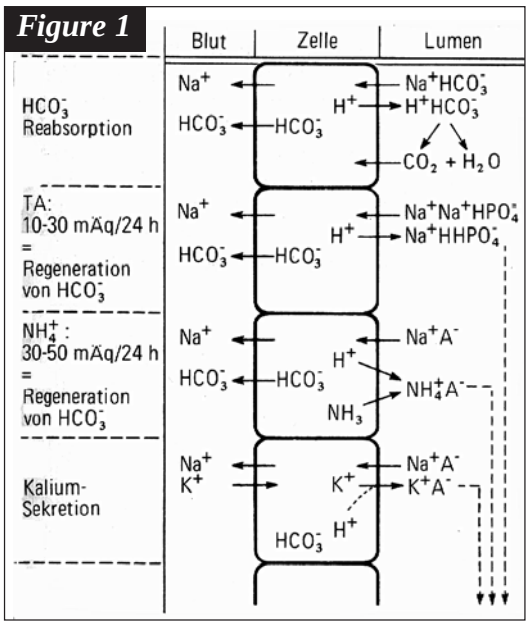
1. The elimination occurs predominantly in buffered form as $H_2PO_4^-$ and bound to NH_4^+: The secretion of H^+ in the renal tibuli is a process catalyzed by carboanhydrase: Na^+ via an active H^+ - dependent transport mechanism is reabsorbed from the tubular lumen into the cell. H^+ in exchange is secreted into the tubular lumen. There it is buffered by HCO_3^-.

   The carbonhydrase located at the tubular cell membrane transforms carbonic acid into CO_2 and H_2O. CO_2 diffuses back into the cell, where carboanhydrase transforms CO_2^+ H_2 to HCO_3^- and H^+. The HCO_3^- via a Na^+ coupled carried system is carried back into the blood stream. This exchange process also brings back HCO_3^- from the tubular lumen so that at the end of the Henle loop there remains a concentration of only 5meq/l HCO_3^- (Schmidt-Thews 1990).

   Eventually the H^+ secretion serves for the reabsorption of the $NaHCO_3$. The passive reflux into the urine remains approximately constant, the active reabsorption according to the necessities of the acid base metabolism is increased or decreased.

2. Due to the H^+ secretion the pH of the urine decreases approximating the pK of $H_2PO_4/H_2PO_4^-$ buffering system, so that an increasing amount of H^+ ions is buffered.

Figure 1



3. H^+ ions by 50%-65% are eliminated through neutralization by NH_3. The NH_3 by 85 to 65% is formed by the tubular cells from amino acids, predominantly glutamine, 15-35% are of hepatic origin. The NH_4^+ secretion system in the situation of longer lasting acidosis is activated within 5-6 days and this adaptation does not occur before the second or third day of acidosis. This is an explanation for the phenomenon of fasting acidosis. After adaptation the NH_4^+ system is able to eliminate up to 500meq acid/day (normal daily production: 60-100meq, Schmidt-Thews, 1990). NH_4^+ in acidosis helps to save K^+ and Na^+: elimination of $H_2PO_4^-$ and strong acids like acetoacetate simultaneously depends on the excretion of kations (negative ions) to maintain electron neutrality. This causes depletion of Na^+ and later K^+. The electron neutrality also is achieved by NH_4^+ (see figure 1).

Intracellular pH
The blood pH and its constancy must not be confused with acid base values of specific organs, which physiologically differ quite significantly from pH of the blood.

According to Devlin (1997), the intracellular pH of erythrocytes is about 7.2, that of most other cells approximately 7.0, of muscle cells as low as 6.0.

Salivary Glands
The optimal pH of saliva is between 7.0 and 7.4. According to Hawkins (Goodheart, 1969, The ileocaecal valve syndrome) it should be as high as 7.8. According to Thomas (1998) the normal values for parotid saliva are between 5.1 to 6.25, for saliva of the submandibular gland 5.9 to 7.3. Factors which influence the pH of saliva: more acid pH values are measured with B-complex deficiency as well as increased phosphate content in food.

Decrease of salivary pH caused by amalgam fillings is speculative, perhaps a suppression of the carboanhydrase by zinc deficiency, which is caused by heavy metal toxicity, may cause lack of alkaline potential.

Stomach
The physiological stomach pH is 1.5 - 2.5. The HCO_3^- which is produced by the activity of the carboanhydrase during HCl production is carried to the basophilic organs, liver and pancreas; the HCl production therefore is a necessary step for bicarbonate production, which is needed in the respective organs.

Small Intestine
The contents of the duodenum are alkaline due to the biliary secretion (pH 7.55; Thomas 1998), the pancreas and the glands of the small intestine. In the ileum, the pH is approximately 6.5 due to the activity of the acidifying flora.

Large Intestine
Normal pH values of the contents of the large intestine are between 5.8 and 6.5. Higher values occur with putrification and cause decreased secretion of ammonia which can be of toxic relevance in suppressed liver function.

Urine
Important oscillations of urine pH are physiological. Changes of pH between 4.5 and 8.0 reflect the input of acids and bases with foods (Thomas, 1998). According to Sander (1985), the pH of the urine in the morning should be about 6.2 to 6.5 with normal acid load. Excessively high pH values can cause a tendency for infections and precipitation of calcium salts.

The urine has a relatively small buffering capacity, so that the actual acid load reflects quite directly in urine pH. Acid ash food, fermentation processes and fast bowel passage cause low urine pH, which in chronicity of these conditions can cause a general hyperacidity of the organism.

Joints
The pH in normal joints is 7.64 - 7.31, in osteoarthritis the pH is 7.54 - 7.25, rheumatic articular disease as low as 7.41 - 6.85 (Thomas 1998). The measurement of the pH in synovial liquid is possible.

Cerebral Spinal Fluid
The pH of the CSF is 7.35 (Thomas 1998).

The So-Called Chronic Latent Acidosis
(Sander, 1985 and Worlitschek, 1994)

According to the definition by the respective authors this is not a manifest acidosis which represents a pathological condition (metabolical acidosis in uraemia, diabetes, cardiac arrest).

Rather is the pH of the blood maintained within the normal range of 7.37 - 7.43 by the buffering capacity of the organism. In latent acidosis there is a decrease of the base reserve through increased utilization of the buffering capacity.

The metabolism produces predominantly acids: carbonic acid in the citric acid cycle, keto acids, lactic acid, uric acid etc. are intermediate or end products of metabolism. The production of $NaHCO_3$ by the carboanhydrase, which via the blood stream, is carried through the basophilic organs, liver and pancreas, occurs with production of equal amounts of HCl: $NaCl + H_2O + CO_2 <—> HCl + NaCO_3$.

Due to the increased acid load, which according to the respective authors, is caused by the wrong, that is acidifying diet, the buffering capacity of the blood decreases (the relation of bases and acids normally is like 20:1). In a later phase the excess acid, bound to protein structures, is supposed to deposit in connective tissue and bradytrophic tissues like cartilage.

The degree of dissociation of acids is pH dependent; a clinical example for acid deposit in the form of salts is the deposit of sodium urate crystals in gout. The more acid in the tissue, the less uric acid is disassociated and therefore precipitates.

Etiology

1. **Increased uptake of acid or acidifying foods** in the form of protein, refined carbohydrates, eating too fast and too late, with the consequences of maldigestion and the production of fermentation acids, as well as the excess consumption of coffee and alcohol. Interestingly, the salts of organic acids are an alkaline source: sodium lactate is a source of bicarbonate.

   Organic acids in fruits and vegetables therefore have no negative impact on acid base metabolism as they are metabolised to CO_2 and H_2O. The salts of organic acids are a physiological source of bicarbonate.

   $Na^+ + CH_3CHOHCOO^- + 3O_2 —> Na^+ + HCO_3^- + 2CO_2 + 2H_2O$.

2. **Decreased uptake of alkaline food.** Industrially processed foods like refined carbohydrates are low in minerals and therefore basic valencies. As a tendency agricultural products due to the depletion of minerals of the soil caused by overfarming and the over acidity of the soil, no longer have the same mineral content and therefore the same alkaline value as in former days (Worlitschek, 1994).

3. **Decreased acid elimination.** Sweating is a means of acid elimination via the skin. Sedentary lifestyle goes along with less sweating. The decreased circulation adds to a decreased mobilization of acids in the muscular, connective and cartilaginous tissues. A pathological condition with decreased acid elimination is renal retention, acidosis of different etiology (Thomas, 1998), which may cause manifest metabolic acidosis.

4. **Increased acid production in metabolism.** The human metabolism is principally acid producing. Hypersympathetic tone due to the related vasoconstriction and any other kind of circulatory disturbance causes an anaerobic tendency. Substrate deficiency and enzyme blockages of the mitochondria by toxic substances negatively influence the respiration chain and anaerobic metabolism is the consequence. Each anaerobic metabolic condition goes along with a shift of pH towards acidity. Anaerobic training via the production of lactate causes lactate acidosis. Repeated or habitual anaerobic training (oftentimes with the intent to work out stress) cause chronic latent acidosis which increases the stress of work overload by an important metabolic stress component. Immune deficiency, proneness to trauma, exhaustion are some of the clinical signs of this condition. In fasting metabolism there is production of acetoacetate and beta-hydroxy butyrate, which supply energy for many tissues including the brain, yet are of acid nature.

5. **Increased toxic load.** The liver plays a key role in the acid base metabolism. In the condition of increased toxic load of the liver by exotoxins (alcohol, solvents, medicaments) and endo-toxins (phenol derivates,

indikan through putrification, alcohol through fermentation in the intestine), there is an inhibition of gluconogenesis and, therefore, there is an accumulation of lactate. Through inhibition of the formation of ketone bodies, there is an accumulation of fatty acids (Devlin, 1997).

6. **Stress.** Increased sympathetic tone is associated with stress, no matter of what kind. Emotional stress causes latent acidosis. The clinical experience that many patients with increased emotional stress level, even with intensive deacidifying therapy, do not reach neutral pH cell metabolism, is an indicator for this phenomenon.

A truly causal therapy of acid base disturbances must correct the above mentioned metabolic and emotional factors and alimentary errors.

Symptoms of Latent Acidosis

According to Sander (1985) and Worlitschek (1994), unspecific health problems like chronic fatigue, concentration problems, chronic conjunctivitis, muscle and joint problems, pruritis and chronic cephalea can be the consequence of a latent but persistent acidosis.

According to Goodheart (1969) symptoms like shortness of breath, frequent sighing, insomnia, tight feeling of the throat, cold sweat, alternating with dry skin and hard dry stools, are associated with acidosis. An important symptom of persistent acidosis is osteoporosis: the calcium salts are relatively alkaline in comparison to plasma and in chronic acidosis are immobilized for the purposes of buffering. Improved bone healing in fractures could be demonstrated using long term application of $NaHCO_3$ (Devlin 1997).

Acidosis raises the membrane rest potential of nerve cells, thereby lowering neuronal activity; coma being an extreme condition. The causes are raised calcium levels and decreased sodium conduction. The resting tone of the central and peripheral nervous system in general is raised. Acidosis lowers the pain threshold through sensitization of pain receptors by H^+ ions.

Wiley (1987, 1989) describes the type which, under stress, shifts towards acidosis as follows: nervous, anxious, hyperkinetic type, workaholic, feels worse in the morning, especially after excess of alcohol and nicotine the night before and after too little sleep. Has pronounced hangover feelings. Tendency of hypoglycaemia, feels bad fasting and skipping meals. Is frequently hypoadrenergic. Supplementation of vitamin B5, zinc, adrenal supplements may help. Chemical toxins, (heavy metals, pesticides etc.) are prone to deposit in the CNS and have a tendency to contribute toward bad heeling of acute inflammations like bursitis, tendonitis. Always seems to have a cold, dripping nose or other flu like symptoms which are stress related.

The So-Called Chronic Latent Alkalosis

Etiology

Little can be found regarding this condition in German speaking literature. Chronic loss of acid and chloride (vomiting, also induced, for instance in bulimia, anorexia nervosa) can cause latent alkalosis. Increased alkaline supply in the form of bicarbonate or other alkaline salts and acids, lactate, citrate, can cause alkalosis. The 'milk alkali syndrome' with alkalosis and hypercalcaemia as well as hyperphosphataemia is caused by excessive intake of milk and well absorbable antacids. Hypopotassaemia is associated with alkalosis, although there is only a shift of H^+ ions in exchange with K^+ ions into the intracellular compartment. The acid base situation in the intracellular space is crucial for the metabolic processes.

Physicians who follow Enderlein's (1981) theories state that there may be alkalosis in the blood of chronically ill patients. The hemoglobin which has a high buffering capacity moves out of the erythrocytes into the plasma when the buffering capacity of the bicarbonate is insufficient. The microcytic anaemia with an anisocytosis in chronic diseases like tumours may be considered an evidence for this.

Iron in this situation is deposited in the mesenchyme due to the decreased bonding in hemoglobin, and the globin of the hemoglobin is visible in the blood as so-called synprotides. These are protein like corpuscles, which are the beginning of a so-called cyclogenic row (Enderlein, 1981). The buffering by globin seems to overshoot in the plasma, as pH compensation is unequally fast in different compartments (plasma, inter- and intra-cellular space), and therefore, the alkalosis occurs.

Symptoms of Chronic Latent Alkalosis
According to Goodheart (1969), alkalotic conditions are associated with slow pulse, itching and creeping sensations, joint stiffness, nocturnal muscle spasms, and high haematocrite.

The alkaline type according to Wiley (1989) moves slowly, is rather melancholic, sleeps many hours, suffers from general lassitude, is frequently choleric. Has a tendency to hyperglycaemia, hyperlipidaemia, tolerates alcoholic beverages well, has little hangover symptoms after nicotine and alcohol excess, tolerates fasting well and feels better doing so. Has a tendency to hyperadrenic stress reactions, a high need for vitamin C. High doses of adrenal extract can help. Chemical toxins are deposited in the fatty tissues. Frequently chronic arthritic conditions. Calcium deposits, hyperuricaemia. Is a type who is never sick, never has fevers or colds.

This type, in opposition to Wiley's opinion, rather than being characterized by an alkaline shift seems to have a tendency of disturbed intermediate metabolism (similar to the metabolic syndrome). Wiley's statement and theory seems to be based on erroneously interpreted measurements (see below).

Hyposympathetic conditions are associated with alkaline tendency. This characteristic of the autonomous nervous system certainly is associated with a certain type of individual.

Organ Acidity - Systemic Acidity
The relationship between hyperacidity of the stomach and latent acidosis according to Sander (1988) are as follows. Sander sees the function of the gastric parietal cells much less in the sense of hydrochloric acid production than in the sense of bicarbonate production for the basophilic organs (liver, pancreas). Under physiological conditions the hydrochloric acid is neutralized by bicarbonate in the duodenum following the equation $NaHCO_3 + HCl \longrightarrow NaCl + CO_2 + H_2O$.

In latent acidosis, in which the tissues of basophilic organs already suffer a lack of bases, whereas the blood still has its physiological reserve, at the end of the so-called alkaline flood during a meal there is an exhaustion of the neutralizing capacity of acid chymus by the pancreas. Bicarbonate is used for the buffering of acid tissue and is no longer available for the neutralization of gastric juice. The low pH in the duodenum is the adequate stimulus for the secretion of alkaline pancreas juice into the duodenum, which according to Sander, in the case of the exhaustion of the base reserve of the pancreas, causes further stimulation of the bicarbonate production in the gastric parietal cells. During this process hydrochloric acid is formed as a waste product, and the hyper secretion of the healthy stomach therefore is suggested to be a consequence of base depletion of basophilic organs. The application of protone (H^+) pump blockers and H2 blockers, therefore, seems to be a purely symptomatic measure which can suppress the pain caused by hyperacidity, yet the hyperacidity remains unchanged.

More physiological, therefore, seems to be the application of antacids. Here again the prescription of physiological absorbable alkaline substances like bicarbonate etc. would be the causal measure rather than the application of unabsorbable buffering substances which are used as antacids sold on the market. According to Sander, hyperacidity of the stomach therefore is supposed to be a symptom of general hyperacidity or in other words a more or less manifest acidosis. He hereby explains the occurrence of associated gastritis in metabolic and hormonal diseases like Basedow, Addison, diabetes, nephritis, and leucaemia.

This model is very seducing yet the theory which has been proposed in 1953 and is hardly compatible with the physiological regulation mechanisms which are known today. Hydrochloric acid secretion is stimulated by gastrine following chemical as well as vagal stimuli (Schmidt-Thews, 1990). According to the three receptor

model, histamine and acid acetylcholine stimulate the parietal cell. Both also act mediated by a histamine receptor. The stimulation of the pancreatic secretion also is driven by the vagal nerve and gastrine (gastric and cephalic phase). When the chymus enters the duodenum during intestinal phase it stimulates secretine and cholecystokinine (CCK) and the HCO_3^- secretion of the pancreas.

The adequate stimulus for the secretine liberation is a pH below 4.5, the one for CCK liberation is protein and fat as well as their metabolic products. Secretine, on the other hand, inhibits the secretion of the stomach and is liberated due to acid, fat, and hyperosmolar solution in the small intestine.

There is no established feedback mechanism which in the condition of alkaline depletion of the pancreas, causes increase hydrochloric acid production in the stomach for the purpose of bicarbonate production for the pancreas. On the other hand, there are conditions in gastroileostomia in which a continuously hyperalkaline content of the small intestine is responsible for a continuous stimulation of the G cells which may have remained in the antrum during operation with the related hyperproduction of acid (Schmidt-Thews, 1990).

Nevertheless, it is worth a therapeutic attempt to reduce the hyperacidity of the stomach by balancing the acid base metabolism, apart from treatment of acute conditions with H2 receptor and proton pump blockers. The criticism that application of bicarbonate causes compensatory hypersecretion of hydrochloric acid due to disturbance of the described inhibitory mechanism of acid pH in the antrum and duodenum, also applies for the generally used unabsorbable antacids (Forth, 1996). The danger of alkalosis caused by antacids, especially bicarbonate, rather has a positive aspect when the alkaline therapy is applied in a condition of chronic latent acidosis. Other mechanisms for the development of gastritis and gastric or duodenal ulcers (emotional causes) naturally have to be considered.

Diagnostic of Acid Based Disturbances

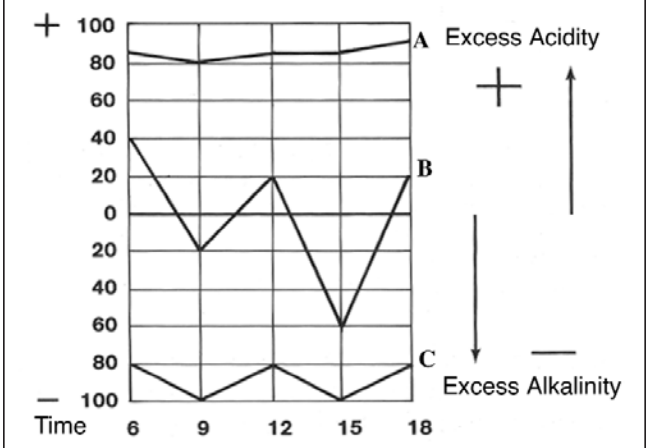
Urine and Saliva pH Measurement
The simplest screening method is the measurement of the saliva and urine with an indicator paper. As in healthy individuals, after 2-3 hours following each meal, due to the digestive processes, there is a so-called alkaline flood (Sander, 1985), for the screening of the urine pH the first urine in the morning is used, which reflects the acidity which is eliminated during the night. The morning urine pH should normally be 6.2 - 6.5. (Sanders) According to Thews (1990), it varies between 4.5 and 8 according to food intake.

The pH of saliva can be measured between meals to avoid the impact of the recently ingested food. The normal value of saliva should be between 7.0 - 7.4. Hawkins (1977) states that fresh saliva normally contains more CO_2 than ambient air and it is reasonable to take two measurements (one minute after sample collection and one hour later). According to the experience of Hawkins (1977) the patient has more alkaline reserves the more the pH of the saliva can increase by the elimination of CO_2.

This to the author seems little relevant in practice, as it is only of screening value.

Acid Base Titration According to Sander (1985)
Five, or according to the original method by Sander, eight urine samples are taken at specific hours of the day. Using a titration with 0.1n NaOH or 0.1n HCl respectively, the buffering capacity of the urine samples is measured in the acid and alkaline range. The morning urine is normally acid, as the acid metabolic waste products occurring during the night are secreted with the 6 o'clock urine. After the meal there normally is a base flood (9a.m and 3 p.m.). Taking the measurements a day line is drawn from which the acidity ratio is calculated. The shape of graph and the acidity ratio is taken for interpretation (see figure 2). In patients whose acid base metabolism is disturbed the compensation capacity of the organism is missing which is indicated by the flattening of the amplitude of the graph which indicates acid and base floods.

Figure 2



The Mean Acidity Ratio

A ratio of +10% and -10% can be considered normal. Acidity ratio between +10% and +30% indicate slight acidity, an acidity ratio between +30% and +50% indicate medium hyperacidity, and an acidity ratio of +70% and +100% a severe hyperacidity.

Slight alkalosis is indicated by a medium acidity ratio of -10% to -60%. The very rare severe alkalosis condition has a medium acidity ratio of -60% to -100%.

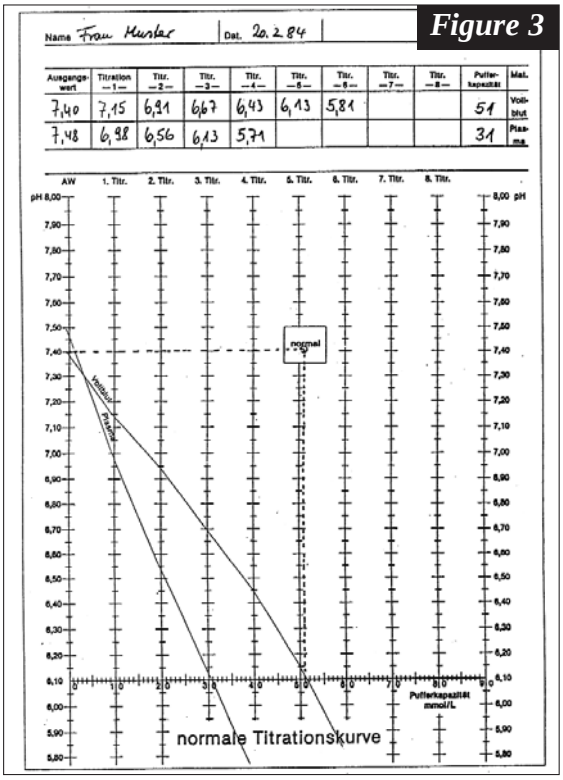
Figure 2: Acid base titration graph modified according to Sander

A (above): Rigid acidity, severe hyperacidity

B (middle): Normal graph, alternation of acid and alkaline floods

C (below): Rigid alkalosis, severe alkalosis for instance in excessive alkaline therapy

Figure 3



Blood Titration According to Jörgensen (1985)

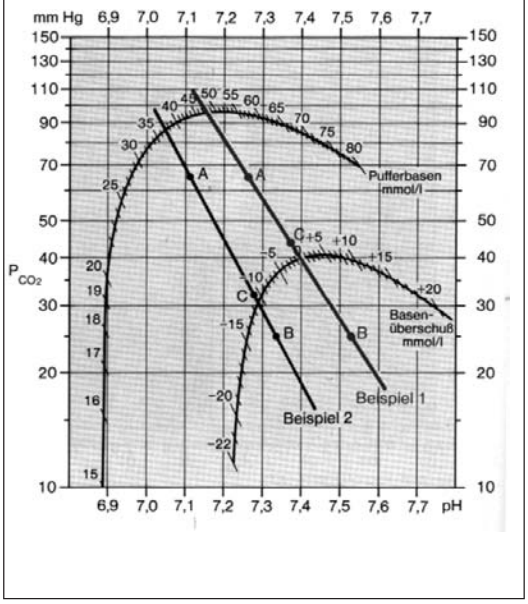
Using pH measurement electrodes, the pH value of venous full blood is measured and then a titration of one ml full blood with 0.1n HCl is performed. The pH values then are reported in a nomogram (Jörgensen, 1985). At the intersection of the thereby defined measurement graph with the abscissa of pH 6.1 (=pK of the blood) the crucial value for buffering capacity can be read (fig. 3). The buffering capacity of full blood should be 47 - 56 meq/l (see above). The same titration is performed with plasma whose original pH is considered without importance, as between the two measurements CO2 has been eliminated. The buffering capacity of plasma should be 27 - 36 meq/l. The difference between buffering capacity of full blood and the one of plasma should at least be 20 meq/l. High plasma and low full blood values indicate intra-cellular acidosis.

The position of the intersection of the values for pH and buffering capacity in another nomogram indicates the character of a disturbance, metabolic or respiratory. The two above-mentioned methods are not commonly used or accepted in established medicine.

Method According to Astrup

This is the standard method in intensive care and pneumonology. After calibration of a blood sample with two mixtures of gas of different pCO_2 the related pH is measured and thereby one gets two pH - pCO_2 pairs which then can be drawn into an acid base nomogram. The line connecting the two points in the nomogram cuts a curve for buffer base concentration and another one for base excess, from where the actual value of the blood sample can be achieved. The pH of the actual sample is reported on that very line and the pCO_2 can be read. (fig. 4). Today it is possible to measure the CO_2 partial pressure in blood samples directly with pCO_2 electrodes. Using the measured pCO_2 and pH values the base excess is derived from a ladder diagram or calculat-

Figure 4



ed electronically. It has to be taken into consideration that the only directly measured values are the pH and the pCO2 . The buffering base and the base excess, which are used to describe increase and decrease of non-volatile acids, are purely calculatory values.

A and B are points which are obtained by a calibration of gas with a known pCO_2 and consecutive pH measurement.

C: reading of an unknown arterial pCO2 on the connecting line after pH measurement.

Example 1: pCO_2 = 44mmHg,. pH = 7.37.
Base excess = 0 meq/l
Diagnosis: normal acid base status.

Example 2: pCO_2 = 32mmHg, pH = 7.28.
Base excess = 11 meq/l
Diagnosis: partially compensated non-respiratory acidosis.
(From Schmidt-Thews, 1990)

Measurement of Acid Metabolic Products

Lactate measurements in practice are possible with simple means, yet only furnish an insight in carbohydrate and potentially amino acid derived acid production. Other non-volatile acids remain unconsidered. The measurement of ketone bodies is not a routine method and only in fasting metabolism or in diabetes furnishes important indicators regarding acid production from acetyl-CoA in hyperlipolysis. Commercial indicator strips are sensitive only to acetone and acetoacetate in urine and diluted serum and serve for a semiquanittative measurement (Thomas, 1998).

Applied Kinesiology

Applied kinesiology is a clinical method of diagnosis and therapy, which combines elements of complementary medicine like acupuncture, nutritional therapy and manual medicine, and relates them to established medical concepts of neurology, biochemistry and physiology. The clinical basis of the method is the manual assessment of muscle function as a means to detect changes in central integrative state of alpha motor neurones.

Normal muscular function is characterized by a muscle which in the test appears strong, yet can be inhibited by specific techniques. This muscle reaction is called normoreaction. A muscle which does not react normal as described above is called disreactive. It appears weak in the test. Also a muscle which in the test appears strong, yet fails to inhibit by the respective measures, is disreactive. Muscular disreaction in the test indicates dysfunction of the muscle and/or the body structures and functions which are associated anatomically or physiologically according to experience.

A muscle can change its reaction in the test from disreaction towards normal reaction and vice versa by diagnostic provocations (challenge). The changes are caused by sensory provocation (challenges), whose effects are mediated by central and peripheral neurone mechanisms.

Diagnostic conclusions are drawn from the nature of the provocation and the change in reaction in the muscle test.

There is no specific muscle which is correlated with acid base disturbances as those types of imbalances are a sign of a systemic problem. Any disreactive muscle can be used.

However, it is recommended that the choice reflects the health disturbance being presented: disturbances of the immune system with suspicion of acid base dysbalance can be tested with a disreactive infraspinatus or adren-

al disturbance might be tested with the associated muscles (sartorius, gracilis, tibialis posterior). It is suggested that no muscle is used as an indicator which is associated with a specific acid or base producing organ such as pectoralis major clavicular (stomach), latisimuss dorsi (pancreas), popliteus (gall bladder), pectoralis major sternal (PMS, which is related to the liver) as this might reflect an examination of a corresponding organ (a hypo or hyperacid state of the stomach, insufficiency of the exocrine pancreas etc).

Diagnostic Provocation of Hyperacidic Condition

In the presence of an over acidity, the alkaline salt mixture, the more simple being sodium bicarbonate NaHCO3, an isopathic potentiation D6 or D12 of lactic acid, acetoacetate and beta-hydroxybutyrate could all produce a normoreactive sensory diagnostic provocation (challenge) to the chosen muscle. The alkaline challenge can be enhanced if the patient hyperventilates (this produces a respiratory alkalosis). An appropriate normoreactive muscle will become disreactive when provoked with betaine hydrochloride, lactate, acetoacetate and beta-hydroxybutyrate in molecular form. The acidic challenge is intensified if the patient holds his breath which promotes a respiratory acidosis (in neutral respiratory phase in order to prevent respiratory diagnostic provocation of the craniosacral system) (fig.5). Short breath holding time, according to Goodheart, can also be the effect of Vitamin B1 deficiency. In this instance a disreactive challenge occurs without the presence of an acid base disturbance and will be further differentiated.

Diagnostic Provocation of Hyperalkaline Condition

Oral application of betain HCl (trimethylglycine hydrochloride is an acid supplement used as a digestive aid in hypoacidity of the stomach) will promote a normoreactive challenge in an appropriate disreactive muscle if a hyperalkalosis is present. The challenge can be intensified if the patient holds his breath, (respiratory acidosis: apnea will lead to a normoreactive challenge). Correspondingly, an appropriate normoreactive muscle will become disreactive if the patient is provoked with an alkaline mixture. The alkaline challenge is intensified if the patient hyperventilates (a respiratory alkalosis). Hyperventilation produces disreaction. (fig. 6)

Figure 5 **Sensory provocation for hyperacidic conditions.**

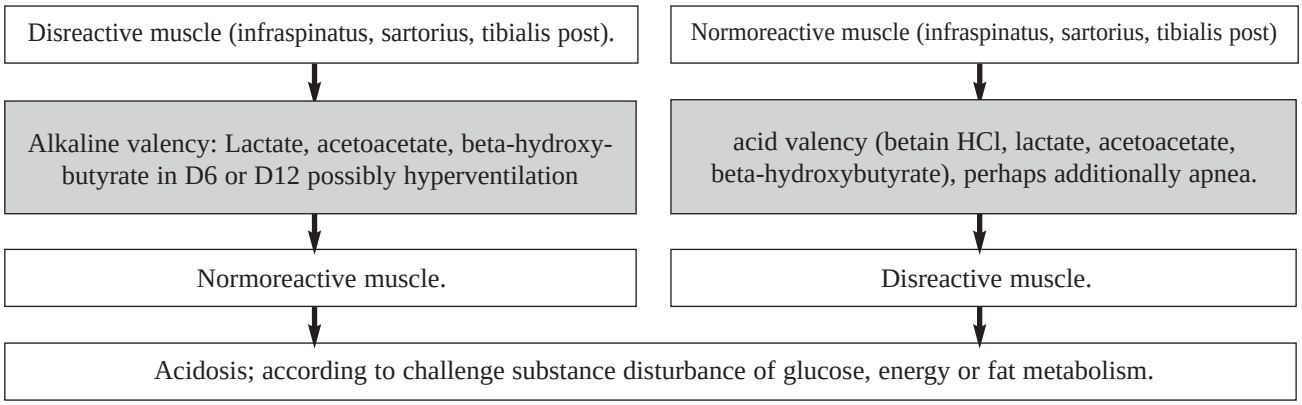
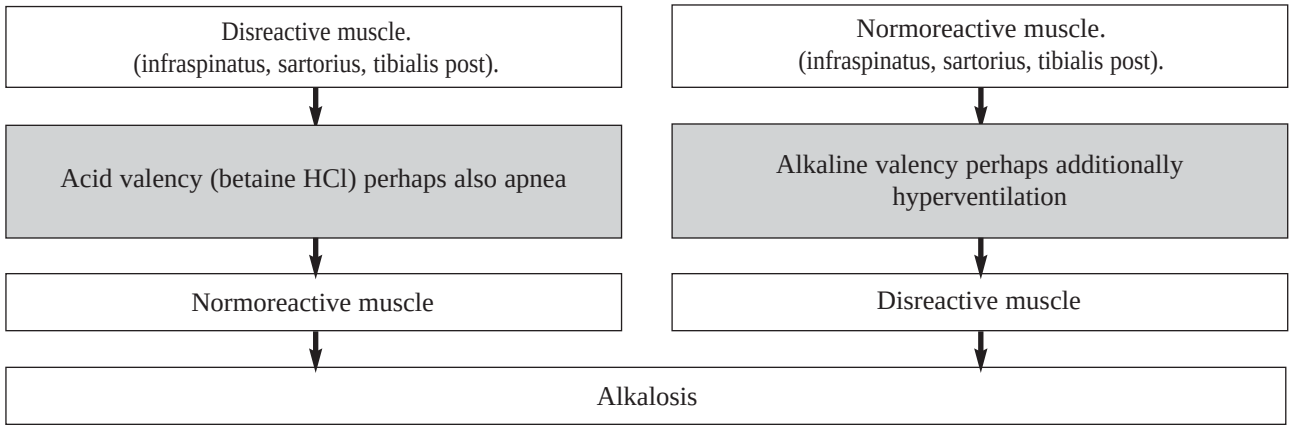


Figure 6 **Sensory provocation of hyperalkalotic condition.**



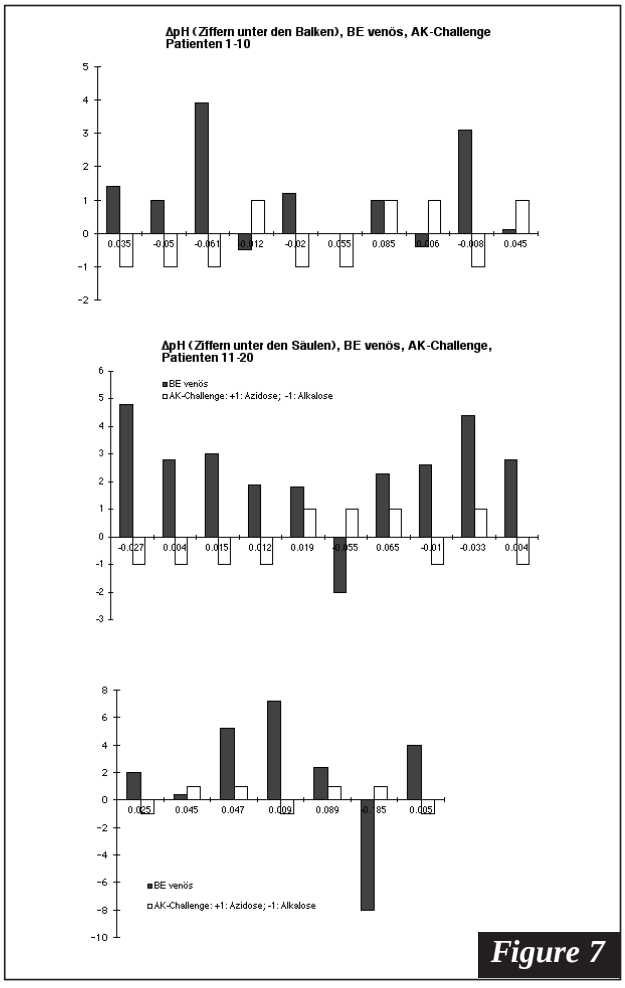


Figure 7

Methodology of the Study

Group 1:

The AK challenge of acid base valency and a blood gas analysis according to Astrup were performed on 27 patients. In order to limit the influence of various diets, these patients were placed on a standardized neutral acid base diet during the 12 hours before examination. Patients presented with the following disorders.

Pain in the musculosceletal system, rheumatic conditions, cephalea (headache), eczemas, bronchial asthma and psychosomatic disturbances. The AK challenge included provocation with betain hydrochloride as an acid substance and alkaline powder (sodium phosphate 10.0, potassium bicarbonate 10.0, sodium bicarbonate 80.0, calcium bicarbonate 100.0 as in alkaline mixture). A comparison was made of pH, base excess from blood gas analysis and the AK challenge. In addition, an arterial blood gas analysis was performed on 19 of the patients in this group. The study was ended with this relatively small number of parallel examination (see group 4 below) which revealed that the parameter of the entire valuation needed to be adjusted.

Group 2:

Acid titration of the urine according to Sander was performed on a total of 20 patients as well as an acid base challenge using applied kinesiology as described above. The mean acidity ratio and the AK challenge outcome were compared.

Group 3:

Acid titration of the urine according to Sander, an acid base challenge as per applied kinesiology and a blood gas analysis were performed on a total of 9 patients. A comparison was made of the results of mean acidity ratio, base excess of the blood gas analysis and the AK challenge outcome.

Group 4:

Venous pH value, base access, standard bicarbonate according to the Astrup method as well as measurement of lactate were performed on 190 patients at the pain therapy section of the department for anesthesiology and operative intensive care medicine of the Justus-Liebig-University in Giessen. Patients were divided into eight subgroups according to the following symptom pictures.

4.0 Healthy associates of the department were used as control group (u = 23).
4.1 Locomotor system pain (n=71)
4.2 Headache (n=31)
4.3 Facial pain (n=20)
4.4 Fibromyalgia (n=8)
4.5 Circulatory disturbances (n=9)
4.6 Neuralgia (n=26)
4.7 Sympathetically mediated pain syndrome (n=16)
4.8 Various pain conditions (i.e. tumour) (n=9)

Results

Group 1:

24 out of 27 of the venous pH values (89%) were found to be in a normal range (n= 7.32 - 7.43). Two were found to be below normal value (7.19 and 7.314 respectively), 2 were found to be above the normal value (7.44 and 7.46). Values of the base excess varied between +7.2 and -8meq/l (MW = 1.79 mmol/l) (fig. 7). The correlation between ΔpH (pH of blood sample - pH of normal value) and the base excess was 63% (17/27) (fig. 7).

Against expectations, in nearly all cases in which the artial and venous base excess was measured, the arterial base excess was found to be lower than the venous (18 out of 19, or 95%) mean value= -2.54 mmol/l, min -0.1; max -9.8 mmol/l). The values were identical in only 1 case.

Group 2:
A positive acidity ratio was found in all 20 cohorts on the second group: found between 17% and + 76%. However, 10 of the 20 patients responded to a positive AK challenge as per alkalosis revealing a truly arbitrary correspondence between the AK challenge and the acid titration.

Group 3:
8 out of 9 patients (89%) demonstrated a positive base excess in venous blood. 9 out of 9 patients in this group showed a positive acidity ratio. 5 out of 9 patients (56%) had a positive AK challenge showing alkalosis. Only 2 out of 9 patients (22%) demonstrated a correlation between base excess and acid titration, 6 out 9 patients (66%) demonstrated agreement between the base excess and the AK challenge.

Group 4: (4.0 - 4.8)
The results of the measured base excess, the breakdown of positive and negative base excess, as well as total lactate values (minimum, maximum, median value) can be found on tables 1, 2 and 3. With reference to the value of base excess it is worth noting that 17 out of 23 cases within the control group (74%) demonstrated a positive base excess in venous blood. The entire patient group held on average a lower base excess than the control group. However, with the exception of 5 fibromyalgic patients, most still demonstrated a positive base excess in venous blood.

The lactate values for the control group correlated well with the reference range of 0.4 - 0.8 mmol/l in laboratory medicine (Thomas, 1991). None of the control cohorts reached the threshold value for lactate (2-4 meq/l) for anaerobic metabolism as described in the literature (Marees 1989, Schmidt-Threws 1990). In the individual disease groups a large percent of the cohorts had a value higher than the average value in the control group. The highest values (i.e. largest difference to the median value of the control group) were found in the groups of patients with circulatory disturbances (4.5) and with sympathetic mediated pain syndrome. (4.7 respectively).

Table 1: Group 4: Base-Excess: median value (m), minimum (min.) and maximum (max.), pH outside normal (7,32 bis 7,43): pHon

	BE (m)	BE(min)	BE(max)	pHon
Control group (4.0)	1,047	-2,7	3,3	0
Total patient groups (4.1.-4.8):	0,393	-6,3	7,8.	27

Tab. 2: Group 4: Base-excess in venous blood: Patient groups 4.1 to 4.8) and control group (4.0)

	total	BEpositive. (%)	BE negative (%)	median
control group (4.0)	23	17 (74%)	6 (26%)	1,05
locomotor system pain (4.1)	56	41 (73%)	13 (23%)	0,81
headache (4.2)	22	9 (40%)	11 (50%)	0,10
facial pain (4.3)	16	7 (44%)	9 (56%)	0,17
fibromyalgia (4.4)	5	1 (20%)	4 (80%)	-0,56
circulatory disorders (4.5)	7	6 (86%)	1 (14%)	0,46
neuralgia (4.6)	22	14 (64%)	8 (36%)	0,12
sympathetically med. pain syndromes (4.7)	13	7 (54%)	6 (46%)	0,24
other pain conditions (4.8)	7	4 (57%)	3 (43%)	0,46

Table 3: Group 4: Lactate: Median value (m), minimum (min.) and maximum (max.), Δ lactate (lactate patients – lactate control group), ratio of patients with lactate value greater than median lactate value of control group (>mc).

	m	min	max	Δlactate	>mc (%)
control group (4.0)	0,659	0,09	1,26		
total patient groups (4.1-4.8)	1,456	0,09	4,42	0,79	88%

Discussion

In spite of the small cohort number for group 1 through 3, the following statements are still possible:

1. The established assumption that pH values have only limited value in a diagnosis of a latent acidosis is confirmed. In group 1, 3 out of 27 (11%), in group 4, only 27 out of 190 (14%), showed a deviation from normal pH (table 1) whereas 24 out of 27 (89%) in group 1 as well as 168 out of 190 (98%) in group 4, had no normal base excess (table 2). This and the great mean deviation of the pH values without correlation to base excess is a result, which is important for the publications of Wiley (1987) who bases his statements regarding acid base shifts exclusively on pH measurements in venous blood. These conclusions drawn from pH measurement are irrelevant for latent (that is compensated) disturbances of the acid base metabolism. According to the statements of Wiley (1987) pH shifts of as little as 0.01 is of importance, which corresponds to the methodical errors of pH measurement machines.

2. Acid titration of the urine apparently practically always results in a positive acidity ratio according to Sander. If the interpretation given by Sander (1985) were correct all these patients were hyperacidic. This correlates neither with the base excess in blood gas analysis (group 2), nor with the AK challenge (group 3). Experience shows that a considerable number of patients react unfavourably to the ingestion of alkaline substances (see below).

   Many patients, despite elementary changes in nutrition and base supplementation, show no normalization of acid titration values, if the alkaline supplementation is followed by a control measurement without this supplementation. Acid titration according to Sander, therefore, seems to be a little reliable instrument for the measurement of acid base situations. As a principle the organism produces acid in excess which has to be eliminated with the urine. Therefore it cannot be expected that along the course of the day a neutral urine is eliminated as an average (acid base ratio = 0 according to Sander); also refer to the discussion on the acid elimination of the kidney. The prescription of bicarbonate and other alkaninizing salts in cases of positive acidity ratio as a sole criteria in this light seems to be totally inappropriate. The bicarbonate buffer (as well as the other buffering systems) in most cases with positive acidity ratio apparently have sufficient capacity apparent through positive to normal buffering bases, base excess and AK challenge.

3. 21 out of 27 patients (78%) (mean value 1.79meq/l) in group 1 had a positive base excess in venous blood. Also in the measurements of group 4, in 89 out of 148 patients (62% - mean value +0.39meq/l) a positive base excess in venous blood was measured. This means also in this light that the buffering systems in the great majority of patients are sufficient. A prescription of bicarbonate or mixtures of alkaline substances seems to be unreasonable in the majority of cases. After the definition of latent, that is compensated acid base disturbance, in all patients with positive BE, that is the majority of patients, there should be a latent alkalosis.

4. The fact that the arterial base excess as a general tendency was always higher than the venous one, needs explanation. A higher BE is significant for increased capacity of buffering systems. As venous blood should contain a higher quantity of nonvolatile acids from metabolic processes than arterial blood, a lower BE should be expected. The higher BE, which was found in contrast to this may be due to the higher buffering capacity of reduced hemoglobin than oxygenated hemoglobin (0.3meq/10g Hb - that is approximately 0.42meq/l).

   On the other hand this conflicts with the established opinion that the measurement of total buffer bases and base excess is independent of the partial pressures of the blood gases O2 and CO2.

5. The AK challenge showed a merely random correlation with acid titration (50%) the correlation with base excess is slightly better (66%). AK challenge with the chosen mixture of alkaline salts could be too little specific for the diagnostic provocation of buffer systems. To be more specific, apart from bicarbonate, phosphate and proteinate as well as hemoglobin should be used for sensory provocation purposes, which is not practicle. As an alternative a mixture of NaHCO3 and Na2HPO4 in a ratio of 3:1 could be used.

6. None of the control cohorts in group 4.0 reached the limit for normal lactate (2-4meq/l) in aerobic metabolism as documented in the literature (Marees 1989, Schmidt-Thews, 1990). Nevertheless, 6 out of 23 individuals (23%) showed a negative base excess. The base excess apparently can be negative despite normal lactate values. This is not surprising as lactate only is a small part of nonvolatile acids. On the other hand the question should be raised why 74% of healthy individuals showed a positive base excess.

7. In group 4.1 to 4.8, it was remarkable that the great majority of health disorders showed a lactate above the one of the control group. This may be an indication that in pain conditions, due to the increased sympathetic tone with the resulting vasoconstriction, there is a tendency towards anaerobic metabolism. As a rule nociception increases reflexively the alpha motor neurone activity and the activity of the presynaptic sympathetic ganglion, which primarily cause an arterial constriction and capillary dilation, which at a later stage develops into a capillary constriction. Blood lactate levels are an indicator for tissue oxygenation (Vincent 1998, Marees 1989, Schmidt-Thews 1990). Tissue hypoxia according to this literature is indicated by lactate values above 2meq/l. The measurements of this study seems to indicate a tissue hypoperfusion already with lactate values below 2meq/l.

   The highest lactate values were found in a group of patients with circulatory disturbances and sympathetically mediated pain syndromes. Lactate acidosis does not correlate with decreased buffering capacity as a rule. The buffering system compensates sufficiently, because in 84.7% of the patients there were normal pH values (n=7.32 - 7.43). 11.6% had low pH, 3.7% were above normal range. 72 (37.9%) of the patients had a negative base excess, 113 (59.5%) a positive, 5 (2.6%) a BE of 0. This also demonstrates that there is no correlation between BE and lactate acidosis or tissue oxygenation. The lactate value represents a small section of the overall load with nonvolatile acids, yet seems to be a significant parameter in all conditions with increased sympathetic tones which correlates with an aerobic metabolic tendency.

Discussion of the Different Acid Base Measurement Methods

Saliva pH Measurement
Measurement of saliva pH accesses a body's secretion or excretion respectively, yet does not furnish any information about the actual metabolic situation intra- or extra-cellulary. This measurement therefore is controversial, even as a screening method, because it is potentially misleading. The secretion of saliva is controlled by the Nc. salivatorius sup. and by Nc.salivatorius inf. (parasympathetic). This control is not primarily pH dependent. The base production is also dependent on the carboanhydrase which requires zinc as a co-factor.

Urine pH Measurement
The measurement of pH in the morning urine, in which the acid valencies, which accumulated during the night are eliminated, can supply a general information about the acid production. This can be useful in fasting situations. On the other hand no conclusions can be drawn regarding the acid deposit in tissues.

Acid Titration According to Sander
The advantage of this method seems to be that the result of metabolic stress, that is the acid which is produced during metabolic processes and eliminated thereafter, can be assessed. The method does not give any insight into potential compartment differences of the acid base state (blood - plasma - tissue, different organs). The disadvantage certainly is the indirect interpretation of the tissue situation based on the acid elimination in the urine. The acid elimination enzymatically is adapted to metabolic needs. A positive acidity ratio means that an increased amount of acid needs to be eliminated. It does not mean that there is excess acid in the organism which cannot be buffered. The proposed normal value of 0% acidity ratio according to the this study seems to be questionable. A negative acidity ratio, which means metabolic alkalosis can only occur in situations like chronic vomiting (see above).

Acid titration according to Sander on the other hand seems to be valuable for the documentation of the need for elimination of acid valencies at a given moment. The acid elimination is enzymatically increased when

there is increased acid production, which occurs during metabolic stress (fasting metabolism, diabetic ketoacidosis and also in increased alimentary acid intake) but also after emotional stress and during painful conditions. Lactate measurements in pain patients in this study give an indication for this. Acid titration therefore is useful to control the success or failure of a causal therapy like the ones described below.

Measurement According to Astrup
A disadvantage which can be overcome is the little availability of the method which is only used by pneumologists and in intensive care units. Unlike the usual practice of blood gas analysis, where arterial blood is measured, in the diagnostic of metabolic stresses which cause acid base imbalances the "finish line" of metabolism seems to be important, that is venous blood should be measured. The fact that BE, a calculated value, in the same patient in arterial blood is consistently more negative than in venous blood seems to be related to the different buffering capacity of oxygenated and deoxygenated blood.

The BE apparently is influenced by partial pressures of gases, somewhat in contradiction to established opinion. It seems like the BE is not a relevant parameter for the diagnosis of compensated acid base disturbances because in the majority of patients with increased acid elimination (urine titration) a positive base excess was found. A negative base excess on the other hand can be used as an indicator, that in correlation with a specific pattern of symptoms alkaline substances should be prescribed. The same applies for positive base excess. With a respective symptomatology (for instance periarthritis with calcium deposit) acid calcium can be prescribed.

Blood Titration According to Jörgensen
The difference of buffering capacities depends upon haematocrite and hemoglobin concentration. Each measurement therefore needs to be corrected by the deviation of the actual Hb of the blood sample from normal. The acid titration according to Jörgensen suffers from the variable of volatile acid which can not be controlled easily, yet contributes 50% of the total acidity. The outcome of an acidosis therefore is programmed by this imminent error. H_2CO_3 contributes 50% of the total acidity of the blood, the denominator of the Van-Slyke equation therefore differs between 1.2 - 2.meq/l, which can cause a pH value differing between 7.4 - 7.7 with the respective influence the calculated buffering capacity. The total acidity, which is crucial for the diagnostic picture, therefore, is variable by the factor 2. The measurement method according to Jörgensen should only be applied by an experienced laboratory which does numerous measurements. Jörgensen suggests always "the same rhythm of measurement" (Jörgensen, 1985), which supposedly maintains the influence of the factor CO_2 very low! Comparable yet free of these uncertainties is the laboratory method of measuring the base deviation in extracellular fluid (see p. 27). Here with constant pCO_2 of 40mmHg a titration towards pH 7.4 is done.

The measurement of bicarbonate and total CO_2 offers more reliable values too. After addition of a strong acid, the preformed and the expelled CO_2 is measured with a pCO_2 electrode. The bicarbonate of the pCO_2 system is measured with an enzymatic test after the addition of alkaline substance (Thomas 1998).

The proponents of the method have a tendency to overestimate acidotic states, not only due to the methodological problems of the blood titration, according to Jörgensen.

According to Worlitschek (personal communication), in alkalotic states there is a "constant counterregulation," which is "paradoxically pathological." The mechanism of the counterregulation seems to be unknown.

It is also questionable if the assessment of the intra-cellular situation of blood cells can be extrapolated to the intracellular pH of organs and connective tissues, which seem not to have the same buffering systems. The normal pH of bone cells is 6!

Lactate measurement in blood
Lactate measurement is a good assessment for tissue hypoxia and therefore the source of acidosis. There are simple bedside measurement devices. A further possibility to measure tissue hypoxia is the pulse oxymetry by which the oxygen saturation of the blood is measured with a small device. The optimum value for oxygen saturation is 98%.

Diagnosis with Applied Kinesiology
Applied kinesiology, a system which uses manual muscle testing as measurement device, naturally is subject to subjective errors, which can be minimized based upon the experience and education of the examiner. The cooperation and effort of the patient also reflect on the neutral outcome. With applied kinesiology, there is the possibility to assess a great number of parameters in a simple and cheap way, which afterwards can be reassessed and controlled with lab measurement methods.

The more parameters afterwards are assembled to draw an overall conclusion, the more exact the assessment is (provocation with bicarbonate, betain HCl, lactate, acetoacetate, beta-hydroxybutyrate as well as orthomolecular substances which are closely connected to acid base disturbances). The method also offers the advantage that a causal therapy can be designed. Functional assessment with applied kinesiology therefore seems the method of choice.

Therapy of Acid Base Disturbances

1. Diet modifications.
The most important factor in the therapy of acid base disturbances is the adaptation of alimentary habits according to the specific condition and type of the patient. There is extensive literature in natural health care which more or less states which foods acidify and which alkalinize. All these lists are based on the acid base characteristics of the food ashes which are the product of drying and burning of those foods. In analogy it is thought that the respective foods in the human organism cause the same acid or alkaline metabolic products. This is only true in very rough outlines. The works of Wiley (1987, 1989) are the only ones which consider the effect of foods in the organism of patients based on acid base measurements after food exposure in the sense of a mono diet, for instance exclusively wheat after a fasting period. Because of the difficult interpretation of the blood pH for acid base analysis this information is not valid. The commonly used attributes and suggestions regarding the acid base value of foods can be summarized very simply: foods high in protein including milk products are acidifying, vegetables including potatoes and fruits are alkalinizing, carbohydrate rich foods (bakery, noodles etc.) are less acidifying to the extent that they are rich in minerals. Fat and milk are neutral, whereas milk products due to higher protein contents are acidifying.

Intolerances of foods have to be considered! Foods to which the patient is intolerant cause metabolic stress and acid base disturbances mostly in the sense of acidosis.

Favorable dietary habits comprise careful chewing and insalivation. Individuals should not eat too late at night and not include too much roughage because the fermentation processes build acids. The ingestion of whole grain products and specific vegetables especially in the raw form should be limited. A significant indication for this is flatulence.

2. Prescription of alkaline salts.
The simplest way for a short-term alkaline therapy is sodium bicarbonate. This is only a therapy to balance already existing disturbances, not a causal therapy. Urine pH must be maintained in an optimum range in the case of hyperuricaemia, urate stones, cystine stones, oxalate stones, calcium phosphate stones, in order to guarantee optimum solubility of these salts: above 6.5 - 6.8 in urate stones and cystine stones, using potassium bicarbonat, in calcium stones between 5.5 - 6.2.

In oxalate stones, calcium citrate or lactate can be used to bind calcium in the intestinal tract to inhibit formation of oxalate stones (Harrison, 1998).

Otherwise the application of bicarbonate in the case of acid urine seems to be reasonable only for the treatment of acute symptoms (like in fasting, with the respective complications; headaches and overall aches and pains) when the acid elimination has not yet been adapted to the present demands. In acute and massive hyperacidity, the i.v. application of 250ml $NaHCO_3$ 4.2% (infused over at least 30 minutes) can be useful and achieves a fast reduction of symptoms. Herewith the base excess is increased by 6 units in an individual of 70 kilograms.

Other mixtures of alkaline substances are the following:
Alkaline powder according to Sander:
Sodium phos. 10,0
Potassium bicarb. 10,0
Calcium carb. 100,0
Sodium bicarb. ad 200

Alkaline powder 2 according to Rauch:
Sodium phos. 10,0
Potassium bicarb. 10,0
Sodium bicarb. 80,0
Calcium carb. 100,0

Alkaline powder 3 according to Rauch:
Sodium bicarb. 85,0
Calcium carb. 60,0
Potassium bicarb. 10,0
Potassium citr. 15,0
Magn. citr. 20,0
Sodium phos. 10,0

The dosage according to the degree of acidity is one teaspoon in one quarter litre of warm water before bedtime. Otherwise, up to three times daily between meals, an interval of 1.5 hours to the meal should be respected in order to avoid neutralization of gastric acid.

Another possibility is the application of alkaline enemas; one tablespoon $NaHCO_3$ in one half to three quarters of litre of water is used as a colonic irrigation in inflammatory diseases of the colon and rectum. The optimum pH of the colon of 5.8 - 6.5 should be respected.

Alkaline baths: This can be used for pruritis, the water should have a pH of 8.5.

Alkaline ointment for inflammatory skin and mucous disturbances with pruritus (Rauch, 1994).
Sodium bicarb. 3,0
Oleum rosac. 0,2 g
Aqua dest. 22,0
Eucerin anhydr. ad 50,0

The author defends the opinion that therapy with alkaline salts is an acute therapy and the metabolic situation has to be changed using an adapted food intake and supplementation. Generally speaking, the principle of reducing metabolic stress has to be adopted. The organism is able to reabsorb bicarbonate, the principle mechanism of acid elimination is via the phosphate buffering system and ammonia. When there is a bicarbonate overflow in the urine and relatively higher pH the phosphate buffering system with its low pK is less efficient. Clinical experience shows that in many cases where alkaline supplementation has been applied over months, after the therapy the acid elimination of the urine, which otherwise is used as a measure for the acidity has not changed.

3. Enhancement of H^+ ion elimination through facilitation of ammonia synthesis.
As the kidney regulates the H^+ elimination by titration of phosphate and ammonia in the tubular filtrate the H^+ elimination is essential for the reabsorption of HCO_3^-. In metabolic acidosis within about 5 days the ammonia forming enzymes of the kidney are induced: glutaminase, glutamate-dehydrogenase, phosphoenolpyruvate-carboxykinase, the mitochondrial glutamine transporters. The necessary glutamine is supplied by means of reduced urea synthesis of the liver. The mechanism is convertible: in metabolic alkalosis there is increased formation of urea from glutamine in the liver, the renal enzymes dispose of less substrate and lose some of their activity (Devlin, 1997, Thomas, 1998).

This mechanism is suggested to be used therapeutically, supplying a surplus of glutamine to override the regulating mechanism of glutamine. Within a few days the H+ elimination hereby should be increased, which represents a more physiological therapy than of alkaline salts, as these inhibit the induction of ammonia forming enzymes. In the context of applied kinesiology, l-glutamine should be tested in the sense of a normoreactive challenge.

4. Application of Acid Supplements.
This is a thought which in German speaking natural health care is unthinkable in accordance with the belief that all evil lies in acidity. Goodheart already in the 60's mentioned that systemic alkalosis could cause calcium deposits and that this situation could be treated with acid calcium. A possible formula is the following:
Betain-HCl 100,0mg
NH_4Cl 100,0mg
$CaCl_2$ 100,0mg
(incapsulated 300mg)

5. Metabolic therapy in acid disturbances.
Basically, one of Wiley's concepts seems to be correct; acid base disturbances can be linked to specific metabolic types and lifestyles (alimentation) which may be incompatible with these types. These types may be predetermined by a genetic component (for instance, heredity hypercholesterolaemia, hyperuricaemia etc). A truly causal acid base therapy should modify the intermediate metabolism of these types. Even if the conclusions drawn by Wiley (1989) do not seem to be correct, his classification into hypermetabolic (with high tendency of hypoglycaemia and functional hypoadrenia) on one hand, and hypometabolic types, which resemble the metabolic syndrome type (with tendency to hypoglycaemia with insulin resistance, hypercholesterolaemia and hyperuricaemia) offers a therapeutic concept.

A similar classification would be disturbances of glucose metabolism, fat metabolism, and the protein metabolism.

Furthermore, conditions with decreased detoxification capacity as well as deficits in aerobic energy production are important for acid base metabolism.

All the above mentioned disturbances require an alimentary counseling (along with the elimination of food intolerances). Additionally, orthomolecular therapy that has been designed specifically for the patient. Laboratory tests for the relevant metabolic parameters are necessary and useful. In the following paragraph, the substances which should be tested and prescribed in the respective conditions are outlined.

Dysinsulinism
These patients have increased insulin levels through carbohydrate hyperalimentation with peripheral insulin resistance. The condition can develop into a non-insulin dependent diabetes type 2 with increased blood glucose levels and a tendency to hyperlipidaemia. Obesity always coincides with a certain degree of insulin resistance. No ketoacidosis develops in this condition, as the lipid cells remain to be sensitive to the inhibitory effect of insulin on lipolysis.

A provocation with insulin (Schmitt, *The Uplink*, No. 11, 1998), which can be done orally, or using an ampoule on the skin under a 3000 gauss magnet (*proposed by Lebowitz, per personal communication.*) causes muscle disreaction in these patients, which indicates a prediabetic tendency with increased insulin levels. The test results correlate well with a genetic predisposition. Along with caloric reduction and carbohydrate reduction the following supplements can be useful:
Chromium (200-800 mg/d)
Copper (4-6 mg/d)
Magnesium (200-500 mg/d)
Manganese (10-20 mg/d)
Vit. A (25000-50000 IU/d)

Vit. B6 (P-5-P) (100 bis 300 mg/d)
Vit. E (400-800 IU/d)
Omega-6 EFA, (ex. evening primrose oil 3x2g/d)
Omega-3 EFA, (EPA, 3x2g/d)
Betain-HCl (2-3x500 mg/meal)

Insulin Dependent Diabetes Type 1.
There is always a tendency towards ketoacidosis caused by increased lipolysis and an increased beta-oxidation in the liver. A provocation with beta-hydroxybutyrate D12 and acetoacetate D12 produces a normoreactive muscle test outcome. (Potentized substances as above act as anti-substance, according to the principle of isopathic; coffee D12 is an "antidote" to too much coffee intake).
The following supplements should be tested using a disreactive muscle and an oral challenge:
Chromium (200-800 mg/d)
Copper (4-6 mg/d)
Magnesium (200-500 mg/d)
Manganese (10-20 mg/d)
Zinc (30-50 mg/d)
Vit. A (25000-50000 IU/d)
Vit. B6 (P-5-P) (100 bis 300 mg/d)
Vit. E (400-800 IU/d)
Niacin (30-50 mg/d)
Vit. C (1-3g/d)
Bioflavinoids: ex: acerola plus (PE)
Omega-6 EFA, (ex. evening primrose oil 3x2g/d)
Omega-3 EFA, (EPA, 3x2g/d)

Hyperuricaemia
The best indicator is the laboratory test. In alkaline conditions uric acid is better soluble than in acid conditions (Harrison, 1998). The measure of alkalinizing the urine is based upon this fact and also the alkaline therapy in hyperuricaemia. In hyperacidosis, that is in circumstances of increased organic acids (diabetic ketoacidosis, hunger ketoacidosis, lactate acidosis), there is a competition of urate with the other acids for renal elimination. Besides ideopathic hyperuricemia, disease related (enzyme defects, neoplastic blood diseases) and diet related overproduction of uric acid are of importance. In hyperuricaemia the following supplements (along with purine reduced diet, that is reduction of meat, shellfish, beans, spinach, peas and the avoidance of fructose, sucrose, coffee and alcohol) can be tested. Alkaline salts (see above) vitamin C (3 – 6g a day) mobilize urate from the tissues. The dosage has to be increased slowly starting with 500mg per day, as a renal elimination of Vitamin C must be avoided. This would cause urine acidification and a subsequent inhibition of urate elimination with the urine. Buffered Vitamin C therefore should be considered. Folic acid (25 – 50mg per day) inhibits the activity of xanthine-oxidase, which catalyses the next to last step in uric acid synthesis.

Hypercholesterolaemia
The best diagnostic method is laboratory measurement. The diet should be protein rich, fat reduced and carbohydrate reduced, refined flour and sugar should be avoided. The following co-factors of the cholesterol metabolism should be tested.
Niacin 3x100 bis 3x500 mg.
Niacitol (inositol hexaniacinat) is less effective with regard to lowering cholesterol levels.
Vit. C (2-3 g/d)
Magnesium (300 -500 mg/d)
Chromium (400-800 mg/d)
Pantothenic acid (1000 mg/d)

Vit. E (400-800 IU/d)
EPA (5-10 g/d)
l-Carnitine (700 mg/d)
The last two can cause paradoxical reactions. Thyroid function has to be observed.

Functional Hypoadrenia (Except Addison's Disease)
A hyporeactive sartorious, gracilis, or tibialis posterior frequently are associated. This disturbance frequently is accompanied by chronic hypoglycaemia.

The diet should contain sufficient complex carbohydrates, adequate salt, preferably in the form of vegetable soup, zucchini, carrots, celery etc. Absolute restriction of sugar and refined carbohydrates. The following supplements can be tested, which cause a normoreactive challenge outcome.
Vit C (1 bis 3 g/d)
Pantothenic acid (500-1000 mg/d)
Zinc (60-90 mg/d)
Vit. B6 (P5P, 3x50 mg/d)
Vit. B3 (3x100 mg/d)
Radix Ginseng
Radix glycyrrhizae
Nebennierenextrakt
aerobic excercise.

Hypothyroidism
The best muscle to test is the teres minor which is associated to the thyroid. In hypo-thyroidism, this muscle frequently is hyporeactive and the following factors may cause normoreactive muscle test outcome.
Iodine (200 bis 500 mcg/d)
l-Tyrosine (500 mg/d)
Selenium (200-400 mg/d)
Zinc (30 bis 60 mg/d)
Copper (4-6 mg/d)
EFA (EPA, GLA, 3x1-3x2g/d)
Coenzyme Q10 (50 -120 mg/d)

Anaerobic Metabolism
Alkaline valencies and lactate D6 or D12 cause a normoreactive challenge. In excess anaerobic exercise, there is frequently a functional hypoadrenia and hypoglycaemia associated. The following substances are tested.
Bicarbonate
Magnesium (3x100 mg/d)
Molybdenium (100 mg/d)
Vit B2 (riboflavine, 2x100 mg/d)
Vit B1 (thiamine, 2x100 mg/d)
Vit B5 (pantothenic acid, 3x100 mg/d),
Vit B6, P-5-P (2x50 mg/d)
Vit B3 (niacin, 2x50 mg/d)
Biotin (2x10 mg/d)
l-Carnitin (100mg/Kg BW) (Hoffmann, 1997)

Manganese (20 mg/d) is a cofactor of the pyruvate carboxylase, which katalyses a step in gluconeogenesis. According to this study, practically all painful conditions are associated with increased lactate, therefore an anaerobic metabolic situation can be suspected, caused by the increased sympathetic tone.

Disturbance of Energy Production
This is equivalent to disturbed ATP production. Energy production from food, principally takes three steps. Fats, polysaccharides and proteins in step 1 are broken down to fatty acids and glycerine, glucose and amino acids, from which in step 2, acetyl-CoA is produced. In step 3, with the oxidation of acetyl CoA 3 NADH+H+, 1 FADH2 und 1 GTP are produced which in the respiration chain give rise to 12 ATP.
Lactate D12 causes a normoreactive challenge in disturbed oxidative metabolism.
Glycolysis needs the following co-factors.
Niacin (3x50 mg/d)
Magnesium (2x80 mg/d)
Potasssium (2x50 mg/d)

Disturbances of the oxidative metabolism globally can be tested for by respiration of oxygen which causes a normoreactive challenge. Perform the AK test. Then the following supplements can be tested.

Acetylation requires:
Vit. B1 (2x50 mg/d)
Vit. B2(100 mg/d)
Niacin (NADH; 2x50 mg)
Magnesium (1x80 mg)

The Krebs cycle requires:
Niacin (NADH; 2x50 mg)
Magnesium (1x80 mg)
Manganese (20 mg/d)
Calcium (3x100 mg/d)
Vit. B1 (2x50 mg/d)
Vit. B2 (100 mg/d)
Vit. B6 (2x50 mg/d)
a-Lipoic acid (3x100 mg)
Iron (10 mg/d)
Sulfur (for example N-acetyl-cystein 2x250 mg)
Biotine (2x10 mg)

The respiration chain requires the following co-factors:
Coenzym Q10 (5 mg/Kg BW)
Vit. B2 (2x50 mg/d)
Copper (2mg/d)
Sulfur (for example N-acetyl-cystein 2x250 mg)
Magnesium (1x80 mg)
L-Carnitine (100 mg/Kg BW)
Iron (10 mg/d)
Phosphorus (no supplementation necessary)
a-Lipoic acid (3x100 mg)

Structural Corrections
As already mentioned above according to Schmidt and Lebowitz (1989), there is a correlation between certain conditions of dural tension and acid base imbalances. The correction of a condition, in which the positioning of the patient in left side bending curve (C-curve) causes a normoreactive muscle test outcome (hyper acidosis) is done with a so-called Logan - basic correction at the sacrotuberous ligament. If right side bending positioning (counter-C-curve) causes normal reaction (hyper alkalotic state) the Logan basic correction is done on the left. Logan basic is a contact at the sacrotuberous ligament in cephalad direction which has an impact on the entire sacrospinal system. Any kind of joint and muscle disturbance through decreased mechanoreceptor

afferent causes a decreased cortical activity and a decreased inhibitory control of the intermediolateral cell column, that is the pre-ganglionic sympathetic neurone. The resulting increased sympathetic tone causes tissue hypoxia and acidotic tendencies. Therefore joint disfunctions and any kind of disfunction of the motor system must be corrected.

Figure 8 Acid titration Case 1 before and 9 weeks after supplementation.

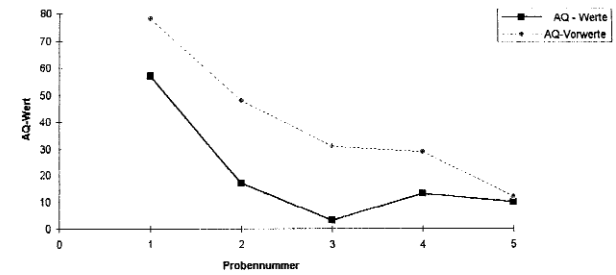


Figure 9 Acid titration case 2 before and 8 weeks after. Reduction of acidity ratio by 11%.

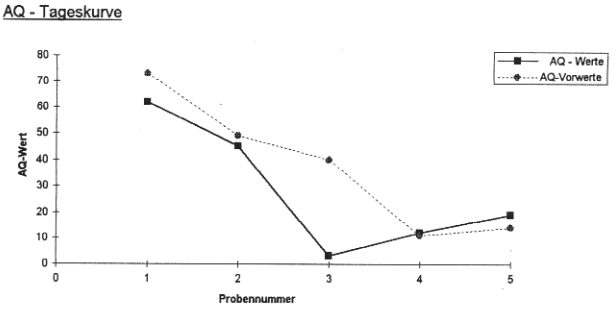
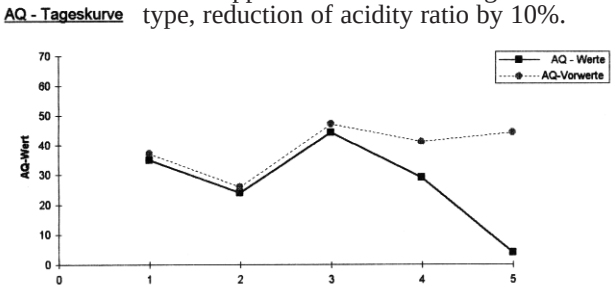


Figure 10 Acid titration case 3 before and 8 weeks after. Supplementation according to alkaline type, reduction of acidity ratio by 10%.



Emotional stress

Emotional stress also causes increases sympathetic tone with the respective lactate acidosis (and lowering of the pain threshold). Emotional stress can be treated with techniques of psychotherapy, NLP and hypnotherapy.

Case Study 1:

55 year old female patient: fibromyalgia, multiple food intolerances. Alkaline therapy, dietary therapy according to Worlitschek and Rauch; no improvement of clinical condition, constant acid values of urine titration 1.

9 week supplementation or according to AK testing, no further alkaline therapy, no dietary measures.

(B12/Folic acid 1x1, Zinc 30 mg 1x1, Phosphatidylcholin (420 mg) 1x1, Vitamin A (25000 IU) 1x1,

Vitamin E 400 mg 1x1, Pantethine (250 mg) 1x1, Algasan 2x1, all according to AK-test.

Result: Clinical improvement, decrease of the acidity ratio by 11%. See fig 8.

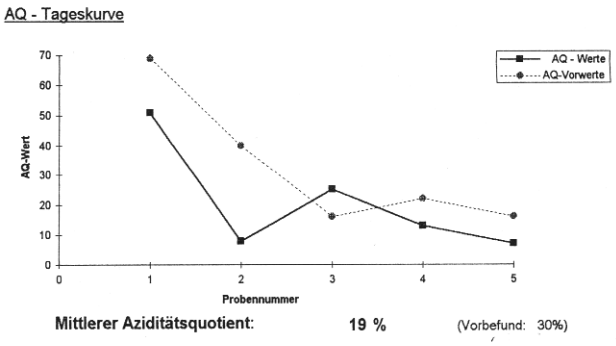
Case Study 2:

26 year old female patient. Chronic reoccurring cystitis, chronic reocurring intestinal candidosis.
Supplementation : Vitamin A (25000 IU) 1x1, D-Mulsin¨, Vit. C 1x500 mg,
B-complex plus[a] 1x1, Potassium/Magnesium (100/70 mg) 2x1, Manganese (15 mg) 1x1, Iron-C (25 mg) 1x1.
Urine titration before and after 8 weeks supplementation. See fig 9.

Case Study 3:

50 year old male patient, medial disc herniation.
Supplementation. Urine titration before and after 8 weeks supplementation.
Vit. A 1x1, D-Mulsin¨, Vit. C 1x 500 mg,
B-complex plus 1x1, Potassium/Magnesium 2x1, Manganese 1x1. See fig. 10.

Figure 11 Acid titration case 4 before (…) and 8 weeks after (…) supplementation according to acid type, reduction of acidity ratio by 11%.

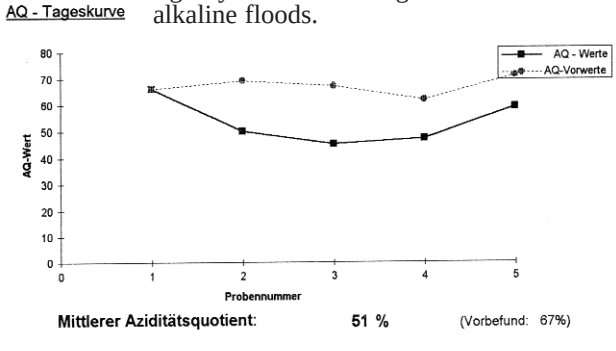


Case Study 4:

74 year old female patient. Osteoporosis, several vertebral fractures, spinal pain syndrome, immune deficiency. Alkaline therapy not tolerated (gastro intestinal problems). Nevertheless, followed during 6 months. 8 weeks supplementation, B12/Folic acid 1x1, Zinc 30 1x1, Phosphatidylcholin 1x1, Vitamin A 1x1, d-alpha-tocopherol 1x1, Pantethine 1x1, Algasan 2x1, all according to AK test.

No further alkaline therapy, no dietary change. Acid titration before and after supplementation. See fig. 11. Because of the importance of acid base metabolism for the calcium metabolism in osteoporosis this result is of special importance.

Figure 12 Acid titration case 5 before (…) and 8 weeks after (…) supplementation. Reduction of acidity ratio by 16% but still rigidity that is no change between acid and alkaline floods.



Case study 5:

39 year old female patient, allergy of room dust, asthma, eczema, allergic rhinitis. Food intolerances with gastro intestinal problems, stiffness of joints. Heavy metal toxicity. Supplementation with B12/Folic acid, Pantothenic acid, Phosphatidylcholin, Algasan, Calcium during 8 weeks according to AK. Thereby normalised abdominal finding and skin, almost total disappearance of stiffness.
See fig. 12.

Conclusion

The study and case histories described in this article seem to suggest the following.

1. The clinician must not examine and perceive latent acidosis exclusively but also latent alkalosis.

2. Acid titration according to Sander does not furnish any insight to the acid-base state of a patient as the urine always has an acid tendency. The elimination of acid valencies is a normal function of urine elimination. Acid elimination is subject to a metabolic control in which the liver plays a crucial role by means of the glutamine metabolism. A latent acidosis as described in German speaking natural health care (Sander, 1985, Worlitschek, 1994, and Jörgensen, 1985) has to be understood as an increased need for elimination of acid valencies caused by metabolic, structural and emotional stress.

   Acid titration according to Sander therefore has to be considered as a parameter for the amount of metabolic stress and therefore can be used as a measure for success or failure of a metabolic treatment.

3. None of the lab measurement methods described in this article alone can furnish a comprehensive understanding of the acid base situation of the patient; according to applied kinesiology the use of $NaHCO_3/NaHPO_4$ mixture, HCl, lactate, acido acetate, hydroxy butyrate is very useful as a screening methods. The metabolic stressors which potentially cause acid base imbalances must be tested individually and specifically.

4. Applied kinesiology is the method that seems to be best capable to evaluate the specific acid-base-imbalance of a patient and to design a therapeutic concept.

   Using a specific nutritional therapy it is possible to reduce several types of metabolic stresses which leads to decreased acid elimination as a sign of correction of acid-base imbalances. This is a truly causal therapy as opposed to supplementation of alkaline or acid substances.

Practical Recommendation

1. Use Challenge as by applied kinesiology with alkaline salts or betaine-HCl respectively. If there is a normoreactive challenge outcome, do the following:
2. Use urinary acid titration as a baseline measuremant before therapy.
3. Use blood gas analysis as by Astrup. If base excess is not normal and there is a respective clinical picture treat with alkaline salts or betaine HCl according to challenge with AK.
4. Use lactate challenge as by applied kinesiology; if positive: lactate lab measurement and supplementation accordingly, improve aerobic metabolism with adequate training, supplementation and physical measures which can improve oxygenation (thoracic mobility!).
5. In fasting metabolism, diabetes type 1; use AK challenge with acetoacetate and beta-hydroxybutyrate. Supplementation as described in the article under diabetes type 1.
6. In all other metabolic disturbances, use orthomolecular supplementation according to tests with applied kinesiology and laboratory measurement.
7. Urinary acid titration as a control of the therapy

References

Devlin, T. M.: Textbook of Biochemistry, Wiley-Liss., New York, 1997.

Forth, W. et al.:Pharmakologie und Toxikologie, BI Wissenschaftsverlag, Mannheim, 1997.

Enderlein, G.: Bakterien-Cyclogenie, Semmelweis-Institut, Hoya, 1981.

Goodheart, G.: Collected Published Articles and Reprints. 1 ed. Detroit: Privately published, 1969.

Goodheart G.: You`ll be Better. Privately published, Detroit, 1988.

Harrison, T.R. (editor): Principles of Internal Medicine,14. ed., McGraw-Hill, New York, 1998.

Hawkins, H. P.: Applied Nutrition, Mojave Books, Resada, 1977.

Hoffmann, G. F. und Lehnert, W.: Leitlinien zur erblichen Laktatazidose. 1997. http://gopher.rz.uni-duesseldorf.de/www/AWMF/II/pstwe012.htm.

Jörgensen, HH.: Säure-Basen-Haushalt - Ein praxisnahes Messverfahren zur Bestimmung der Pufferkapazität. Erfahrungsheilkunde 1985;5:372-7.

Jörgensen HH.: Zur Klärung einer medizinischen Grundfrage, Antworten zur Problematik des Säure-Basen-Haushaltes. Sanum-Post, 1989;7:22-4.

Lebowitz, M., Steele M.: Correcting chronic health problems, Selbstverlag, 1989.

Marées, H., Sportphysiologie, Tropon, Köln-Mühlheim, 1989.

Rauch, E.: Diagnostik und Therapie nach F. X. Mayr, Heidelberg, Haug, 1994.

Sander F.: Der Säure-Basen-Haushalt des menschlichen Organismus, Hippokrates, Stuttgart, 1985.

Schmidt, R. F. und Thews, G.: Physiologie des Menschen., Springer, Berlin, 1990.

Thomas, L. (Herausg.): Labor und Diagnose, Medizinische Verlagsanstalt, Marburg, 1992.

Vincent, J. L., Blood lactate levels: index of tissue oxygenation, International Journal of Intensive Care,Vol. 5, 98, p. 48-52.

Walther DS. Applied Kinesiology, Synopsis. 275,West Abriendo Av., Pueblo, Colorado 81004: Systems D.C., 1988.

Walther, DS. Applied Kinesiology, Synopsis, Pueblo, Colorado, Systems DC, 2000.

Walther DS. Applied Kinesiology Vol. I. Pueblo, Colorado: Systems D.C., 275,West Abriendo Av., 1972.

Wiley R.: The Effect of Acid/Alcaline Nutrition on Psychophysiological Function. Int Journ Biosocial Res 1987;9 (2):182-202.

Wiley R.: Biobalance. Life Science Press, Tacoma, 1989.

Worlitschek M.: Der Säure-Basen-Haushalt, Haug Verlag, Heidelberg, 1994.

Worlitschek M.: Praxis des Säure-Basen-Haushaltes. Haug Verlag, Heidelberg, 1994.

Case Study: Chronic Severe Constipation Caused by Asymptomatic L3-4 Intevertebral Disc Syndrome and Closed Ileocecal Valve

William Maykel, D.C., DIBAK

Abstract

The case of a thirteen year old boy who had never moved his bowels is discussed. Normalization of the biomechanics of his lumbosacral spine and ileocecal valve function corrected the case of a young man who had a once a week enema for all of his life. After his third visit he was moving his bowels daily.

Key Words: Applied Kinesiology, Ileocecal Valve, Challenge, Constipation.

Introduction

As a result of a passing conversation with a mother of a thirteen year old boy, I found out that he had never begun to move his bowels since birth. He had extensive workups at many of the Worcester and Boston area hospitals to no avail. He had gone through a series of treatments which included repeated cycles of x-rays and mineral oil - none of which worked. For the better part of his life, his mother had given him a weekly enema. Constipation is defined as less than three bowel movements per week.[1] Constipation is common and significantly impairs health-related quality of life.[2, 3] The incidence range in the pediatric age is 0.3 to 8% and in 90-95% of cases it is functional, often due to an exclusively milky diet.[4]

Discussion

Through my own past experience in treating people with closed ileocecal valves[5] I thought he may have an asymptomatic low back problem that may be related to a dysfunctional gastrointestinal tract from a mechanical basis. Therefore I suggested that she bring him in.

Physical examination revealed that he in fact had a bilateral sacroiliac sprain/strain (right posterior externally rotated, left anterior) with a compression of the L3-4 intervertebral disc. Additionally L3 was anterior and that there was a positive challenge suggestive of a closed ileocecal valve. Other findings included a bilateral tarsal tunnel syndrome with laterally displaced talli and externally rotated tibias. The muscles relating to his stomach (pec major claviculars) were weak bilaterally suggestive of hypochlorlydria. There was also tenderness on palpation of the gallbladder so we suggested that he use beet greens. They have lipotrophic effect . After his second office visit, he had two small bowel movements on his own and after the fourth office visit, seven weeks from his first visit, he was moving his bowels on a daily basis. Treatment consisted of some lumbar intersegmental traction to the L3-4 disc along with corrective stretching exercises. He avoided several foods that he was found to be sensitive to using muscle testing. With a gustatory challenge, he was found to be sensitive to milk, corn, soy and wheat. We also gave him some nutritional advise to increase the water soluble fiber in his diet by incorporating the use of papaya and apples in his diet.

Conclusion

Normalization of the lumbosacral plexus outflow to the gastrointestinal tract specifically the ileocecal valve should be one of the first lines of approach to normalizing bowel function. It is both a non-invasive and cost effective (given the fact that poor quality of life is an important predicator of healthcare utilization in these patient types[6]) approach as well as represents a perfect example of the relationship between structure and visceral function. The tool of "challenge" utilizing applied kinesiology through muscle testing represents a technology which has awesome clinical applications that are unfortunately highly under utilized.

References

1. Cayaran S, Dorule E, etal. "The Assessment of constipation in monosymptomatic primary nocturnal enuresis." Int Uro Nephrol 2001;33(3):513-6.
2. Irvine EJ, Ferrazzi S, Pare P "Health-equated quality of life in functional GI disorders:focus on constipation and resource utiliztion. Am J Gastroenterol 2002 Aug;97(8):1986-93.
3. Walter S, Hallbook O, etal "A population-based study on bowel habits in a Swedish community:prevalence of faecal incontinence and constipation," Scand J Gastroenterol 2002 Aug;37(8):911-6.
4. Dito L. "Constipation in Children." Clin Ter 2002 Jul-Aug:153(4):281-7.
5. Walther, David S DC, "Ileocecal Valve Syndrome" - Applied Kinesiology Synopsis-Second Ed. 1988, p494-500.
6. Irvine EJ, Ferrazzi S, Pare P "Health-equated quality of life in functional GI disorders:focus on constipation and resource utiliztion. Am J Gastroenterol 2002 Aug;97(8):1986

Case Study: Correction of Severe Hiatal Hernia Complaints in a Patient with a Congenital Failure of Skeletal Muscle Growth with Resultant Severe Scoliosis

William Maykel, D.C., DIBAK

Abstract

A thirteen year old boy with Werdnig-Hoffman Disease presented for treatment. He had an inability to hold down food for a five month duration. A hiatal hernia was reduced utilizing the applied kinesiology tools of surrogate testing, respiratory adjusting and muscle balancing. After one session, the patient was able to swallow an entire meal without side effects.

Key Words: Applied Kkinesiology, Werdnig-Hoffman Disease, Hiatal Hernia, Surrogate Testing, Respiratory Adjusting, Muscle Balancing, Origin Insertion, Muscle Spindle, Neurolymphatic Reflex.

Introduction

The Spinal Muscular Atrophies (SMA) are a group of inherited disorders in which there is degeneration of the anterior horn cells in the spinal column resulting in weakness. There are three clinical variants based on varying degrees of severity and onset.

- Type I – Infantile Type – Werdnig-Hoffman Disease (severe);
- Type II – Intermediate form (intermediate);
- Type III – Juvenile Type – Kugelbers-Welander Disease (mild).

Kevin had the type I (infantile) form with a prognosis of death before age two. Kevin's mother was the reason for his survival. Her great love, caring and spirituality had allowed Kevin to nuture and survive against all odds.

Kevin had previously gotten chiropractic care for his craniosacral system with an applied kinesiologist (Dr. Richard Herzig) at eighteen months. This care helped his extreme weakness. This allowed him to hold his head up, start to have normal bowel movements and turn around the general overall failure to thrive in Kevin. At age four she started him on deep muscle therapy with once a month visits. At four and one half months, he developed severe pneumonia and Dr. Herzig once again helped him through this episode.

A thirteen year old boy presented for treatment of symptoms related to a severe hiatal hernia. For the previous five months, he had been able to swallow his food without difficulty, but would regurgitate his food and copious amounts of liquid upon eating just a few bites. During this time frame, from May through October, he was virtually unable to eat and allow food to enter his stomach. He sometimes had been able to eat one muffin or one meal within a two day period.

Discussion

Prior to his visit to this office, Kevin had undergone two endoscopies without good effect. Kevin was not your typical child. In fact, it was a miracle he was alive to begin with. He was suffering with complications of a usually fatal neurological disorder called Werdnig-Hoffman disease. This polygenic disorder causes premature death of the anterior horn cells with resultant hypotonia and weakness.

When Kevin's mom brought him in for treatment he had an electric wheelchair which he could operate with digital controls. He wore a molded body cast made out of semi-dense foam. This served as his muscles, as there are no muscles with this condition.

Due to the lack of muscles, Kevin's spine bent and twisted as he grew with a severe scoliosis (88 degrees). Upon presentation it had grown forward and twisted so as to crush his esophagus-diaphragm against his sternum, blocking the passage of food. On May 25, 1995 a policy statement on surrogate testing by ICAK-USA was passed. It is as follows "Surrogate Testing is not a usual method of diagnosis in Applied Kinesiology. It is used only when the subject can not respond appropriately, such as with a comatose individual, an infant, or with an otherwise incapacitated person." An excellent review of the subject may be found in the ICAK-USA 1996-97 Proceedings by Dr. Hans Boenke.

Hiatal hernia and reflux esophagitis have been found to occur together.[1,2,3,4] They have also been associated with respiratory manifestations.[5]

Surrogate testing[6, 7] allowed the evaluation of his skeletal misalignment to proceed quickly and accurately. A bilateral sacroiliac sprain/strain (right posterior/left anterior) was found along with the usual concomitant findings (right inferior sacral base, L3 right, L4 left, L5 right and C1 right, C2 left, C3 right). Additionally he had T9 through L1 anterior with ribs bilaterally lateral from T9 through T12. Challenge was positive for a diaphragm strain.

Origin-insertion and muscle spindle technique along with related neurolymphatic reflexes were performed to facilitate the abdominals, diaphragm, and major pelvic muscles.

Conclusion

Kevin responded well to the interventions and treated at this office through age eighteen. Kevin moved to Colorado, graduated from the University of Colorado with a major in psychology and minor in special education. Kevin's mother credits Kevin's extraordinary longevity to the remarkable benefits of applied kinesiology.

References

1. Sonnenberg A, Everhart JE "Hospitalization with respiratory disease following hiatal hernia and reflux esophagitis in a prospective, population-based study." Ann Epidemiology 2001 Oct; 11(7):477-83.
2. Koike T, Ohara S, etal "Increased gastric acid secretion after heicobacter pylori eradication may be a factor for developing reflux esophagitis." Alimentory Pharmacology Ther 2001 Jun;15(6):813-20.
3. Hamada H, Haruma K, etal "High incidence of relux esophagitis after eradication therapy of Helicobacter pylori: impacts of hiatal hernia and corpus gastritis." Aliment Pharmacol Ther 2000 Jan;14(6):729-35.
4. Penagini R, Carmagnola S, Cantu P. "Review Article:Gastro-oesophageal relux disease-pathophysiological issues of clinical relevance." Aliment Pharmacol Ther. 2002 Jul;16 Suppl 4:65-71.

5. Dal Negro R, Pomari C, Micheletto C, etal "Prevalence of gastro-esophageul reflux in asthmatics: An Italian Study," Ital. J Gastroeterol. Hepatol 1999 Jun-Jul;31(5):371-5.
6. McCord, Kerry M. DC, "Surrogate Testing Discovery and Application," Collected Papers of ICAK-USA, Winter 1988-89;257.
7. Boenke, Hans W. DC, DIBAK, "Surrogate Testing - It's History, Controversy and Recommended Uses" Collected Papers of ICAK-USA 1996-97;27-34.

Case Study: Cryptorchidism Correction with Conservative Chiropractic Applied Kinesiology

William Maykel, D.C., DIBAK

Abstract

A nine month old infant diagnosed with a congenital right inguinal hernia and undescended testicle (cryptorchidism) was brought for conservative care prior to surgery. A successful outcome utilizing applied kinesiology surrogate testing and respiratory correction of a bilateral sacro-liac sprain/strain is discussed. The author believes this non-invasive, cost effective approach should constitute the primary initial approach if future validation studies are positive.

Key Words: Applied Kinesiology, Surrogate Testing, Cryptorchidism, Respiratory Adjusting.

Introduction

The parents of a nine month old male infant sought a second opinion for their child who was diagnosed one week earlier as having a right hernia with undescended testicle. Their pediatrician referred them to a general surgeon who recommended surgery. Cryptorchidism is the most frequent abnormality of male sexual differentiation and is the main risk factor for testicular cancer, which is the most frequent cancer in young men. It is also a major risk factor for male infertility.[1]

At the same time as a wide spread increase in the incidence of testicular cancer, a tendancy toward an increase in the incidence of cryptorchidism in industrialized countries has been observed in recent decades.[2] Low birth weight (for equal gestational age) is the main risk factor along with first birth and cesarean section or toxemia.

Additionally there have been ecoepidemiological studies performed in various animal species which show a relationship between environmental pollutants (especially organochlorine pesticides) and the unexpected high incidence of cryptorchidism. DES (diethylstilbestorol) has a well-documented impact on undescended testes in male offspring. No formal conclusion can be drawn presently that industrial chemicals known to be potential endocrine disruptors are responsible for the recent increase in the number of cases of cryptorchidism.[3]

Discussion

Physical examination revealed a healthy infant with a small palpable lump in his right mid inguinal area. He had been a normal vaginal delivery without consequence, although he was six weeks premature.

Surrogate testing[4] was utilized to determine a misalignment of his pelvic complex. In this case, the child was held by one of his parents (who was seated on the exam table) with his head over one of their shoulders while being held with one arm under his pelvis. A standard muscle test was performed on the parent's intact anterior serratus muscle. The child's sacroiliac joints were challenged immediately after which the patient's intact muscle was rechecked. This is called “surrogate testing.” In essence, since there is a melding of the energy fields

of the parent and the child, the parent's arm responds to changes in the child's nervous system. Specifically in this case, a bilateral sacroiliac sprain/strain was found with a right posterior, left anterior sacroiliac sprain/strain. Correction was achieved with gentle respiratory adjustments. This involves having the parent consciously inhale and exhale deeply and with the child held against the parent, entrainment of their breathing ensues. Gentle force is applied on inspiration to correct the misaligned segments. Inspiration is chosen since there is straightening of the spinal curves allowing easier correction of the vertebral subluxation complex.

The specific complex that arises with this type of distortion involves an inferior sacral base on the side of the posterior inferior ilium (in this case the right). An inferior sacral base causes a compensatory rotation of the last three lumbars – right, left, right and as well C1, 2, and 3, right, left, right. The thoracolumbar junction was also found to be rotated T10, 11, 12, right, left, right. These patterns are found to be ubiquitous for this type of pelvic lesion. This was performed on two occasions one week apart with complete resolution of the problem after the second visit.

Conclusion

The child was seen four years later for other unrelated conditions and he had no further difficulties with the right inguinal area or his testicles. Since surgery is the only current approach further validation studies should be undertaken especially due to the global increase in this condition. Surrogate testing represents a quick, accurate diagnostic technique and respiratory adjustments a gentle correction of musculoskeletal lesions.

References

1. Thonneau Patrick F, Candia Peggy and Mieusset Roger, "Cryptorchidism:Incidence, Risk Factors, and Potential Role of Environment; An Update." Journal of Andrology, Vol 24, No.2, March/April 2003.
2. Kurahashi N, Kishi R, "A Review of epidemiological studies about the incidence and etiological factors of cryptorchidism-relevence to endocrine-disrupting chemicals." Nippon EiseigakuZassh:2003 Jan;57(4):636-44.
3. Thonneau Patrick F, Candia Peggy and Mieusset Roger, "Cryptorchidism:Incidence, Risk Factors, and Potential Role of Environment; An Update." Journal of Andrology, Vol 24, No.2, March/April 2003.
4. Boenke, Hans W. "Surrogate Testing - It's History, Controversy and Recommended uses." Collected Papers of ICAK-USA 1996-97;27-34.

The Pineal Cranial Fault

Paul T. Sprieser, D.C., DIBAK

Abstract

An examination of a new cranial fault that associates specifically with the pineal gland and melatonin metabolism.

Introduction

Two years ago, while researching switching and the Figure "8" energy patterns, I received a referral from a registered nurse, who has a very astute psychic ability in the area of health matters.

The woman that she referred had a myriad of health problems that associated with some sleep disturbances and symptoms that occurred during the night. During the examination of this patient, the R.N. was present to help with any intuitive information she could perceive that might assist me with my treatment program.

One of this R.N.'s special abilities was seeing the energy in the meridian system. I have worked with her for more then 25 years and I know what she describes to be accurate through AK muscle testing and meridian energy diagnosis.

I asked the referred patient, would she mind me checking her and collecting data for a research paper that I was working on. I described to her briefly about neurological disorganization and its connection to the Figure"8" energy pattern.[1] As I described this pattern the R.N. agreed about how the energy appears. She commented to me that she saw the same pattern in the roof of the mouth on the palate.

Discussion

Not knowing what this might mean I had the patient T.L. the palate for cranial faults are associated with the region. I checked for Sphenobasilar Fault, Palatine Sutural Faults, possibly Frontal faults,[2] and Learning Disability Fault[3] all of these had T.L. patterns to the palate region.

The Sphenobasilar Faults T.L. to the area of the cruciate suture with the thumbs are localizing on both the right and left side of the maxilla on the palate, with breathing differentiating the type of fault.[4] The Palatine suture would be challenged by using the index fingers near the first molar and pressure laterally to separate the suture with respiratory challenge to assist correction.[5] The Learning Disability Fault is T.L. with the index finger at the cruciate suture will be positive but inspiration or expiration will not negate indicator muscle weakness, but inspiration is required for the correction.[6]

What I came upon was a new pattern of T.L. to the palate. I had the patient cross her thumbs and place them on either side of the cruciate suture similar to the Sphenobasilar fault T.L. except the right thumb was on the left side and left thumb was on the right side. This produced a weakness to the indicator muscle. I believed I had discovered a new cranial fault.

Method

Patient was supine muscle testing was done with TFL, with cross thumb localization to the cruciate suture. A positive weakness to the indicator muscle was noted a phase of respiration that negated weakness was used.

I then tried various vectors both to the palate and on the occiput and found the one that created a weakness to a strong indicator muscle. I found that a contact to the center of the palate with the index finger following a figure "8" pattern of motion would cause a positive response. With my other hand I used a contact to the occiput with my hand spread so I could contact both mastoid processes simultaneously. Using a challenge both clockwise and counterclockwise similar to the Universal Fault correction I went in direction of greatest weakness.

Following this pattern of motion with both hands as I described in the phase of respiration that negated the muscle weakness. I found that it took about 40 seconds of this repeated action to achieve a correction. I was being assisted by the R.N. (psychic) who noted when the energy balanced.

At this point I did not know what this fault associated with, but the R.N. said she thought it might have to do with the pituitary or pineal glands. Over subsequent visits with this patient I saw the pattern return. I tried having the patient chew both Standard Process and Biotic Research nutritional products. What I found negated the positive cross thumb T.L. to the palate was melatonin and pineal gland extracts. I also tried muscle testing in totally dark room that caused weakness to and indicator muscle and have the patient cross T.L. this would negate the weakness. This confirmed the connection to the pineal gland for this fault.

Conclusion

I have been following and examining for the fault for the past two years and, at the present time, I have treated 78 patients with this fault.

References

1. Sprieser, Paul, T., D.C.-A New Slant on Switching and the PRYT Technique, The Collected Papers of ICAK, 2002-2003 p.p. 197-202.
2. Walther, David, S., D.C., Applied Kinesiology, Synopsis 2nd Edition, System DC, Pueblo, CO, 2000, p.p. 387-388, 392-393.
3. Sprieser, Paul, T., D.C.-Learning Disability Fault, Part I & II, The Digest of Chiropractic Economics, May/Jun 1987, Nov/Dec 1987 and The Collected Papers of ICAK, Winter 1984 p. 265.
4. Walther, David, S., D.C., Applied Kinesiology, Synopsis 2nd Edition, System DC, Pueblo, CO, 2000, p.p. 387-388, 393-393.
5. Walther, David, S., D.C., Applied Kinesiology, Synopsis 2nd Edition, System DC, Pueblo, CO, 2000,
6. Sprieser, Paul, T., Learning Disability Fault-The Collected Papers of I.C.A.K., Winter 1984, p. 265.

The Importance of Applied Kinesiology in Treating Children

Juli Tooley, D.C., D.I.C.C.P.

Abstract

Children should receive chiropractic adjustments from the time of conception until the time of transition. Applied kinesiology (AK) should play an important role in the health care of children. AK is both a diagnostic and treatment approach to total healthcare. The three sides of the triad of health, along with the five factors of the intervertebral foramen are instrumental in optimizing both the possibility and probability of increased resistance to disease, along with above average immunity. Thereby all but insuring that the individual will proceed through life in the most advantageous manner.

Introduction

Ideally, children should receive chiropractic adjustments throughout their entire lifetimes, from the time of conception until the time of transition (death). Not only should the children receive this extremely important preventive care, but BOTH parents should also be experiencing the benefits of optimal health before the conception ever occurs.

Applied kinesiology should play an integral role in both the prospective parents' health, as well as, the unborn fetus, infant, toddler, pre-schooler, school-aged child, adolescent, and young adult's life plan. As a supplement to specific chiropractic adjustments, AK affords the individual an opportunity to embark upon the game of life with a distinct advantage. The triad of health is balanced at the beginning of life, thereby all but eliminating the many inequities that arise due to structural, chemical, or mental factors. The five factors of the IVF (**N** - nerve supply/nutrition, **NL** - neurolymphatic reflexes, **NV** - neurovascular reflexes, **CSF** - cerebrospinal fluid, **AMC** - acupuncture meridian connector) are used both diagnostically and therapeutically to enhance the adjustment of any subluxated or fixated articulation. The adjustment allows for optimal function and thus enhances the overall health of the patient throughout her/his entire lifetime.

Discussion

What does it mean that children should receive chiropractic, including AK, from the time of conception … if not before? It simply means that if both parents, the father with his sperm carrying their portion of the DNA material, and the mother with her ovum supplying the other portion, are functioning optimally, the probability for any abnormalities or anomalies is drastically reduced. The fertilized ovum now has both maximum potential for its proper development into a viable fetus and an ideal environment in which to grow and flourish.

As we have all been taught in the various AK courses throughout the world, functional physiology is routinely restored using not only the five factors, but also integrating all three sides of the triad of health, barring any pathology. It is important to remember this concept during the child's development throughout the pregnancy. As published research studies have shown, semantic muscle testing is now a fact. (To verify this information, see the reference to the article by Monti, et. al. at the end of this paper.) Thus giving even more credibility to Dr. George Goodheart's adage to "ask the body to ask the body."

In utero concerns can now be addressed with a greater degree of assurance that the actual problem is being ascertained. This also allows for many emotional issues to be dealt with before the time of delivery, both for the parents and the unborn child/children. Just a reminder, AK is an adjunctive approach to in utero concerns and should not be used as a "stand alone" therapeutic approach.

Once the delivery approaches, the infant now has a much better chance of lessened complications due to adequate nerve, vascular, and hormonal supply to both the mother's and child's bodies. Balanced uterine musculature, thereby allowing for organized and polarized contractions, assists the baby in exiting the birth canal in the most efficacious manner.

Once the infant arrives and the umbilical cord has been severed, who better than a chiropractor should attend to that little person? Utilizing surrogate muscle testing, the AK practitioner can not only check infantile automatisms (reflexes), adjust vertebrae and clavicles, restore muscular balance, but also administer the often much needed cranial remolding which is so beneficial for the newborn.

Now that the child is born, doesn't it make sense to give it the most beneficial way to grow and develop with both chiropractic adjustments and AK supportive procedures? In my experience, children that receive regular and ongoing AK and chiropractic care during the first two years of their lives develop a stronger resistance to dis-ease than their untreated siblings. In utilizing AK procedures adjustments are longer lasting and body systems are more in balance.

Children have an amazing capacity for understanding why they should do certain things. It is a perfect time to turn them into lifetime referring patients when you demonstrate simple concepts like how a weak muscle is "turned back on" by its NL, NV, alarm point, or by holding their breath in or out. They will look at you first in disbelief, then in awe and wonder, then in delightful amusement (usually via giggling) and beg you to do more.

When confronted with the various childhood ills, these children suffer less virulent attacks because their bodies are more in balance. This includes infants who suffer from colic, children who have otitis media (ear infections), and those with cold and flu symptoms (fever, runny or stuffy noses, vomiting). The school age children seem to get over things quicker and more easily than their classmates who have not received any chiropractic care whatsoever.

What makes AK important in treating children, especially those under the age of 3 or 4 (pre-schoolers), is that the muscle test can often locate a problem that the child cannot adequately verbalize. Since these little bodies are like fine-tuned athletes, inasmuch as their bodies and minds are not yet as polluted as adults, they respond greatly to very little therapeutic intervention.

One of the most difficult aspect in treating children, is getting the parents and grandparents to comply with your recommendations. Dietary constraints are the most difficult. Not enough water … and too much sugar. The average (whatever "average" is??) American now consumes 165 pounds of sugar per year (and I think that's conservative when it comes to children). The public school system is a huge contributing influence. Snacks in preschool and kindergarten consist of cookies and milk, lunch programs provide pizza and hot dogs with soda machines installed at several locations across the campuses.

Have you ever wondered why such a huge percentage of kids are now being labeled ADD or ADHD? Is it merely that the drug company which makes Ritalin has a foothold in the schools? I think not. Is it that theses conditions have now reached epidemic proportions? Or is just that time has finally borne out the fact that the older kids who have been fed a constant diet of sugar, white flour, and fast foods have been severely damaged? I recently read that children are now actually mutating their DNA and have altered livers to counteract the ill effects of the fast foods they have ingested for such an extended period of time. Is it really more conve-

nient to shove non-nutritional foodstuffs down your kids' throats or will you both pay in the long run with medical interventions and hospitalizations due to organ systems which rebel and cease to function adequately by the time the child becomes a teenager?

Conclusion

Education at a very young age is a key factor to a child's health. AK not only fixes their problems but is also an excellent educational vehicle for those young minds to soak up amazing amounts of valuable information. We have all heard the old adage, "A picture is worth a thousand words". Demonstrate simple AK procedures and educate your young patients and they will not only grow into more healthy adults, but their children will reap the benefits in the future. They really do want to know and understand what it is you are doing and how good it is for them to maintain a healthy lifestyle.

References

1. Walther, David, Applied Kinesiology Synopsis, Second Edition, Systems DC, Pueblo, CO, 1988-2000.
2. Monti, Daniel, et.al., Muscle Test Comparisons of Congruent and Incongruent Self-Referential Statements, Perceptual and Motor Skills, 1999.
3. Phillips, Carol, Hands of Love, Seven Steps to the Miracle of Birth, New Dawn Publishing, St. Paul, MN, 2001.
4. Fysh, Peter, Chiropractic Care for the Pediatric Patient, International Chiropractors Association Council on Chiropractic Pediatrics, Arlington, VA, 2002.
5. Goodheart, George, Being a Family Doctor, Self-Published, Grosse Pointe Woods, MI.
6. Goodheart, George, You'll Be Better, Self-Published, Grosse Pointe Woods, MI.

The Triad of Health, Expanded

Juli Tooley, D.C., D.I.C.C.P.

Abstract

What is the triad of health and why is it so important? The triad has deep historical roots in the chiropractic profession which are often either overlooked or not remembered. The triad has applications in many facets of life. Included will be those components which affect life, in general; those which pertain to the practice of chiropractic; and those which specifically apply to applied kinesiology (AK).

Introduction

Since this forum is primarily chiropractic kinesiologists, I will concentrate on a limited number of pursuant aspects of this phenomenon. The triad of health, as applied to life, in general, leads to numerous conclusions. One of those conclusions which applies to our quest for healing via total integration of the body, mind, and spirit, is the triune brain theory, as introduced by Dr. Paul MacLean in 1973. He identified three distinct components: reptilian, mammalian, and neocortex. Many other correlations can be extracted which apply to life, in general. Another of these is: man, woman, child. Still another is: hot, cold, warm. Why are these concepts important in our lives?

If we apply this mode of thinking to Chiropractic, in general, we arrive at another triunal theory: science, philosophy, art. Why is this correlation so important in restoring total health, according to many of our founding fathers?

Still another application of the triad of health appears in the applied kinesiology triangular analogy: structural, chemical, mental. Why is this triad at the crux of healing, according to Dr. George Goodheart?

Discussion

Triad, according to The American Heritage Dictionary, is a noun that means:

1. A group of three.
2. Music. A chord of three tones, especially one built on a given root tone plus a major or minor third and a perfect fifth.
3. A section of a Pindaric ode consisting of the strophe, antistrophe, and epode.

If we access the synonyms from The Original Roget's Thesaurus of English Words and Phrases, we include:

1. three: three, triad, trine
2. musical note: chord, common chord, triad, tetrachord, arpeggio
3. Trinity: Trinity, triad, Hindu triad, Brahma, Shiva, Vishnu

So, as you can see, the topic of the triad of health EXPANDED can take many multidimensional turns. I will attempt to limit this paper to the most apropos applications whenever possible.

As health care providers, we must not only be concerned with the patient's physical body as she/he enters our office, but also her/his entire life as she/he proceeds on the journey. This life consists of mental and spiritual components, as well as physical aspects of health. Patients cannot remain healthy in an unhealthy environment, be that body, mind, or spirit. According to Dr. R. W. Stephenson in his Chiropractic Textbook (1948), "Life is a Triunity having three necessary united factors; intelligence, force, and matter. Without intelligence, matter could not even exist. Without matter, intelligence could not be expressed. Then there is a bond between intelligence and matter that cannot be dispensed with. The bond is called force."

As Paul MacLean points out in The Triune Brain in Evolution (1990), the reptilian portion of our brain is related to survival (the most basic of human drives), the mammalian or limbic section has to do with emotions (the further evolved endocrine and conditioned response functions), and the neocortex has to do with thinking and intellect (the most highly evolved concepts such as reasoning, decision-making, morality, etc.). Or, as Sigmund Freud would put it, the unconscious, subconscious, and conscious portions of the mind.

What is it about three that is so important to us as humans? Could it be that we are currently inhabiting a three-dimensional plane of existence? Many people in the past have discussed the possibility of duality: up/down, hot/cold, large/small, mother/father, in/out. But is that really what we are all about, or is there another aspect which must be addressed? That middle ground … the gray area … the "glue" which holds both extremes together. To continue with the above examples: up/down/middle, hot/cold/warm, large/small/medium, mother/father/child, in/out/center. If you ascribe to contemporary philosophy, nothing is an accident. Things fit perfectly if we only know how to assemble them in the correct alignment.

This leads to the relevance of this triune approach to chiropractic. What were we taught in first-year philosophy class in chiropractic college? Chiropractic is a triad of philosophy (3 T's, homeostasis, disease vs. dis-ease hypothesis, subluxation concept, etc.), science (anatomy, physiology, biology, biochemistry, embryology, pathology, diagnosis, etc.), and art (correction via adjustment or alignment of vertebral /extraspinal articulations). Or, to more correctly quote Dr. Palmer, "Chiropractic is a philosophy, science and art of things natural; a system of adjusting the segments of the spinal column by hand only, for the correction of the cause of disease".

An important piece of the proverbial puzzle is described above … a triad within a triad … the "3T's" which cause a subluxation, according to Palmer (Trauma, Toxins, Thought or Autosuggestion). There are varying causes which create dis-ease in our patients, but the majority fall into one of these three categories. Were our founders more in tune with the existence of Universal Intelligence and hence the concept of Innate Intelligence being able to heal the body from within was considered normal? Or have we merely become overburdened with mechanical, biochemical, and emotional stressors to the point of dis-ease manifesting as disease is the accepted norm? Maybe truth is present in both viewpoints, but we are here in the twenty-first century and must understand and deal with all three aspects if we are to both educate and heal our patients, as well as ourselves.

Applied kinesiology (AK) uses the triad concept to explain the basics of balance in health. If the equilateral triangle whose sides are comprised of the structural, chemical, and mental aspects of health become imbalanced, either over or under, the entire organism suffers and eventually breaks down. Thankfully, Dr. Goodheart developed muscle testing and therapy localization so that we are able to "Diagnose the need, Supply the need, and Observe the results". We have been handed a tremendous legacy to assist us in treating our patients with the five factors approach of AK, thereby giving us a huge advantage over many other health professionals. If we fail to recognize the whole person appearing before us as such, we iatrogenically create an imbalance in her/his triad of health with a resultant state of disease or disease and a lack of homeostasis.

Conclusion

The triad of health EXPANDED can continue indefinitely. There are so many applications that can be included, I have only chosen a few. As you treat your patients, remember to address them as three-dimensional beings, living in a three-dimensional world. It is often so easy to overlook any one side of the triad of health and concentrate on that which we feel most secure or knowledgeable, but don't be misled … all three are essential for the miraculous results of which we all are capable. I hope this will inspire some of you to "get back to the basics" of chiropractic and applied kinesiology and realize the power which we, as chiropractors, literally hold in our hands.

To quote Dr. Goodheart:

> "Man possesses a potential for recovery through the innate intelligence of the human structure. This recovery potential with which he is endowed merely waits for your hand, your heart, and your mind to bring it to potential being and allow the recovery to take place, which is man's natural heritage. This benefits man, it benefits you, and it benefits our profession. Do it, do it with knowledge, do it with physiologic facts, do it with predictable certainty, do it because it has to be done, and we as a profession are the only ones who can do it effectively."

References

1. The American Heritage Dictionary of the English Language, Third Edition, Houghton Mifflin Company, 1996.
2. The Original Roget's Thesaurus of English Words and Phrases, Longman Group UK Limited, 1994.
3. Walther, David, Applied Kinesiology Synopsis, Second Edition, Systems DC, Pueblo, CO, 1988-2000.
4. Goodheart, George, Collected Published Articles and Reprints, Self published, Grosse Pointe Woods, MI, 1992.
5. Palmer College of Chiropractic Catalog, Palmer College, Davenport, IA, 1999.
6. Stephenson, Ralph W., Chiropractic Textbook, Palmer School of Chiropractic, Davenport, IA, 1948.
7. Walker, Scott, NET Certification Seminar Manual, N. E. T., Inc., Encinitas, CA, 1996.
8. MacLean, Paul, The Triune Brain in Evolution, Plenum Press, 1990.

Division II

Critical Review

Criteria for Accurate Manual Muscle Testing as used in Applied Kinesiology Practice

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Abstract

This is a proposal of a possible description of the manual muscle testing methods used in the practice of applied kinesiology that attempts to satisfy all of the criteria considered important by the practitioners of this art and science and who have published information on this topic. Please understand that this is open for revision with appropriate referenced suggestions.

Introduction

There are a number of descriptions of the basic manual muscle test in applied kinesiology. To get a description with a consensus would be have to be a description that lists all factors important to the professionals utilizing the technique. A descriptive test would appear to be the best solution. We should likely start with the two basic types of testing found in the neurology text by Chusid and Macdonald[1] 1967, and Chusid[2] 1985. The description is as follows: "Two techniques of testing may be used: active motion against the examiner's resistance and resistance against a movement performed by the examiner."

I will therefore call them:
Examiner Started Manual Muscle Test – EsMMT
Patient Started Manual Muscle Test – PsMMT

There are certain conditions considered important to these types of testing.

A. Patient conditions

A_1. The patient should maintain normal respiration unless he or she is asked to hold a phase of respiration by the examiner.

A_2. The patient should be instructed to leave the teeth slightly separated with the temporomandibular muscles in a relaxed state unless specifically asked to contract them in a certain way as during a challenge test.

B. Examiner conditions

B_1. The examiner should be consistent in his or her testing procedures when the test is repeated on the same or other patients.
The testing pressure should be with a soft contact not causing pain at the contact or stabilization point.

B_2. The examiner should place the patient's body, or parts thereof to be tested in a position that maximally isolates the muscle to be tested.The examiner should demonstrate the tangent of the movement through which the patient is expected to move the body part so that the patient understands what is expected during the test.

The other type, i.e. Schmitt's G-2 Submax test,[4] is included as an addendum to the PsMMT. This is referred to as PsMMTsm.

Discussion

EsMMT *(Also known in the AK literature as Type 1, or G-1 Testing).*[4][5][6][7][10][13][15][17][22][23][31][32]
This is a muscle test in which the examiner directs the patient to place the body or its parts in a position that isolates the muscle to be tested from its synergistic muscles to the greatest degree possible. The patient is then directed to hold the body part in that position without changing the position in any way. While the patient is holding the body part in that position, the examiner directs a gradually increasing force against the body part in a direction that lengthens the muscle separating origin from insertion or insertion from origin as is practical for the test. During this test the examiner senses the patient's ability to resist the test with a firm locking sensation. The examiner then increases the testing force slightly to determine if the locking sensation maintains, and if so the muscle test is considered as facilitated and or strong. If the patient's resistance cannot meet the examiner's increasing force and the tested muscle lengthens while the patient is actively contracting the tested muscle, (eccentric contraction), the muscle is considered Functionally Inhibited or Pathologically Weak according to additional criteria (see below). There is one additional condition that is considered important to this type of testing; the patient should resist the examiner's force with full effort.

With EsMMT, the examiner should gradually increase his or her testing force without the test duration exceeding 3 seconds. In most cases the test should be complete in 2 seconds or less to prevent a fatigue factor from influencing the test. The test should not be less than 1 second as there may not be enough time for the patient to prepare his musculature to resist the testing pressure.

PsMMT *(Also known in the AK literature as Type 2, or G-2 testing).*[5][6][7][8][10][11][12][13][15][22][23][25][26][28][31][32]
This is a muscle test in which the examiner directs the patient to place his or her body or its parts in a position that isolates the muscle to be tested from its synergistic muscles to the greatest degree possible. Contact is made by the examiner on the part to be tested in a manner, which is not painful to the patient. The patient is instructed to maximally contract the muscle in the vector that isolates the muscle, a vector that would approximate the origin and insertion or vice versa if the movement created was allowed to proceed without resistance. The examiner resists this increasing pressure, until the examiner can no longer sense an increase in pressure against his or her hand. At this point, the examiner adds an additional, slowly increasing vector of force that is directly opposite to the arc of motion of the body part that would be created by the muscle if no examiner resistance prevailed. If the patient's resistance cannot meet the examiner's increasing force and the tested muscle lengthens while the patient is actively contracting the tested muscle, (eccentric contraction), the muscle is considered to be Functionally Inhibited or Pathologically Weak according to additional criteria (see below). There are both patient and examiner conditions to be met for this test as listed below. There is one additional patient condition for PsMMT; the patient should exert his or her pressure against the examiner's stationary contact with as much force as he or she can muster in a controlled manner.

In PsMMT, the patients increase in force. The patient's increase in force should not exceed 1.5 seconds and the examiners counterforce should not exceed an additional 1.5 seconds.

PsMMT (addendum).
PsMMTsm (Patient started sub-maximum Manual Muscle Test) (Also known in the AK literature as Type 3, or G-2s).[3][7][9][5][22][31][32]
Schmitt describes a test that he associates with the withdrawal reflexes following injury, allergy and hypersensitivity reactions, systemic functional endocrine imbalances, and visual motor problems such as functional problems with accommodation reflexes. In this test the patient and doctor are positioned to do the muscle test as above and the doctor instructs the patient to push against his testing hand. As soon as force is felt from the patient, the doctor applies his testing force. This is a concentric to eccentric patient contraction. This we can name PsMMTsm (*Patient started Manual Muscle Test submaximum).* The timing of the examiner's pressure is the only difference in this test. All other factors related to the PsMMT listed in this document apply to this form of testing as well.

Functional muscle inhibitions found by the manual muscle tests used in AK testing are graded as 4 or 3 on the traditional six level scale. Grades 2 and 1 are observed phenomenon rather than an actual test as described above. When muscles are pathologically weak (see below) they may be rated anywhere from grade 0 to 4. The full six level scale is given below.

There are various criteria, regarding the current state of the tested muscle, which will be referred to as follows: Normally Facilitated Criteria (NFC), Hyperfacilitated Criteria (HFC), Functionally Inhibited Criteria (FIC) and Pathologically Weak Criteria (PWC).

Results of muscle testing**:** *(The traditional scale of muscle test grading can be applied to the result of any type of muscle test).*[5] [24] [25] [26] [27] [28]

5. Normal, or full motion with full resistance
4. Good, or full motion against gravity and some resistance
3. Fair, or full motion against gravity only
2. Poor, or full motion possible but only with gravity eliminated
1. Trace, or evidence of contractility but no motion
0. No contractility

Normally Facilitated Criteria (NFC) (*Also known in the AK literature as normotonic, normoreactive, strong, or intact muscles).*

- If the tested muscle in either an EsMMT or a PsMMT does not give way or in fact overpowers the examiner, this is referred to as normally facilitated, or hyperfacilitated according to further criteria.
- A muscle that is normally facilitated will demonstrate inhibition when one of the following procedures is used to influence the muscle. Therapy localization to the sedation point or tapping of the sedation point of the ipsilateral associated meridian
- Manually running the examiners hand along the meridian in reverse. Digital pressure approximating the muscle spindles in the belly of the muscle involved.
- Placing either of the two poles of a strong axially polarized magnet (minimum 2000 gauss), centrally placed on the belly of the muscle.

Hyperfacilitated Criteria (HFC) (*Also known in the AK literature as hypertonic, hyper-reactive, frozen, or hyper muscles).*[6] [11] [12] [13] [23]

- A muscle that when tested is not inhibited by the NFC above.
- If the tested muscle (either EsMTT or PsMTT) goes into eccentric contraction (lengthens while contracting), it is considered as either a Pathologically Weak or a Functionally Inhibited muscle, according to further criteria:

Pathologically Weak Criteria (PWC).

- A muscle is considered pathologically weak when its failure is due to pathological peripheral nerve injury, central nervous system injury or pathology, or local muscle injury.

The types of peripheral nerve injuries are as follows:[19] [30]

- Neurapraxia: Nerve injury (often times mechanical) of a minor degree involving only a temporary loss of conduction without loss of axon continuity. There is no Wallerian degeneration. The muscles affected by this will return to a facilitated strong state immediately upon the appropriate correction being made. This would cause a functional inhibition as described below under Functionally Inhibited Criteria.
- Axonotmesis: Nerve injury, which involves the axon and includes axonal disruption without loss of the neural connective tissue. This usually comes from stretch and crush injuries. This type of injury will experience Wallerian degeneration 12-48 hours post injury. In this type of injury, if the damaging condition is relieved a gradual return of function can be expected over a number of months as the nerves heal at a rate of 3-4 cm per month. This is considered a pathological injury.

- Neurotmesis: Nerve injury that is severe and involves loss of axon and neural connective tissue continuity. Severe crush, penetrating wounds, or rapid stretch/avulsion injuries cause it. Wallerian degeneration is present in these cases and they are surgical cases. This is considered a severe pathological injury.

Central Nervous System Problems.
Examples of central nervous system pathology or injury are multiple sclerosis, spinal cord injury from trauma, or cord compression by tumor or other space-occupying lesion.

Muscle Injury.
A muscle is also considered Pathologically Weak when it suffers from local pathology such as muscle strains of a severity to make the muscle unable to meet the demands of a manual muscle test. Such local pathology can include muscle tears, partial avulsions (not including microavulsions commonly treated with origin and insertion technique), and complete avulsions. Pathologically Weak muscles can be graded on the six level scale as grade 4 or lower (See the scale above)

Functionally Inhibited Criteria (FI-PsMMT-C or FI EsMTT-C) *(Also known in the AK literature as, functionally weak, weak, hypotonic, hyporeactive, conditionally inhibited, or non-intact muscle).*

- A muscle that cannot resist a manual muscle test, either EsMMT or PsMMT, without evidence of actual nerve interference of a pathological nature is considered to be Functionally Inhibited. *(Most muscles which cannot resist an AK muscle test are in the Functionally Inhibited category, either as a FI-EsMMT or a FI-PsMMT.)*
- Functionally inhibited muscles can be graded on the traditional six level scale as grade 4 and occasionally, grade 3. (See the scale above)

Important Additional Factors that may Influence the Accuracy of Any Manual Muscle Test:

- The patient's hands should be off his or her body so that random therapy localization is not done changing the test results.
- The patient's eye position during the test should be considered as this can influence the test. In some cases a challenge test can be with the eyes in a specific direction and can influence the test with either facilitation or inhibition.
- The examiner should observe the patient for the holding of a phase of respiration, which may influence the manual muscle test.
- The examiner should observe the patient for possible facial grimacing, clenching of the teeth or gum chewing, which may influence the manual muscle test. These actions should be avoided unless the examiner asks the patient to specifically contract certain stomatognathic muscles as a challenge test. *(If gum chewing is noticed, the patient should be advised to avoid gum chewing before appointments.)* If the above are noticed, the patient should be advised to let the temporomandibular muscles to relax with the teeth slightly apart.
- In some cases where postural indications of possible inhibition are not borne out by the testing, having the patient take the tongue away from the roof of the mouth may make an underlying inhibition evident.[18]
- Adequate stabilization is important to accurate manual muscle testing so that the tested muscle can function from a stable base.
- The examiner should not contact the meridian pulse points on the wrists of a patient during the test, unless he or she has verified that these points do not demonstrate therapy localization before proceeding with the test. *(Therapy localization is covered elsewhere in the applied kinesiology literature)*
- The body position of the patient during the test, such as side bending and torsion, can influence the test. At times, the examiner may find useful clinical information by comparing the results of muscle testing in a neutral position with the results of other positions, such as weight bearing, and or habitual, slouched, or work postures.

- The examiner must develop timing consistency so that repeated tests are consistent.
- Although it has been mentioned before in this document, the timing of the test has been found on objective studies to be very important.[20] Therefore, the test should not be of less duration than 1 second or more duration than 3 seconds. *Some researchers in Russia [38] on a small sample of 3 patients used prolonged muscle contraction increasing pressure every 3 seconds for a total of 9 seconds. They postulate different physiological mechanisms of support of prolonged muscle contraction. This is however a specialized case which does not represent the usual test done by the majority of those using manual muscle testing in applied kinesiology practice.*
- The evaluation of force produced by the patient is only one factor of accurate muscle testing. It is imperative that the examiner observes the changes the patient makes to change the parameters of the test. Changes the patient makes can be very subtle and easily missed, e.g. holding a phase of respiration. Often more information about the patient can be learned by these observations than from the actual muscle test.
- Any changes of parameters in the test have to be done in a controlled manner. This is called a diagnostic provocation or challenge. Structural, chemical, and or emotional challenges can be used. They may show a change in muscle function on a manual muscle test. These are described elsewhere in the applied kinesiology literature.
- Examiner prejudice should be avoided. Goodheart and others, have demonstrated that certain individuals are susceptible to the thought patterns of the examiner at the time of the manual muscle test. As this can influence the test findings, it is advised that the examiner keep his or her mind in a state of wonder as to what the outcome of the test will be during a manual muscle test rather than expecting the muscle to respond in a particular manner.
- Tonic labyrinthine reflexes, which relate to head position in relation to gravity, although not strong enough to cause a muscle to have a weak response to a manual muscle test, can have a facilitation or inhibition effect on the muscle being tested.[22] [29] [32]
- Dehydration in some cases is a very important consideration especially if the majority of muscles tested appear to demonstrate inhibition and or weakness. In these cases, having the patient drink water will cause facilitation of many of the inhibited muscles resulting in clinical findings that follow a more understandable pattern.
- Darkness or artificial light during the test has been found to influence a manual muscle test in certain patients.
- Medications may interfere with accurate manual muscle testing. Special procedures may be necessary in these cases to get an accurate MMT.
- Electrical, magnetic, or other energy sources such as laser light, homeopathic remedies etc., can influence manual muscle tests if the body or parts thereof are directly subjected to these energy sources.[12]

Other factors will in all probability be discovered that will increase this list and will likely be included at a future date.

According to Dr. Schmitt, all muscle tests are in eccentric contraction. The differences exist in what pre-load activity takes place prior to the eccentric contraction. The EsMMT has no pre-load, the PsMMT has maximum concentric to isometric pre-load and the PsMMTsm has submaximal concentric pre-load.[32] He also points out that the *use* of the terms "hypertonic" and "hypotonic" as used in some AK literature is *inherently incorrect* as these terms are defined differently in the medical literature.[32]

The Neurological Model of the Manual Muscle Test.[9] [10] [14] [16] [17] [21] [31] [32]
The current neurological explanation of a weak response to any muscle test is that the motor neuronal pool is biased too far towards hyperpolarization. This means that the central integrative state (CIS) of the alpha motorneurons (AMNs) of the involved muscle is inhibited. This state does not allow adequate muscle contraction resulting in the examiner's force taking the muscle into eccentric contraction (the muscle lengthening while still contracting).

A muscle is contractile tissue that is depolarized by an efferent signal from the motor neuron. Muscle testing is simply a test of the functional state or bias of the motor neuronal pool of the tested muscle. The weak response of the tested muscle should then be referred to as neurologically inhibited muscle. No matter what technique is used, whether it is acupuncture meridian stimulation, Chapman's reflex stimulation (also known as neurolymphatic technique), or osseous manipulation etc., if it returns the muscle to normal strength and function, it can be assumed that the technique has brought to anterior horn cells (both alpha and gamma motor neurons) associated with the muscle involved to a normal state of function.

Schmitt goes further to define a model for the various types of muscle testing EsMMT, PsMMT, and PsMMTsm. He associates the EsMMT weak response as being associated with local reflexes from mechanoreceptors mediated by the gamma motorneuron loop. He suggests that neurological factors affecting the dynamic (gamma 1 motorneuron system) originate at spinal levels.[8] He associates the PsMMT weak response as being also mediated by the gamma motorneuron loop but that it is associated with suprasegmental influences on these motorneurons.[8] He associates the PsMMTsm weak response with possible suprasegmental influences, which are significantly different from those influencing the PsMMT. For a full description refer to the ICAK-USA website.[17]

The concept of Deafferentation refers to a loss of afferent input to an area. This can result from various causes including pathology such as a cut nerve or dead nerve following a stroke, or joint dysfunction anywhere in the body etc. It is defined below as used in the treatment of any joint dysfunction as described in AK literature. As the definition below is limited to joint dysfunction, the term will be defined as Functional Deafferentation.

Functional Deafferentation.[19] [21]
This refers to reduced mechanoreceptor activity due to any joint dysfunction. This would include the spinal joint dysfunction (also known as the Chiropractic subluxation, or Osteopathic lesion). When the joint dysfunction is corrected, normal afferentation is restored resulting in immediate restoration of central facilitation and inhibition or the muscles affected.

The Physiological Model of the Manual Muscle Test.[11] [12] [13]
The connective tissue called "the ground system" according to Pischinger is the substrate of the "ground regulation" which comprises all neural, hormonal and humeral regulating mechanisms of the body. It is the histological and biochemical substrate of the regulation mechanism of the human body. There is a concept of electrochemical connection between capillary, ground substance, and cell, which was developed by Nordenstroem. A lesion or a mechanical stimulus, such as a structural challenge, will activate this system, as the electromagnetic potential of this area will become different from the surrounding tissue. It is felt that there is no neuromuscular interaction at all without participation of this Matrix System.[13]

Electrical stimulation or other energy stimulation can also influence the ground regulation system and thereby the neuronal pool referred to in the Neurological Model above.[12] This model appears to explain the influence of homeopathic treatments and other electromagnetic effects on the manual muscle test.

Muscle Strength and Power Versus the MMT in Applied Kinesiology.
The descriptions given above for the EsMMT and PsMMT are more of a test of a dynamic quality of neuromuscular response than a test of strength.[33] Schmitt has used the phrase "muscle testing as functional neurology".[8] There have been a number of attempts to correlate manual muscle test findings with muscle strength using various forms of dynamometers which have demonstrated poor correlation. Indeed, Blaich and Mendenhall in 1983 concluded that "The manual muscle test and the Cybex machine muscle test are probably independent phenomena, at least as the results are currently being measured".[34] Liesman et al. in 1995, referred to a number of studies that support the manual muscle test as used in applied kinesiology.[35] He made the following points:

a) Muscles identified as "weak" using applied kinesiology manual muscle testing methods are in a fundamentally different state than those identified as strong.

b) Muscles testing "weak" using AK are fundamentally different than muscles that are fatigued. In other words, "weakness" is not attributable to fatigue.

c) AK muscle testing procedures can be objectively evaluated via quantifying the Neurologic electrical characteristics of muscles.

d) The cause and effect of applied kinesiology treatment can be plotted over time objectively.

In a later study, Caruso and Leisman[36] determined the following:

a) Examiners with over five years of clinical experience using AK procedures were shown to have reliability and reproducibility when their outcomes were compared.

b) The perception of inhibition or facilitation was made in the initial pressure exerted by the examiner and this was corroborated by test pressure analysis using the instrumentation developed.

Guyton[37] gives the following descriptions relating to strength and power:

a) "The strength of a muscle is determined mainly by its size with a maximum contractile force between 3 and 4 kg/cm (squared) of muscle cross section area."

b) "The holding strength of muscles is about 40% greater than the contractile strength.

c) "The power of muscle contraction is different than muscle strength because power is a measure of the total amount of work that the muscle does in a unit of time. This is determined not only by the strength of contraction but also by its distance of contraction and the number of times that it contracts in one minute.

Conclusion

Baker[33] on terminology states the following: "You cannot evaluate a dynamic process (e.g., dF/dx, the rate of change of force with respect to displacement) with a single static measurement like that produced by a force transducer. You cannot measure the dynamic AK muscle test process without using either instrumentation that measures the dynamic nature of the process or a combination of instrumentation that allows you to calculate the dynamic characteristics of the process." As the contractile strength as defined by Guyton is a single value not descriptive of a dynamic process, the terms "strength," "strong," and "weak" do not accurately depict the dynamic nature of the AK muscle test and should not be used as such. He recommends alternative terms such as conditionally inhibited, conditionally facilitated and conditionally overfacilitated or facilitated, normal, and over-facilitated.

Commentary

It can be seen that there is much training necessary for an examiner to be an accurate - precise muscle tester. The EsMMT (Examiner Started Manual Muscle Test) is a very refined test, which requires much skill, accuracy and knowledge. It is a very sensitive test, which can be very useful and objective when used by a skilled examiner. However, because of the many influencing factors in this type of testing, it can become very subjective in the hands of an inexperienced and or biased examiner.

The Patient Started Manual Muscle Test PsMMT on the other hand does not require as much skill and can be easily done by an examiner with minimal training. Any studies of the manual muscle test that do not consider and apply criteria as listed above, without skill, accuracy and knowledge, cannot be considered as truly credible.

I wish to express my appreciation for the help and suggestions given me by both David Walther and Katharine Conable over the many revisions of this document. I would also like to thank Hans Garten and Walter Schmitt for their input.

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References

1. Chusid, J., McDonald, J., Correlative Neuroanatomy & Functional Neurology (New York, Lange Medical Publications, 1967).
2. Chusid, J., Correlative Neuroanatomy & Functional Neurology19th edition. (Los, Altos, CA Lang Medical Publications. Published by Appleton-Century-Crofts, East Norwalk CT 1985).
3. Schmitt, Walter H., "Three Variations of Manual Muscle Testing" (ICAK-USA Website, 1998) Proceedings of ICAK-USA Boston 1995-1996.
4. Haldeman Scott, Principles and Practice of Chiropractic 2nd edition (Norwalk, Connecticut 1992) ISBN 0-8385-6360-0.
5. Walther, David S., Applied Kinesiology Synopsis 2nd Edition (Pueblo Colorado. Systems DC, 2000) ISBN 0-929721-03-9.
6. Gerz, Wolfgang, Lehrbuch der APPLIED KINESIOLOGY in der naturheilkundlichen Praxis. (Munchen, AKSE – Verlag, 1996) ISBN 3-00-000616-8.
7. Boehnke Hans W., "The Science and Art of Manual Muscle Testing," ICAK-UK Collected Papers 1998 Windsor.
8. Schmitt W.H., Jr., "Muscle Testing as Functional Neurology differentiating functional upper motorneuron and functional lower motorneuron problems" (Proceedings of Winter Meeting, ICAK Maui HI, 1985.
9. Schmitt W.H., Jr., "A Neurological Model For The Three Types of Muscle Testing" ICAK-USA Proceedings 1996-1997.
10. Belli, Richard, "Manual Muscle Testing And The Motor Neuron" ICAK-USA Website 1998.
11. Frost, Robert, Grundlagen der Applied Kinesiology (Kirchzarten bei Freiburg VAK Verlags GmbH, 1998) ISBN 3-932098-27-7.
12. Garten, Hans, "The Mechanisms of Muscle Test Reactions and Challenge in Applied Kinesiology: an Attempt to Explain the Test Phenomena. This was received by members of the IBS by email. Can't locate journal yet. (Townsend Letter No 161, December 1996).
13. Gerz, Wolfgang, "Quality Muscle Testing" (ICAK-USA Proceedings ICAK-USA Proceedings Volume 1, 1995-1996).
14. Allen, Michael, "Insurance Report Manual Muscle Testing" (Collected Papers of ICAK Winter Meeting 1984).
15. Lebowitz, Michael, Steele, Mark, Correcting Chronic Health Problems A Doctors Manual (Privately Published by the authors 1989).
16. Belli, Richard, March, Randall "Manual Muscle Testing and the Motor Neuron" (ICAK-USA Website neuropapers).
17. Schmitt, Walter H. Jr. "A Neurological Model for the 3 Types of Muscle Testing" (ICAK-USA Website neuropapers).
18. Sandweiss, Jay, (Jay is an osteopath and a martial arts master who uses AK and demonstrates it at The American Back Society Meetings) (AK seminar).
19. Schmitt, Walter H. Jr. (Session 1 Changes for the 100 hour course page 2 on Peripheral Nerve Entrapment Concept).
20. Nicholas J. A. et al., "Factors influencing manual muscle tests in physical therapy – the magnitude and duration of force applied" (Journal of Bone and Joint Surgery, Vol 60 A No 2).

21. Allen, Michael D., Brook, Robert T. “Deafferentation vs Pinched Nerves” (ICAK-USA Website neuropapers).

22. Maffetone, Philip., Compementary Sports Medicine (Human Kinetics Champaign IL 1999) ISBN 0-8801-869-5.

23. Utt, Richard, Deal, Sheldon., “The Seven Conditions of Muscle Balance” (ICAK Collected Papers Summer Meeting 1986).

24. Schafer, R.C., Chiropractic Management of Sports and Recreational Injuries (Baltimore, Williams & Wilkins 1986) ISBN 0-683-07583-7

25. Hoppenfeld, Stanley., Klinische Untersuchung der Wirbelsaule und der Extremitaten 2. Auflage (Stuttgart, Gustav Fischer Verlag 1992).

26. Hoppenfeld, Stanley., Physical Examination of the Spine and Extremities (Norwalk, Connecticut., Appleton & Lange 1976) ISBN 3-437-00673-8.

27. Kendall, Kendall, Wadsworth., Muscles Testing and Function second edition (Baltimore, Williams and Wilkins 1971) SBN 683-04574-1.

28. Janda, Vladimir., Manuelle Muskelfunktions-diagnostik (Berlin, Ulstein Mosby GmbH & Co. 1994) ISBN 3-86126-516-8.

29. Schmitt, Walter, Jr., “Tonic Labrinthine Reflexes In The Weight Bearing Position” (ICAK Collected Papers Summer Meeting 1986).

30. Hearon, Kevin., Advanced Principles Of Upper Extremity Adjusting (Forks WA Olympic Graphic Arts Inc. 1991).

31. Schmitt, W. H. , & Yanuck, S.F. Expanding the neurological examination using functional neurological assessment part II : neurologic basis of applied Kinesiology. (Intern J Neuroscience, 1999, 97, 77-108).

32. Schmitt, W. H. Personal Communication via email June 2001.

33. Baker, Donald, “Review of the AK Muscle Testing Process” (ICAK-USA Experimental Observations of Members of the ICAK Volume 1, 2002-2003).

34. Blaich, Robert, Mendenhall, Edward,. “Manual Muscle Testing And Cybex Machine Testing, A Search For A Correlation” (ICAK Collected Papers Winter Meeting 1983).

35. Leisman, Gl, et al. “Electromyographic Effects of Fatigue and Task Repetition on the Validity of Estimates of Strong and Weak Muscles in Applied Kinesiology Muscle Testing Procedures. (Perceptual and Motor Skills 1995; 80:963-977).

36. Caruso, B., Leismann, G. “A Force/Displacement Analysis of Muscle Testing in Applied Kinesiology” (Perceptual and Motor Skills 2000 91, 683-692).

37. Guyton Arthur, Hall John,. Textbook of Medical Physiology 9th edition (Philadelphia, Pennsylvania, W. B. Saunders Company 1996 ISBN 0-7216-5944-6).

38. Vasilyeva L. F., et al. “About Peculiarities of the Effect of Muscle Functional Weakness” (ICAK-USA Experimental Observations of Members of the ICAK Volume 1, 2001-2002).

The Sacral Distortion – Some Additional Observations

Hans Boehnke, D.C., DIBAK

Abstract

The sacral distortion as taught by Goodheart and described in Synopsis 2nd edition by Walther is a valuable and good technique which I have used to help many patients. As health and disease are all on a continuum, it is reasonable that there are cases, which are of a sacral distortion nature that are at a stage where the classic symptoms do not all show. This I have observed and will attempt to describe below.

Introduction

Goodheart in his 1983 manual[1] made reference to nuchal tension in sacral problems especially with regard to an inferior sacrum on one side. In the ICAK – U.S.A. Annual Meeting in 1996 the handout for Dr. Goodheart's lecture[2] elaborates on the sacral distortion as well being involved with a sacral misalignment (subluxation), which is often inferior and anterior on one side or both sides and sometimes-superior posterior on the contralateral side. He indicates that it stems from an old S.O.T. technique that Dr. DeJarnette used some time before. In a textbook on Sacro Occipital Technique published in 1952,[3] Dr. DeJarnette shows a diagram of an inferior sacrum with a description of it. In a book titled Scoliosis Manual by Mawhiney, an inferior and anterior sacrum is again described in its relationship to scoliosis.[4]

Since I have studied and used the technique on the sacral distortion described by Goodheart, it has proven to be a very helpful and useful technique, giving much relief to patients. As most of us have learned, disease or dysfunction do not just occur but that we are on a continuum between good and bad health and that intermittent stages of any condition are present before all the classical signs are there. I have noted a few observations with regard to diagnosing an inferior sacrum that I feel are beneficial and should be shared.

Discussion

The Sacral Distortion

The classical diagnostic signs according to Goodheart and Walther[5] are as follows when relating to an inferior sacrum.

- Unilateral or bilateral palpatory pain at the nuchal ligament.
- Lower buttock (gluteal fold) on the side of the inferior sacrum.
- Usually a skin crest fold under the scapula contralateral to the inferior sacrum unless the patient is thin.
- Therapy localization will be negative at the ipsilateral nuchal ligament or the ipsilateral sacrum when done individually unless some other lesion is present needing correction. If so it should be corrected before proceeding with the examination.
- Therapy localization will be positive when the ipsilateral nuchal ligament and sacrum ala are therapy localized simultaneously.

- Challenge testing is best done by a superior challenge on the lateral aspect of the sacral apex while keeping the ilium from moving superiorly by restraining its movement by the examiner or someone else.
- Leg length checked in the prone position by measurement at the inferior aspect of the medial malleoli will show a long leg on the side of the inferior sacrum unless there is either a bilateral inferior sacrum or an anatomical leg length difference.

The additional findings that I have noted over a period of time regarding this condition are as follows:

- If the doctor kneels behind the patient and contacts the sacral apex bilaterally and has the patient flex forward, the sacral apex on the side of the inferior sacrum will drop inferiorly.
- If the doctor kneels behind the patient and has the patient side bend to the right and to the left while holding a contact on the sacral apex bilaterally, he or she will note that the apex of the sacrum on the side of the inferior sacrum will not rise whereas in a normal case it would rise.
- If the classical signs of the inferior sacrum such as a lower buttock (gluteal fold) is not present in a case where the doctor suspects the he may find the inferior sacrum, the following will often bring out the postural finding of the lower buttock (gluteal fold). Have the patient side bend to the right and then to the left alternately for a few times with as clean a side bend as possible minimizing other movements and then have the patient stand erect normally. Often if the inferior sacrum is present, the observation of the inferior buttock will now be present.
- On forward flexion I have commonly seen the paraspinal muscles in the lower spine on the ipsilateral side of the inferior sacrum contract giving a hump like rise of the paraspinal area. This is similar to the screening test often done for scoliosis when one looks for the imbalance of the paraspinal tissues on forward flexion. Indeed in cases of scoliosis, I find that the sacrum is usually inferior on the side that the paraspinal muscles contract when the patient flexes forward.
- In some cases, especially if they do not have excess fat the usual skin crest fold under the scapula contralateral to the inferior sacrum as described by Walther, will not be present. I have found that the doctor can press into the soft tissues immediately under the 12th rib bilaterally in a medial (spinal) direction and feel the resistance of the tissues. He will find that the soft tissues on the contralateral side will allow a deeper penetration of the palpating hand toward the spine. The palpation should be gentle enough to not elicit pain.
- In cases where the usual therapy localization for the inferior sacrum is not found (that is the bilateral contact with one hand on the ipsilateral nuchal ligament and the other hand on the ipsilateral sacrum), just have the patient increase dural tension and retest. That is, have the patient flex their neck bringing the chin closer to the chest (usually done prone) and re do the therapy localization. The therapy localization will often become positive causing weakening of the indicator muscle. If the therapy localization, still is not positive, add slight inferior traction on the coccyx while the chin is toward the chest and maintain the inferior coccygeal traction while the therapy localization is done. The doctor, the patient, or support person may do the inferior traction to the coccyx.

Conclusion

The treatment of the inferior sacrum is still done as described by Goodheart and Walther and is not part of this paper. My object is to add to the diagnostic signs for the sacral distortion, when an inferior sacrum is present. It is my impression that not only is the nuchal ligament under tension when the sacrum is inferior, but the entire complex of ipsilateral paraspinal tissues, including the muscles and fascia are under some tension. I have found that after correction of the inferior sacrum, that not only is the tension and tenderness relieved in the ipsilateral nuchal ligament, but the ipsilateral paraspinal muscles do not contract on forward flexion of the spine, or at least do not contract as much as before the correction. It is as if the paraspinal tissues are trying to pull the inferior sacrum up when the patient flexes forward, because it will not easily rise, as it should when positioned normally.

References

1. Goodheart George, Applied Kinesiology 1983 Workshop Procedure Manual 19th edition. (Page 83).
2. Goodheart George, Handout notes for 1996 Annual Meeting “Further Forays into Healing through AK” (pages 21-38).
3. DeJarnette Major B. Sacro Occipital Technique Privately published by author 1952, P. 143.
4. Mawhiney R. Scoliosis Manual (fourth printing) Waukesha WI, Roberts Publishing Company 1984 P. 13.
5. Walther David S. Applied Kinesiology Synopsis 2nd edition (Pueblo, CO., Systems DC, 1988-2000 P. 78-80.

Anteriority Screening by Simultaneous Supine Middle and Lower Trapezius Testing, Refined

Richard M. Burger, D.C., C.C.T.P., DIBAK

Abstract

Use of the bilateral simultaneous supine middle and lower trapezius testing can be an effective screening test for anterior thoracic subluxations, and depending on the starting level of the test, can give a good indication of the level of lesion.

Key Words: Anterior Thoracic, Anteriorities, Middle and Lower Trapezius, Simultaneous Testing, Screening Test.

Introduction

Early in 1980 I was shown a test for screening of anterior thoracic subluxations by a fellow applied kinesiologist using a bilateral supine "arm-pull-down" test during one of our weekly workshop sessions held in the evenings at my office. I apologize for the fact that I cannot give the original source from which this doctor learned the technique and passed it on to me. While I do not take credit for the discovery of this method, I have refined the test and found these refinements to yield consistent and reliable results.

The test is performed with the patient supine, both arms extended vertically at 90° with full external rotation at the shoulders, elbows locked in extension and full supination of the forearms with the medial edges of the hands touching one another. The examiner applies the testing force in the caudal direction below the wrists in the direction of shoulder extension with a broad one-hand contact. A failure to lock is a screening indication for anterior thoracic subluxations. It appears that the test involves primarily the lower and middle trapezius muscles bilaterally as a group. If there are anteriorities, then the contraction of the middle and lower trapezius muscles pulls the segments further into flexion and anterioriority causing a positive test response due to a dynamic/static challenge.

I have used this test successfully ever since learning of it, however I noticed at times that it would fail to give a positive result in the presence of obvious anterior thoracic subluxations. In order to understand this, I tried testing at angles below the 90° vertical position and found that there was a good correlation between the angle at which the test failed and the spinal level(s) of the anterior thoracic subluxations.

Discussion

As stated above, the test is performed with the patient's arms in full external rotation at the shoulder, elbows locked in extension and the forearms in full supination with the medial edges of the hands together. In some large-chested patients it may not be possible to have the hands touch, so greater care is needed in observing the body-language of compensation. The test is begun at 90°, and if negative, the arms are brought down

approximately 15° and retested, and so on until they reach the lowest possible position for testing, at about 30° of flexion from the horizontal. It may be necessary to allow the arms to separate at the lower testing levels. If there is a positive test, meaning a failure from isotonic to isokinetic, then it is very likely there are anterior thoracic subluxations present. The lower the positive test, the lower in the thoracic spine will be the anteriorities. The body-language of compensation will be an attempt for the patient to flex the elbows, or to internally rotate the shoulder or pronate the forearm. Attention to these attempts and care in testing will yield good results.

The test can also be performed in the standing position, but extra care must be taken to stabilize the patient's torso in order to prevent trunk flexion as a compensatory response to hide a positive test since the patient will be more concerned with locking the shoulders.

Conclusion

In a busy practice, quick and easy screening tests can be very useful in allowing the doctor to "work smarter and not harder." This test itself has passed the test of time and been a very effective means for anterior thoracic screening, both before and after correction. False positives are rare, and if present, are most likely due to bilateral middle and lower trapezius weakness in the clear. The only false negative tests I have encountered were simply due to a need to adjust the starting test angle.

References

1. Walther, David, S., Applied Kinesiology Synopsis, Pueblo Colorado, Systems DC, 1988-2000.

Mandibular Torsion (Category I of the Jaw)

Richard M. Burger, D.C., C.C.T.P., DIBAK

Abstract

It has been found by this author that mandibular torsion may be responsible for recurring cranial fault patterns and their attendant symptoms and that correction of this pattern can yield lasting correction in sometimes difficult and challenging cases. The challenge and therapy localization of this condition are similar in nature to that found for a pelvic Category I.

Key Words: Jaw, TMJ, Category I, Jaw Torsion, Cranial Fault, Mandibular Torsion.

Introduction

Any doctor who has been treating patients with cranial faults for any length of time has come across resistant, recurrent or difficult cases that do not respond as expected or hoped for with resolution of symptoms and improved or restored function. In the treatment of these patients with jaw or head pain, or recurring cranial faults and their associated far-reaching effects, one may look to the dura, the pelvis, the feet, and anywhere in between to find the solution. These cases may be particularly frustrating to the doctor and the patient due to the less than desired for response.

In dealing with just such a case, I recalled that in making the correction for the Pineal Cranial Fault which was first described by Goodheart in 1979,[1] it was necessary to correct the mandible by spreading, in order to prevent it from re-compressing the sphenoid. It occurred to me, that if the mandible could be compressed medially and effect the cranium, then perhaps it could be torqued and have a similar detrimental cranial effect. The patient in question had unremitting and recurring trigeminal neuralgia in the V2 and V3 and occasionally in the V1 dermatomal areas which had not responded to usual cranial technique. I challenged the mandible for torque by pressing inferior on the left while simultaneously pressing superior on the right, causing a clockwise (my view) torsion around the menton. There was an immediate weakening of the indicator muscle. Following the "rebound challenge" rule of the cranium and axial skeleton, I attempted correction using firmer pressure with my gloved left thumb on the left lower molars pressing inferiorly and my right hand contact under the right ramus pressing superiorly, enhanced by inspiration, four or five times until I sensed resilience. Immediately, the intractable pain was gone, as were all of the previously identified and recurring cranial faults. Happily for the patient and me the correction was lasting. Subsequent to that initial observation, I have found the problem of mandibular torsion to be fairly common and to resolve a number of cranial faults.

Discussion

The challenge for this Mandibular Torsion fault is to press inferiorly on the bony ridge of the inferior aspect of the mandible, approximately 2.5 cm anterior to the angle of the mandible on one side, while simultaneously pressing superiorly from under the same landmark on the opposite side, using the pressure one would normally use for cranial challenge (0.5 lb. or less). It is important that the teeth not be clenched and that the jaw is in a relaxed state during challenge. A positive response is indicated by a change in the indicator or previously asso-

ciated weak muscle. So far, I have only observed the positive torsion challenge to be in a clockwise direction, even though I consistently screen for counter-clockwise as well if I do not find it in the clockwise direction. This may have to do with handedness, as my subjects to this point have all been right-handed. It may also have to do with the same common patterns of dural torsion which cause the Universal Cranial Fault to be predominantly one direction as well.

Therapy Localization will be positive with the patient's index fingers over both TMJs simultaneously, one on each side, and over only one side when done separately. The TL is also positive with one index finger over the positive side TMJ and the other index finger on top of the nail of that finger. Once again, the pattern has demonstrated only on the left in the patients I have found it in so far, but variation cannot be ruled out. This pattern is reminiscent of that found in the Category I fault of the pelvis, which is also due to torsion of the pelvic structure, and this is why I have referred to this pattern of mandibular torsion as Category I of the jaw.

Correction is achieved by using a gloved thumb over the mandibular molars on the side which challenged inferiorly, applying approximately three to four pounds of pressure inferiorly, while simultaneously applying similar pressure in a superior direction from under the opposite mandible with the intent to cause torsion around the menton. This is repeated three to five times on inspiration, or until resilience is palpable.

Conclusion

It has been well documented that malocclusion can be an important factor in cranial dysfunction, and dental equilibration has been used both successfully and unsuccessfully to attempt correction.[2] The technique discussed in this paper has been found useful for resolution of recurring cranial faults, particularly the rotational faults (internal and external frontals, temporal bulge, parietal decent and universal cranial faults).[3] Using the same logic applied with the need to separate the mandible in order to gain lasting correction for a compressed sphenoid (Pineal Cranial Fault), it has been found that torsion of the mandible must be corrected to gain lasting correction for the cranium. The challenge and therapy localization are similar in nature to that found for the Category I of the pelvis, and as Dr. Goodheart has been heard to say, "the jaw is the pelvis of the skull", I feel comfortable with the "Jaw Category I" reference and designation. I have also seen resolution of a Pelvic Category I after this procedure, even though no corrections were made to the pelvis. The body truly is an integrated structure and everything is connected to everything.

References

1. Goodheart G. 1979 Applied Kinesiology Manual. p. 13.
2. Walther D. Applied Kinesiology, Volume II, Head Neck and Jaw Pain and Dysfunction—The Stomatognathic System. Pueblo: Systems DC; 1983. p. 270-271.
3. Walther D. Applied Kinesiology, Advanced Approach in Chiropractic. Pueblo: Systems DC; 1976. p. 148.

Occiput Screening by Tongue Protrusion, Refined

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Abstract

Lateral tongue protrusion has a long history of use in applied kinesiology as a means of screening for lateral occiput subluxations. By refining the use of this test to include protrusion between full lateral and the midline, it can also indicate other degrees of occiput subluxation and correlates well with palpatory tenderness on the side of subluxation.

Key Words: Occiput Subluxation, Tongue Protrusion, Tongue Extension, Lateral Occiput, Inferior Occiput.

Introduction

The use of lateral tongue protrusion as a means of "lateral occiput" screening has been described early in the history of applied kinesiology[1, 2, 3, 4] and has been used by this author to good success in the evaluation and treatment of patients for over 23 years. However, in the early 1980s, I found that in some patients who had an obvious occiput subluxation which would respond to standard challenge and corrective procedures, the tongue protrusion screening test failed to elicit a positive response. In these patients, by having them change the angle of tongue protrusion, the expected positive response would be obtained. The angle of tongue protrusion correlated well with the degree of laterality of occiput tenderness and the point of optimum contact for correction of the subluxation.

Discussion

When an occiput subluxation is suspected, but does not show up with lateral tongue protrusion, have the patient point their tongue at your outstretched finger, which is held at an intermediate angle between the midline (0°) and full lateral protrusion (90°) on the side of suspected subluxation, while testing an indicator muscle (the pectoralis clavicular is convenient). Typically, the positive response in the indicator muscle will show up using approximate angles of 30°, 45°, or 60°.

Palpation in the suboccipital region will reveal a point or region of tenderness associated with the occiput subluxation. Walther has stated, "This is the optimum point of contact for correction."(1) I have found that there is a consistent correlation between the degree of laterality of the tender point and the degree of laterality of tongue protrusion which elicits a positive response in the indicator muscle test. In other words, the greater the angle of tongue protrusion from the midline, the further lateral the tender point will be, and vice versa. When the test is positive closer to the midline, the tenderness and hence the contact for correction, will also be closer to the midline. Therefore, the tongue protrusion-screening test can indicate not only a "lateral" occiput but an inferior occiput subluxation as well.

Conclusion

Quick and easy screening tests can be a big help in a busy practice, allowing the doctor to serve more patients more effectively in a shorter period of time. It should be pointed out that weakness on tongue protrusion is not pathognomonic for an occiput subluxation. When indicator muscle weakness is found on tongue protrusion in the absence of suboccipital tenderness or challenge, then other factors in the stomatognathic chain, such as hyoid or mandibular imbalance should be ruled out. It is wise to remember that all shortcuts have shortcomings.

References

1. Stoner F. The Eclectic Approach to Chiropractic. 2nd ed. Las Vegas: FLS Publishing Co; 1976. p. 57.
2. Walther D. Applied Kinesiology, Advanced Approach in Chiropractic. Pueblo: Systems DC; 1976. p. 30.
3. Walther D. Applied Kinesiology, Volume I, Basic Procedures and Muscle Testing. Pueblo: Systems DC; 1981. p. 62-63.
4. Walther D. Applied Kinesiology, Volume II, Head Neck and Jaw Pain and Dysfunction—The Stomatognathic System. Pueblo: Systems DC; 1983. p. 394-395.

Body Out of Distortion (BOD)

John M. Corneal, D.C., DIBAK

Abstract

Applied kinesiology, a discipline within Chiropractic, has been using aids like Body into Distortion (BID) and Eyes into Distortion (EID) to uncover hidden disturbances of body function. Both are used in conjunction with manual muscle testing. Body Out of Distortion (BOD) is a way of uncovering hidden problems using posture and patient verbal input.

Key Words: Applied Kinesiology, Manual Muscle Testing, Chiropractic, Posture

Introduction

Postural analysis is one of the diagnostic tools of applied kinesiology.[1] Poor posture is a reflection of disturbed body function and will change when functional anomalies are corrected, assuming no birth or traumatic defects are present. Sometimes the simplest Chiropractic correction makes a dramatic change in a patient's posture, while other times the key to the problem is evasive and multiple corrections yield little change. Using postural analysis as the criteria of patient response to treatment sometimes leads me to wonder whether I am making any progress. Out of desperation I moved a patient into the correct posture using the plumb line as a guide. Satisfied that they looked normal I asked them how they felt. Their feedback of how their body felt was instrumental in locating the evasive problem.

Discussion

Analyze the patient's postural distortion using postural analysis. Next, the doctor moves the patient to correct any postural distortion viewed both laterally and posterior to anterior. Instruct the patient not to move and ask them to describe what they feel. I often ask them where in their body their brain/thoughts are drawn while holding that position. Most times they will note an area of the body and you can ask for a description of the sensation, re: weak, tight, painful, etc. If they describe a sensation of being off balance I will ask them to locate the area of the body that feels strained or weak. In other words, if they say they feel like they are falling backward I ask if there is a sensation of strain or weakness anywhere. They may describe a feeling of strain in the quadricep muscle. Once the area of involvement is found proceed with standard diagnostic procedures to evaluate and treat the problem.

If you are at the end of your therapy, the objective indicators are resolved and the symptoms are abated, but the posture is still abnormal, using BOD may illicit no ill response from the patient. If this is the case it is an indication that the remaining postural distortion is not neurological but habitual and requires stretching, muscle strengthening, or just a little time for the body to correct the remaining postural imbalance.

Conclusion

BOD differs from BID and EID in that it requires no muscle test. Instead, it relies on the verbal input of the patient. BOD offers the practitioner a means to uncover distortions that may otherwise be difficult to find. It is another tool in understanding the simply complex, and complexly simple workings of the human body.

References

1. Walther D: Applied Kinesiology Synopsos (System DC, 1988).

Indirect Inguinal Hernia Non-Surgical Repair - A Case Study

John M. Corneal, D.C., DIBAK

Abstract

Inguinal hernia is one of the most common conditions treated by a general surgeon. The cost of medical treatment is in the billions of dollars per year, without factoring in lost worker productivity. Yet, very little is known about the causes of inguinal hernia in the adult population. Much has been written about the different surgical procedures, anesthesia, recurrence, and the like, but the literature is quiet in the area of cause or prevention. A case involving a middle-aged male with indirect inguinal hernia is presented. Evaluation and management involved a Complimentary and Alternative Medical (CAM) approach incorporating applied kinesiology, chiropractic therapy, and nutritional evaluation. In this case a nonsurgical approach resolved the hernia. Discussion examines some theories of cause, prevention, and alternative treatment of inguinal hernia.

Differential Diagnosis Using Applied Kinesiology Methods In Cases of Long-Term Head Pain: A Case Study

Scott C. Cuthbert, D.C.

Abstract

This case demonstrates the importance of developing accurate differential diagnostic thinking and skills in order to solve the problems producing long-standing head pain. The most common error in chiropractic diagnosis of head pain is an over focus on a favorite hypothesis. This of course is an inherent limitation of accurate diagnostic recognition – that is, when you try to put things and people into discrete categories, the categories themselves become the focus of your attention and it is difficult to see any patterns outside those categories. Care is needed to avoid a preoccupation with one diagnosis, one structure or one system at the expense of the others, as this will be reflected in the management of the patient.

This patient's case was selected to outline the principles of physical examination and treatment using the full range of specific differential diagnostic methods available in applied kinesiology. For reasons of clarity, an abbreviated description of the main information is presented.

Introduction

In March of 2001, a 56-year-old mental hospital nurse presented with constant, worsening headaches for the past seven years after several severe automobile accidents.

Materials and Methods:

Presenting Symptoms

Constant, daily headaches for the past seven years that have been increasing in severity. The left side of the head hurts the worst, with a constant grinding ache in the suboccipital, retro-orbital, and temple areas bilaterally. Low back pain with sciatic neuralgia in the right leg for the same period of time. The headaches produce stomach sickness. She has TMJ problems also, and numerous mouthpieces have been fitted for her but they haven't helped. She has muscle weakness throughout her body. On the visual analog scale she rates her low back pain as 5/10 and headache as 7/10 at the time of her first appointment, but the headache pain is frequently 10/10. Frequency of headaches was marked at 10/10.

Onset

This patient has had daily headaches since an auto accident in February 1995, and they worsened after a second auto accident in February 1999. The first accident in 1995 occurred because the patient, a nurse and a Good Samaritan, stopped her car to help a driver who had pulled off the freeway holding his head. As she was unbuckling her seatbelt to get out of her car on the shoulder of the freeway her car was struck from behind, knocking it forward 60 to 70 feet and back onto the road. She heard a loud popping in her spine that made her think she had broken her neck. She heard squealing tires and smelled smoke and was afraid her car might

catch fire. Although dazed she got out of her car, and was later told by a friend who witnessed the accident that she was wandering about on the highway in an aimless manner. She remembers walking around looking for her hat. She was not taken to the hospital; a police officer called for someone to take her home. She was off work for 7 1/2 months after this accident. For much of that time she was unable to lift her head off the pillow. Unfortunately, this was only one of five automobile accidents the patient has endured.

Previous History

She takes Synthroid and has low energy. There is a history of thyroid disease in her family. She has difficulty swallowing. She has occasional diarrhea since her gallbladder was removed two years ago. Cataract surgery in 1994. She takes Effexor for chronic fatigue syndrome. Several D & Cs have been performed. The patient comes from an illustrious family of chiropractors (even bonesetters are in her family history, from the era before 1895), but she had found that HVLA (high velocity/low amplitude) corrections to her cervical spine since the auto accident of 1995 were unbearable for her any longer.

On Examination

This woman had a variety of mechanical restrictions throughout her body. There was dramatic weakness on manual muscle testing, with hypertonicity in other postural muscles.

The patient had an excessive overbite (a class II malocclusion), as well as a narrow maxilla compared to her mandible. Pain was acute in the left cervical facet joints to palpation and with all ROMs tested, and the Soto-Hall test was positive for cervicodorsal junction pain. The deep neck flexor and SCM muscles, as well as the cervical extensors when tested prone and seated, were inhibited bilaterally. Pincer palpation of the rectus capitus posterior minor muscle was positive, suggesting a possible myofascial-dural connection in this area.[1, 2] Positive therapy localization to the left TMJ was elicited with clenching of the teeth. Gluteus maximus and hamstrings were weak bilaterally. The rectus abdominus muscles were weak also. The shock absorber test was positive on the right foot, indicating problems in the gait mechanisms and the positive support system.

A category II pelvic fault was present. Yeoman's flexed knee and hip extension tests were positive for pain in the lumbosacral and sacroiliac areas. Ely's knee flexion and hip extension caused pain in the left lumbosacral and sacroiliac area, as did the Hibbs' test.

Assessment of visual, proprioceptive, vestibular system and cerebellar function and integration indicated neurologic disorganization in these areas. Finger-to-finger testing was inexact, near-pointing was inexact, and the ocular lock test of applied kinesiology showed disorganization in the use of the extraocular eye muscles, indicating neurologic disorganization of cranial nerves III, IV, and VI. Saccadic motion was evident during movement of the eyes through the cardinal fields of gaze.

Freeman Wycke's one-leg standing test was also very imbalanced on initial examination, and TL to the cervical spine immediately improved her balance. (Patients admire and learn from this test! Older patients especially appreciate this demonstrable, very evident change in their balance before and after treatment. It is an excellent evaluation tool, and shows how much you can do to immediately change such a critical body function.) The patient is asked to stand upon one foot in front of the doctor, to find their balance if they can, and then to close their eyes. If they lose their balance, the test is positive. The eyes are closed in order to eliminate the effect of the optical righting reflex. Upon asking the patient to therapy localize to the cervical spine, they may immediately improve their balance. That occurred in this patient's examination.

There is evidence that a stable equilibrium comes significantly from proper cervical mechanoreceptor function. Frequently after the proper cervical or cranial correction or a category correction to the pelvis, the Freeman Wycke one-leg standing test is passed, with eyes open or closed, which persuades both the patient and the doctor of the effect of your efforts. TL to the cervical spine improves the stability of the cervical mechano-receptors during the Freeman Wycke testing, and may account for the improvement in the patient's balance while

standing on one foot with their eyes closed. Many times a foot subluxation may be producing the imbalance during this test. Structural corrections to areas of dural attachment create the most immediate and permanent changes in the Freeman Wycke test in our experience.

Tests of standing balance should not be the sole criterion for evaluation of degree of integration of postural mechanisms. There is a fairly high natural variability in standing balance (especially among young children and older adults), and most patients have a large amount of practice balancing in the biped position.[3, 4]

Treatment

Treatment started in March 2001 with an attempt to correct the causes of the muscular and proprioceptive deficits throughout the body. Gentle cranial corrections (discovered by challenge) were performed to return proper neurologic control to the muscles of the neck and jaw that tested weak. Right inspiration and left expiration assist cranial faults (producing temporal bone rotation) strengthened the SCM and deep neck flexor muscles to normal function. This strengthening indicates improved motor function of cranial nerve XI. These corrections eliminated the positive ocular lock, and changed the TMJ finding from clenching weakness to sagittal opening weakness. Weakening of indicator muscles also occurred with swallowing, and muscle spindle cell treatment to the jaw opening and hyoid muscles corrected these findings. Sagittal suture separation strengthened the abdominal muscles to normal function. A slight weakening of the left SCM occurred with respiratory challenge, and temporal bone mobilization in sync with respiration abolished this finding. Release of these cranial faults eased a lot of the cervical muscle tension and made all the cervical manipulations very easy. The patient remarked that she had not had her cervical spine adjusted with such ease in years.

A category II pelvic fault was found by challenge and corrected. A left posterior occiput correction strengthened the gluteus medius and psoas muscles bilaterally, and improved the cervical facet joint tension on the left from C1 through C6. Bilateral deltoid weakness was strengthened with correction of T1-T2. However, an upper cervical fixation correction was still required to strengthen the gluteus maximus muscles, which were weak bilaterally.

Treatment of the foot was accomplished with HVLA thrusts, and these corrections abolished the shock absorber test and all directions of challenge to the foot.

Ocular lock, finger-to-finger, near-pointing, and Freeman Wycke testing were now negative. Provocative tests at the sacroiliac articulations were now negative. This indicated that the causative factors for a significant portion of the cerebellar, equilibrium, proprioceptive, vestibular, and visual dysfunction had been found and corrected using applied kinesiology structural methods. Cranial corrections not only improve the dural tension upon the cranial nerves to the eye, but also strengthen the cervical flexor and extensor muscles. The neck muscles are closely associated with the medial longitudinal fasciculus and through it the cranial nerve nuclei that control oculomotor functions.[5-8]

Results

The patient said at the end of her first treatment that her headache was gone…the first time she had felt like that in over seven years. Over the next two weeks the headaches stayed at a 1 or 2 level, in the retro-orbital area only. After eight visits all of her symptomatology was gone. She and her large family have remained under care at one visit a month for the past 2 1/2 years.

Discussion

Intervention procedures derived from applied kinesiology neurological disorganization theory are hypothesized to effect change in the nervous system. This is based on the assumption of applied kinesiology chiropractic that there is *plasticity within the CNS.* Plasticity refers to the ability of brain, spinal cord, and other neural structures to change or to be modified in their function. This assumption, which is central to the theoretical

basis of neurological disorganization intervention procedures, makes it feasible to speculate that enhancement of the function of the nervous system is possible through the restoration of proper ocular, joint and muscle receptor, vestibular, tactile, and proprioceptive inputs.

The cranial nerves, together with the cranial connective tissue, operate as a continuum and are powerfully linked to vital organs (eyes, ears, cerebrum, cerebellum, brainstem) and pain-sensitive structures (e.g., cranial nerves and dura mater), and injury or impaired physiology or motion to these structures can make cranial nerve tissue dysfunctional. One of the more important principles is that any major neural structure receiving sensory input from many areas is also apt to have widespread influence over the rest of the brain. This is why cranial nerve corrections may have such widespread influence on the nervous system. Multiplicity of input usually means convergence of input. The brainstem and midbrain (where the cranial nerve nuclei are located) are good examples of structures to which the principle of convergence is applicable. The nonspecific systems in these areas receive sensory input from many sources (visual, vestibular, proprioceptive, and mechanoreceptors, among others) and in turn have a widespread influence over organization of nervous system function by the rest of the brain.

Conclusion

This case was interesting because it demonstrated that mechanical faults, especially when present for long periods, can disturb proprioceptive signaling from the eyes, the cervical spine, and the vestibular mechanism. When proprioception from the muscles, joints, ligaments, and other soft tissues is impaired for a long time, the overall tone of the nervous system may be reduced. This creates weaknesses in both the anterior and posterior muscles of the body, and perpetuates pain.

Dural sensitivity is another significant contributor to head and neck pain.[9] A neuroanatomical link between the cervical spine and cranium is formed by the trigeminal nucleus (often referred to as the trigeminocervical nucleus), and is of particular significance when considering possible mechanisms of pain in the region of the head and neck. Returning the dura to a physiological range of tension by using specifically applied cranial corrections is a major goal of AK treatment.[5, 10] AK cranial evaluation and treatment seeks to achieve zero defects both within and without the cranium. In 1991, Goodheart stated, “We, as a profession, are divided by the dura and its attachments. We, as a profession, can be united by an understanding of the dura and its attachments.”[11]

The previous treatments (extensive physical therapy and chiropractic) did not deal directly with the craniosacral system, nor evaluate all of the sensory input into the nervous system to discover persisting neurologic disorganization in the patient following her injuries. This case serves to show that examination must include all structures and not just those that are simply the most easily accessible. It also shows the relationship between structural factors and proprioceptive and sensory tests that help to identify neurologic disorganization. The complex links between different body parts involved with daily living are coordinated and integrated and may explain why dysfunction in one part of the body may lead to dysfunction in another part.

AK manual muscle testing procedures take great care to evaluate for these interactive factors in any patient’s condition. This point is emphasized in this case report because applied kinesiology theory believes that to resolve biomechanical, locomotor, postural and sensory problems, many areas of the body must be examined and corrected in order to achieve long-lasting symptomatic relief. Our global view of integrated biomechanics and neurophysiology is one of the major differences between chiropractic (and especially applied kinesiology) practice and that of many of the other manipulative and medical professions.

References

1. Kenin S, Humphreys BK, Hubbard B, Cramer GD, "Attachments from the Spinal Dura to the Ligamentum Nuchae: Incidence, MRI Appearance, and Strength of Attachment" *Proceedings of the 2000 International Conference of Spinal Manipulation* Sep 2000: 202-4.
2. Hack, GD, Koritzer RT, Robinson WL, Hallgren RC, Greenman PE, "Anatomical relation between the rectus capitis posterior minor muscle and the dura mater" *Spine* Dec 1995; 20(23): 2484-2486.
3. Goodheart, G. J. *Applied Kinesiology 1997/98 Workshop Procedural Manual* (Privately published, 1998).
4. Ayres, A.J. *Sensory Integration and Praxis Tests*. (Los Angeles: Western Psychological Services, 1989).
5. Walther DS, *Applied Kinesiology Vol. II, Head, Neck, and Jaw Pain and Dysfunction—The Stomatognathic System.* (Pueblo, CO, 81004: Systems D.C., 1983).
6. Blum, CL, "Cranial Therapeutic Approach to Cranial Nerve Entrapment Part I: Cranial Nerves III, IV, and VI" *ACA Journal of Chiropractic Jul* 1988;22(7): 63-7.
7. Upledger J.E., *Craniosacral Therapy II: Beyond the Dura* (Seattle, Washington, Eastland Press, 1987) pg.115-30.
8. Crisera, P. *Cranio Sacral Energetics, Vol. 1*. (Rome, Italy: Privately published, 1997).
9. Butler, DS. *Mobilisation of the Nervous System*. (Churchill-Livingstone, 1991).
10. Goodheart, G.J. *Applied Kinesiology Workshop Procedure Manuals*. (Detroit, MI: Privately published, 1982).
11. Goodheart, GJ. *Being a Family Doctor*, videotaped seminar, 1991.

The Anterior-Inferior Sacrum: Sutherland's Depressed Sacrum Revisited

Scott C. Cuthbert, D.C.

Abstract

There is a sacral subluxation that I am finding repeatedly that seems to have greater repercussions on the patient than many others we correct every day.

Introduction

I found this subluxation described in the writings of Dr. William G. Sutherland. My purpose here is to describe this sacral situation in light of his writings, and my findings using applied kinesiology diagnostic and treatment methods.

In these cases the patient frequently describes a traumatic incident that injured his sacrum, pelvis, or low back. A fall on one or both feet for example, or a fall on the buttocks is common. Dr. Sutherland points out that the subluxation might occur during delivery when the pelvic diameter is increased and the ligaments of the pelvis are relaxed. The displacement of the sacrum downward and anterior follows the mother's exertions to expel the child during labor with maximal expiration while bearing down, against resistance, with her knees or feet spread apart in the lithotomy position. Traction applied to the baby's head by the obstetrician during delivery, without adequately following the natural curve of the pelvis with its anterior concavity, can strain the base of the mother's sacrum anteriorly and inferiorly while the ilia are maintained in a posterior position by the fixed flexion of the hips in the stirrups. A number of female patients have related that their lumbosacral pain began after delivery of a child. With the legs externally rotated, abducted, and flexed, a strain on the sacroiliac and lumbosacral ligamentous apparatus may occur. If these subluxations are not corrected, subsequent births may be much more painful for the mother.

Discussion

A charming anecdote given by Dr. Sutherland in the 1950s that explains his mechanical insight into the "sacral sag" problem may be of interest.

> "I had an experience in the days when I drove out into the country with a little team of horses and a buggy. Automobiles could not travel on those roads in mud season because the blue clay would roll up onto the wheels. The horses and the buggy were more reliable. One day, however, the axle on the buggy broke when I was on the way to a house call. It was only 25 miles, but it took me quite a while to get there, riding one horse and leading the other.
>
> Along the way I found my patient coming to meet me. I found her in a mentally-strained, distraught state following the birth of her child. After helping her onto the horse I was leading, we finally reached her house. By the time we did arrive, her disturbed state had disappeared, and she was her usual calm self.

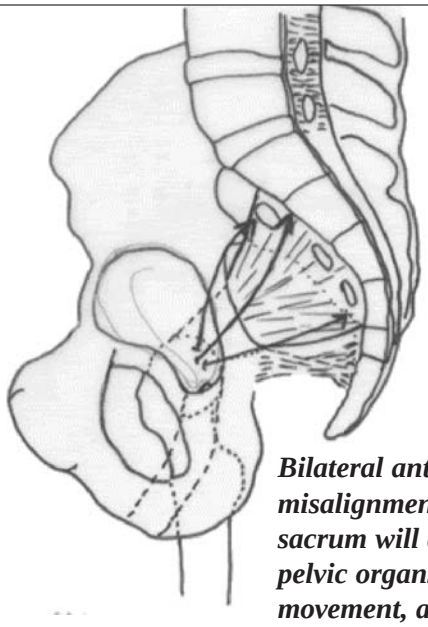
After examining her and thinking about her history, I concluded that her sacrum had sagged following delivery and thus created a membranous articular strain in the cranium that especially locked the cerebellum down upon the brain stem, the fourth ventricle, and the cisterna magna in the posterior cranial fossa. But what accounted for the change after riding horseback?

It seemed that the sides of the horse held the femurs laterally so as to provide a traction on the pelvic ligamentous articular mechanism. Then, with the movement of the walking horse, the sacroiliac ligaments could allow the sacrum to become reseated and functional, thus fluctuating the cerebrospinal fluid in the primary respiratory mechanism and relieving the reciprocal tension membrane so that the fulcrum could shift."[1]

The common injury of bending forward and lifting a heavy object or bending forward for a long time and suddenly coming up, creating the strain-counterstrain problem in the psoas, gluteus maximus, or piriformis muscles, may also account for this type of sacral fault when the muscle injury occurs bilaterally.

My experience confirms Sutherland's that this subluxation produces serious consequences. The sacral displacement is not always extremely painful in itself, but its effect on the entire spine, muscles and metabolism of the body can be profound. Dr. Sutherland states that there is a very interesting relationship between this sacral fault and the psyche, with the appearance of neurosis and psychological disturbances. In post-partum women, he attributes the post-partum depressions frequently seen to this problem of craniosacral mechanics. He calls this depressed sacrum a "sacral sag" producing "fascial drag," and postulates that it produces an alteration in the dural membranes at the base of the cranium and within the cranial membranes. The pull of the spinal dura mater upon the falx and tentorium produces this cranial stress.

Any sacral subluxation produces dural tension, but this one is especially profound. Dural tension results in occipital and upper cervical muscle dysfunction. This dysfunction seems to powerfully misalign the occiput in relationship to the atlas. All the dural structures at the base of the skull are under extreme tension in this situation. Correction of this sacral depression is essential for primary respiration to be normal, for dural tension to be equal, and ventricular function to be normal. Once this subluxation is corrected, many post-partum depression cases are treated very successfully. In infancy, this traumatic sacral subluxation may be quite painless and asymptomatic.



Bilateral anterior misalignment of the sacrum will disturb the pelvic organs' position, movement, and tone.

This sacral subluxation may have a particularly detrimental effect upon an expectant mother and her developing fetus. Prominence of the sacral base in an anterior inferior maternal sacrum may obstruct the descent of the fetal head on one or both sides, and this asynclitism (if the child can be born vaginally under these conditions) may distort the child's cranial mechanism. Any structural fault of the mother's pelvis may cause the fetus to assume a degree of extension or lateral cervical flexion greater than the ideal. This may result in the fetus presenting a portion of the head greater than the minimum occipitobregmatic diameter of the mother. This may create a presentation of the posterior occiput, a transverse arrest, a brow presentation, or a complete extension of the fetal head making a vaginal delivery impossible. These presentations may make cesarean section necessary if the baby is to survive.

The difference between the "depressed sacrum" and the category I or II sacrum is that the axis of sacral motion at S2-S3 has dropped anterior and inferior on both sides.

- It is a very rigid anterior sacrum, with the entire sacrum being forward. The sacrum is lowered en masse, with the exception of its apex. This fault lowers the axis of rotation of the sacroiliac auricular surfaces, or lowers the sliding "boot" mechanism of the craniosacral propulsion system bilaterally. This is called a sacral inspiration assist fault, or what is called in S.O.T. the sacral base in extension or SB+. This one, however, has both sides of the sacrum forward and depressed, with very little motion present. This sacral fault will directly affect the gluteus maximus, gluteus medius, hamstring, piriformis, perineal, psoas and iliacus, obturator internus, gemelli, levator ani and coccygeus muscles.

The sacrum sits mechanically like this:

- The sacroiliac sulci are situated deep between the wings of the ilia.
- This is a definitive sign, and easily seen. On obese individuals (who frequently have this type of sacral fault) this can be seen but is more easily felt.
- L5 is in an anterior position.
- Lumbar lordosis is increased.
- The iliolumbar ligaments are in extreme bilateral tension.
- Overall body mobility will be much reduced; stiffness will be a large part of the clinical picture in these patients.
- Sensory integration disorders and neurologic disorganization of many types will be present in these patients, and so applied kinesiology challenge methods will be more revealing to the doctor than therapy localization in many instances.
- Myofascial gelosis is frequently present in the lumbo-pelvic and occipito-cervical areas. Any portion of the "superficial back line," elegantly described by Thomas Myers, may be compensating for this sacral fault.[2]
- The ligamentum nuchae is very tense, dense, painful, and ropy.
- Only the sacral apex is prominent – posteriorly.
- The sacrosciatic notch is very tight bilaterally.
- The posterior-superior iliac spines are close together to the median line of the pelvis.
- The total mobility of the sacrum is reduced, with a little more latitude however in anterior rotation than in posterior rotation.
- Upledger has found a similar type of lumbo-pelvic "compression" associated with "endogenous depression."[3] This correlated in his examinations with sphenobasilar compression of the cranium. However many patients with chronic dural tension from many different sources may demonstrate the spectrum of symptoms in "endogenous depression."

Cranial faults associated with the anterior inferior sacrum:

- The sacral subluxation, through its connection with the meninges, will dissipate its tension up through the cranial and spinal membranes, producing most typically a compensation or a superimposed sphenobasilar subluxation in extension.
- The basilar surfaces of the occipital and sphenoid bones will be lowered, with innumerable consequences upon the cervicocranial area.
- The pituitary gland will be lowered.
- Hormonal and gynecological disorders may be present (pituitary-ovarian axis).

Dural tension in the sacrum may be transmitted to the cranium. Pelvic tension is thereby mechanically related to cranial respiratory function.

- With sacral respiratory faults, the tone and angulation of the pelvic floor is changed. If sacroiliac breathing movement is restricted, the continual massage of the pelvic fascias, which occurs with sacral respiratory motion, may not take place, thereby allowing venous and lymphatic congestion to occur in the pelvis. Neither lymph nor blood should be allowed to pool or stagnate anywhere in the body.
- An internal rotation of peripheral cranial bones (parietal, temporal, frontal, maxillary, palatine) will accompany the extension of the midline cranial bones.
- Careful examination for cranial nerve dysfunction and disturbances of the senses of taste and smell may be found.
- The central line of gravity of the body and neuraxis will be changed.

AK testing reveals:

- Lindner's added to Bechterew's test may be positive producing lumbo-sacral pain, as will Valsalva's test when added to these.
- Cervical and lumbar spine range of motion is frequently limited in rotation, flexion and extension.
- PiLUS testing will usually be positive.
- An expiration fault of the sacrum is usually present.
- Discovery of respiratory faults of the sacrum in these cases will be improved by adding a vector of challenge pressure during the respiratory challenge. Press firmly forward and headward on the sacral base during an expiration challenge to the sacrum, and press forward and caudalward during an inspiration challenge.
- The inferior sacral base challenge will be positive bilaterally. Challenge pressure should be forceful to identify this fault.
- Leg-length inequality may be minimal.
- Spondylogenic reflexes on the sacrotuberous ligaments are usually active; C1-T4 are especially responsive to treatment of the sacrotuberous and sacrospinous ligaments in these patients.[4]
- Brachial problems may be present.
- Tension around the thoracic inlet and shoulders may be prominent.
- The ischial tuberosities will often have painful nodules on them from the original trauma to the sacrum, affecting the sacrotuberous and sacrospinous ligaments as well as the hamstrings, which have a continuity of myofascial fibers with these ligaments.[2]
- Category I, II, or III may be superimposed upon this fault.
- Iliolumbar ligaments may need treatment.
- Pincer palpation (myofascial gelosis) may be present at the sacrococcygeal, lumbosacral, and cervicothoracic junctions bilaterally, and these should respond to the percussor instrument.
- The piriformis muscle originates from the anterior surface of the sacrum by fleshy digitations between the 1st to 4th anterior sacral foramina. The muscle will frequently be dysfunctional with this sacral fault. Definite pain, faltering, or weakness on resisted lateral hip rotation may be elicited. This piriformis pain on testing may also be elicited by pulling the bent knee across the body. This kind of pain may be found in female patients who complain of dyspareunia while in the so-called missionary position. Pain is produced in this position because of dysfunction in the piriformis muscle.

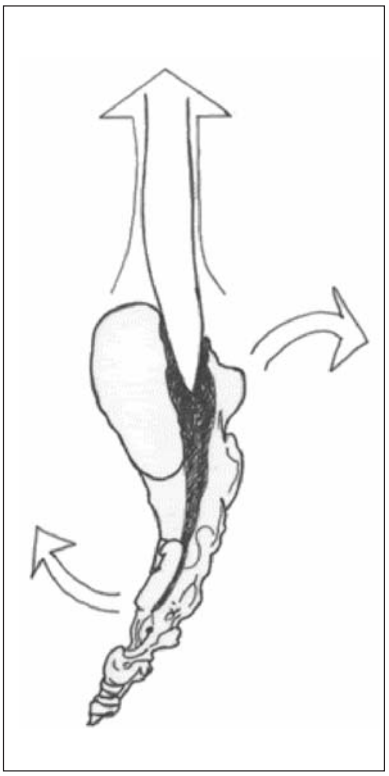
Pudendal nerve entrapment by the piriformis muscle may cause impotence in both men and women.[5]

- The piriformis muscle may have maximum muscle contraction testing weakness and be in need of the strain/counterstrain treatment described by Lawrence Jones.[6]
- Gluteus maximus will often have trigger or tender points, and may need strain-counterstrain or percussion treatment (the muscles are fatigued from holding that sagging sacrum back).
- Inferior mechanical consequences may follow this fault in the hip, knee and foot.
- Compression of one or both feet may cause weakening of the gluteus maximus, indicating the need for proper treatment of the foot.
- Imbrication testing, however, is frequently negative.
- SOT testing will show an SB + cough test, but there is usually limited excursion of the L5 spinous with the cough, and often times the cough is painful to the patient, especially noticeable "in the center of the sacrum."
- Bilateral gluteus maximus weakness may be present in the clear, but more frequently will be found with cervical flexion, extension, rotation, or TMJ movement added to the test.
- The sphenobasilar is in extension and may be felt as compressed.
- The cranial mechanism is frequently very rigid, with very little motion of the mechanism detectable.
- Muscular dysfunction may be found in the temporomandibular mechanism and hyoid areas.

This subluxation is one of many in chiropractic that fail to respond to corrective techniques or, having responded, recur. More effective and lasting results might be ours if we give consideration to the total cranial-sacral mechanism and its attachments (which reflect, or produce, the sacral subluxations).

Treatment

- DeJarnette blocks placed under the anterior superior iliac spines of the prone patient elevate the 5th lumbar.
- Logan Basic[7] contacts with a sacral inspiration line of drive will also help lift the sacrum out of its extension position.
- Percussion of the S2 segment as advocated by Fulford has been effective in improving movement of the femur, lumbosacral and sacroiliac joints. If this is followed by proper cranial and cervical spine releases, good increases in ROM and mobility often result.
- Category involvement, though almost always present, may only be found by specific challenge. The stiffness, tension, and spasm in the area can make positive therapy localization unrewarding in diagnosis.
- The sacral base needs to be moved up and back, and so with the patient prone, the doctor can hold the sacrum in the palm of the hand and gently nudge the sacral apex cranially as the patient inhales, drawing the sacral base posteriorly. DeJarnette blocks under the ASIS bilaterally will speed this correction. This correction may need to be prolonged as much as five minutes to effect change in the sacral depression subluxation.
- The small thoracic spine block may be used on the sacral apex to encourage respiratory flexion of the sacrum. The femurs can be abducted-adducted, externally-internally rotated in order to encourage the sacrum to reseat itself higher within the auricular boot of the sacroiliac joints.
- Using the Dutchman's roll and the flexion-distraction instrument will help "lift" the sacrum out of its divot.
- Dr. Sutherland's method of treatment is as follows. The patient is seated in front of the doctor on the table. The patient is asked to bend forward and rest upon the doctor's shoulders. The doctor then wraps his arms around the patient's pelvis and places his fingers into the grooves of the sacroiliac



joint, pulling the wings of the ilia toward him while the patient returns to the upright position on deep inspiration. This serves to pull the sacral base up and back. This procedure must be done slowly to maintain contact on the sacral alae. This usually needs to be repeated.

- A similar method has the doctor behind the patient. He takes a firm hold on the iliac wings at the back while the patient bends forward and exhales deeply. The doctor then asks the patient to straighten his spine while breathing in deeply and raising his shoulders, while the doctor pushes the two wings of the ilia into anterior rotation. The sacral base is moved little by little upward and backward.
- Many osteopaths describe a method for lumbosacral decompression that I have found effective, especially in pregnant mothers. I place one hand between the legs and spread my fingers on either side of the sacrum. The other hand can touch one ASIS as the fleshy part of the forearm holds the other ASIS. The contact on the sacrum encourages sacral respiratory flexion, while the contact on the ASISs encourages a spreading of the posterior sacroiliac joints. This is the anterior movement encouraged in the Sutherland method. As the sacrum goes into flexion, encourage it also to rise up gently between the ilia. This correction helps in many types of low back pain.
- All other factors found by AK examination should be corrected.

Conclusion

I have found this condition many times in new mothers. The post-partum neurosis and blues are frequently dramatically improved by correction of this situation. Correction of this sacral fault has significantly improved the lumbo-pelvic immobility in patients I have treated in this way.

References

1. Sutherland, W.G. *Teachings in the Science of Osteopathy*, p. 204 (Rudra Press, 1990).
2. Myers, T.W. *Anatomy Trains: Myofascial Meridians for Manual and Movement Therapists*, p. 74-76 (Churchill-Livingstone, 2001).
3. Upledger, J.E., Vredevoogd, J.D. *Craniosacral Therapy*, pg. 141, 240 (Eastland Press, 1983).
4. Dvorak, J., Dvorak, V. *Manual Medicine: Diagnostics*, pg. 130-133 (George Thieme Verlag, 1984).
5. Travell, J.G., & Simons, D.G., *Myofascial Pain and Dysfunction – The Trigger Point Manual*, Volume I & II. (Williams & Wilkins, 1992).
6. Jones, L.H., *Strain and Counterstrain* (American Academy of Osteopathy, 1981).
7. Coggins, W.N. *Basic Technique: A System of Body Mechanics*. (ELCO Publishing Company, 1983).

The Piriformis Muscle and the Genito-Urinary System: The Anatomy of the Muscle-Organ-Gland Correlation

Scott C. Cuthbert, D.C.

Abstract

Chiropractic and the Genito-Urinary System

As used in applied kinesiology (AK), manual muscle testing (MMT) procedures are helpful diagnostic tools in the examination of a system frequently ignored and unexamined by traditional physicians today: the reproductive system of both men and women. Using AK MMT we can work directly with the position, motion, innervation, nutritional needs, tissue tone and functional capacity of the reproductive organs as well as their adjacent and supportive tissues. AK MMT procedures permit us to restore proper tone and structure/function relationships throughout this critical area, and to improve its potential for health. Within chiropractic the genito-urinary system seems to be viewed differently than other body systems, i.e., it is usually ignored as a specific clinical entity.

This paper will argue that it makes no sense for the chiropractic profession to deliberately shy away from the genito-urinary system in its patient recruitment, history taking, functional examinations or therapeutics. We possess methods within our therapeutic system that permit us to evaluate and treat the genito-urinary system – with respect, reverence, sensitivity, and skill. Visual and laboratory inspection of the tissues and fluids of the reproductive organs is only one aspect of the proper evaluation of this system.

Introduction

The relationship of applied kinesiology to the endocrine system is one that creates success for the clinician where other manual modalities might fall short. Each of the endocrine organs has been given diagnostic tests, therapeutic protocols, nutritional correlations, and treatment monitoring methods. The endocrine glands are of course controlled by the nervous system, and this is why chiropractic has been helpful throughout its history for endocrine-related disorders.[1] But applied kinesiology offers an exhaustive, exemplary, and repeatably accurate way to monitor both the endocrine symphony and the effect of our natural therapies upon it. Our system of chiropractic offers us as much *endocrinology for the general practitioner* as can be found anywhere.

It is very difficult to localize and distinguish between the various palpated and tested tissues in the pelvic area. Only by having a thorough knowledge of both the external and internal anatomy of the pelvis can this be accomplished. Most states in the U.S. do not allow chiropractors to palpate the internal anatomy of the genito-urinary system. However the pelvic tissues can be specifically tested using non-invasive AK MMT procedures, and the muscle inhibitions found can be anatomically interpreted by the physician as to the location of the primary involvement: segmental level, pelvic articulation, muscle, organ-gland, fascia, nutritional deficiency, lymphatic or vascular pooling, cranial-sacral involvement, and so forth.

If there is a connection between the genito-urinary system and the muscles of the pelvis we should find an *immediate improvement* in pelvic muscle strength and lumbar and femoral ROM upon application of the appropriate sensory-receptor challenge or correction. We should discover in a moment the difference this correction will make upon the tissues of the pelvis. To immediately improve (by a particular therapeutic trial) the

tissue tone surrounding the sacral plexus, for instance, or the tissue tone of muscles through which the pudendal arteries and veins pass, or the tissue tone of the suspensory ligaments of the uterus, ovaries, bladder, rectum, or vagina is a real benefit in the evaluation and treatment of any genito-urinary involvement. Demonstrating this change to a patient, and making sure that they understand this improvement in tissue tone, is an excellent way to increase your practice and your reputation.

The Genito-Urinary System's Eminence

All too often women who suffer from pelvic or gynecological disorders see themselves condemned to the conventional regimen of painkillers and anti-inflammatory drugs until the inevitable trip to the surgeon. This is so common in the patients who come to our practice that a large book could only begin to cover the subject. The 560,000-plus hysterectomies performed *annually* in the U.S. indicate the severity of these problems.

Thirty to forty percent of menstruating women are affected by premenstrual syndrome. The use of antidepressant drugs such as Prozac is quickly becoming the dominant medical treatment for PMS.[2] Large pharmaceutical companies have realized the huge potential market in this area and have sponsored clinical trials using drugs to treat PMS symptoms (e.g., antidepressants such as Prozac and Zoloft, anti-anxiety drugs related to Valium, and gonadotrophin releasing hormone), despite the fact that risks due to side effects far outweigh the benefits.[3]

Added to these statistics would be the 46 percent of post-menopausal women who take or have taken hormone replacement therapy (HRT). Continental Europeans are notably less enthusiastic about medication, the figures there falling into the teens, while in Japan a mere 6 percent of post-menopausal women take HRT – perhaps because they bring enough estrogen into their bodies through the foods they eat, notably that well of phytoestrogens, soy. The most common complaints of menopause are hot flashes, headaches, atrophic vaginitis, frequent urinary tract infections, cold hands and feet, forgetfulness, and an inability to concentrate. In the U.S., 60 to 80 percent of menopausal women experience hot flashes to some degree. In most cases, hot flashes are most uncomfortable in the first and second years after menopause. As the body adapts to decreased estrogen levels, the hot flashes typically subside.

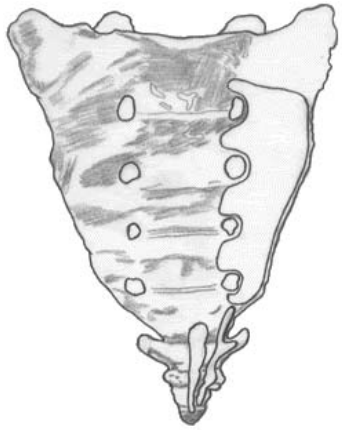
There are fifty million women in the United States over the age of fifty, and all of them are potential candidates for hormone therapy. If every one of them were to take hormone pills for the next thirty years – to the age of eighty, which is close to the current female life expectancy – that amounts to 1.5 billion woman-years of drug consumption for genito-urinary related problems. Never before has a drug regimen been proposed on such a scale.

Discussion

Piriformis: The Anatomy of the Muscle-Organ-Gland Correlation

The anatomy and physiology of this area is wonderfully complex and wonderfully simple (to borrow a thought from Dr. Goodheart). The anatomical inter-relationships between the pelvic organs and the pelvic muscles are of great importance in the analysis and treatment of tissue malfunctions in the pelvis and genito-urinary system. Some of these relationships will be discussed in this paper. I hope that this will enable us to comprehend and visualize the extent of the interdependence between the musculoskeletal system and the functioning of the pelvic organs.

The piriformis muscle originates from the anterior surface of the sacrum by fleshy digitations between the first to fourth anterior sacral foramina. Some fibers may attach to the margin of the sciatic foramen at the capsule of the sacroiliac joint and some fibers may attach to the sacrospinous ligament. The muscle inserts into the superior border of the greater trochanter, just posterior to the obturator internus and the gemelli. This tendon of insertion often blends with these two muscles. The fifth lumbar and first and second sacral nerves innervate the piriformis. Because of its sacral insertions (S1-S4), its triangular shape, and the way it runs outward and downward and its anterior position, the piriformis muscle has an immediate stabilizing action on the sacroiliac articulation. The piriformis muscle is an abductor of the thigh, an external rotator, and a minimal extensor of the femur. As far as the low back and sacrum are concerned, it is a major source of trouble. By itself this one muscle, when hypertonic, can extend sciatica down to the popliteal space at the knee. Taut bands in this muscle may entrap the peroneal part or all of the sciatic nerve.[4, 5]



Muscle attachments to the sacrum: Piriformis, levator ani, coccygeus.

In chiropractic practice, the frequency with which piriformis muscle problems are confused with sacroiliac subluxations is due to the fact that the muscle lies deep to this area. When the muscle is dysfunctional and engaged, especially when moving or lifting the leg, pain will be found at the points of attachment of the muscle. These points of attachment are on either side of the sacroiliac joint, thus causing the confusion in diagnosis for many therapists. Referred pain from trigger points in the piriformis may radiate into the sacroiliac region, producing confusion once again. The piriformis muscle and its contiguous fascia are often the site of painful inflammatory processes involving the toxic products of the circulating blood and lymph. The muscle itself will produce some of this irritating chemistry when it is dysfunctional.[6]

A unilateral weakness of the piriformis induces the sacrum to make a torsional movement on its oblique axis. The base of the sacrum moves backward and upward on the weakened side, while the apex of the sacrum on the opposite side is displaced forward and upward. (This movement will be determined by the geometry of the auricular surfaces of the patient's sacrum.) This sacral deviation will usually be mirrored by the occiput. Another finding that results from an oblique rotation of the sacrum may be a rotoscoliosis of the lumbar vertebrae. Numerous other distortion patterns will follow that affect the entire spinal column, including the shoulders, upper cervical and cranial areas. A "scoliosis capitis" may correlate with the oblique sacrum produced by a unilateral weakness of the piriformis.

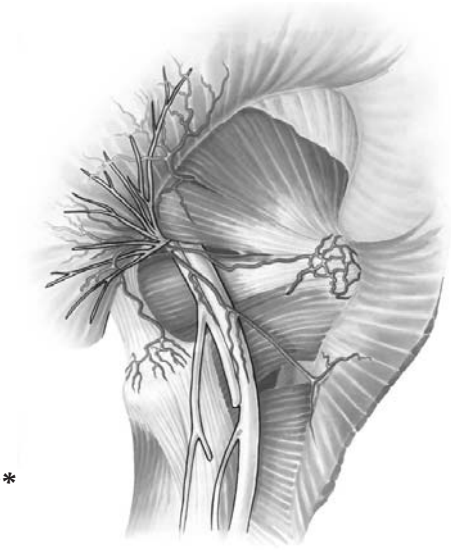
Specific MMT offers us the best way to differentially diagnose a piriformis muscle problem from other problems in this area. AK MMT, palpation of the muscle, and ROM testing are effective means for differentiating the source of a sacroiliac dysfunction. As we know from AK MMT, the following can produce piriformis muscle dysfunction:[7, 8, 9, 10]

- Subluxation, or pelvic category I through III involvement
- Local muscle injury (trigger or tender points; origin-insertion microavulsions)
- Cranial-sacral respiratory faults
- Visceral reflex weakness
- Gait mechanism imbalances
- Foot and ankle subluxations
- Lymphatic congestion
- Nutritional deficiencies

- Vaso-constriction of the blood supply
- P.R.Y.T. (pitch, roll, yaw and tilt) body imbalances
- Oculo-Basic faults

With piriformis pain, it is usually possible to palpate the taut rope of the muscle through the overlying gluteus maximus. Neuromuscular spindle cell problems, microavulsions, trigger points or tender points in the muscle will usually have positive therapy localization on testing and will also be palpable. The muscle stretch reaction or the maximum muscle contraction tests from AK will reveal these dysfunctions if present in the muscle.

Another method for the discovery of piriformis involvement has been described by Pace and Nagle.[11] They use a functional test for the piriformis by placing the hands on the lateral aspects of the patient's flexed knees and asking the patient to push the hands apart. Definite pain, faltering, or weakness on resisted lateral hip rotation will be elicited. This piriformis pain is local to the hip, does not radiate, and can also be elicited by pulling the bent knee across the body. This kind of pain may be found in female patients who complain of dyspareunia while in the so-called missionary position. Pain is produced in this position because of dysfunction in the piriformis muscle. Pudendal nerve entrapment by the piriformis muscle may cause impotence in men as well.[12] (These are further validations of the muscle-organ-gland correlation described in AK for many years.)



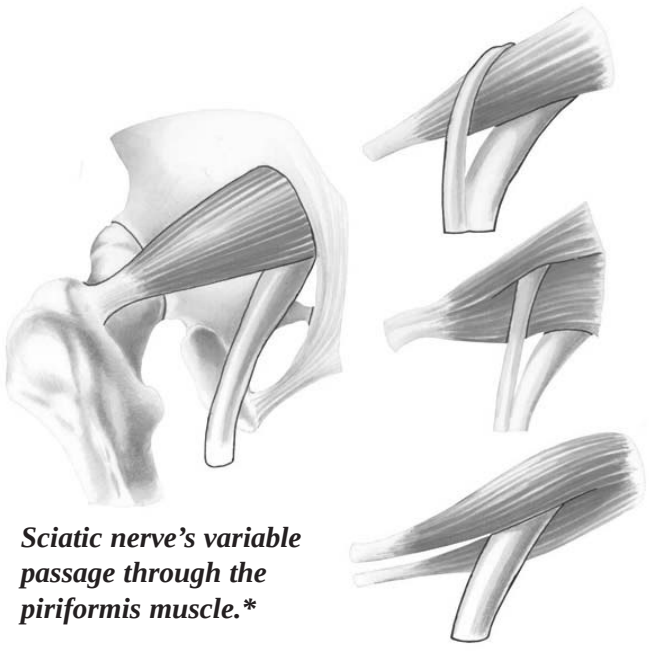
Piriformis muscle test and myofascial complex*

Another critical area of importance in any functional evaluation of the pelvis must be mentioned: the lumbosacral junction. This area must be examined thoroughly for any patient with genito-urinary involvements. The upper two nerves that supply the piriformis muscle pass through this area, which is sometimes called Marcelle's triangle – lateral to the body of the fifth lumbar vertebrae. The medial boundary of this triangle is formed by the body of the fifth lumbar vertebra, the lateral by the medial border of the psoas major, and its base by the upper part of the sacral ala. The floor is formed by the anterior portion of the transverse process of the fifth lumbar vertebra and the iliolumbar and lumbosacral ligaments. The lateral sympathetic chain ganglia pass through this triangle, as well as a large branch passing to the hypogastric plexus and the genito-femoral and obturator nerves from the lumbar plexus. Within the triangle on the left side are the common iliac lymphatic glands, the ureter, the internal spermatic or ovarian vessels, and the terminal portion of the inferior mesenteric artery that becomes the superior hemorrhoidal. The internal and external iliac veins unite to form the left common iliac vein, while the left common iliac artery divides to form the external and internal iliac arteries, and the ilio-hypogastric artery. These arteries directly supply the genito-urinary system.[13]

All of the rotators of the hip attach to the ilium, ischium, sacrum, and femur. Although they are small, short and deep, they powerfully influence the basic structural relationships of the pelvis when they are weak or in spasm. The fascial sheets of the erector spinae, quadratus lumborum, psoas, piriformis and pelvic floor are all continuous. This myofascial blending travels laterally to form the connective tissue covering of the abdominal oblique muscles and the rectus abdominus. The myofascia of the psoas is continuous with those of the lower border of the diaphragm. The fascia of the psoas via the iliacus is also continuous with that of the obturator internus, which then blends into the pelvic diaphragm.

Even this partial list of the muscles of the pelvis makes it obvious that they and their fascia form a web; the muscles are not layered like a cake, but interwoven like a mesh. A reasonable working knowledge of local anatomy gives us an understanding of the tissue layers and direction of muscle and fascial fibers producing the body distortions we find by measurement, palpation, challenge, and TL. This knowledge also allows us to determine which nerve, organ, lymph, blood, or bony distortion is most likely implicated in the muscle imbalances that we find by testing.

Drs. Travell and Simons,[12] in their excellent chapter on the piriformis muscle, review the critical importance of this muscle's functional tone to the nervovascular complex that passes through the greater sciatic foramen with it. The superior gluteal nerve and blood vessels pass between the superior border of the piriformis and the upper sacroiliac rim of the foramen. This nerve supplies the gluteus medius, gluteus minimus, and tensor fascia lata muscles. The sciatic nerve that we are all familiar with exits between the piriformis muscle and the rim of the greater sciatic foramen. It supplies the skin and muscles of the posterior thigh and most of the leg and foot. Also exiting the pelvis along the lower border of the piriformis muscle are the pudendal nerve and vessels. Proper function of this nerve and the structures it supplies is critical to normal sexual function. The inferior gluteal nerve to the gluteus maximus muscle, the posterior femoral cutaneous nerve, and the nerves to the gemelli, obturator internus, and quadratus femoris muscles also pass through the greater sciatic foramen with the piriformis.



Sciatic nerve's variable passage through the piriformis muscle.*

Drs. Travell and Simons close this section of their chapter on the piriformis muscle by saying, "It is apparent that chronic compression of these nerves would cause buttock, inguinal, and posterior thigh pain, as well as pain lower in the limb."

A) Pelvic Nerve Supply and the Myofascia of the Piriformis

It must be understood that most of the nerves of the pelvis that emerge from the spinal canal actually pass through and are surrounded by skeletal muscle. The contractile forces of these skeletal muscles of the pelvis exert a profound influence on the metabolism and excitability of the nerves. In this environment the nerves are subject to considerable mechanical and chemical influences of various kinds, including compression, stretch, angulation, torsion, edema, and many others.

Enmeshed in the myofascia of the piriformis lies the sacral plexus, which is formed by the anterior primary divisions (ventral rami) of L4 and L5 (lumbosacral trunk), S1-S3, and part of S4. The anterior primary division of S4 also contributes to the coccygeal plexus. The critical neuroanatomic importance of this fact will be described on the following page.

The branches of the sacral plexus are listed below (contributing spinal cord segments appear in parentheses).[13]

- Posterior cutaneous nerve of the thigh (S1-3)
- Pudendal nerve (S2-4)
- Sciatic nerve (L4-5, S1-3)
- Superior gluteal nerve (L4, 5,S1)
- Inferior gluteal nerve (L5, S1, S2)
- Nerve to the obturator internus and superior gemellus (L5, S1, S2)
- Nerve to the quadratus femoris and inferior gemellus (L4, L5, S1)

If we study the course and final innervation of these nerves, we will find that the pelvic organs have a shared nerve supply with the muscles described in the applied kinesiology muscle-organ/gland correlation to the reproductive system: the gluteus maximus, medius, minimus, piriformis, anterior tibial, and adductors.[7, 8, 9, 10] The nerves listed above are extremely important in maintaining normal genito-urinary function.

To cite just one example: the pudendal nerve gives off a branch called the inferior rectal nerve that supplies a muscular branch to the external anal sphincter and sensory fibers to the lower portion of the rectum, the skin surrounding the anus, and the distal third of the vagina. The pudendal nerve then divides into the dorsal nerve of the penis (or clitoris), and into another branch called the perineal nerve. From its point of origin in the pelvic floor, the dorsal nerve runs anteriorly to supply sensory fibers to the distal half of the penile shaft and the clitoris. The perineal nerve then divides into the posterior scrotal or labial nerve, which sends sensory fibers to the posterior two thirds of the scrotum or labia majora and a muscular branch that innervates the muscles in the anterior half of the pelvic floor that include the bulbospongiosus, ischiocavernosus, deep and superficial perineal muscles, the urethral sphincter, and portions of the external anal sphincter and levator ani.

Mechanical interference or compression of the pudendal nerve and blood vessels (along any portion of its course through the pelvis) can produce real trouble in the functioning of the genitalia of both sexes. These facts should be kept in mind the next time you MMT any muscle that crosses the pelvis.

Many disturbances have been attributed to mechanical interferences to the sacral nerve roots. These include pelvic pain (inguinal, pubic, anal, coccygeal, rectal), urinary frequency, urgency, dribbling, incontinence, difficulty, sluggishness, retention, nocturia, enuresis, dysuria, repeated infections, inability to sense vesicle filling, constipation, diarrhea, excessive gas, painful anal sphincter or urethral spasm, fecal incontinence, mucorrhea, inability to feel rectal filling, spontaneous miscarriage, painful and irregular menstruation, vaginal spotting, persistent vaginal discharge, menstrual migraine, genital pain and/or paresthesias, decreased genital sensitivity, anorgasmy, dyspareunia, deficient lubrication during coitus, pelvic pain during orgasm, loss of libido, and impotence. Throughout our profession's history, chiropractors have reported improvement in chronic pelvic pain and in genito-urinary system dysfunction with chiropractic treatment of pelvic and lumbar subluxations in countless case reports and studies.[1]

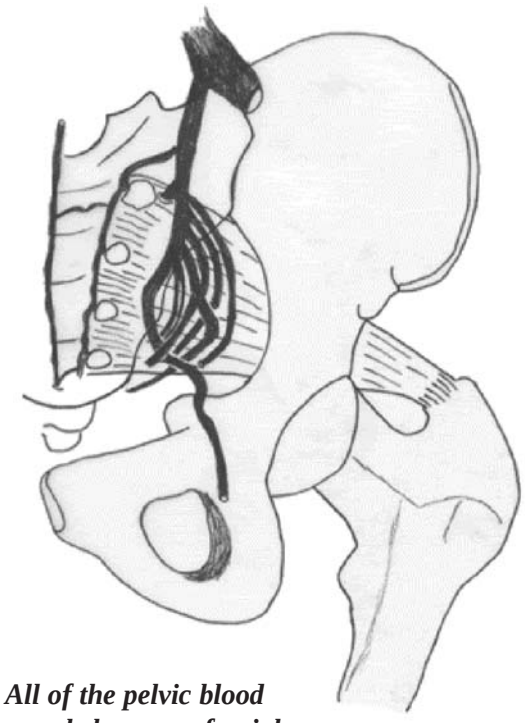
A genuine understanding of the neurology of the pelvis should encourage us to increase our scope of practice!

B) Pelvic Blood Vessels and the Myofascia of the Piriformis

Although very slight pressure on an artery may not produce occlusion, it does produce turbulence to flow or other serious hemodynamic consequences. With respect to venous supply and drainage we have a critical physiological point to make because the venous pressure is extremely low, and slight congestion of the vessels as they pass through the sacral foramina or the sacral muscle masses can cause some damming back into the areas of venous drainage. Deformation of a nerve or spinal root almost inevitably produces deformation and even occlusion of the vessels supplying the nerve, and vice versa. The degree of ischemia and neural dysfunction vary, but changes in the blood supply to the nerves of the pelvis may not be salutary. We are not only con-

cerned here with the damage resulting from vertebral trauma or herniated discs, but also subtle deformations, pressures, and circulatory changes (edema, congestion, compression, angulation, and ischemia, to name a few.). These mechanical disturbances may be due to very slight forces exerted by relatively slight tissue changes near the intervertebral foramina and in the paraspinal muscles.

The piriformis muscle of the pelvis forms part of the pelvi-trochanteric musculature and blood supply (iliolumbar, sacral and gluteal arteries) and therefore plays a part in pelvic and gynecological circulatory disturbances. This arterial and venous system supplies blood to the uterus and ovary blood system; consequently it is of great importance in the physiology and physiopathology of the uterus and ovaries.



All of the pelvic blood vessels have myofascial sheets anchoring them to nearby structures. The arterio-venous system of the pelvic organs is the same system that supplies and traverses the piriformis muscle masses. The myofascial complex of the piriformis is traversed by the following arteries: the internal iliac; lateral sacral; internal pudendal; middle rectal; umbilical; superior and inferior vesicle; inferior gluteal; and obturator arteries.

I want to emphasize that the only muscles in the body possessing an arterial and venous system linked with the genito-urinary system are the muscles attaching to or passing in front of the anterior portions of the sacrum or ilia:

- The piriformis
- The psoas
- The iliacus
- The obturator internus
- The gemelli
- The levator ani and coccygeus

The arteries and veins listed below supply the reproductive system and are embedded in the myofascial matrix of the muscles listed above.[13]

1) The iliolumbar arteries irrigate the general lumbosacral mass and arise at the level of L4 (Marcelle's triangle) from the abdominal aorta.
2) The left and right internal iliac arteries bifurcate at the L4 level also and supply all the pelvic organs.
3) The internal iliac artery also branches to become the lateral sacral artery and is a major source of blood to the sacrum, the sacral foramina, and nerve roots. This artery provides spinal branches that enter the vertebral canal through the IVF; it vascularizes the meninges, ligaments, osseous structures, roots, and rootlets.
4) The internal iliac artery branches to become the superior gluteal artery and supplies the gluteal region.
5) The inferior gluteal artery also supplies buttocks and thigh.
6) The internal pudendal artery has a long, tortuous course through the pelvis, and divides into the deep and dorsal arteries of the penis.
7) The inferior vesical artery in the male is called the vaginal artery in the female; it supplies the inferior aspect of the bladder.
8) The obturator artery has a long intrapelvic course before exiting the pelvis at the obturator foramen. It then supplies the adductor region of the thigh. (Another relationship in the muscle-organ-gland correlation of AK: the adductor muscles and the reproductive system.)

9) The uterine and vaginal arteries are found only in the female. The uterine artery, veins, and lymph vessels pass through the pelvic diaphragm and can be disturbed by imbalances in the pelvic floor muscles.

10) The ovarian artery and the uterine artery are complementary because they anastomose at the level of the horn (cornu) of the uterus and irrigate the utero-ovarian system. Their course is long within the abdomen and pelvis.

11) The ovarian artery begins at the level of the abdominal aorta, just below the renal artery. It then passes towards the ovaries, and one of its branches anastomoses with the uterine artery. It passes to the uterus, which it reaches at the level of the cervix of the uterus, then ascends to the base where it divides into two terminal branches (fallopian tubes and ovarian artery).

12) The median sacral artery courses along the anterior surface of the sacrum and sends branches to the anterior sacral foramina.

The venous drainage of the sacrum, coccyx, and the pelvic organs flows in the opposite direction as the arterial supply and drains into the internal iliac vein.

All of the pelvic blood vessels have myofascial sheets anchoring them to nearby structures. Of particular interest are two structures primarily associated with the internal iliac artery, called the sacrogenital folds. These ligaments run from the middle of the sacrum (S2, S3, S4) to the pubes, through the subperitoneal space. They are called the uterosacral ligaments. These sacrogenital folds attach to the rectum, isthmus of the uterus, superior vagina, and bladder, and contribute to the strong connection between these organs. Local tissue problems in this area may bring about local vasoconstriction as well as visceral spasms, with an associated slowing down of arterial, venous, and lymphatic flow.

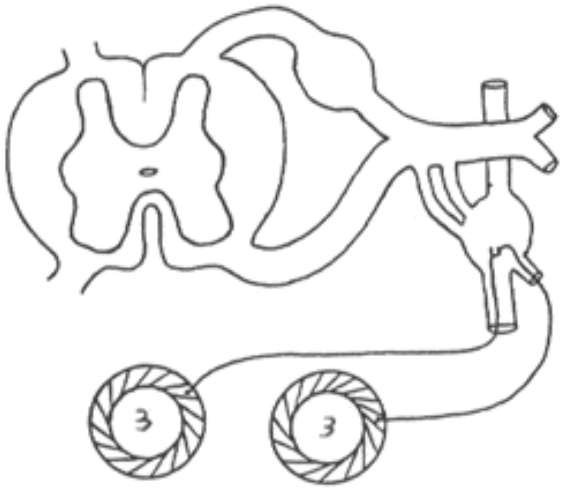
The close approximation of the internal iliac, iliolumbar, inferior vesical or vaginal arteries and veins to the anterior sacral muscles and to the osseous structures of the pelvis makes the possibility of mechanical interference with optimal blood flow through the pelvis a definite reality. A muscle when working mobilizes 6 to 10 times the quantity of blood compared to the same muscle at rest.[13] Poorly perfused tissues succumb to infection far more frequently than tissues with good nutrition. It has been shown that it takes very little improvement in circulation and only small increases in tissue oxygen levels to achieve large increases in the resistance of body tissues to infections.[14, 15] For proper blood flow to occur in the pelvis, muscular balance is necessary.

Therefore what has to be done is to act on the pelvic muscle masses, the nerves, arteries and veins that are linked with those of the utero-ovarian system. Treating the tonicity of the pelvic muscles is the best way of affecting the pelvis' nerve, vascular and fluid systems. With improved balance of muscle tone here, an improvement in the smooth muscle tonicity of the pelvic organs and blood vessels should be expected, as well as a reduction in the perfusion imbalances that occur with hypo- or hypertonicity. The result will be an increased blood flow, that is to say a decongestion spread over a potential area of 2,400m^2 of capillaries, thus influencing the local vascular condition of the pelvis and subsequently the general state of health.[13]

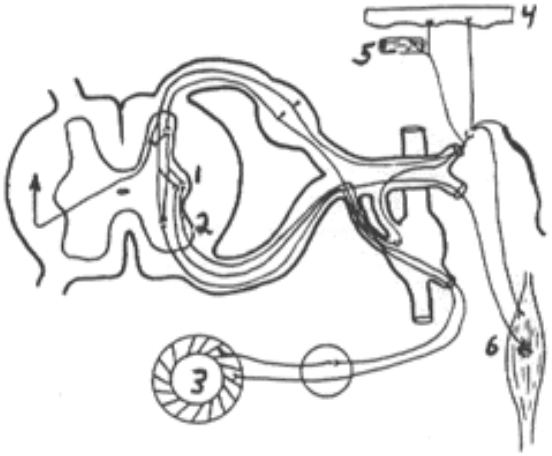
A note should be made about the sacral lymph system. The sacral lymph nodes are separated on each side of the rectum on the anterior surface of the sacrum. Some are always found on the median line, between the piriformis muscle's insertions, along the middle sacral artery. The pelvis is full of loose connective tissue that is capable of holding excessive fluid. Many of the pelvic plexuses described elsewhere in this paper pass through this loose connective tissue during part of their course through the pelvis. Retardation in the lymphatic circulation may occur, creating chronic passive congestion of the pelvic tissues. Varicosities, hemorrhoids, swelling in the legs, ankles, or knees, frequent urination due to mechanical pressures on the bladder, and disturbances in the elimination of fecal material can all result from this situation. Retrograde and anterograde lymphatic findings may be present in these cases. Muscle and fascial tone, as well as fluid content, once again becomes important.

C) Viscerosomatic Reflexes

The existence of polysynaptic viscerosomatic and somatovisceral reflexes implies that visceral afferent fibers are involved not only with the mediation of visceral functions, but also with the functions of somatic effectors, that is, ***skeletal muscles***.[13] Visceral pain fibers are mainly associated with the sympathetic nerves (for example, the hypogastric). The inputs from a malfunctioning viscus can come to dominate a segmental zone of the spinal cord, traversing the ganglia without synapse and entering the cord through the dorsal roots along with somatic sensory fibers. The facilitation produced here extends to the neurons supplying the somatic structures, producing muscular weakness, spasm, vasomotor and sudomotor changes, referred pain and tenderness. Many physicians have mapped these "visceral referred pain zones" over the past 100 years. One example of this type of viscerosomatic reflex would be the contraction of the abdominal skeletal musculature after excessive distention from a large meal or the inflammation of peritonitis or the prevalent "open" ileo-cecal valve.



Diagrammatic representation of the visceral reflex arc. The neural system between one visceral organ and another are indicated.



Schematic representation of the possible neural connections involved in the chiropractic subluxation complex. 1) mediolateral autonomic cells, 2) anterior horn of the spinal cord, 3) visceral organ, 4) skin over the paravertebral musculature, 5) blood vessels of the connected skeletal musculature, 6) the connected muscles of the body.

Experiments on rabbits have shown that stimulation of organs such as the renal pelvis and small intestine cause reflex paravertebral muscle contractions. In addition, some pathologic conditions (e.g., coronary artery disease) cause stimulation of afferent fibers that produce not only skeletal muscle contractions, but also concurrent activation of autonomic effectors in somatic tissue that results in cutaneous vasomotor and sudomotor changes. From these experiments involving the viscerosomatic and somatovisceral reflexes, it has been concluded that poor health and physiological dysfunction may be ascribed to a breakdown in the communication between the two major components of the body, the visceral and the somatic (musculoskeletal).[16]

Pelvic Muscle Tone and Organic Function

In a patient who presents with subluxations of S1/S2, for instance, all of the tissues in the corresponding dermatomes, myotomes, and sclerotomes are likely to have *abnormal tone*. This affects the nerve supply to the pelvis, the facilitation of the autonomic system, arterial, venous, lymphatic circulation, and the chemical balance of the area as well. In this way any trauma, chemical or emotional irritating agent to the subluxated area may provoke a physio-chemical response out of proportion to the cause.[16]

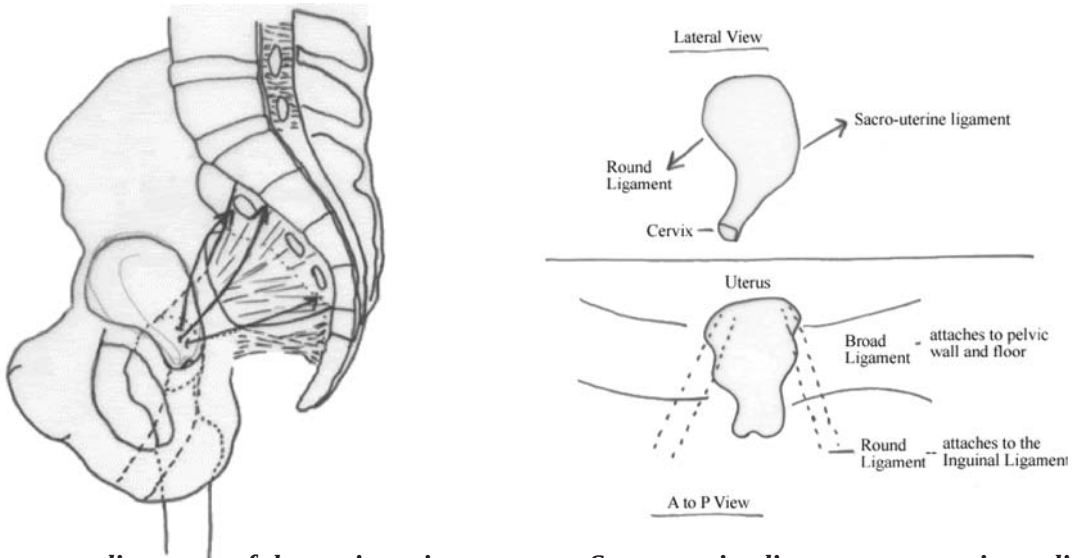
A satisfactory structural environment for an organ requires that the tissue tensions surrounding it be well balanced. Disturbance of a single component can change the overall balance and produce widespread visceral effects. As with all the organs of the body, the pelvic organs are held together by reciprocal tensions. In the pelvis, no muscle weakness or tissue injury can be isolated; the consequences can be surprisingly far-reaching anatomically. The slightest disturbance of a tissue may destabilize the reciprocal tension of all the supple (and adaptive) elements of the organism.

With any rotational, oblique, inferior or superior misalignment of the sacrum, the nerve supply to the pelvic organs will be disturbed. Further, the utero-sacral suspensory ligament system, which also supports the ovaries, rectum, and bladder, will produce an abnormal position and mobility in these organs due to disturbed articular relationships. The uterus is anchored tightly into the bony pelvis by eight distinct ligaments. The uterus lies just below the pelvic brim, is almost 3 inches long (including the cervix), and is hollow and muscular and lined with endometrium.

To cite one example: the round ligaments connecting the uterine horns to the inguinal canal may be disturbed when the category II subluxation is present. Tension in the inguinal ligament will be transmitted through to the round ligament and on to the uterus by the same subluxation process. It might be that the greater the leg length inequality found with the category II subluxation, or the greater the inguinal tension found with the category II subluxation, the greater will be the corresponding tension placed upon the round ligaments supporting the uterus.

Normal genito-urinary functioning is also impaired if the uterus is anteverted and anteflexed or if its surrounding myofascia is restricted. Uterine anteversion or anteflexion will force the body and cervix to press hard against the bladder, increasing bladder pressure and lessening sphincter resistance. The “uterine lift technique” has been helpful for many women in the clinic over the years.[8]

In order for the pelvic floor to function properly with movement, in visceral support, in the propulsion of the cranial-sacral rhythm, and in digestive and sexual functions, there must be good tone and elasticity of the sacrospinous and sacrotuberous ligaments, the coccygeal muscles, levators, perineum, and the muscles that attach the pelvis to the femur. If the pelvic floor is hypotonic, the uterus, bladder, vagina, rectum, and other pelvic tissues will tend toward prolapse and descend toward their orifices. The cervix is secured within the

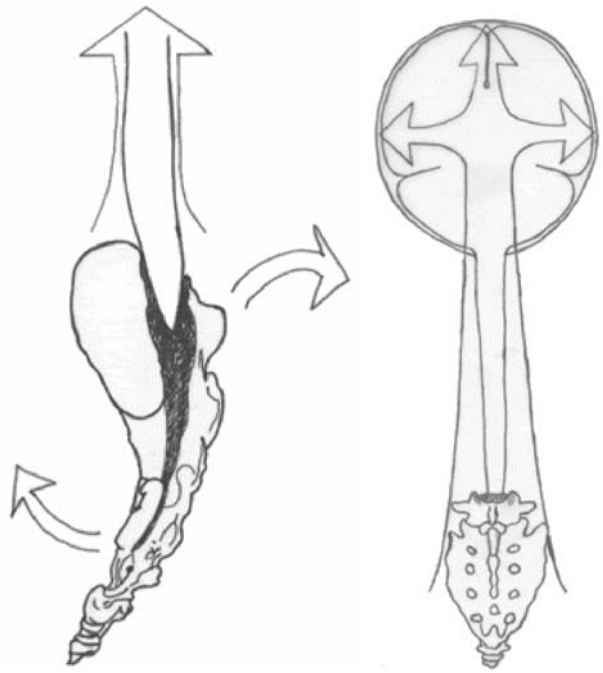


Suspensory ligaments of the genito-urinary system. Sacro-uterine ligament; sacrospinous ligament; broad and round ligaments; sacro-vaginal ligament. Articular misalignments of the sacrum or ilia will disturb the pelvic organs’ position and movement. The pelvic organs are supported by the peritoneum, by the ligaments spanning the pelvis, and by muscles, aponeuroses, and other related connective and myofascial tissues. The ligaments of the pelvis also contain contractile fibers.

vagina by the concentric muscles of the mother's pelvic diaphragm and by ligaments that secure it to the sacrum. With any imbalance in the uterine ligaments, the cervical portion of the uterus, or the cervix, may tip away from the center of the vaginal canal. The pelvic floor muscles also guide the baby into the mother's pelvis and through the birth canal. If the pelvic floor musculature is too weak or too tight, it will be difficult for either the mother or the baby to force the cervix open in the last stages of labor. A baby that experiences in-utero constraint, due to imbalances in the ligaments that hold the uterus in place, will have to fold his body to accommodate the reduction in available space. The molding of the child's cranium during birth will be deeply affected by the muscle and ligament tone surrounding the uterus.

With sacral respiratory faults, the tone and angulation of the pelvic floor is changed. If sacroiliac breathing movement is restricted, the continual massage of the pelvic fascias, which occurs with sacral respiratory motion, may not take place, thereby allowing venous and lymphatic congestion to occur in the pelvis. Neither lymph nor blood should be allowed to pool or stagnate anywhere in the body. Sacral base extension (a sacral inspiration assist fault) is the most common problem found clinically. To correct this, the doctor encourages a movement of respiratory flexion in the sacrum that will help the pelvic diaphragm to be lowered. At the same time, the doctor asks the patient to draw in a breath in order to lower the thoracic diaphragm. The doctor can ask the patient not to breathe for a few moments while finding the point of equilibrium in the tissues beneath the hand. The tension within the anterior sacral tissues and the intra-spinal membranes beneath the hand are improved by this correction.

Prolonged weakness of the piriformis muscle(s) will change the angle at which the sacrum rests within the pelvis between the ilia; thus long-term imbalances here may be transmitted to the coccyx. The effects of coccygeal subluxations upon the cranial-sacral mechanism have been written about extensively. Dr. Fulford called this the "recto-respiratory reflex" and considered it fundamental in the treatment of many disorders of the central nervous system, especially those of childhood. The ramifications of a malpositioned coccyx may also manifest through the ganglion of Impar, which lies just in front of the coccyx and has influence over the hypogastric plexus and the sympathetic tone throughout the pelvis.



The sacral subluxation, through its connection with the meninges, may dissipate its tension up through the cranial and spinal membranes, producing a compensation or a superimposed sphenobasilar subluxation.

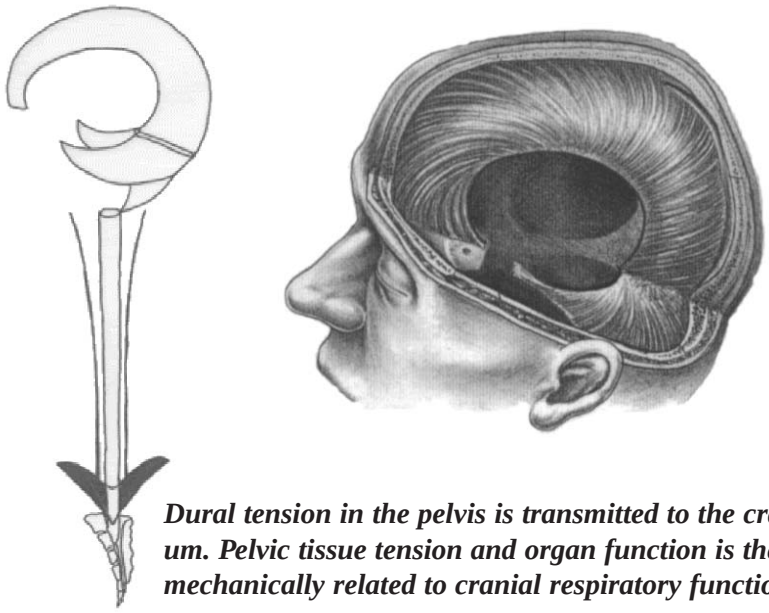
Coccyx subluxations or respiratory faults influence the bilateral tone of the pelvic floor muscles and their associated tissues. A misalignment of the coccyx will change the length and tension of the coccygeus, levator ani, and the myofascia of the perineal floor bilaterally. The bladder, external urethral orifice, muscles and sphincters in the rectum, vagina, and uterus will all be impaired in their function if the coccyx is. The coccyx may also be impaired from coccygeus muscle imbalance, from some fibers from the piriformis, internal obturator, the sacrotuberous ligaments, and perineum. The common myofascial gelosis problems of the lateral sacrococcygeal area are part of this coccyx pattern. With lateral or side-bending subluxations of the coccyx, you can release the coccyx externally with your thumb in order to balance the para-coccygeal tissues. These restrictions are often unilateral and require appropriate correction (a Logan Basic evaluation and treatment[17] is a very effective method to balance the pelvic floor).

Brieg states the primary source of adverse mechanical spinal cord tension is "set up directly by virtue of its anchorage at its two extremities, namely the brain and cauda equina."[18] Balanced elasticity of the sacrotuberous and sacrospinous ligaments is also critical, and a good indicator of the balance in the pelvic floor musculature.

Craniosacral and Endocrine Involvement

The relationship between the piriformis muscle's strength on AK MMT and the respiration of the cranial-sacral system is demonstrated on a daily basis in clinical practice. The sacrum and ilia exert a strong influence on the bones of the head, especially those that make up the cranial base, namely the occiput, sphenoid, ethmoid, temporals, and frontal. The upper cervical spine is also frequently implicated in sacral problems. Tension around the upper cervical nerve roots as they exit posteriorly from the upper cervical joints can be felt to dissipate during pelvic corrections. With proper pelvic corrections we have seen remarkable effects on the cranium, such as headaches of several weeks' duration clearing up immediately. Sinus congestion may clear up within an hour of proper sacroiliac treatment. TMJ muscular involvements can be eliminated with proper treatment to the pelvis.

Another remarkable interconnection is found occasionally (a phenomenon possibly related to endocrine interactions within the muscle system). Weak cervical flexor or extensor muscles will occasionally strengthen dramatically with therapy localization to the neurolymphatic reflex for the piriformis muscle. Almost any variety of body pain can be implicated in the numerous sacral deviations.



Dural tension in the pelvis is transmitted to the cranium. Pelvic tissue tension and organ function is thereby mechanically related to cranial respiratory function.

The sacrum moves in a compound, sinuous, and multi-dimensional way as we walk, and it is affected by the reciprocity between the anterior and posterior lumbar and pelvic muscles, as well as the tension imparted to the pelvis by the thighs. The piriformis and gluteus maximus muscles both cross the sacroiliac articulation and are drawn out in the direction of the oblique axes of the sacroiliac's motion and play an important part in establishing this motion. This "gyroscopic" movement is in harmony with the anatomy of the auricular surfaces of the sacrum. The sacrum moves with the craniosacral fluctuation, with every movement of the legs, and with every breath. The P.L.U.S. testing technique evaluates the relationship of the piriformis muscle to the cranial-sacral system's tensions very effectively.

Dr. Sutherland suggested that any limitation in the movements of the pituitary body in the sella tursica brought about by mechanical, membranous restrictions of the reciprocal tension membranes that hold the pituitary in place would be a primary cause of pituitary secretory disturbances. Normal sphenoid flexion and extension movements act to "milk" the pituitary of its excessive venous blood, optimizing hormonal function.[19, 20, 21, 22, 24] The mechanics of a sphenobasilar distortion may play a role in the passage of fluid from the pituitary system into the rest of the brain. The applied kinesiology approach to pituitary gland function and its mechanical and chemical correction has been effective for many patients, and has been a genuine advancement in the treatment of this area. Pituitary function is very important in gynecological conditions. Any encroachment at the

borders of the gland – especially at the cavernous sinus or diaphragma sella – can disturb the menstrual cycle, menopause, the mechanisms of labor, in fact the entire endocrine system.

The temporal bulge cranial fault, the inspiration and expiration assist faults, the sphenobasilar and frontal faults, the universal, glabellar, and naso-sphenoid faults from applied kinesiology, which includes the side-bending and torsion faults of the osteopathic world…any of these faults in the cranial mechanism may place undesirable effects and stresses upon the hypothalamic-pituitary relationship (with consequences throughout the endocrine system). This mechanical fault at the center of the autonomic and endocrine systems is crucial to normalize in the genito-urinary patient's physiological makeup, if possible.

The pituitary is the commander-in-chief of the endocrine system. The subject of endogenous endocrinology is so vast, however, and has been covered so extensively in books devoted to the subject, that I only hope to touch on it here.

Drs. Harrower,[25] Rubel,[26] Barnes,[27] and many others in the world of endocrinology have demonstrated the interconnections that exist between the pituitary-hypothalamus and the ovaries, uterus, prostate and testes. This is why MMT frequently demonstrates a relationship between certain cranial faults and weaknesses of the piriformis (and reproductive system) muscles in cases of hypothalamic-pituitary-reproductive system imbalances. These inter-connections are very important to a patient with genito-urinary imbalances, but often require astute clinical investigation to discover them. Unless someone spends years studying the endocrine system and knows the multiplicity of signs, symptoms, and indicators to look for, discovering these connections between the parts of this vast system would be intimidating.

Using AK MMT procedures, the primary dysfunctional organ in the endocrine complex can be found during an office visit. Drs. Goodheart, Schmitt, Walther, and others have written about this extensively. [7, 8, 9, 10] In brief: To find the primary endocrine gland, therapy localize to each of the endocrine neurolymphatic reflex points to find which one strengthens the other inhibited endocrine gland related muscles. This point of therapy localization indicates the primary organ or gland needing treatment. With a degree of understanding of the interrelationships of the endocrine glands and their connection to particular muscle-organ-gland relationships, the clinical value of these considerations can be shown clinically.

The uterus and ovaries are major parts of the endocrine system, that macramé of glands, organs, and brain structures that secrete and respond to hormones. These organs are enmeshed biochemically with the adrenals, the hypothalamus, the thyroid, and the pituitary. The value of strengthening an inhibited piriformis muscle goes beyond improving the function of the nerves and vessels supplying the reproductive organs: the entire endocrine system may benefit. Two-point therapy localization helps you "find the major" for the most effective treatment of this complex system.

In the 1930s, Dr. Charles Owen stated in his book on Chapman's Reflexes, "an innominate lesion always indicates an endocrine disturbance."[28] Later in his work he shows how the endocrine secretions of the pelvic organs influence the thyroid, induce acidosis, affect the urine, and set up digestive disturbances. The piriformis muscle is very frequently involved in the innominate lesion, and it may be that this muscle frequently tests weak due to disturbances in the endocrine and more particularly the genito-urinary system.

An estrogen/progesterone imbalance may also adversely affect the proper functioning of pelvic region. Once again, Drs. Goodheart, Schmitt, Walther, and many others within AK have discussed the relationship of the reproductive system with hormonal imbalances and the muscles of the pelvis for the past 30 years.[7, 8, 9, 10] The female's reproductive system is an even contest between two well-matched, muscled dames: estrogen and progesterone. Their harmonious apposition keeps this very fertile and complex system working well month after month, between 450 and 480 cycles in one lifetime. If for some reason an imbalance occurs between these two reproductive sisters and estrogen becomes the dominant force, then the hormone will throw the muscle cells of the uterus and pelvis into a state of electrical excitation. Estrogen makes them twitch. A uterus that twitches too much is a uterus that expels a fetus. And so, even as the uterus is urged to expand by estrogen, the

myometrium must also be tranquilized. That is the job of progesterone, the so-called hormone of pregnancy. Progesterone means progestation. Progesterone inhibits the contractibility of muscle cells. Throughout the whole nine months of baby making, as well as during the monthly menstrual cycle, the negotiation between estrogen and progesterone is a dynamic one.

I have had 3 cases where the patients had been trying to have a child for quite some time, not using any type of contraception, but who were unable to conceive. In 2 others, miscarriages had occurred repeatedly. In each of these cases, the patient became pregnant after chiropractic treatment for pelvic and lower back pain. Two of the first group of patients were found with inhibited muscles in the reproductive area that responded to both Wheat Germ Oil and Chlorophyll (S.P.[29]). Pelvic, cranial, and other spinal subluxations were also corrected as needed. Menstrual irregularities are constantly found in the patients we treat, and improvement in their problem is a regular outcome for our patients. This kind of evidence is of course "anecdotal," but it has been one of many reasons for my own continuing interest in the genito-urinary system and its relationship to our chiropractic procedures.

The Genito-Urinary System and AK Research

Because of the communication systems in the body between the nervous, circulatory, and muscular tissues, a disturbed portion of the musculoskeletal system can impair the function of other tissues and organs, especially those with which it is neurologically most closely related. In this paper the focus has been on the genito-urinary system and its communication with the nerves and blood vessels of the piriformis muscle area.

Conclusion

From the diagnostic viewpoint AK MMT has great significance because it makes possible the detection of a "dis-ease" process far in advance of the emergence of symptoms in the genito-urinary system. Whether the muscle inhibitions we find on AK MMT in the pelvis are primary (as in a postural subluxation) or of secondary reflex origin (as in a visceral disturbance), we must recognize that this component in the genito-urinary system's function is a contributing, exacerbating, and perpetuating influence, that must be given effective treatment regardless of the primary etiology. Because it is the most recognizable and responsive component in the disease process, it is the one through which a capable physician can influence the disease process in the genito-urinary system itself, and thereby interrupt the vicious cycle of somato-visceral and viscero-somatic impulses.

The genito-urinary or reproductive system is not often high on the list of priorities in many chiropractic clinics. Yet we know that our skills can help people with genito-urinary problems, but we must convince others of this. Objective, carefully applied AK MMT can convince ourselves, our patients, and those who watch us work that what we are doing is effective, reproducible, scientific, and occasionally miraculous.

It is very difficult, however, to produce definitive proof of the effectiveness of our techniques, even though applied kinesiologists have been actively researching and effectively treating the genito-urinary system for many years. The kind of immediate improvements we achieve in MMT evaluation (using sensory receptor stimulation) of the genito-urinary system, however, and the improvements we achieve clinically with these patients are both good signs of the effectiveness of our AK procedures as they have been developed up to this point in time. But this research has been done privately in our clinics and thus is of little use in persuading skeptics in the scientific community. We have been able to demonstrate to our patients' (and our own) clinical satisfaction the effects of our work on many of the problems in the genito-urinary system, but our research into this fascinating area must continue.

Chiropractic work on the genito-urinary system for both women and men requires great subtleness and respect for the patient. Our work in this area, using the procedures described in AK over the past 30 years to relieve problems that might otherwise require H.R.T. or surgery, makes us indispensable for the health, happiness, fertility and longevity of women everywhere.

References

1. Masarsky, C. S., and Masarsky, M. T. *Somatovisceral Aspects of Chiropractic: An Evidence-Based Approach*. (Churchill Livingstone, 2001).
2. Barnhart, K.T. A Clinician's Guide to the Premenstrual Syndrome, *Medical Clinics of North America, 79*. p. 761-767, 1995.
3. Mortola, J. F. A Risk-Benefit Appraisal of Drugs Used in the Management of Premenstrual Syndrome, *Drug Safety 10,* p. 160-169, 1994.
4. Freilberg, A.H. Sciatic pain and its relief by operations on muscle and fascia. *Arch. Surg., 34*: 337-350, 1937.
5. Hallin, R.P. Sciatic pain and the piriformis muscle, *Postgraduate Medicine, 74*: 69-72, 1983.
6. Ochoa, J. Histopathology of common mononeuropathies. In: Jewett, D.L., McCarroll, H.R., Jr., Eds. *Nerve Repair and Regeneration.* (St. Louis, CV Mosby, 1980: 36-52).
7. Goodheart, G.J. *Applied Kinesiology Workshop Procedure Manuals,* (Detroit, MI: Privately published) .
8. Walther, D.S., *Applied Kinesiology*, *Vol. I, Vol. II*. (Systems DC, Pueblo, CO, 1981 & 1983).
9. Walther, D.S., *Applied Kinesiology, Synopsis, 2nd Edition*, pg. 575. (Systems DC, Pueblo, CO, 2000).
10. Schmitt, W.H., Jr. *Common Glandular Dysfunctions in the General Practice – An Applied Kinesiological Approach*, and *Compiled Notes on Clinical Nutrition Products* (Chapel Hill, NC: AK Study Program, 1981 &1979).
11. Pace, J.B. Commonly overlooked pain syndromes responsive to simple therapy. *Postgraduate. Medicine, 58*: 107-113, 1975.
12. Travell, J.G., & Simons, D.G., *Myofascial Pain and Dysfunction – The Trigger Point Manual*, Volume I & II. (Williams & Wilkins, 1992).
13. Cramer, G. D., Darby, S.A. *Basic and clinical anatomy of the spine, spinal cord, and ANS.* (Mosby, New York. 1995).
14. Ninikoski, J. *Medical Biology*; Helsinki, Vol. 58, No. 6 1980: 303-309.
15. Hohn; Mackay; *Surgical Forum*; Vol. 27, 1976: 18-20.
16. Korr, I. M. The sympathetic nervous system as mediator between the somatic and supportive process *The Collected Papers of Irvin M. Korr*. (American Academy of Osteopathy, Indianapolis, IN, 1979).
17. Coggins, W.N. *Basic Technique: A System of Body Mechanics*. (ELCO Publishing Company, 1983).
18. Brieg, A. *Adverse Mechanical Tension in the Central Nervous System*. New York: John Wiley & Sons, 1978.
19. Magoun, H.I., "A Pertinent Approach to Pituitary Pathology," *The D.O., 11*, July, 1971.
20. Magoun, H.I. *Osteopathy in the Cranial Field*, Third Edition. (The Journal Printing Company, Kirksville, MO, 1976).

21. Stretch, O.M., "The Pituitary and the Ageing Process in relation to the Cranial Concept," *Lectures on Cranial Osteopathy*, Denis Brookes. (Thorsons, 1981).

22. DeJarnette, M.B. *Cranial Technique*. (Nebraska City, Nebraska: Privately published, 1979, 1978, 1977).

23. DeJarnette, M.B. *Technic and Practice of Bloodless Surgery*. (Nebraska City, Nebraska: Privately published, 1939).

24. , W.G. *Teachings in the Science of Osteopathy.* (Rudra Press, 1990).

25. Harrower, H.R. *Practical Endocrinology*; *Endocrine Pointers*; *An Endocrine Handbook*. (The Harrower Laboratory, Glendale, CA, 1932, 1933, & 1939).

26. Rubel, L. *The GP and the Endocrine Glands* (Decatur, IL: Privately published, 1959).

27. Barnes, B.O. *Hypothyroidism: The Unsuspected Illness*. (Thomas Y. Crowell Company, 1976).

28. Owens, C. *An Endocrine Interpretation of Chapman's Reflexes*. (American Academy of Osteopathy, Indianapolis, IN, 1937).

29. Lee, R. *Collected Papers and Reprints* (Standard Process Labs: Palmyra, WI).

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Learning the Law of Five Elements

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Abstract

Here is a method of learning the command points of The Law of Five Elements. This is a follow-up to shortcut number six in my paper, Acupuncture Shortcuts, that was presented in Atlanta in 2001 and printed in the AK Journal issue number 14 in 2002.

Introduction

The acupuncture system is one of five major energy systems in the body. Since energy can neither be created nor destroyed, whenever there is an imbalance in the acupuncture system, there will always be at least one over and one under meridian. The shortcut is to treat the command point on the under meridian that represents the element of the over meridian. Thus, you are drawing energy to the under from the over as opposed to treating the over, which merely scatters energy, some of which arrives at the under and produces temporary results at best. The energy may have to jump through hoops to get there via the Sheng cycle or the Ko cycle, but it does not change the above procedure.

In I.B.E., when we give a Diplomate exam, we allow the applicant to say they would look up the exact command point on a chart hanging on the wall of their treatment room. However, treatment of the acupuncture system is so powerful, you don't want to be at a loss to treat someone when your not in your office, such as, being at your mother's home or the like and she needs help.

Most of us have memorized the neurovasculars and neurolymphatics, the meridian pathways and origin and insertions. So, here is another method to help remember the command points of The Law of Five Elements.

Discussion

The command points, of which there are five for each meridian, relates that meridian to each of the five elements. There are sixty of these junction points to remember, since we do not include the conception vessel or governing vessel. There are many, many laws of acupuncture, such as, mother, child, grandmother, etc., methods of tonifying, and methods of sedating. This shortcut of treating the under meridian command point that represents the over element will take the place of most of these laws.

Referring to diagram one, each element is represented by a large circle. Within that circle are two medium sized circles (four for fire), one for each meridian in that element. The inside circle are the yin meridians, the outside circles are the yang meridians. Inside each meridian circle are five more circles - one for each of the elements. Notice that the location of the five circles are arranged in the same order as the five large element circles, Fire at the top, followed by (clockwise) Earth, Metal, Water and Wood. Within each of the small circles of the meridian circles are numbers. These numbers represent the point on that meridian that are related to the elements of the large circles.

Now, look for a pattern. Notice the number for the wood command points in the six yin meridians. You'll see that three of them are the number one. Those three happen to be the yin meridians of the leg (kidney, spleen and liver), which all begin on the feet. The other three yin meridians (lung, circulation sex, and heart) end on the hand. If we know that the lung meridian has 11 points, the circulation sex meridian has 9 points and the

heart meridian has 9 points, then we see the wood points of the yin meridians are either the beginning or end points of each yin meridian respectively. All six are the most distal point of their respective meridian.

Now look at the five element command points for each of the yin meridians. You'll see that the kidney, spleen and liver meridians all move up to Ki-2, Sp-2 and LIV 2. Also notice that the lung, circulation sex and heart meridians all move back one to LU-10, CX-8 and HT-8.

Now look at the earth element command points. You'll see they simply move in another point from the extremity. So, the earth command points are K1-3, Sp-3 and LIV-3 on the foot and LU-9, CX-7 and HT-7 on the hands.

So, the pattern is for the yin meridians, the distal point is the wood element command point and then continuing around the five element wheel in a clockwise direction, the fire element command point is the next point on the meridian towards the trunk of the body from the distal point, and the earth element command point is the third point on the meridian in from the distal end of the meridian.

Now look at the metal element within the yang meridians. Again, there are three number ones. These are for the large intestine, triple heater and small intestine, the yang meridians of the arms and hands. For the yang meridians of the legs and feet, we find bladder 67, gallbladder 44 and stomach 45, which are the end points for those three meridians. So, the distal point of each of the six yang meridians is the metal command point.

Moving clockwise to the water element, each meridian changes by one number. This is the second point in from the distal end of each yang meridian. Moving clockwise once more shows a similar pattern for the wood element, except the gallbladder skips a number and becomes our one exception for the three in a row pattern that we have seen both the yin and yang meridians, if you can remember that one exception; you have now learned 36 command points.

For the yang meridians, the distal point is the metal element command point. Continuing around the five element wheel clockwise, the water element command point is next point on the meridian towards the trunk of the body, and the wood element is the third point in from the distal end of the meridian with one exception. The gallbladder meridian wood command point skips one and is the fourth point in from the distal end, gallbladder 41.

This is the end of the numbering patterns. However, there are two meridians that have four points in a row. The lung and liver meridians, the metal points lung 8 and liver 4, respectively since they are both yin meridians, their numbering pattern started with wood and going four points in from the distal end, you end up at the metal point.

We know that all the command points are located between either the elbows and the hands or the knees and the feet. Because we've looked at the sequence of the elements starting at the distal end of each meridian, there is a pattern that evolves from what you've seen so far.

Because the yin meridians start with the wood point, we know that the points go no higher than the elbows and the knees. We can safely say that the water points are located either around the elbows or the knees.

For the yang meridians, we use the same comparison, but we start at the metal point. Therefore, we find that the earth point for all of the yang meridians are located at either the elbows or the knees.

The Chinese look at the energy flow from the extremities in towards the center of the body as a flow of water. Starting with the distal point as a well, the well becomes a spring. The spring becomes a stream. The stream becomes a river. The river ends up in the sea. Thus, the well points are the distal points and the sea points are around the elbows or knees. This is summarized in diagram two.

Conclusion

The purpose of learning these patterns is twofold. First, it will help you to memorize the 60 command points and second, it gives you a way to think about the points. Using the water analogy you have a way of figuring out which point and its likely location, even if you don't have your chart with you. It is useful to learn the numbers of the command points as 12 different sequences. For example, spleen is 1,2,3,5, and 9. Heart is 9,8,7,4 and 3. By learning these, it is easy to overlay the numbers on the five element chart, knowing that the yins begin with the wood element and the yangs begin with the metal element. It's not as hard as it sounds. You've probably memorized at least as many phone numbers, and they have seven numbers each.

References

Deal, S.C., *Advanced Kinesiology.* New Life Publishing Co. Tucson, AZ. 1999.

Deal, S.C., *Acupuncture Shortcuts.* ICAK Annual Meeting. Atlanta, GA. June 2001.

Lehman, Adam. *Patterns of Chinese Command Points.* TFH Annual Conference. July 2002.

Worsley, J.R., *Acupuncture Charts.* South Carlton, N.Y. Acupuncture. 1983.

Diagram 1

Law of the Five Elements

Luo Points • Energy Direction • Mid-Day Mid-Night Law • Superficial and Deep Pulses

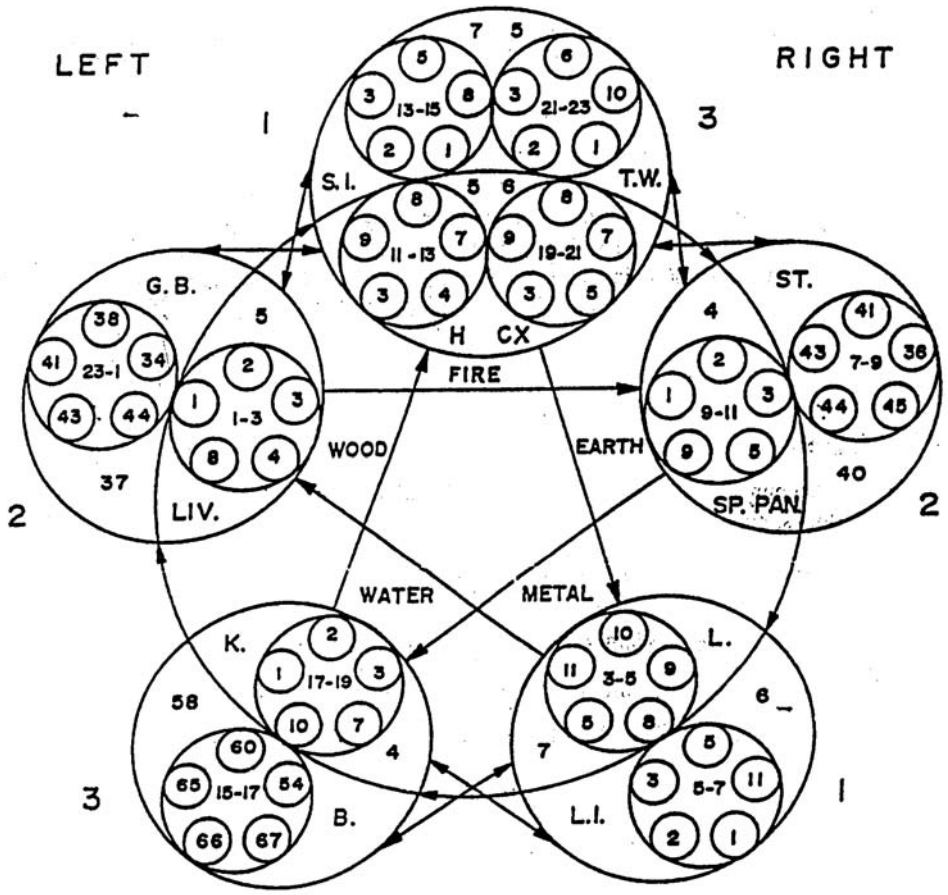

Diagram 2

Command Point Pattern Chart

YIN COMMAND POINTS

ELEMENT	WOOD	FIRE	EARTH	METAL	WATER
MERIDIAN	WELL	SPRING	STREAM	RIVER	SEA
KIDNEY	1	2	3	7	10
SPLEEN	1	2	3	5	9
LIVER	1	2	3	4	8
LUNG	11	10	9	8	5
CIRCULA-TION/SEX	9	8	7	5	3
HEART	9	8	7	4	3

YANG COMMAND POINTS

ELEMENT	METAL	WATER	WOOD	FIRE	EARTH
MERIDIAN	WELL	SPRING	STREAM	RIVER	SEA
STOMACH	45	44	43	41	36
GALL BLADDER	44	43	41	38	34
BLADDER	67	66	65	60	54
LARGE INTESTINE	1	2	3	5	11
TRIPLE HEATER	1	2	3	6	10
SMALL INTESTINE	1	2	3	5	8

Extremity Subluxation Correlation Part I: Spine Dysfunction

Timothy Francis, M.S., D.C., DIBAK, D.H.M.

Abstract

Spinal dysfunction may display itself in a variety of ways: visceral symptoms, extremity symptoms, neurological dysfunction, mental/emotional symptoms, and in a numerous other patterns. Dysfunctions in the spine are occasionally caused from extremity subluxations. This paper presents the author's personal observations regarding the relationship between the extremity subluxations and spinal dysfunction patterns.

Introduction

In two previous papers by this author: Upper Extremity Subluxation/Muscle-Syndrome Correlations and Lower Extremity Subluxation/Muscle-Syndrome Correlations, each extremity subluxation was matched with certain muscles. This paper will expand that concept to linking those particular extremity subluxations to various spinal dysfunctions. These spinal patterns may take the form of gait, P.L.U.S., fixations, passive range of motion, P.R.Y.T. (pitch, roll, yaw and tilt), and/or failure to pass the deep tendon reflex test. Correction of the appropriate extremity subluxation will then correct the matching spinal dysfunction if that is the ultimate cause.

Discussion

Gait testing via manual muscle testing may be broken down into basically upper and lower gait. As one steps forward with one foot and opposite arm, the (in relation to the forward leg) ipsilateral upper trapezius-contralateral SCM should be conditionally inhibited (manually muscle test weak). This is considered a normal response. If the subject does not respond in the aforementioned fashion, extremity subluxations to consider are: an acromioclavicular joint separation, posterior tibia, and/or a posterior calcaneus.

Lower gait analysis may be done via the piriformis gait inhibition test. In a weight bearing posture, the piriformis is tested, then the opposite arm is brought forward; the piriformis should now manually muscle test weak. If not, check for a scaphoid, posterior tibia, posterior calcaneus, and/or a talus subluxation.

People walk and people bend daily. According to Goodheart, in 30 degrees spinal flexion and 15 degrees spinal extension, the right piriformis, left latissimus dorsi, left upper trapezius, and right SCM should be inhibited. He based his observations on the prior work of Illi. Aberrant patterns check for a femur head, talus, cuboid, and superior second cuneiform subluxations.

Spinal fixations are a locking together of two or more vertebrae creating specific bilateral muscle weakness patterns. The limbic fixation is a locking of either C7 and first rib head and/or L1 and twelfth rib head displaying in the body as weak ankle muscles all on one side only. (peroneal and tibialis muscles). Extremity causes may be a proximal clavicle, posterior tibia, posterior calcaneus, talus, and/or an inferior navicular.

Pelvic categories were developed by Dr. DeJarnette in Sacral – Occipital Technique. Generally three categories are talked about. Category III is an intact pelvis, which is rotated as a unit in reference to L5. An extremity correlation is a superior first cuneiform. Category II is an osseous misalignment of the ilium to the sacrum.

Extremity causes are the talus, superior third cuneiform, and/or laterally rotated metatarsals. Category I is a dural torquing of the pelvis. Extremity considerations are the scaphoid and/or a posterior fibular head.

Pitch, roll, and yaw patterns in the spine have been correlated by Goodheart using his background in aeronautics. Another pattern called tilt was added by Beardall. Pitch patterns (flexion and/or extension) may sometimes be corrected by a medial olecranon subluxation. Roll patterns extraspinally consider a dropped scapula and /or jammed carpals. There are three basic types of yaw patterns. Yaw occiput may involve the a-c joint, posterior fibular head, and /or cuboid. In yaw sacrum look for a posterior radial head, posterior calcaneus, talus, and/or a cuboid subluxation. yaw thoracolumbar (number 3) consider extraspinally, jammed carpals, talus, cuboid, superior second and/or third cuneiform, and/or dropped metatarsal heads. In a positive tilt test look for an a-c joint problem.

Deep tendon reflexes according to Belli, can be tested for proper neurological function utilizing manual muscle testing. For example, if one elicits a patellar reflex; the ipsilateral hamstring should be inhibited once and the opposite quadriceps once. If not, check extraspinally for a dropped scapula, a femur head, and/or a posterior tibia subluxation. Likewise, if one elicits a biceps reflex; the ipsilateral triceps should inhibit once and the opposite biceps once. If it doesn't, check for a medial olecranon, posterior tibia, talus, cuboid, and/or a superior second cuneiform subluxation.

Dr. Goodheart in the last few years has been studying and applying Fulford's work and observations utilizing passive range of motion tests along with pincer palpation of muscles. Part of Goodheart's protocol with the subject supine is to check for passive range of motion in the shoulders, knees, torso (right to left and left to right), and with the patient sitting with her/his feet flat on the floor rib cage rotation clockwise and counterclockwise. Beginning with the shoulders, decreased passive range of motion in abduction on the right check for talus and/or superior third cuneiform. The left shoulder one may consider a dropped scapula, and/or a superior third cuneiform. Decreased passive range of motion in the left leg in Fabere Patrick's style, check for a medial olecranon, a superior second cuneiform, and/or dropped metatarsal heads. This author has not correlated any extraspinal subluxation for a loss of passive range of motion for the right leg. Torso decreased range of motion from left to right consider the femur head and/or a superior second cuneiform. If from right to left consider a posterior radial head, jammed carpals, posterior calcaneus, and/or a superior sdecond cuneiform. For rib cage decreased passive range of motion in a clockwise fashion with the subject seated look for a scaphoid subluxation. For counterclockwise decrease check for a posterior radial head, scaphoid, posterior calcaneus, talus, inferior navicular, and/or a cuboid subluxation. In a positive pincer palpation test of any muscle (pinch the muscle across its belly and then manually muscle test) check for a posterior fibular head and/or a talus subluxation.

In summary the following patterns have been observed.

Spinal Dysfunction	Extremity Subluxation(s)
Upper Gait	A-C joint Tibia Calcaneus
Piriformis Gait Inhibition	Scaphoid Tibia Calcaneus Talus
P.L.U.S.	Femur head Talus Cuboid 2nd cuneiform

Spinal Dysfunction	Extremity Subluxation(s)
Limbic Fixation	Clavicle Tibia Posterior calcaneus Talus Navicular
Category III	1st cuneiform
Category II	Talus 3rd cuneiform Metatarsals
Category I	Scaphoid Fibular head
Pitch	Olecranon
Roll	Scaphoid Carpals
Yaw – Occiput	A-C joint Fibular head Cuboid
Yaw Sacrum	Radial head Calcaneus Talus Cuboid
Yaw Thoracolumbar	Carpals Talus Cuboid 2nd cuneiform 3rd cuneiform Metatarsal heads
Tilt	A-C joint
DTR's (lower)	Scapula Femur head Tibia
DTR's (upper)	Olecranon Tibia Talus Cuboid 2nd cuneiform

Decreased Passive ROM	
Shoulder R	Talus 3rd cuneiform
Shoulder L	Scaphoid 3rd cuneiform
Legs R	N/A

Decreased Passive ROM	
Legs L	Olecranon 2nd cuneiform Metatarsal heads
Torso L – R	Femur head 2nd cuneiform
Torso R – L	Radial head Carpals Calcaneus 2nd cuneiform
Pincer palpation	Fibular head Talus

Conclusion

These particular extremity subluxations affect the spine in many ways, some of which are discussed here. Correction of the extraspinal subluxation will then correct the spinal dysfunction, if that is the cause of that aberrant pattern. Remember there is no rebound phenomenon in the extremity when challenging for these subluxations. Fix what you find and then observe the results to obtain the optimum potential for each patient.

References

1. Francis, Timothy D., Upper Extremity Subluxation/Muscle Syndrome Correlations. Experimental observation of the members of the I.C.A.K. Volume I (1999 – 2000).
2. Ibid, Lower Extremity Subluxation/Muscle Syndrome Correlations. Experimental observations of the members of the I.C.A.K. Volume 1 (1999 – 2000).
3. Ibid, Spinal – Rib Subluxation/Muscle Syndrome Correlations. Experimental observations of the members of the I.C.A.K. Volume 1 (1999 – 2000).
4. Ibid, Spinal Subluxation / Bilateral Muscle Syndrome Correlations. Experimental observations of the members of the I.C.A.K. Volume 1 (2000 – 2001).
5. Ibid, Additional Fixation Patterns. Experimental observations of the members of the I.C.A.K. Volume 1 (2001–2002).
6. Goodheart, George J. You'll Be Better, The Story of Applied Kinesiology. AK Printing; Geneva, Ohio.
7. Leaf, David. Applied Kinesiology Flow Chart Manual 3rd Edition. Privately Published (1995).
8. Walther, David. Applied Kinesiology: Synopsis, Systems DC. Pueblo, Colorado (1998).
9. Belli, Richard. ICAK-U.S.A. Proceedings of the Annual Meeting (1989–1995).

Extremity Subluxation Correlation Part II: TMJ

Timothy Francis, M.S., D.C., DIBAK, D.H.M.

Abstract

Temporomandibular joint (TMJ) dysfunction may display as symptoms almost anywhere in the body. This aberrant TMJ motion may at times be related to extraspinal subluxations.

Introduction

According to Penfield and Rasmussen, 35 – 40% of all motor and sensory nerves of the body are related to the TMJ. Thus TMJ dysfunction has far reaching effects on overall body function. A few of these would include but are not limited to cranial-sacral primary respiratory motion, leg length discrepancies, pelvic categories, gait imbalances, upper cervical subluxations-fixations, P.L.U.S. patterns, etc. Goodheart, Leaf, Gelb, and many others have been researching and demonstrating the relationship between the TMJ and remote body function for over twenty-five years via applied kinesiology. TMJ dysfunction may be caused by a variety of different things, including extremity subluxations.

Discussion

There are basically two different methods of diagnosing TMJ dysfunction via manual muscle testing; without T.L. (therapy localization) to the TMJ and with T.L. to the TMJ. Starting with a strong indicator muscle the subject is asked to clench his/her teeth together. If this creates a weakening of the strong indicator muscle, traditionally some type of occlusal and/or tooth problem exists. This author has not correlated an extremity subluxation for this pattern. Next the subject is asked to open wide and again the indicator muscle is tested. If positive, (indicator muscle weakens), then this demonstrates a need to fascial flush one or more of the TMJ closing muscles (masseter, temporalis, internal pterygoid). However, also check the extremities for an extraspinal subluxation including the glenohumeral joint, navicular, and/or cuboid. The patient at this point is usually asked to open/close the mouth slowly and then rapidly without letting the teeth touch each other to check for an aerobic/anaerobic involvement. If positive then iron/pantothenic acid is given to negate the positive test. Extraspinally check for a patellar subluxation.

The subject is now requested to T.L. the TMJ without movement. This should create no weakening of the strong indicator muscle. If positive, then this usually indicates disc pathology or a small intestine involvement. Extremity subluxation involvements may include a posterior tibia, talus, cuboid, and/or a superior third cuneiform. The patient is then requested to clench his/her teeth together while therapy localizing the TMJ. If positive, we then have the patient T.L. to the three closing muscles while biting down. If positive the spindle cells are turned down manually to the muscle belly. Extraspinal subluxations related to this may be a slipped bicipital tendon, and/or femur head involvement. The subject is then asked to open the mouth while continuing to therapy localize to the TMJ. If positive, one side is therapy localized at a time and the appropriate inferior division of the external pterygoid spindle cells are manually turned down. From an extraspinal viewpoint, check for a medial olecranon or a talus subluxation. Once again, continuing to T.L. the TMJ, the subject is asked to lateralize the jaw to the right and then to the left. If positive, to the left, for example, then the TMJ is therapy localized one side at a time. If positive therapy localization to the left TMJ while lateralizing the jaw

to the left, then the left posterior temporalis spindle cells are turned down. If positive therapy localization to the right TMJ while lateralizing to the left, then the right internal pterygoid spindle cells are turned down. Extremity subluxation correlations include a femur head, posterior tibia, and/or a posterior calcaneus. The patient is then requested to protrude the jaw and if positive then each side is therapy localized to determine involvement. The inferior division of the external pterygoid spindle cells are then turned down to the involved side(s). Extremity subluxations to be investigated would be the posterior tibia, posterior calcaneus, anterior distal tibia, cuboid, and/or a superior third cuneiform. Finally, the patient is requested to retrude the jaw. If positive, therapy localize to the masseters and then to the posterior temporalis muscles while retruding the jaw. The appropriate spindle cells are then manually turned down. Extraspinal involvement would include the glenohumeral joint, posterior radial head, fibular head, superior second cuneiform, and/or superior third cuneiform.

In summary the following patterns have been observed.

TMJ Dysfunction	Extremity Subluxation
W/Out T.L. to TMJ	
Closing	N/A
Opening	Glenohumeral Joint Navicular Cuboid
Open/Close w/out Touching teeth (Movement is Slow/Rapid)	Patella
W/ T.L. to TMJ	
Neutral	Tibia Talus Cuboid 3rd cuneiform
Close	Bicipital tendon Femur head
Open	Olecranon Talus
Lateralization	Femur head Tibia Calcaneus
Protrusion	Tibia Calcaneus Tibia (distal) Cuboid 3rd cuneiform
Retrusion	Glenohumeral Radial head Fibular head 2nd cuneiform 3rd cuneiform

Conclusion

The extremity subluxations presented here have an effect on TMJ dysfunction as correlated. When the extraspinal subluxations are the cause of TMJ dysfunction, this has a profound and wide-ranging effect on total body function and harmony. Correction of the extremity subluxation is of paramount importance at certain times in treating a patient from a holistic perspective. To find the cause is the highest good (Hippocrates).

References

1. Francis, Timothy D., Upper Extremity Subluxation/Muscle Syndrome Correlations. Experimental observation of the members of the I.C.A.K. Volume I (1999–2000).
2. Ibid, Lower Extremity Subluxation/Muscle Syndrome Correlations. Experimental observations of the members of the I.C.A.K. Volume 1 (1999–2000).
3. Ibid, Spinal – Rib Subluxation/Muscle Syndrome Correlations. Experimental observations of the members of the I.C.A.K. Volume 1 (1999–2000).
4. Ibid, Spinal Subluxation / Bilateral Muscle Syndrome Correlations. Experimental observations of the members of the I.C.A.K. Volume 1 (2000–2001).
5. Ibid, Additional Fixation Patterns. Experimental observations of the members of the I.C.A.K. Volume 1 (2001–2002).
6. Goodheart, George J. You'll Be Better, The Story of Applied Kinesiology. AK Printing; Geneva, Ohio.
7. Leaf, David. Applied Kinesiology Flow Chart Manual 3rd Edition. Privately Published (1995).
8. Walther, David. Applied Kinesiology: Synopsis, Systems DC. Pueblo, Colorado (1998).
9. Gelb, Harold, DMD. Clinical Management of Head, Neck and TMJ Pain and Dysfunction. W.B.Saunders. (1977).

Extremity Subluxation Correlation Part III: Neurological Disorganization

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Abstract

Neurologic disorganization describes a group of phenomenon whereby a subject's body does not respond to manual muscle testing in a predictable prescribed manner. Extremity subluxations may induce and cause neurologic disorganization from a structural perspective.

Introduction

Neurologic disorganization may manifest itself in body dysfunction in a variety of ways. For example: dyslexia, stuttering, clumsiness, and schizophrenia, just to mention a few. There are various methods in applied kinesiology to test for neurologic disorganization such as ocular lock, hyoid challenge, right–left brain activity, psychological reversal, umbilical reversal, K27 and cross K27 period. For most of the aforementioned, there exists an extraspinal subluxation correlation.

Discussion

Early on in applied kinesiology, a phenomenon was observed by Goodheart and given the label: switching. In those patients displaying this particular pattern, for example, on postural analysis, a high shoulder on the right would be observed, however it was the left latissimus dorsi that would manually muscle test weak. Hence the word switching. These patients would also T.L. (therapy localize) to K27 (junction of clavicle, sternum, and first rib). Right hand to right K27 and left hand to left K27 period. Originally K27 was stimulated (rubbed manually) by the physician while at the same time CV8 (umbilicus) was also stimulated. Both right and left K27 / CV8 were rubbed. This temporarily unswitched the patient. Although this procedure did not derive the cause of switching, it would nevertheless reorganize the patient's body long enough to allow treatment. There is a long list of extremity subluxations that may be involved here. Shoulder, wrist, hip, knee, and foot subluxations have been found to be involved. Please refer to the chart at the end of this discussion.

Later on it was found that some subjects did not T.L. to the right K27 with the right hand and left K27 with the left hand. But would T.L. with the right hand to left k27 and the left hand to right K27. This was called Cross K27 switching. These patients would also display universal muscle weakness after doing a cross crawl pattern and yet would strengthen following a homolateral crawl pattern. Extraspinal subluxations that may have an influence here are the scapula, clavicle, glenohumeral joint, axillary lift maneuver, medial olecranon, posterior radial head, scaphoid, jammed carpals, femur head, posterior tibia, laterally rotated tibia, and a patellar subluxation.

Ocular lock is yet another test in applied kinesiology for neurologic disorganization. The subject's eyes are held either straight superior/inferior, right/left, up to the right/left, down to the right/left and a strong indicator muscle is tested for weakening. This author has previously written a paper correlating spinal subluxations to the various eye positions. Extraspinal subluxations to be considered are jammed carpals, posterior fibular head, and/or the talus.

The hyoid is a bone under the mandible suspended by 16 different muscles. It has also been implicated in the neurologic disorganization phenomenon. Goodheart has correlated the hyoid to many things one of which is right/left brain testing. That is, if there is a positive challenge to the hyoid (usually a static type challenge is done), then the subject will also exhibit weakening to right brain activity (music) and/or left brain activity (mathematics). Typically the spindle cells are turned down to the hypertonic hyoid muscle. Extraspinal subluxations to be screened here is the proximal clavicle.

Right-left brain testing may be performed in various ways. Right brain (music) activity may weaken muscles on both the right and left sides of the body or just the left side or only the right side. Left brain activity testing (math) may weaken muscles on both the left and right sides or only in the right or only in the left (only contralateral or only ipsilateral). There are various nutrients correlated to all of these conditions. Extremity subluxations also play a role here but are so vast and varied that any attempt at a list would prove inadequate.

Psychological reversal is based on Callahan's observations. A statement such as "I totally and completely accept myself with all my faults and shortcomings" is stated and a strong indicator muscle is tested for weakening. If positive, the subject repeats the statement while the appropriate B & E point (yang meridian points ending on the face) are tapped (the point that negated the weakening). Extremity subluxations to be investigated are the scapula, scaphoid, jammed carpals, posterior fibular head, talus, and/or superior 3rd cuneiform.

Umbilical reversal was originally Diamond's observation and development. A strong indicator muscle is tested, then the patient's fingers are inserted into his/her umbilicus (test should be negative), then the doctor's fingers are inserted into the patient's umbilicus (test should be negative), then the patient's palm is placed on the doctor's palm (should be negative) but when the doctor places his/her fingers into the patient's umbilicus, and then the patient places his/her palm on the doctor's palm the test if positive, is labeled umbilical reversal. The patient will weaken to a belief statement stated such as god is good. Diamond recommends a nutritional approach including RNA, choline, and a brain tissue extract. No extraspinal subluxation has been correlated by this author at this time.

In summary the following patterns have been observed.

Neurological Disorganization	Extraspinal Subluxation
K27	Shoulder Wrist Femur head Knee Foot
Cross K27	Shoulder Elbow Wrist Femur head Knee
Ocular lock	Jammed carpals Fibular head Talus

Neurological Disorganization	Extraspinal Subluxation
Hyoid	Clavicle
Rt/Lt brain activity	Shoulder Elbow Wrist Femur head Knee Foot
Psychological Reversal	Scapula Scaphoid Carpals Fib head Talus Third cuneiform
Umbilical Reversal	N/A

Conclusion

Extremity subluxations have an influence in body organization as exhibited by the various applied kinesiology tests for neurologic disorganization. These extraspinal subluxations are often times the structural cause of a patient's body dysfunction and must be corrected for return to optimum health and balance within the nervous system.

References

1. Francis, Timothy D., Upper Extremity Subluxation/Muscle Syndrome Correlations. Experimental observation of the members of the I.C.A.K. Volume I (1999 – 2000).
2. Ibid, Lower Extremity Subluxation/Muscle Syndrome Correlations. Experimental observations of the members of the I.C.A.K. Volume 1 (1999 – 2000).
3. Ibid, Spinal – Rib Subluxation/Muscle Syndrome Correlations. Experimental observations of the members of the I.C.A.K. Volume 1 (1999 – 2000).
4. Ibid, Spinal Subluxation / Bilateral Muscle Syndrome Correlations. Experimental observations of the members of the I.C.A.K. Volume 1 (2000 – 2001).
5. Ibid, Additional Fixation Patterns. Experimental observations of the members of the I.C.A.K. Volume 1 (2001- 2002).
6. Goodheart, George J. You'll Be Better, The Story of Applied Kinesiology. AK Printing; Geneva, Ohio.
7. Leaf, David. Applied Kinesiology Flow Chart Manual 3rd Edition. Privately Published (1995).
8. Walther, David. Applied Kinesiology: Synopsis, Systems DC. Pueblo, Colorado (1998).
9. Callahan, Roger. Five Minute Phobia Cure: Dr. Callahan's Treatment for Fears, Phobias, and Self-Sabotage. Enterprise Publishing, Inc., Wilmington (1990).
10. Diamond, John. ICAK-U.S.A. Proceedings of the Annual Meeting (1976-2002).

Extremity Subluxation Correlation Part IV: Chart Summary

Timothy Francis, M.S., D.C., DIBAK, D.H.M.

Abstract

Extremity subluxations do exert a profound influence on total body organization and function.

Introduction

The following chart is given as a quick reference summary to the prior three papers.

Discussion

Jammed Carpals
- Ocular lock
- Psychological reversal
- Roll
- Yaw Thoracolumbar
- Rib cage decreased passive ROM R -> L

Scaphoid
- Psychological reversal
- Piriformis Gait Inhibition
- Category I
- Roll
- Shoulder passive ROM decreased L
- Rib cage passive ROM decreased clockwise

Medial Olecranon
- Pitch
- Passive ROM decreased left leg
- DTR's (upper)

Posterior Radial Head
- Yaw Sacrum
- Jaw retrusion
- Passive ROM decreased torso R –> L
- Passive ROM decreased rib cage counterclockwise

A-C Joint
- Upper Gait
- Yaw Occiput
- Tilt

Scapula
- Psychological Reversal
- Roll
- DTR's (lower)

Proximal Clavicle
- Hyoid
- Limbic fixation

Glenohumeral Joint
- TMJ open w/out TL
- TMJ retrusion w/ TL

Bicipital Tendon
- TMJ closing w/ TL

Femur Head
- TMJ closing w/ TL
- P.L.U.S.
- TMJ lateralization w/ TL
- Passive ROM decreased torso L –> R
- DTR's (lower)

Tibia
- Upper Gait
- Piriformis Gait
- Limbic fixation
- TMJ neutral TL
- TMJ lateral W/ TL
- TMJ protrusion w/ TL
- DTR's (upper / lower)

Fibular Head
- Ocular lock
- Psychological Reversal
- Category I
- Yaw Occiput
- TMJ retrusion w/ TL
- Pincer Palpation

Patella
- TMJ aerobic/ anaerobic

Calcaneus
- Upper Gait
- Piriformis Gait
- Limbic fixation
- Yaw Sacrum
- TMJ lateralization w/ TL
- TMJ protrusion w/ TL
- Passive ROM decreased rib cage counterclockwise

Talus
- Ocular lock
- Psychological Reversal
- Piriformis Gait Inhibition
- P.L.U.S.
- Limbic fixation
- Category II
- Yaw Sacrum
- Yaw Thoracolumbar
- TMJ neutral w/ TL
- TMJ open w/ TL
- TMJ protrusion w/ TL
- Passive ROM decrease rt. shoulder
- Pincer Palpation
- Passive ROM decreased rib cage counterclockwise
- DTR's (upper)

Distal Tibia
- TMJ protrusion w/ TL

Navicular
- TMJ open w/out TL
- Passive ROM decreased rib cage counterclockwise
- Limbic Fixation

Cuboid
- P.L.U.S.
- Yaw Occiput
- Yaw Thoracolumbar
- TMJ open w/out TL
- TMJ neutral w/ TL
- TMJ protrusion w/ TL
- Passive ROM decreased rib cage counterclockwise
- DTR's (upper)

1st Cuneiform
- Category III

2nd Cuneiform
- P.L.U.S.
- Yaw Thoracolumbar
- TMJ retrusion w/ TL
- Passive ROM decreased left leg
- Passive ROM decreased torso left to right
- DTR's (upper)

3rd Cuneiform
Psychological Reversal
Category II
Yaw Thoracolumbar
TMJ neutral w/ TL
TMJ protrusion w/ TL
TMJ retrusion w/ TL
Passive ROM decrease rt. shoulder
Passive ROM decrease lt. shoulder

Laterally Rotated Metatarsals
Category II

Dropped Metatarsal Heads
Yaw Thoracolumbar
Decreased passive ROM left leg

Conclusion

Both upper and lower extremity subluxations have a deep and profound effect on overall body function. It is of utmost importance to diagnose the need, supply the need, and observe the results (Goodheart).

References

1. Francis, Timothy D., Upper Extremity Subluxation/Muscle Syndrome Correlations. Experimental observation of the members of the I.C.A.K. Volume I (1999–2000).
2. Ibid. Lower Extremity Subluxation/Muscle Syndrome Correlations. Experimental observations of the members of the I.C.A.K. Volume 1 (1999–2000).
3. Ibid. Spinal – Rib Subluxation/Muscle Syndrome Correlations. Experimental observations of the members of the I.C.A.K. Volume 1 (1999–2000).
4. Ibid. Spinal Subluxation / Bilateral Muscle Syndrome Correlations. Experimental observations of the members of the I.C.A.K. Volume 1 (2000–2001).
5. Ibid. Additional Fixation Patterns. Experimental observations of the members of the I.C.A.K. Volume 1 (2001–2002).
6. Ibid. Extremity Subluxation Correlation Part I: Spine Dysfunction. Experimental observation of the members of the I.C.A.K. (2002–2003).
7. Ibid. Extremity Subluxation Correlation Part II: TMJ. Experimental observation of the members of the I.C.A.K. (2002–2003).
8. Ibid. Extremity Subluxation Correlation Part III: Neurologic Disorganization. Experimental observation of the members of the I.C.A.K. (2002 – 2003).
9. Goodheart, George J. You'll Be Better, The Story of Applied Kinesiology. AK Printing; Geneva, Ohio.
10. Leaf, David. Applied Kinesiology Flow Chart Manual 3rd Edition. Privately Published (1995).
11. Walther, David. Applied Kinesiology: Synopsis, Systems DC. Pueblo, Colorado (1998).

Ligament Laxity Technique

G. Kingman Fung, D.C.

Abstract

Ligament laxity has been most often treated through a nutritional protocol. Ligament interlink technique has been successful as a non-direct, structural treatment for acute ligament problems. This paper explains a direct, structural technique which will further contribute to the healing of ligamentous laxity in joints.

Introduction

The main treatment for ligament laxity has been supplementing with whole adrenal, manganese, minerals and other adrenal nutrients. In exploring techniques that could make a difference in the ligament laxity condition, structure, emotion and meridians were investigated. The resulting technique combines approximating the ligament in question while stimulating the Hsi cleft point related to the organ or gland. This is achieved while adding an emotional component.

Methods and Summary of Procedures

Pre-testing for Ligament Laxity Technique

1. The doctor tests the joint for ligament stretch reaction. (Joint should be able to stretch in all motions without a strong indicator weakening.) If there is a strong indicator weakening, then the joint suffers from ligament laxity.

2. After determining the presence of ligament laxity, there is a small window of time in which the strong indicator will be inhibited. The doctor must immediately T.L. (therapy localize) to a neurolymphatic of an organ or gland that will strengthen the previously weakened indicator.

3. Using the emotions related to the Chinese meridians, the doctor then verbally runs the emotions related to that organ or gland until the indicator re-weakens.

4. At this point, the Dr. disengages the T.L. from the neurolymphatic organ or gland and the doctor re-tests the joint for ligament stretch reaction. Upon the weakening of the indicator muscle, the doctor has the patient T.L. to the Hsi cleft point related to the earlier organ.

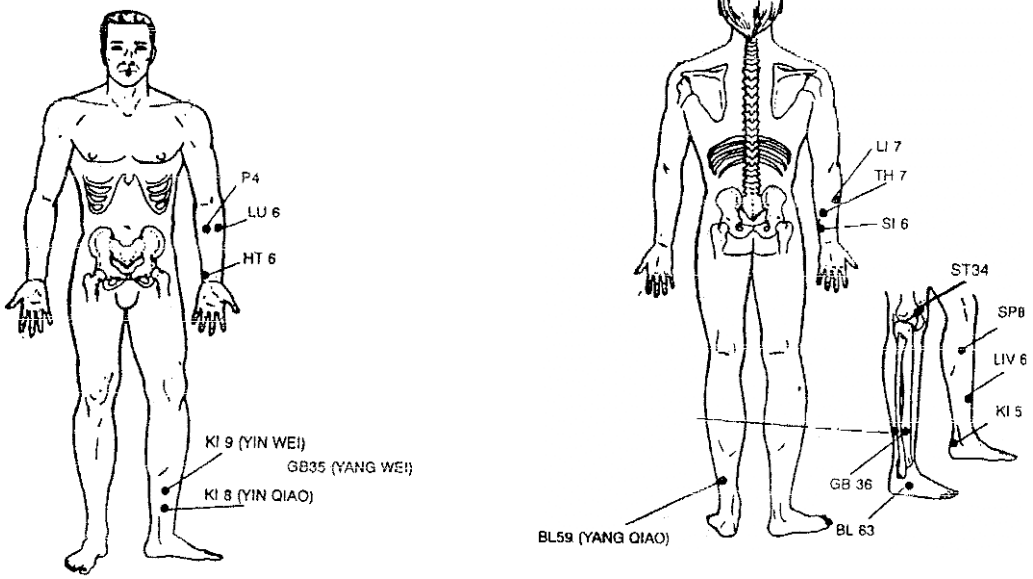
Treatment

The doctor approximates the joint while patient or doctor stimulates the Hsi cleft point and the patient visualizes or thinks about the *positive emotion*, or *the opposite emotion* to that which negated the earlier T.L. This is held for 30 to 60 seconds. A re-test of the ligament stretch reaction should now be negative.

Discussion

Ligament Laxity causes a diffuse, sometimes deep aching in the surrounding muscles related to that joint, because these surrounding muscles are constantly working to assist the ailing joint. This condition can also cause these overworked muscles to become spastic, sometimes resulting in severe, restrictive pain. Approximating the joint induces a 1A afferent stretch and allows the body to be aware of that joint neurologically. The positive

Xi–Hsi Cleft Points



thought related to the meridian increases the frequency of firing of cortex and further depolarizes, or pushes it farther away from threshold and the Hypothalamic-Pituitary-Adrenal axis. In Chinese medicine, the Hsi cleft points are stimulated in some life-threatening situations when the electrical resistance of the body is extremely low. My belief is that the electrical resistance of the body is low in effected joints. By stimulating the Hsi cleft point, we are able to rebalance the electrical resistance in that meridian or area.

Conclusion

The competent AK practitioner is constantly in search of ways of utilizing additional treatments for Ligament Laxity. With this combination and the nutritional approach, you may find that you will be able to resolve ligament stretch reaction more efficiently.

References

1. Walther, David, *Applied Kinesiology Synopsis, 2nd Edition,* Pueblo Colorado Systems D.C. 2000, p. 211 - 213.
2. Walter, Scott Neuro Emotional Technique Master Card, Privately Published, Encinitas, CA (1993).
3. Worsley, J.R. *Acupuncture Charts*, South Colton, NY, Acupuncture (1983).

The Other 49% of the 51%er

Stephen C. Gangemi, D.C.

Abstract

A 51%er in applied kinesiology muscle testing is when the patient therapy localizes (TL) to the correlating muscle Chapman's neurolymphatic reflex (NL) to reveal a hidden muscle inhibition.[1] This paper will propose that a 51%er is actually a need to first treat an injury or immune issue to resolve the 51%er problem.

Introduction

When a 51%er is found during a muscle testing procedure, the known treatment has been to address that muscle as if it were "weak in the clear," even though the patient has to TL to corresponding muscle/organ NL. While the patient then holds the TL to the respective NL, the appropriate treatment to the organ or muscle would be identified and applied, thereby causing the muscle to function normally. However, the question arises as to why the involved muscle needs this extra receptor stimulation to get a neurological inhibitory response.

Discussion

There is a logical reason why a patient will have a muscle 51%er and why he/she will not. A 51%er indicates a need to address another underlying condition first. The patient will have either an injury that needs to be treated using Injury Recall Technique (IRT),[2] and/or an immune circuit that needs to be treated. After all the injuries and immune issues are resolved, the muscle will either be inhibited without the TL to the NL, ("weak in the clear"), or will show normal facilitation. Treating the immune system and/or the "hidden" injuries is the missing 49% of the 51%er. Therefore, when the physician identifies a muscle 51%er, he or she needs to take a step back and fix the pattern causing the 51%er first, rather than treat the 51%er.

One of the most common reasons for a 51%er is an injury somewhere in the body. The injury or injuries must be corrected using IRT. As the need to perform IRT is indicated by a muscle that does not strengthen with autogenic facilitation, (stretching the spindle cell), the same rule applies to this 51%er principle. When a 51%er is identified, simply stretch the spindle cell of the muscle being tested. If the muscle does not strengthen, then there is an injury that needs to be treated using IRT. If that was the only involved injury and there is no immune involvement, then the 51%er will either now show regular inhibition, or the muscle will be functioning normally (facilitated). Therefore, a 51%er often indicates that a "hidden" injury is present.

The other reason for a 51%er is an immune involvement, either to the thymus (at the upper sternum), lower sternum (chemical hypersensitivities area), or spleen. The need to investigate the immune system will occur when there is a 51%er and the muscle shows normal facilitation with stretching of the spindle cell. As Schmitt notes, the only exception to this would be if there is an injury to the muscle itself which is being tested. There are a few ways to go about finding out which immune circuit is involved in the 51%er.

One way to check for immune involvement is to check the involved muscles relating to immune system – infraspinatus for the thymus, lower-middle trapezius for the spleen, and pectoralis minor for the [lower sternum] chemical sensitivities.[3]

Another way to check for immune involvement is to simply check the visceral referred pain (VRP) areas for the correlating immune circuits. The VRP area for the thymus is over the skin of the right first rib.[4] There are many references for the spleen's VRP area to be above the left shoulder, over the skin of the AC joint. There isn't a documented VRP area for the lower sternum, but rubbing or pinching over the NL[5] has been found to elicit a response. Rubbing (parasympathetic) and pinching (sympathetic) over each area will indicate which immune circuit, or circuits, needs to be treated.[6] Screening for an immune involvement this way lets the physician use the same 51%er muscle.

A third way to check for immune involvement is to perform pre-test imaging.[7] Schmitt has shown that pre-test imaging signals the presence of a cranial fault. This is usually secondary to an immune system problem and the need for immune treatment.[8] If pre-test imaging is positive, then therapy localizing to the involved cranial fault will in turn lead the physician to the immune system. While the patient TLs to the involved cranial fault with head in extension, (as if checking for an IRT problem), the physician can tap over the upper sternum for the thymus, lower sternum (over the NL), and spleen (over the NL) to see which immune circuit is involved. Tapping the involved immune circuit will negate the positive TL with the head in extension.

After the appropriate therapy is applied towards the immune system and there are no injuries (at least affecting the 51%er), then the 51%er will now either be inhibited without therapy localization to the NL or the muscle will no longer be inhibited at all. In other words, the missing 49% has been addressed.

The "occasional" exception to this finding is if the 51%er is of an immune system related muscle – the infraspinatus, low/mid trap, or pec minor. If this is the case, and AF does not facilitate, then the muscle should be treated with IRT. If there is no injury, (AF strengthens), then there will be a TMJ involvement. Have the patient TL to each of the TMJs. Usually the right TMJ will TL for the thymus and the left TMJ will TL for the lower sternum or spleen.[9] The treatment to the TMJ is most often a need to perform origin-insertion (with IRT) to either the internal or external ptygeroid muscles. Sometimes there is a tooth involvement that must be treated. After correcting the TMJ and/or tooth problem, the immune related muscle will either now no longer be inhibited or it will no longer need the therapy localization to be inhibited. The absence of the 51%er means that it is now appropriate to treat the muscle or organ (in this case the immune system).

Conclusion

A 51%er indicates that the muscle/organ should not be treated until the reason for the 51%er is resolved. The reasons for 51%ers are immune issues and injuries. There could also be a jaw involvement. Finding and treating the injuries and/or immune system will either resolve the 51%er muscle, or resolve the muscle inhibition altogether.

Procedure

1. A 51%er is found
   a. AF (spindle cell) has no effect
      i. Correct injury or injuries
      ii. Muscle is still a 51%er
         1. AF strengthens (if negative then there is still an injury)
            a. Check the immune system (1b)
      iii. Muscle is not a 51%er
         1. If still inhibited, treat as you normally would

   b. AF strengthens – immune involvement (unless the muscle itself is injured)
      i. Use the VRPs, [correlating] immune related muscles, or pre-test imaging to find the involved immune circuit to treat
      ii. After the immune system is treated, muscle is not a 51%er
         1. If still inhibited, treat as you normally would
      iii. After the immune system is treated, muscle is still a 51%er
         1. Check for another immune involvement
         2. Check for another injury with AF
         3. Check for TMJ involvement

References

1. Walther, David S. Applied Kinesiology: Synopsis, 2nd edition, Systems DC. 2000. p. 613.
2. Schmitt, Walter H., Jr. Injury recall technique. Chiropractic journal of NC Vol VI, 1990. pp. 25–30.
3. Walther, David S. Applied Kinesiology: Synopsis, Systems DC. 1988. pp. 306–323.
4. Gangemi, Stephen C. The Thymus Visceral Referred Pain Area. Proceedings of the ICAK - U.S.A. 2003–2004.
5. Walther, David S. Applied Kinesiology: Synopsis, Systems DC. 1988. p. 313.
6. Schmitt, Walter H., Jr. The somatic window on neurological function, part 1. Proceedings of the ICAK - U.S.A. Volume 1, 1999–2000. p.141.
7. Schmitt, Walter H., Jr. Pre-test imaging: a screening test for cranial faults, I.C.A.K. collected papers. Summer, 1987.
8. Schmitt, Walter H, Jr. The Uplink Issue #17, Spring, 2002.
9. Ibid.

The Thymus Visceral Referred Pain Area

Stephen C. Gangemi, D.C.

Abstract

Every organ has a cutaneous area which it refers pain to. Visceral referred pain (VRP) areas exist throughout the body. The location of the thymus VRP will be proposed.

Introduction

Visceral problems will result in, (most commonly), a muscle inhibition pattern.[1] Activating a VRP area with some type of sensory stimulation, usually rubbing (mechanoreceptor stimulation) or pinching (nociceptor stimulation), will elicit a muscle response if the VRP is related to the organ with a problem. The VRP areas are extremely useful in guiding the physician whether to perform more sympathetic (pinching strengthens) or parasympathetic (rubbing strengthens) activity.[2] While [most] all major organs have identified referred pain areas, there is none currently known for the thymus gland.

Discussion

The thymus, along with the spleen and gut associated lymph tissue, (GALT), account for the majority of an individual's immune health. Although there are many references for the spleen's VRP area to be over the left shoulder/acromioclavicular joint, one does not [currently] exist for the thymus. Knowing when to provide more sympathetic or parasympathetic activity to the immune system is important. The author has found a referred pain connection for the thymus over the skin of the right first rib, both anterior and posterior.

The Chapman's neurolymphatic reflex (NL) for the thymus as reported by Schmitt is over the right ribs, 4-6, from the axillary line anterior to the midmamillary line.[3] Therefore, if a thymus involvement is present, this NL should therapy localize (TL). Whether to rub the NL to create a net parasympathetic response or to perform visceral challenge technique[4] to create a net sympathetic response requires the use of the VRP. Rubbing or pinching over the skin over the right first rib will guide the physician towards the treatment necessary to improve the thymus, and therefore immune, function.

If there is an inhibition of the infraspinatus and rubbing over the VRP causes facilitation, then there is a need for more parasympathetic activity. Rubbing over the NL and/or providing the necessary supplement(s) to support the thymus will accomplish this. If pinching over the thymus VRP negates the inhibition, this would indicate a need for more sympathetic activity. Visceral challenge technique with the appropriate offender will correct this problem. Having the patient TL to the thymus NL with the offender on the tongue, and performing IRT to the taluses bilaterally will negate the infraspinatus inhibition. The most common irritants to the thymus gland are cortisol, trans fats, sugar, allergies, and caffeine.[5]

Conclusion

There is an apparent thymus visceral referred pain area which exists over the right first rib area both anterior and posterior. This area may be used to determine the need to rub the thymus Chapman's NL reflex or to perform visceral challenge technique to the thymus.

References

1. Goodheart, George J. Applied kinesiology 1968 workshop procedure manual. Detroit: privately published, 1968.
2. Schmitt, Walter H., Jr. The functional neurology of referred pain. ICAK collected papers, Summer, 1989. p. 229.
3. Schmitt, Walter H., 100 Hour AK Course Session 6 notes. February 2003.
4. Schmitt, Walter H., Jr. The somatic window on neurological function, part 1. Proceedings of the ICAK - U.S.A. Volume 1, 1999-2000. p. 141–148.
5. Schmitt, Walter H., Jr. Common Glandular Dysfunctions in the General Practice. Chapel Hill, NC: privately published, 1981.

Injury Recall Technique Revisited

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Abstract

The Injury Recall Technique (IRT) was first introduced to the chiropractic profession and ICAK in 1990[1, 2] by Walter Schmitt. This technique addresses withdrawal reflex muscular imbalances that may persist for years or decades after the original injury. This paper discusses the great efficacy the author has noted in difficult chronic cases. Also presented are observations regarding the effect of IRT on wounds and scar tissue that have incompletely healed even years after the original injury or surgery.

Key Words: Injury recall, Surgery, Laser, Withdrawal Reflex, Scar Tissue

Introduction

The Injury Recall Technique was first introduced to ICAK in 1990 by Wally Schmitt.[2] He presented it as a variation on a technique he had learned from an innovative podiatrist. The IRT has been invaluable in my practice for addressing neuromuscular consequences of various injuries and surgeries. These consequences, as described below, frequently persist for years or decades after the initiating event.

Discussion

We can easily observe there is a withdrawal reflex which is activated in response to injuries. For instance, if you burn your hand, you don't need to consciously recognize that your hand is in a flame, smell it cooking and make a decision whether to leave it there or not. The withdrawal reflex acts immediately to contract muscles such as the rhomboids, latisimus dorsi, middle trapezius and posterior deltoid and inhibit associated antagonists to whisk your hand out of harms way. Of course it is still possible to make a conscious decision to leave your hand in the flame but you have to work to override the withdrawal reflex to do it. This withdrawal reflex becomes activated even if the individual is anaesthetized as during surgery. I had a chance to observe this myself several years ago when my wife was anaesthetized to set a bad fracture and displacement of the radius. Even though she was completely unconscious, her arm and shoulder contracted in response to the nociception associated with the setting maneuver.

The above paragraph describes a normal reaction to injury. Normal, that is, *if* the withdrawal reflex extinguishes once the involved body part is out of harms way. The IRT was developed to deal with an abnormal situation wherein the withdrawal reflex persists for a prolonged period after it is no longer needed. Thus we may see, using the above example, that there is a subtle over-facilitation of the rhomboids, latisimus dorsi, middle trapezius and posterior deltoid and corresponding antagonist inhibition that persists for years or decades after the initiating injury. This creates imbalances in the joints and other musculoskeletal problems that will not respond in any lasting way to our many otherwise wonderful therapeutic interventions. Why this reflex should persist in some cases is not clear but it seems likely that the severity or invasiveness of the injury (I find IRT needed after well over 95% of surgeries) and the overall vitality of the patient at the time of the injury are important determining factors. I routinely check for the IRT in any difficult structural problems. I also follow Dr. Schmitt's advice[2, 3] and have new patients mark the site of any surgeries, fractures, burns or serious contusions on a body diagram.

IRT can often be the key to dramatic improvement in a difficult case. It is common for me to see cases that have plateaued at an unsatisfactory level, start making good progress again after the IRT injury sites have been cleared. One aspect I greatly appreciate is that IRT rarely has to be performed more than once for a particular injury. This makes IRT particularly appropriate for patients who travel a long distance or who may be seen only a few times for some other reason.

In addition to clearing out an inappropriate withdrawal reflex that is interfering with structural balance, I have observed that, in many cases, IRT has allowed more complete healing of scar tissue. Three cases were especially impressive.

The first example was my wife, Karen. When I learned about the IRT at the Summer, 1991 meeting, Karen was there with me. She had a scar across her lower abdomen from a caesarian section sixteen years previously that was thick, ropy and tender on palpation. I remember having her lay down on the floor of the meeting room after Dr. Schmitt's presentation, testing and treating her for the IRT. Not only did we see immediate and long-lasting improvement in her abdominal muscle strength but six months later the scar was barely palpable and no longer tender to digital pressure. We had done nothing else that could account for a change in her scar and it seems unlikely that, after sixteen years, some process that "would have happened anyway" caused the scar to finish healing.

The second example was a twenty three year old woman who had extensive scoliosis surgery ten years earlier. The surgery had left a scar from the third thoracic down to the fifth lumbar vertebra. The patient was a student from out of state visiting her parents on the school break and seemed like a perfect candidate for the IRT. I found the need for IRT and treated it with gratifying improvement in several muscle groups. When I next saw her, three months later, she informed me that she had experienced numbness along the length of the scar for the last ten years. She went on to tell me that the numbness was now gone and that she had normal feeling along the scar path for the first time since the surgery!

The third example is a 44 year old man. He suffered from an autoimmune condition which had resulted in multiple (three) corneal transplants. After the last surgery, he related that the surgeon had told him he would probably have a permanent "wrinkle" across his field of vision from the surgery. I found that the IRT was needed for both eyes and treated them. Three weeks later he told me that a few days earlier his vision had gone blurry for about thirty minutes and then cleared. He was very pleased to report that the "wrinkle" across his vision was gone and his eyesight was better than it had been since the surgery.

Below you will find a review of the basic IRT protocol as presented by Schmitt with an addition from my own experience denoted with an asterisk.*

I. Starting with an area of obvious injury

   A. Stimulate the injury site/scar
      1. Pinching to stimulate nociception
      2. Therapy localize to the area
      3. Shine a laser beam over the area*

   B. Immediately after stimulation, test a previously strong muscle (all tests are doctor initiated unless otherwise specified)
      1. If inhibition (weakening) is noted, treat to re-facilitate
      2. If no inhibition is noted, proceed to step C below

   C. Repeat the above stimulation, followed quickly by a light cephalward tug on the ipsilateral talus bone (the purpose is to approximate the talo-maleolar articulation)
      1. Test a previously strong muscle. Weakening = need for IRT

D. IRT Treatment
   1. Stimulate the injury site as above
   2. Immediately tug gently caudally on the ipsilateral talus bone

E. For head injuries
   1. Have the patient extend the head on the table and test strong muscle
      a. If inhibition is noted, treat for extension fixation of occiput on atlas or other structural fault
   2. If step 1. Above does not cause inhibition:
      a. Stimulate the head injury site and immediately have the patient extend their head
         (1) inhibition of strong muscle = need for IRT
   3. Treatment
      a. Stimulate the injury area followed by gentle passive flexing of the patient's head, 2 or 3 times

F. Repeat test to verify correction

II. Starting with a weak muscle or inhibition caused by therapy localization or challenge

A. Repeat the test with patient initiation and doctor response as soon as patient pressure is felt (sub-maximal contraction)
   1. If muscle changes to strong or facilitated, treat with other techniques
   2. If muscle stays weak or inhibited = IRT *may* be cause of the weakness
      a. Rub various injury areas or suspected injury areas and retest above muscle
      b. Temporary re-facilitation = need for IRT to site that was rubbed

B. Proceed as in I. A.>F. Above

C. Retest original area of weakness or inhibition to verify correction

In the above outline, I mention use of a laser as an alternative method of stimulation to an injury site. I have found that a laser in the 630-650nm range for stimulating an injury site, provides results comparable to stimulation by pinching or therapy localization. A laser is useful for treating areas that are too tender or unhealed to pinch or therapy localize. Lasers are also useful for stimulating hard to reach areas such as dental extraction sites. Although very sophisticated lasers with variable frequency settings are available, I have found that a simple laser pointer is effective for IRT therapy, if it produces the proper wavelength.

There may also be an additional advantage to using a laser for IRT stimulation. Lasers have the ability to stimulate at a deep level. Some sources state that laser energy penetrates far deeper than the visible fraction of the laser beam.[4] I have found that, after treating a deep injury site with surface stimulation via pinching, I will sometimes be able to elicit the IRT pattern again by stimulating the area with a laser. This despite a negative test if I try pinching again. I have found the use of laser stimulation to be frequently effective in surgeries where several layers of tissue have been traumatized. Often in micro-surgery the superficial scar may be several decimeters or more from the actual area of internal surgery. In these cases, playing the laser beam over the site of internal surgery will often demonstrate a positive IRT response despite unblemished overlying skin. *Please follow the safety precautions that come with laser documentation.* In particular, care must be taken not to expose the retina to laser energy.

Conclusion

I have found the Injury Recall Technique (IRT) as originally presented and elaborated by Schmitt[1, 2, 3] to be a valuable approach to treating structural problems associated with certain types of trauma. In addition to restoring proper muscular facilitation and inhibition patterns, I have found the IRT to be tremendously useful when applied to some poorly healed areas of scar tissue, facilitating a more complete healing.

In addition to stimulation by pinching and therapy localization as per Schmitt, I have found laser stimulation in the 630 to 650 nm range to be effective both diagnostically and therapeutically. In the original IRT, as per Schmitt, stimulation of the old injury area, via pinching or therapy localization is used diagnostically along with either the talus challenge or head extention. Therapy is accomplished with re-stimulation of the injury area followed by either caudal talus tug or passive head flexion. Due to depth of penetration, laser stimulation may have the added advantage of clearing a persisting withdrawal reflex resulting from injuries at deeper layers such as occur during surgery.

References

1. Schmitt, Walter H., Jr. Injury recall technique. Chiropractic journal of NC Vol VI, 1990. pp. 25-30.
2. Schmitt, W.H., Jr., Injury recall technique: Dealing with the history of injury and trauma. Proceedings of Summer Meeting, ICAK, 1990.
3. Schmitt, W.H., Jr. A neurological rationale for injury recall technique. Proceedings of Summer Meeting, ICAK-USA, 1999.
4. Personal conversation with Majes-Tec representative, Majes-Tec Innovations 888-242-0571.

Adrenal and Insulin Related Disorders: More Complex Than We Thought

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Abstract

This paper will discuss the complex web of physiological alterations that take place with adrenal and insulin related disorders. This paper is divided into four sections. Section I will discuss the vicious cycles developed from insulin and cortisol. Section II will discuss the impact of these hormones in abnormal levels on human physiology. Section III will critically analyze clinical evaluation methods of adrenal related disorders. Section IV is a review of the scientific literature in regard to natural compounds to help support these patterns of imbalance.

Introduction

It is conservatively estimated that 25-35 percent of the population in westernized countries, suffer from some degree of insulin resistance and blood sugar metabolism disorders.[i] Insulin resistance and blood sugar disorders have been found to be a contributing factor to diabetes,[ii] cardiovascular disease,[iii] sleep apnea,[iv] hormone metabolism disorders,[v] obesity[vi] and certain types of cancer.[vii]

Insulin resistance refers to a state in which insulin receptor sites become unresponsive to the binding of insulin. This may be due to defects in ligand/receptor binding, signal transduction, intercellular communication, polymorphisms, etc.

Many different mechanisms have been found to contribute to insulin resistance, such as a vicious cycle created from elevated cortisol, hippocampus destruction, gastrointestinal disorders, etc. The four sections in this paper will discuss the complex web of physiological alterations that take place when hypercortisolemia and hyperinsulinemia exist and provide support for natural therapies to treat these patterns.

Discussion

Section I – Adrenal and Insulin Vicious Cycles

Abnormal insulin and cortisol levels induce many vicious cycles that impact human metabolism and endocrinology in a way that makes it difficult for the patient and clinician to resolve the syndrome. It is this lack of understanding of how alterations in these hormones impacts human physiology that makes resolution of this abnormal pattern difficult for the clinician. Once the vicious cycles are addressed and broken it unlocks the patterns associated with a functional defective endocrine system.

The Hippocampus- Hypothalmus-Pituitary-Adrnenal Axis Vicious Cycle

The first vicious cycle involves the relationship the hippocampus has with the hypothalamus-pituitary- adrenal axis. As we know, the hypothalamus releases corticotrophin releasing factor (CRF) when systemic cortisol levels are low. CRF is secreted into the primary capillary plexus of the hypophysial portal system in the median

eminence of the hypothalamus and then carried to the anterior pituitary gland where it induces adrenocorticotrophic hormone (ACTH) secretion. ACTH then has an effect on the adrenocortical cells to activate adenyl cyclase in the cell membrane. This induces cAMP in the cell cytoplasm and activates the intracellular formation of adrenocortical hormones. Simply stated, cortisol levels are regulated by a negative feed-loop axis that involves the activation of CRF to induce ACTH to induce cortisol synthesis.[viii]

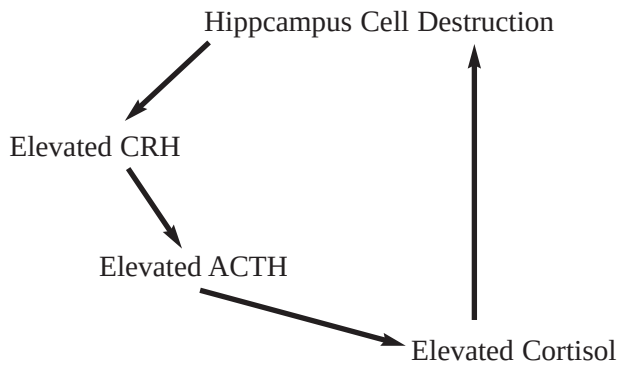
The hippocampus is a region of the cerebral cortex that lies along the medial-most border of the temporal lobe and folds upwards and inwards to form the inferior surface of the inferior horn of the lateral ventricle. The hippocampus in addition to being a cerebral center for memory serves as the "brake" for of the hypothalamic-pituitary-adrenal axis (HPA axis). Thus hippocampal dysfunction leads to loss of feedback inhibition of the HPA axis and causes a disorganized feedback patterns with an elevation of cortisol.[ix] The vicious cycle exists between the hippocampus and the HPA axis because the hippocampal neurons are rich in glucocorticoid receptors and very sensitive to cortisol. Therefore, when the body is placed in a chronic stress response the hippocampus has prolonged exposure to cortisol. Since its cells are sensitive to cortisol prolonged exposure initiates neuronal cell death by excitotoxicity and ultimately leads to hippocampul glucocortoid receptor down regulation, neuronal death and HPA negative feedback insensitivity.[x, xi, xii, xiii, xiv, xv, xvi,] The excitotoxic hypothesis from cortisol in the hippocampus is based on the influence of cortisol on glucocorticoid receptors which have an impact on the neuronal transmembrane gradient rendering the NMDA receptor more receptive to glutamate. The resulting influx of calcium enhances reactive oxygen species generation which further damages mitochondrial activity in a self-propagating feed forward cycle leading to cell death.[xvii, xviii, xix]

This vicious cycle that develops in adrenal stress syndrome may explain why some patients do not respond as effectively to adrenal support as others. If the patient has been in a chronic stress pattern, they may not possess the ability to regulate function. The classical pattern with a loss of HPA axis is the patient that has developed an inability to deal with stress over a period of time. These patients will also be the patients that never seem to stabilize when the adrenals are supported, but always show a need for some type of adrenal support. The chief complaint with patients that have some hippocampul loss of function is loss of short-term memory in addition to unstable and unpredictable adrenal stress syndromes symptoms. These patients my present to the clinician as a difficult case because the clinician is ignoring the importance of re-establishing proper HPA axis and therefore can never unlock the metabolic pattern associated with adrenal stress syndrome.

Objectively, these patterns can be observed from an adrenal salivary index that present with adrenal hyperfunction or a pattern that reflects an elevated level of cortisol at midnight. Elevations of midnight cortisol suggest lack of sensitivity to suppression of the HPA axis.[xx] Applied kinesiology findings clinically demonstrate patients that are either stuck with patterns of adrenal over-activation or patterns that switch to adrenal exhaustion. However, it should be noted any abnormal adrenal profile can indicate loss of HPA axis. Phosphorylated serine and other compounds that have shown to suppress this vicious cycle will be discussed. It should also be made clear that once cortisol levels are lowered, changes in the hippocampus are reversible and the potential to optimize the hippocampus-HPA axis can be restored but the process is not immediate.[xxi]

In addition, once one understands the impact of cortisol on the hippocampus, excitotoxicity, glucocorticoid receptor downregulation, neuronal death and insensitivity of the HPA axis negative feed back loop the use of exogenous cortisol or high dose adrenal concentrates that have trace levels of cortisol to treat adrenal stress syndromes seems illogical. Exogenous cortisol therapy may cause the patient to feel better immediately but it feeds the vicious cycles that will make the appropriate physiological changes that need to be corrected for adrenal stress syndrome extremely difficult down the road. In my personal experience, it is extremely difficult to re-establish proper HPA feedback control with patients that have been on cortisol therapy or long term adrenal concentrate glandulars.

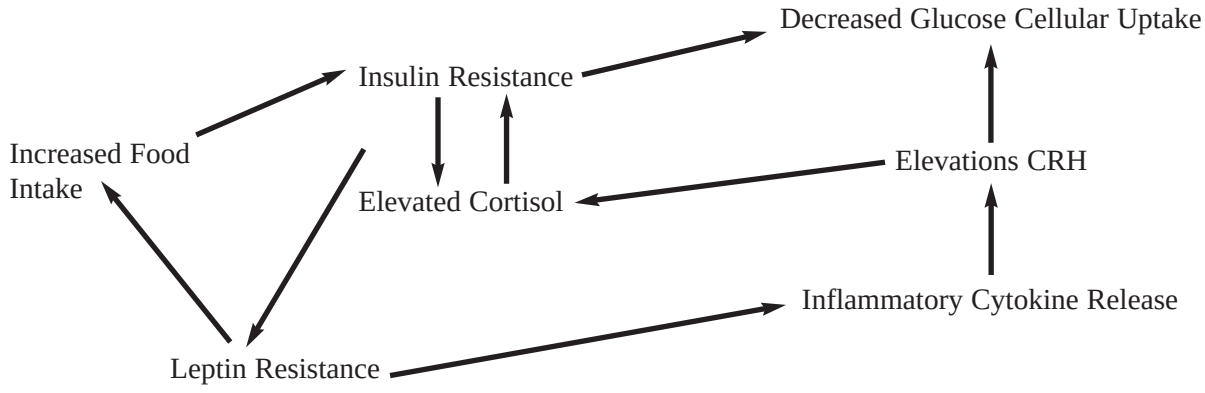
Diagram: **Hippcampus and HPA axis Vicious Cycle**



Adrenal Hypercortical Function – Insulin Resistance – Leptin Resistance Vicious Cycle

Another vicious cycle that occurs with adrenal stress syndrome is the pattern involved with elevated cortisol, insulin resistance and leptin resistance. Elevated cortisol has been found to induce insulin resistance and insulin resistance can produce hypercortisolemia.[xxii] Both hypercortisolemia and insulin resistance have been shown to induce leptin resistance.[xxiii, xxiv] Leptin is a hormone produced by fat cells that help regulate fat storage. Leptin is a newly discovered protein that acts as an intercellular messenger on the brain to decrease food intake. With elevated cortisol and insulin, the receptors for leptin in the hypothalamus are down-regulated and do not respond to leptin, therefore it increases the potential for obesity. Obesity then creates the vicious cycle with leptin resistance the leads to insulin resistance and hypercortisolemia.[xxv, xxvi, xxvii] This happens because if they are not responding to insulin, glucose is unable to enter the cell and promote oxidative phosphorlation for ATP production. Messages are sent by negative feedback loops to increase available glucose to produce ATP so cortisol is released to initiate gluconeogenesis and glycogenolysis. The other pattern that promotes this cycle involves inflammatory intercellular mediators. Leptin induces the inflammatory cascade by its influence on the regulation of transcription factors for cytokine receptors.[xxviii] As we know inflammatory cytokines are adrenal stimulants and therefore promote adrenal hyperfunction and hypercortisolemia.[xxix, xxx, xxxi, xxxii] The net result of this cycle is in increased potential for obesity, increased hunger with cravings for sugar, insulin resistance and elevated cortisol in vicious cycle that feeds itself.

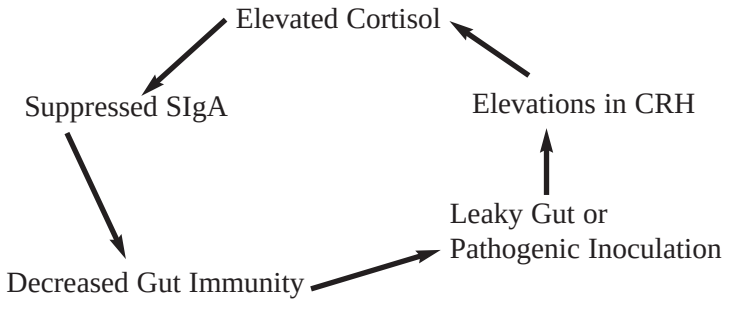
Diagram: **Insulin, Cortisol and Insulin Resistance Vicious Cycle**



The Gastrointestinal-Hypothalmus-Pituitary-Adrenal Axis Vicious Cycle

Gastrointestinal dysfunction in the form of pathogenic inoculation, or gastrointestinal sensitivities to food antigens are major causes of an adrenal stress syndrome, and are usually undetected due to diverse and sub-clinical symptoms. Food sensitivities to giliadin, casein, soy, eggs and others generally do not cause severe symptoms but do place the adrenals in a chronic stress response.[xxxiii] An objective way to identify these sensitivities is to perform a salivary SIgA test for these common foods.[xxxiv, xxxv, xxxvi] Applied kinesiology methods however may be the best and most economical form of testing. It is important when testing potential food sensitivities by AK methods to test for both a conditional inhibition response, as well as to look for an All-Muscles-Strong pattern. In addition, many forms of pathogenic organisms such as parasites, yeast and bacteria or mild food sensitivities do not cause major gastrointestinal symptoms but place significant amounts of stress on the adrenals. Chronic stress, as caused by pathogenic organisms or food antigens, can then create numerous vicious cycles and defects on normal physiology. As discussed earlier, cortisol will suppress SIgA levels. When SIgA levels are lowered, gut immunity is weakened and will be become sensitive to food as well as microbial antigens.[xxxvii] A person may develop an IgA sensitivity to soy for example. Once the SIgA sensitivity to soy is established, exposure to soy in the gut-associated-lymphoid-tissue (GALT) will cause a release of inflammatory cytokines that will further stimulate the adrenals and cause a vicious cycle between the gut an the HPA axis. The same vicious cycle could develop from any other type of antigen such as a candida overgrowth or a pathogenic inoculation.

Diagram: **Adrenal and GI Vicious Cycle**



Section II – The Impact of Cortisol on Human Physiology and Metabolism

In the previous section, we discussed vicious cycles that develop in adrenal and insulin related disorders. In this section we will discuss the impact of abnormal cortisol and insulin on human physiology.

The adrenal stress syndrome adversely impacts hypothalamic-pituitary-axis, binding of thyroid hormones, peripheral thyroid hormone conversion, and thyroid hormone detoxification pathways.

Elevated cortisol has a suppressive impact on the enzyme 5'diodinase which converts inactive T4 into active T3. Many sources have identified the pattern associated with elevated cortisol and its influence of low T3. The mechanism of lowered T3 may be due to multiple biochemical influences. Typically T4 is converted to T3, revereseT3 (rT3), T3 sulfate (T3S) and triiodothyroacetic acid (T3AC) peripherally by the enzyme 5'diodinase. In the past it was believed that the reason for the low T3 state was because it was shunted to the production of irreversible and inactive rT3. This explanation has recently proven to be incorrect since rT3 production rates measured in vivo are generally unaltered in stress patterns. Thus, it has been shown that rT3 concentrations result from diminished rT3 clearance rather than increased production. Current evidence published by LoPresti and Nicoloff have found that increased T3S and T3AC at the expense of T3 are most likely responsible for the lowered T3 state. T3S and T3AC are inactive forms of T3, but they have the potential to become activated into T3 by sulfation and acetylation. The bottom line is that elevated cortisol will decrease active T3 levels and therefore reduce the potential of optimal gene expression by thyroid hormones.[xxxviii, xxxix, xl]

Chronic stress (increased cortisol) or increased steroid hormones can have a negative impact on the hypothalamus-pituitary-adrenal axis. This may be identified with a TSH below the functional range of 2.0 and subjective indication such as: reduced or absent sex drive, increased ability to eat sugars without symptoms, abnormal thirst, lack of menstruation and weight gain around the hips and waist. Patients with chronic hypercortisolemia will demonstrate blunted ACTH responses because of the suppressive effect on the pituitary gland.[xli]

The bodies detoxification system becomes suppressed during adrenal stress syndrome due to chronic elevations of cortisol. Cortisol elevations must be conjugated by the liver in order to change them from fat-soluble substances into water-soluble substances in order for our bodies to eliminate them in our sweat, urine and feces. The liver uses two phases to break down hormones called Phase I and Phase II. Phase I is an oxidation/reduction system that utilizes cytochrome P450 enzyme system. Phase I primarily use antioxidants to detoxify substances. Phase II detoxification pathways for steroid hormone utilize glucoronic acid (a product of a thiamin and magnesium dependent glycolysis) and sulfur amino acids (methionine, cysteine, traurine and glutathione). Patients that have adrenal hyperfunction with elevated cortisol or adrenal exhaustion patients with low cortisol (that went through a preliminary phase of increased cortisol) may need the have their liver detoxification pathways supported. Patients with congested livers or overworked detoxification systems may have the following signs and symptoms: skin blanching with pressure, acne or acne worse at menses, constipation, bloating, sensitivity to medications, unresponsiveness to endocrine support (hormones or supplements), etc.

Elevated cortisol levels have adverse effects on the gastrointestinal tract by suppressing secretory IgA (SIgA), delaying mucosal cell regeneration and by promoting a pro-inflammatory environment.[xlii, xliii, xliv] SIgA is the main immunocyte in the gastrointestinal tract, and forms an immune barrier to protect against gastrointestinal cell infection.[xlv] Elevated cortisol can contribute to dysbiosis (imbalances between beneficial and adverse bacteria) and leaky gut syndrome (increased permeability due to thinning of the gastrointestinal lining).[xlvi] The suppression of SIgA can make the gastrointestinal tract more susceptible to inoculations of parasites and other pathogenic organisms.[xlvii] In addition, SIgA suppression may allow candida and other yeast organisms to overpopulate the gastrointestinal tract.[xlviii, xlix] It is very important to check cortisol levels with patients that have challenging gastrointestinal conditions. If the effects of elevated cortisol are not corrected, it will cause a dramatic delay in normalizing dysbiosis, leaky gut syndrome and other chronic gastrointestinal conditions.

Elevated cortisol levels have suppressing effects on the immune system and can decrease a person's ability to fight infections and other immune pathogens. Chronic levels of increased cortisol, in addition to suppressing SIgA levels, also decrease white blood cells, and induce atrophy of the thymus gland, and decrease interleukin-2 production.[l]

Selye's original research of chronic adrenal stress recognized that the effects of elevated cortisol can induce gastric and duodenal ulcers. Cortisol levels should always be investigated with patients that have reoccurring or chronic ulcers. In addition, hypchlorhydira (decreased levels of hydrochloric acid) and helicobactor pylor inoculation should be investigated. H. pylori can be identified with either a serum antibody test or a stool antigen test. It appears that increased cortisol levels seem to allow the gastric and duodenal lining to thin and become more susceptible to the development of ulcers. Although, increased cortisol is not the primary cause of an ulcer, it does increase the risk due do its thinning impact on the gastric and duodenal lining.

Elevated cortisol has negative impacts on bone metabolism. It appears that either endogenous or exogenous cortisol leads to calcium malabsorption, lower bone mineral density and an increased risk for fractures.[li, lii] One of the most common side effects of patients that take cortisol replacement therapy, such as Addison's patients, is increased fracture rates.[liii]

Metabolic shifts that take place in chronic stress, have been shown to be a related causative factor in the onset of depression. Overactive HPA activity and abnormal circadian rhythms of cortisol have been associated with depression.[liv, lv, lvi] Alterations in the adrenal medulla catecholamine output and neurotransmitter function have also been linked to depression. As our current understanding of neurotransmitter involvement with depression has evolved, we now realize that norepinephrine is a major player in the neurochemistry involved with depression. Theories of its impact have been associated with both decreased norepinephrine production, as would be expected from adrenal exhaustion, as well as norepinephrine overproduction, that leads to neurotransmitter down-regulation.[lvii, lviii] In any case, the management and regulation of the metabolic stress response has its role to play in the depressed patient.

Many people have a hard time falling asleep in today's active society. Medications addressing insomnia carry heavy side effects, and are not tolerated well by most people. In addition, medications do not address the underlying causes of insomnia.

Many times insomnia is directly related to blood sugar imbalances, in particular to adrenal function. The adrenal glands are two small glands located above the kidney that secrete hormones that help the body stabilize blood sugar problems. A common pattern seen clinically with patients that have adrenal hyper-function (overactive), is an inability to fall asleep. With adrenal hypofunction (under-active), the symptom is exactly opposite. Under-active adrenal patients will typically be able to fall asleep, but not stay asleep. Therefore, both hypo or hyper adrenal function impact insomnia.

Adrenal hormone levels can be measured by using an adrenal salivary index (ASI). The ASI consists of having the patient collect several samples of saliva during the day. These samples are then sent to the lab for assessment. The lab will then map out the circadian levels of cortisol and confirm hypo or hyperfunction. This test is very accurate and the cost is reasonable.[lix, lx] However, many times with symptoms of insomnia, subjective indicators are sufficient to apply conservative nutritional and lifestyle therapy.

Let us discuss adrenal hypofunction first. This pattern typically occurs in individuals who frequently miss meals, eat and drink lots of sweets and simple sugars, and are fueled during the day by caffeine. They typically complain of irritability when meals are missed, low energy, crave sweets during the day, and depend on caffeinated beverages during the day to "keep them going". The end result is chronic stress to the adrenal glands with a loss of function to maintain blood sugar levels, especially when they are asleep. The body depends upon a constant supply of glucose (sugar) to keep the body functioning properly. When the body is asleep it requires a constant supply of glucose, retrieved from stored glucose in the form of glycogen that is kept in supply in the liver and the muscle. On average, the body loses 60% of its glycogen (stored glucose) levels after an overnight fast from sleeping. If a person has blood sugar problems, they are not able to tolerate an overnight fast, and their bodies will go into a stress response when blood sugar levels drop. A person with healthy adrenal glands would never drop into a stress response from fasting, because they have of plenty of cortisol to stabilize their blood sugar levels during the night.[lxi, lxii] This stress response will cause the adrenal medulla to secrete epinephrine and norepinephrine to try to mobilize glucose to compensate for lack of cortisol.[lxiii, lxiv, lxv, lxvi] The release of these stimulatory hormones will cause the person to wake up during the night. They present clinically as the type of person that has low energy, craving for sweets during the day and an inability to stay asleep throughout the night. These people need to learn how to stabilize their blood sugar levels during the day, and take some non-stimulatory support for their adrenal glands, as will be discussed.
Now let's discuss the person who cannot fall asleep. These people typically have elevated cortisol, especially at night time.[lxvii] Cortisol is a hormone that is released by the adrenal cortex that helps stabilize blood sugar levels during the day. Cortisol is released during the day in-between meals, when blood sugar levels need to be balanced. When blood sugar levels drop, the body releases cortisol to maintain blood sugar levels.[lxviii, lxix] It acts by releasing glucose from stored glycogen levels. The normal circadian release of cortisol is high in the morning, with levels that drop as the day goes on. An abnormal circadian release of cortisol is when the levels stay high all day. When the levels are high at night instead of normally low, a person will not be able to fall asleep due to the excitatory nature of cortisol on the nervous system.[lxx]

The only reason for a physiologically abnormal circadian rhythm of cortisol, is the body being placed in an alarm or stress reaction. An alarm pattern will place extra demands on the body that the adrenals will have to compensate for. For example, if a person is having severe episodes of emotional or mental stress, the body needs extra glucose to meet the metabolic activity caused by this type of stress, and therefore releases cortisol to increase the systemic glucose levels to produce more ATP to meet the extra demands. Many other physiological patterns can cause elevations in cortisol. For example, food sensitivities can activate the gut associated lymphoid tissue and place the body in an alarm pattern. Pathogenic organisms such as parasites and other inoculations can elevate cortisol levels. The bottom line is, any type of stress; mental, physical or physiological can lead to elevated cortisol levels. Many times it is a combination of factors that cause this dysfunctional pattern.

The impact of cortisol on REM sleep is also a factor not only with the quantity of sleep but the quality as well. Age related increases in cortisol are directly related to sleep fragmentation and reduction of REM sleep. According to a study published in August 2000 in JAMA the researchers concluded," elevated evening cortisol levels in late life probably reflect an impairment of the negative feedback control of the HPA axis in aging. Researchers suggested that there is a relationship between this alteration of the HPA function and decreased amounts of REM sleep that is independent of age.[lxxi] Other studies have also supported the impact of corticosteroids on sleep cycles and the alteration of REM and non-REM sleep cycles with hyprcortisolemia.[lxxii, lxxiii]

Elevated cortisol levels have been associated with neurodegenerative disorders. For example, dysregulation of the HPA axis has been shown to be related to the clinical course of Multiple Sclerosis. The researchers of a study published in Neurology in 1999 stated, "…the degree of HPA axis hyperactivity is related significantly to the clinical course of MS."[lxxiv] Lowered DHEA levels, which is often associated with the pregnenolone steal that takes place in adrenal stress syndrome, has been linked to Alzheimer's disease.[lxxv] Elevated cortisol has also shown to cause hippocampal cell destruction due to excitotoxicity of cortisol sensitive hippocampal cells. Elevated HPA activity has also shown to increase levels of inflammatory cytokines. Upon exposure to inflammatory triggers, the glial cell within the brain's structure will also produce inflammatory cytokines and neurotoxic agents such as nitric oxide, and other oxidants that can damage neurons, and cause neuronal apoptosis (cell death). The term "gliosis" has been generally applied to this process of inflammation induced damage to the glia and subsequently to the neurons. Gliosis has been identified in numerous neurodegenerative conditions including Alzheimer's, Huntington's disease, multiple sclerosis, as well as ischemia, edema and seizures.

Patients suffering from chronic stress impact physiology in multiple ways to increase risk factors associates with cardiovascular disease. The stress response increases blood pressure because of elevations in catecholamines and alterations in mineral corticoid metabolism and suppression of DHEA synthesis. These metabolic shifts increase vasoconstriction and alter sodium/potassium equilibrium leading to hypertension.[lxxvi] Elevated cortisol, at even small quantities, has been associated with increased risk for cardiovascular disease. This may be due to the impact of cortisol on hypertension, insulin resistance, obesity and hypertriglyceridemia.[lxxvii, lxxviii, lxxix]

The Impact of Insulin Resistance on Human Physiology

Hyperinsulinemia is considered an independent risk factor for cardiovascular disease. It adversely impacts physiology to increase risk factors associated with lipoprotein metabolism, coagulation protein synthesis, and blood pressure.

Evidence has demonstrated that insulin and blood pressure have a direct relationship on each other. For example, studies have demonstrated that blood pressure drops as insulin levels are decreased, and elevates when insulin levels are increased in type 2 diabetics.[lxxx, lxxxi] The proposed mechanism for its impact on blood pressure is insulin's role in increasing sodium retention, as well as altering internal sodium and potassium distribution, which leads to increased peripheral vascular resistance.[lxxxii]

Hyperinsulinemia has also shown to alter coagulation proteins involved with fibrinolysis. Fibrinolysis is the process of breaking down the protein fibrin which is involved with coagulation. There is a protein called plasminogen activator inhibitor 1 (PAI-1) which is elevated in hyperinsulinemia. Higher levels of PAI-1 lower the activity of fibrinolysis and may induce a greater risk for coagulation.[lxxxiii, lxxxiv] Insulin directly impacts cholesterogenesis by up-regulating the rate-limiting enzyme in cholesterol synthesis, HMG-CoA reductase.[lxxxv] Statin drugs, which are the most common drugs used to lower cholesterol, are designed to down-regulate this enzyme. Therefore, a more logical step in patients with elevated cholesterol and symptoms of hyperinsulinemia is to consider insulin resistance management. Insulin also imparts influence on cholesterol ester transfer protein (CETP) due to its impact on hypertriglyceridemia. CETP is responsible for the transfer of insoluble cholesterol esters from HDL to other lipoproteins. With elevated triglyceride levels and hyperinsulinemia, shifts in cholesteryl esters on HDL take place with the end result of lowered HDL cholesterol.[lxxxvi]

In summary, insulin resistance elevates blood pressure, increases the rate limiting enzyme HMG-CoA reductase which elevates cholesterol synthesis, impacts CETP to lower HDL levels and increase PAI-I which decrease fibrinolysis.

Insulin resistance appears to play a major role in a vicious cycle that alters female hormone metabolism towards androgen dominance. Androgen dominance may eventually lead into Polycystic Ovary Syndrome (PCOS). It is the most common female hormone disorder in menstruating age women. It is estimated that androgen disorders occur in as many as 4-10 percent of women of reproductive age and it is the most common cause of female infertility in the United States.[lxxxvii, lxxxviii] PCOS is primarily characterized by hyperandrogenism, insulin resistance and chronic anovulation. Symptoms of androgen disorders tend to appear gradually over a number of years and range from the mild to serious. They include irregular periods, infertility, unexplained weight gain, fluid retention, fatigue, mood swings, acne beyond puberty, hair loss and unwanted hair growth. The symptoms of androgen disorders should not be dismissed or ignored. The syndrome is associated with estrogen proliferative cancers,[lxxxix] acanthosis nigricans,[xc] increased cardiovascular disease and abnormal lipid metabolism.[xci]

It appears that insulin resistance creates a vicious cycle that shifts into androgen excess and the androgen excess promotes insulin resistance. Several different mechanisms have been postulated in regards to research, however this disorder is characterized by abnormal gonadotropin dynamics, insulin resistance and androgen excess. This syndrome presents with elevated testosterone, elevated estrogen, decrease, sex-hormone binding globulin, increases androstenidione, increased DHEA, increased 17-hydroxprogesterone and increased LH.[xcii]

Increased testosterone production appears to have the potential to come from the adrenals and the ovaries. Dysregulation of cytochrome p450c17, which is the main enzyme in both the adrenal and the ovaries for androgen synthesis, has been shown to be one mechanism of this disorder. In the ovaries, in addition to dysregulation of cytochrome p450c17, insulin has shown to up-regulate the enzyme 17,20 lyase in the theca cells of the ovaries, and contribute to androgen dominance. In addition to insulin's role in upregulating enzymes that promote insulin resistance, elevated insulin levels decrease the amount of sex-hormone binding globulin.[xciii] When sex-hormone binding levels are decreased from hyperinsulinemia, it causes greater exposure of both estrogens and testosterone to the tissue, which leads to an even greater androgen response.[xciv]

In contrast to insulin's role in causing an androgenic shift in metabolism, elevated androgens appear to decrease insulin receptor sensitivity. The receptor site sensitivity induced by excess androgens appear to be post-binding defects in intercellular communication, responsible for type-4 glucose transporters (GLUT-4), which allow glucose to be transported across the cell membrane.[xcv] Androgen excess also plays another role in its impact on insulin receptor site insensitivity. Excess androgens increase levels of free fatty acids, which inhibit hepatic insulin excretion, and inhibit insulin-stimulated glucose uptake in skeletal muscles.[xcvi, xcvii] These mechanisms create a vicious cycle of elevated insulin, which is fed, and feeds, excess androgen synthesis.

This syndrome is associated with defects in the hypothalamus-pituitary feedback loop that create a pattern of elevated LH and anovulation or menstrual irregularities (oligomenorrhea, amenorrhea, menorrhagia). Different mechanisms may be involved with this increase in LH. It has been demonstrated that chronic patterns of hyperinsulinemia and hyperandrogenism contribute to increased levels of LH.[xcviii] Another mechanism demonstrated that the elevations of LH are due to an abnormal feedback by estrogen.[xcix] This is because LH release, and the LH surge in midcycle, is triggered by elevated levels of estrogen at the end of the follicular phase. Yet another mechanism demonstrated that the elevations in LH were due to elevations in b-endorphin.[c, ci] This intercellular communicating agent is responsible for exerting inhibitory control on GnRH and on pituitary release. This enzyme is increased with elevations of insulin, and by cortisol.[cii]

The conventional pharmaceutical based therapy to treat this disorder does not seem to treat the underlying cause of insulin resistance and adrenal dysfunction. Instead, pharmaceutical agents are used to over-ride physiology to change metabolism. Many agents are being used to treat PCOS and androgen disorders in women.

The most common treatment is the use of oral contraceptives. Oral contraceptives contain high amount of hormones used to take the pituitary feedback loop out of the picture. The birth control pill suppresses the secretion of hormones and therefore decreases ovarian androgen production. Many times patients cannot tolerate the pill because of the high amounts of synthetic estrogens in the pill. This is more common when they already have high levels of estrogen. It should be noted that chronic use of the pill will inhibit the natural hypothalamus-pituitary-ovarian feedback loop. The condition called post-birth control syndrome, which is classified by inability to regain normal menstrual cycles, is increased with chronic use of oral contraceptives.

For women that cannot tolerate the pill, the use of GnRH analogue drugs are used to completely suppress all ovarian hormone production. Many of these patients suffer from symptoms of hormone deficiency which is then treated with exogenous hormone therapy. This type of therapy is probably the most extreme and completely takes over normal endocrine function which obviously comes with its own list of side effects and risks.

If the androgen shift is coming from the adrenals, conventional medical therapy uses drugs such as glucorticoids, prednisone or dexamethasone (decadron) to suppress adrenal function. Once again, this form of drug treatment does not identify the cause of adrenal over-activation and will only inhibit the natural feedback loop with the HPA axis in the future.

Other forms of therapy include androgen antagonist drugs such as ketoconazole and finasteride and insulin sensitizing drugs such as metformin. These drugs come with serious side effects. Metaformin may cause lactic acidosis, malabsorption, and B12 deficiency.[ciii] The anti-androgen drug ketoconazole suppresses cortisol synthesis and healthy adrenal function and has been shown to cause severe liver toxicity.[civ] The list of side effects for these drugs is long and serious. In most cases, the pharmaceutical approach as first line therapy seems illogical, especially when diet and nutritional management can not only change the symptoms associated with androgen disorders, but actually change the abnormal alterations in metabolism and enhance normal physiology.

The natural management of this syndrome includes diet and exercise with nutritional protocols to help insulin sensitivity. A basic protocol in addition to diet and exercise would include the use of nutrients to support insulin receptor sensitivity, phosphatidylserine and adaptagens to decrease adrenal over-activation, chaste berry to optimize the feedback loop with the hypothalamus and the pituitary, as well as possible regulation of androgen hormone synthesis suppression with compounds such as saw palmetto, stinging nettle, EFAs, etc.

Insulin has the potential to alter male hormone pathways in several ways. First, hyperinsulinemia will alter adrenal hormone physiology due to insulin's role in inducing hypercortisolemia. On a male hormone profile, many patterns of altered steriodogenesis becomes evident when this shift takes place. First, the levels of progesterone may be low. This may indicate that progesterone levels are being converted into cortisol. In males, progesterone is only made by the adrenal glands, since men don't have ovaries. Any shifts or alterations in progesterone levels makes the adrenals suspect for evaluation. Progesterone serves many important functions in the male such as its role in protecting the prostate, it impact on neurochemistry, its role in osteoblast activity, etc.

Insulin's role on adrenal function can also be seen with alterations in androstenidione and DHEA. It appears to increase the levels of androstenidione and lower the levels of DHEA.[cv, cvi] When levels of DHEA become compromised, it induces decreased vitality, difficulty in losing weight, and may potentially lead to decreased testosterone levels. When androstenidone levels become elevated, it appears to cause low testosterone type symptoms. This may be due to the high affinity of androstenidione to bind to testosterone receptor sites, with only a slight androgen impact on cells, compared to what testosterone could induce. Finally, it appears that insulin resistance may increase body fat, and the activity level of the enzyme aromatase. Aromatase is responsible for converting testosterone into estrogens. Any patient that demonstrates aromtase upregulation needs to be investigated for adrenal and insulin type dysfunction. An exercise and weight reduction program with a dietary program to manage insulin resistance needs to be considered.

Insulin resistance has a contributing role to obesity. When insulin resistance exists the cells are impaired from transporting glucose from the circulation, into the cells to be used for energy. Glucose is then forced to take an alternative pathway into lipogenesis. Therefore, the person will have a metabolic shift in which the glucose that exists in their cells cannot be used for energy, but will be converted to adipose. This shift explains the subjective complaint commonly expressed by insulin resistance patients, that they put on weight quickly, and have low energy. Their body cannot take advantage of the potential to use glucose for energy. Instead, they have to use energy for altered metabolism of glucose. Therefore, when they eat, it will cause them to put out more energy for the biochemical process of lipogenesis, and at the same time they have less potential for ATP production. This may explain why patients that have insulin resistance feel so fatigued after they eat.

Secondly, there are links to obesity from the relationship developed between insulin, cortisol and leptin. Leptin is a hormone released from adipose tissue that is responsible for decreasing appetite and stimulating thermogenesis. However, when a person has elevated levels of insulin, they also present with a concomitant metabolic shift of cortisol. This shift appears to cause a state of leptin resistance by increasing leptin levels very high at the same time it inhibits the response rate of leptin receptors. Overall this equates to a person that has decreased potential to metabolically burn fat and increased potential to metabolically store fat and be hungry all the time.[cvii, cviii, cix]

Elevated insulin levels will down regulate glucose-6-phosphate dehydrogenase (G6PD) which will then suppress the hexose monophosphate shunt (HMS). Reduced HMS activity will decrease NADPH which is the cofactor required to produce glutathione (GSH). When GSH levels are suppressed, phase II detoxification will be compromised. Decreased production of glutathione will not only impact phase II glutathione conjugation but it will also increase the cellular demands of phase I detoxification since glutathione is one of the most important nutrients for oxidation/reduction activity. The net result is decreased phase I and phase II activity due to insulin's impact in inhibiting glutathione synthesis.

There is suggestive evidence in the literature of the role insulin plays with carcinogenesis, specifically to colorectal cancer and breast cancer.[cx, cxi] The studies are still not conclusive however, due to insulin's role in promoting inflammatory responses, and its numerous adverse impacts on human metabolism. Its contribution to carcinogenesis is of no major surprise to clinicians.

Insulin resistance has profound impacts in altering essential fatty acid metabolism. It appears that low cellular insulin response inhibits the activity of the rate-limiting enzyme, delta-6-desaturase. This enzyme is used to transform linoleic and linolenic acid into longer essential fatty acid metabolites such as GLA, and EPA respectively. Down-regulation of this enzyme would result in less then ideal production of prostaglandins, eicosanoids, and leukotrienes which act as important intercellular mediators. On the other hand, hyperinsulinemia alters another enzyme in fatty acid metabolism called delta-5-desaturase. This enzyme, when up-regulated with excess insulin levels, will alter essential fatty acid metabolism by converting DGLA into arachadonic acid. This shift will alter the PG1 and PG2 balance, and shift the body into an inflammatory state, as well as alter the balance of prostaglandins, eicosanoids, and leukotriens used to modulate intercellular communication. Hyperinsulinemia also alters delta-6-desaturase by up-regulating its activity, as opposed to states of insulin resistance which down-regulate its activity. When delta-6-desaturase is stimulated by excess insulin, it will also increase delta-5-desaturase activity, which will lead to increase arachadonic acid.[cxii, cxiii]

Therefore, it appears both low insulin response and hyperinsulinemia alter essential fatty acid metabolism. Insulin resistance is characterized by surges of elevated insulin with low cellular insulin response. The net effect of insulin on fatty acid metabolism enzymes is inflammatory end products, and an unbalanced state of intercellular mediators important in cell messenger communication and response.

Abnormal insulin regulation impacts energy metabolism in several ways. First, insulin resistance will prevent the cellular uptake of glucose which decreased amount of fuel to be used in the citric acid cycle for energy. Second, since insulin cannot be delivered into the cells it if forces metabolism to shift into an energy requiring

state of lipogenesis. Third, insulin produces an inflammatory state which will not only short circuit the citric acid cycle but it will uncouple oxidative phosphorylation used to produce ATP. Finally, insulin resistance will alter essential fatty acid metabolism which will hinder lipolysis and beta-oxidation.

Section III – Adrenal and Insulin Resistance Evaluation

We have numerous ways to evaluate and identify adrenal and insulin related disorders in clinical practice. This section of the notes attempts to review and critically evaluate these methods.

Probably one of the most common we use in our practice to evaluate adrenal exhaustion is the standard test to identify signs of orthostatic hypotension. The normal response is for the systolic blood pressure to elevate by 8 mm/Hg when the blood pressure is evaluated in a supine, seated, and standing position. If there is inadequate production of catacholamine hormones, there will be no elevation, or a drop in the systolic blood pressure when one goes from a supine to a sitting and then to a standing position. The normal response takes place because catecholamines cause constriction of abdominal veins when one stands, to prevent pooling of blood due to gravity. The problem with this test is related to the fact that the response has no direct impact from cortisol. Cortisol is entirely different than epinephrine and norepinephrine produced in the adrenal medulla. Although cortisol levels will increase with elevations in catecholamine levels, this test cannot identify adrenal levels of cortisol output. It may be a test to help identify decreased production of adrenal medulla output, but it is too big of an assumption to conclude patients who have the ragland effect have adrenal cortical hypofunction. Many times patients will present with adrenal salivary profiles that demonstrate adrenal hyperfunction and also present with the ragland effect. This is because the adrenal salivary index will measure adrenal cortical output, while the orthostatic hypotension test evaluates adrenal medulla output. It should also be noted that patients may have a false positive if they had recently consumed caffeinated beverages before the test. In conclusion orthostatic hypotension helps identify an adrenal stress syndrome, but it does not determine if they are hyperadrenal or hypoadrenal.

Many tests such as the urinary Koeinsburg test, the ligament stretch reaction, and the pupil dilation test indicate mineralocorticoid imbalance. These do not accurately reflect if we have a patient that is in a state of adrenal exhaustion or hyperfunction. This is because of the relationship cortisol plays with mineralocorticoid receptors. Although both aldoseterone and cortisol are adrenal medulla hormones, cortisol has a high affinity to bind to mineralocortioid receptors and induce changes in mineralocorticoid response. In medicine, there is a condition called Pseudo Mineralocorticoid Excess Syndrome. Patients with this syndrome exhibit symptoms of hypertension, hypokalemia, and suppression of the rennin-angiotensin-aldosterone system that would be expected if they were hypersecreting aldosterone. However, tests completed on these patients by serum and urine fail to identify any excess of mineralocorticoids. This syndrome is found to be a result of not excess mineralocorticoids or adrenal cortical output, but rather a result of cortisol inactivation by the enzyme 11-beta-hydroxy steroid dehydrogenase (11-B-HSD). The enzyme 11-B-HSD is a bi-directional peripheral enzyme that converts active cortisol into less active cortisone and vice versa. There are multiple reasons for defects in this enzyme physiologically at lesser extremes then Pseudo Mineralocorticoid Excess Syndrome that cannot be expanded in this paper (wait for a paper in 2004 for further explanations). Therefore, when we use tests that represent mineralocorticoid excess we are looking at responses that cannot specify if we have adrenal hyperfunction or adrenal exhaustion. This is because too many factors outside of mineralocorticoid output are involved.

In applied kinesiology, we have multiple ways of identifying patterns of adrenal exhaustion, adrenal hyperfunction, insulin resistance, hypoglycemia, defects in citric acid cycle, and blood sugar metabolism. However, these tests do not always take into consideration what takes place during an entire day. We are limited in clinical practice to be able to see our patients only once during the day for their appointment. However, blood sugar regulation by the adrenal glands vary during the day due to it own circadian rhythm. The circadian rhythm of cortisol is linked to patterns associated with sleeping at night and working during the day. It is

controlled by the suprachiasmatic nucleus of the hypothalamus. The normal pattern can be observed on a graph with high levels of cortisol in the morning, which gradually taper down until its lowest levels are plotted at midnight. The reason cortical levels are highest in the morning is because the body had just engaged in sleeping. Sleeping is a fasting state on human physiology. When the body is asleep it does not have available glucose from food intake to help with repairs, and to supply cells, such as the brain and blood cells, with the constant supply of glucose they demand. Therefore, the adrenals will release cortisol during sleep to engage gluoneogenesis and glycogenolysis for the needed glucose demand. As a matter of fact, the body will lose 2/3 of its glycogen levels after a typical night of sleep. As the person wakes up there is less demand on glucose from gluconeogenesis and glycogenolysis and therefore cortisol will taper down if the pattern is normal. Many times patients will have alterations in their circadian rhythm of cortisol. They present with patterns of low cortisol output in the morning and high levels of cortisol at night time. If we were to test them via applied kinesiology methods in the morning, we would find adrenal hyperfunction signs and symptoms, but at night time the findings would be consistent with hyperfunction. This is one of the major limitations of muscle testing in a practice setting.

The adrenal salivary index may be helpful in these cases to monitor the circadian rhythm of cortisol during an entire day. Cortisol is secreted in a specific pattern during a 24 hour period. This is why salivary tests that incorporate a collection sample in the morning, noon, afternoon, and bedtime are so useful in tracking adrenal function. Any aberrations in this pattern indicate specific patterns of adrenal stress syndrome.

Section IV – Nutritional Management of Insulin and Cortisol

Many different types of natural compounds can be used in the management of cortisol and insulin related disorders. This section of the paper reviews the scientific literature in regards to adtaptogenic herbs, vitamins, minerals and natural hormones. Methods commonly used in applied kinesiology such as lingual receptor challenges should be used in the clinical setting to identify protocols specific to the patient.

Adaptagenic Herbs

Adaptagens are plant compounds that seem to have a normalizing impact on the Hypothamalus-Adrenal-Pituitary (HPA) axis under times of stress. An "adaptagen" was defined in 1957 by the Russian pharmacologist I.I. Berkman as a substance that fulfills three criteria. First, it must be innocuous and cause minimal disorders in the physiological functions of the organism. Second, it must have a non-specific action to increase resistance to adverse influences by a wide range of physical, chemical and biochemical factors. Third, it is has a normalizing action irrespective of the direction of the pathologic state.[cxiv] Adaptagens seem to be useful for both adrenal hyperfunction as well as adrenal hypofunction. By definition an adaptagen implies the capability for bi-directional or normalizing effects and therefore should be used in cases of adrenal stress syndrome. The most important adaptagens for the adrenals include Panax Ginseng, Siberian Ginseng, Ashwagandha, Rhodiolia, Boerhaavia Diffusa and Holybasil Leaf Extract.

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.[cxv] 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.[cxvi, cxvii, cxviii, cxix]

Siberian ginseng also known as eleutherococcus senticosus is an adaptagen. Most of the studies on Siberia 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.[cxx, cxxi, cxxii] 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.[cxxiii]

Ashwagandha is also known as withania somnifera and Indian ginseng. It is a very popular herbal adaptagen in Ayuredic medicine. Many animal studies have been published on this adaptagen. It apparently has adaptagen-like glucocorticoid activity which makes it so helpful in adrenal stress syndromes.[cxxiv] Studies have found that ashwagandha has similar adaptagenic activity to Pannax ginseng.[cxxv] It also had the ability to counteract some of the adverse physical responses to stress such as changes in blood sugar management.

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.[cxxvi, cxxvii, cxxviii, cxxix]

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.[cxxx, cxxxi, cxxxii, cxxxiii, cxxxiv] Rhodiola has also been shown to enhance cognitive function and mental fatigue, as well as support immune function.[cxxxv, cxxxvi]

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.[cxxxvii]

Nutrients to Support Adrenal Function

Pantethine is a major cofactor for adrenal hormone steroidogenesis and is a useful nutrient in stress conditions. Pantethine has demonstrated the ability to down-regulate the exaggerated secretion of cortisol under times of stress, as well as the ability to enhance adrenal cortical function when needed.[cxxxviii, cxxxix, cxl, cxli] B vitamins are important for adrenal hormone synthesis. Patients that have been placed in an adrenal stress pattern have greater loss of B vitamins.[cxlii]

Phosphatidylserine (PS) is an endogenously produced phospholipid that is embedded in cell membranes, and is the major phospholipids in the brain. Its general functions include supporting cellular chemical signal transmissions, activating cell surface receptors and cellular exchange of nutrients and waste products.

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,[cxliii, cxliv, cxlv] protect cells from oxidative damage,[cxlvi] decrease anxiety, improve mood, motivation and depression,[cxlvii, cxlviii, cxlix, cl] enhance memory and cognition,[cli, clii, cliii] and decrease cortisol.[cliv, clv, clvi, clvii]

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. Elevated cortisol has been shown to induce insulin insensitivity, decrease TSH and T3 production,[clviii, clix] increase inactive reverse T3,[clx] decrease phase II glucoronidation and sulfation, suppress pituitary function,[clxi]

increase the potential for gastric and duodenal ulcers, lower intestinal secretory IgA,[clxii, clxiii] delay intestinal mucosal cell generation,[clxiv] suppress immunity,[clxv] decrease bone density, induce depression,[clxvi] encourage obesity,[clxvii, clxviii, clxix] and increase the risk for cardiovascular and neurodegenerative disorders.[clxx]

Therefore, the use of PS shows great promise in the management of disorders induced by the elevations of cortisol from chronic stress syndromes. Up until now the use of PS was limited in clinical practice because very high doses of oral PS (up to 800 mg a day) are required to blunt the physiological stress response. This therapeutic dose of PS is very expensive and requires 8 or more capsules of intake per day, which makes it difficult for patient compliance. Many of the best responses of PS in clinical studies also used intravenous forms of delivery. This appeared to be the best form of delivery because it bypassed the gastrointestinal tract and was able to be delivered directly into the blood stream.

The new innovative form of PS delivery in a cream has now allowed clinicians to use the required amounts of PS to modulate the stress response. The PS cream allows hundreds of milligrams of PS to enter directly into the blood stream by bypassing the gastrointestinal tract from trans-dermal delivery. Trans-dermal delivery is a mechanism of delivery in which lipid spheres, called liposomes, surround PS and act as transport mechanism through the skin until it reaches the blood supply. Once there, the liposome shell around the PS substance degrades, and makes PS available for active response in the blood stream.

Natural Compounds to Support Insulin Resistance

Gymnema sylvestre in has demonstrated positive impacts in managing insulin resistance. It has demonstrated the ability to reduce insulin requirements, decrease fasting blood sugar, enhance the action of insulin, and even the ability to regenerate pancreas beta-cells.[clxxi, clxxii, clxxiii, clxxiv] It does not encourage the endogenous production of insulin, and if given to healthy volunteers, does not produce any blood sugar-lowering or hypoglycemic effects.[clxxv, clxxvi]

Chromium is an essential nutrient for insulin resistance, especially when one considers the evidence that chromium deficiencies are common in the United States, and that chromium levels are depleted by a diet of refined carbohydrates and sugars. There is evidence that chromium deficiency results in insulin resistance.[clxxvii, clxxviii, clxxix] Chromium, also known as "glucose tolerance factor," appears to optimize the impact of insulin on receptor sites, and therefore improves glucose uptake.[clxxx, clxxxi] Studies have demonstrated that chromium normalizes post-prandial glucose and insulin levels, glycated hemoglobulin, and hypercholesterolemia.[clxxxii, clxxxiii clxxxiv, clxxxv, clxxxvi]

Vandium is an important mineral when it comes to managing insulin resistance. It appears to have insulin-like impact on receptor sites, and improves the transport of glucose transporters to the cell membrane to allow cells to intake serum glucose.[clxxxvii, clxxxviii, clxxxix] This physiologic impact is of great importance because most defects in insulin resistance involve intercellular transduction reactions that vanadium appears to enhance. Numerous studies have demonstrated the positive role vanadium plays in managing insulin resistance.[cxc, cxci, cxcii]

Alpha-lipoic acid is a sulfur-containing substance that seems to improve insulin resistance by increasing the activation of glucose transporters (GLUT1 and 4), which enhance glucose disposal by sensitizing tissues to insulin, and by restoring proper intracellular redox states which then reset signaling and response to insulin.[cxciii, cxciv, cxcv, cxcvi] Alpha-lipoic acid has also shown to improve glucose metabolism, reduce serum lactate and pyruvate and improve mitochondrial oxidative phosphorylation.[cxcvii] Alpha-lipoic acid is also a powerful antioxidant that can help clench insulin induced oxidative stress patterns.[cxcviii, cxcix] Numerous studies have shown the positive impact of alpha-lipoic acid for insulin resistant disorders.[cc, cci, ccii]

Vitamin E (Tocopherols) has been shown to improve insulin sensitivity, improve serum triglycerides and LDL, and aid not only in the oxidative complications of diabetes, but also in the prevention of the disease.[cciii, cciv, ccv, ccvi, ccvii, ccviii]

Magnesium has been shown to improve insulin resistance. It appears to optimize insulin secretion, activate glucose transport for insulin-mediated glucose uptake and to improve insulin intercellular transcriptional response.[ccix, ccx, ccxi] Furthermore, insulin resistance has been reported in individuals with low magnesium status.[ccxii, ccxiii, ccxiv]

Biotin supplementation has been shown to improve insulin response to glucose load, lower post-prandial glucose levels and up-regulate the enzyme glucokinase which is responsible for the first step in glucose utilization by the liver.[ccxv, ccxvi, ccxvii, ccxviii]

Zinc is an important mineral in the management of insulin resistance. Zinc has protective effects against beta-cell destruction, improves insulin sensitivity and plays an important roles in insulin metabolism.[ccxix] There have been strong correlations with low zinc status and increased risk for insulin resistance as well as evidence that diabetics excrete large amounts of zinc and therefore require supplementation.[ccxx, ccxxi, ccxxii]

Inositol has shown the ability to re-establish normal myoinositol levels in deficient neurons and therefore, may be helpful in cases of diabetic neuropathy.[ccxxiii]

Niacin is a component of glucose tolerance factor which helps optimize the impact of insulin on the receptor site.[ccxxiv] Several studies have also shown that niacinimide has the potential to prevent the onset of type I diabetes.[ccxxv, ccxxvi, ccxxvii] Niacinimide has also been shown to inhibit macrophage and interleukin-1-mediated beta cell damage, inhibit nitric oxide, and function as an antioxidant.[ccxxviii]

L-carnitine has the potential to improve insulin sensitivity by enhancing whole-body glucose uptake and increasing glucose storage.[ccxxix, ccxxx, ccxxxi] L-carnitine has been shown to improve both peripheral nerves and vascular function in patients with diabetes.[ccxxxii] In addition, it has been shown to significantly reduce total serum lipid and increase HDL cholesterol levels in diabetics.[ccxxxiii]

Natural Hormones to Support Insulin and Cortisol Related Disorders

DHEA is made by the adrenal glands and is the precursor for testosterone and estrogen. DHEA levels become suppressed with chronic stress. The adrenal's ability to produce DHEA is compromised during chronic stress, because the glands will produce cortisol to stabilize energy and blood sugar levels at the expense of synthesizing DHEA. This can cause many negative impacts on physiology since DHEA has been shown to improve memory, lower cholesterol, strengthen the immune system, prevent bone loss, and optimize insulin receptor function.[ccxxxiv, ccxxxv, ccxxxvi, ccxxxvii, ccxxxviii] However, the incorrect use of DHEA creates more problems than it helps. DHEA should never be used in doses above 20 mg a day for longer then one month. As a matter of fact, 10 mg is a safe standard for most people. Doses from 5-20 mg will vary due to clinical judgment. It should be understood that most cases of low DHEA are from adrenal disorders. When the adrenals are overworked, a biochemical process called the "pregnenolone steal" takes place. This biochemical shift causes the body to shift hormone metabolism into cortisol synthesis instead of DHEA. DHEA overdosing will convert into testosterone in females and into estrogen in males. The best way to support overdosing is to use sublingual troches

Conclusion

In conclusion, we can see that insulin and cortisol related disorders are very complicated. They involve numerous vicious cycles and impact every major aspect of human physiology. A detailed evaluation of blood sugar and insulin related disorders should be considered in all patients.

References

[i].Grimm JJ. Interaction of physical activity and diet: implications for insulin-glucose dynamics. *Public Health Nutr* 1999;2:363-368.

[ii] Moore MA, et al. Implications of the hyperinsulinemia –diabetes-cancer link for preventative efforts. *Eur J Cancer Prev* 1998;7:89-107

[iii] Despres JP, et al. Hyperinsulinemia as an independent risk factor for ischemic heart disease. *N Eng J Med* 1996;334:952-957.

[iv] Tiihonen M, Partinen M. Narvanen S. The severity of obstructive sleep apnea is associated with insulin resistnace. *J Sleep Res* 1993;2:56-61.

[v] Arthur LS, Selvakumar R, Seshardir MS. Hyperinsulinemia in polycystic ovary disese. *J Reprod Med* 1999;44:783-787.

[vi] Benzi L, et al. Intracellular hyperinsulinemia: a metabolic characteristic of obesity with and without type 2 diabetes: intracellular insulin in obesity and type 2 diabetes. *Diabetes Res Clin Pract* 1999:46:231-237.

[vii] Stoll BA. Essential fatty acids, insulin resistance and breast cancer risk. *Nutr Cancer* 1998;31:72-77.

[viii] Guyton and Hall., Textbook of Medical Physiology, ninth edition (Philadelphia, Pennsylvania, W.B. Saunders Company, 1996).

[ix] Perlmutter, David, Stress Aging and Neurodegenerative Disordes, The Eight International Symposium of Functioanl Medicine, May 22-26, 2001. Vancover, British Columbia.

[x] Ericksson, P.S., et al., Neurogenesis in the adult human hipppcampus,*Nature Medicine* 4(11): 1313-17.

[xi] De Kloet, E.R., et al., Brain corticosteroid receptor balance in health and disease. *Endocr Rev* 19(3):269-301; June, 1998.

[xii] Bergh, F.T., et al. Dysregulation of the hypothalamo-pituitary-adrenal axis is related to the clinical course of MS. *Neurology* 53; 772-777,1999.

[xiii] Lyons, D.M., et al. Early environmental regulation of glucocorticoid feedback sensitivity in young adult monkeys. *J Neuroendocrinology* 12: 723-728;2000.

[xiv] Jack, C.R., et al. Rates of hippocampal atrophy correlate with change in clinical status in aging and Alzheimer's disease. *Neurology*; 55:484-489, August, 2000.

[xv] Peterson, R.C., et al. Memory and MRI-based hippocampal volumes in aging and Alzheimer;s disease. *Neurology* 54:581-587;February, 2000.

[xvi] Weaver, S.A., et al. Neonatal handling permanently alters hypothalamic-pituitary-adrenal axis function, behavior and body weight in boars. *J Endocrin* 164(3): 349-359;March, 2000.

[xvii] Perlmutter, David. Functional Therapies in Neurodegeneratve Disease. Monograph published by Great Smokies Diagnostic Laboratory.

[xviii] Halliwell B. Reactive oxygen species and the central nervous system. *J Neurochem* 1992; 59:1609-1623.

[xix] Rothstein JD, et al. Chronic inhibition of glutamate uptake produces a model of slow neurotoxicity.

[xx] Sakakura, M, et al. *J Clin Trials.* 1989; 26:33.

[xxi] Starkman, MN, et al. Decrease in cortisol reverses human hippocampal atrophy following treatment of Cushing;s disease. *Biol Psychiatry* 12: 1595-1602, 1999.

[xxii] Brindley DN. Role of glucocorticoid and fatty acids in the impairment of lipid metabolism observed in the metabolic syndrome. *Int J Obes Relat Metab Disord.* 1995;19(suppl 1):S65-S69.

[xxiii] Koistinen HA, et al. The effect of exercise on leptin concentrations in healthy men and in type I diabetic patients. *Med Sci Sports Exercise.* 199;30(6):805-810.

[xxiv] Havel PJ. Leptin production and action: relevance to energy balance in humans. *Am J Clin Nutr.* 1998;67(3):355-358.

[xxv] Freidman JM. Leptin, leptin receptors, and the control of body weight. *Nutr Rev.* 1998;56(2):S38-S46.

[xxvi] Widdowson PS, et al. Inhibition of food response to intracerebroventricular injection of leptin is attenuated in rats with diet-induced obesity. *Diabetes.* 1997:46:1782-1785.

[xxvii] Luke AH, et al. Leptin and body composition of Nigerians, Jamaicans, and US blacks. *Am J Clin Nutr.* 1998;67(3):391-396.

[xxviii] Baumann H, et al. The full-length leptin receptor has signaling capabilities of interleukin 6-type cytokine receptors. *Proc Natl Acad Sci USA.* 1996;93(16):8374-8378.

[xxix] Kapcala LP, et al. The protective role of the hypothalamus-pituitary-adrenal axis against letheality produced by immune, infectious and inflammatory stress. *Ann Ny Acad Sci;1995:771:364-371.*

[xxx] Sternburg EM, et al. Overview of neuroimmune stress interactions, *Ann Ny Sci, 1995:364-371.*

[xxxi] Marx C., et al. Regulation of adrenocortical function by cytokines-relevance for immune-endocrine interaction. *Hormone Metab Res.* 1998;30:416-420.

[xxxii] Soderholm J.D., et al. Stress and the gastrointestinal tract. II. Stress and the intestinal barrier function. *Am j Physiol Gastrointest Liver Physiol.* 2001;280:G7-G13.

[xxxiii] Johnston S.D., et al. Intestinal permeability and morphometric recovery in celiac disease. *Lancet.* 2001;358:259.

[xxxiv] Kaukinen, K., et al. Small bowel mucosal inflammation in reticulin or gliadin antibody-positive patients without villous atrophy. *Scan J Gastroenterol,* 33(9). 944-949, 1988.

[xxxv] Hakeem, V., et al. Salivary IgA antigliadin antibody as a marker for celiac disease. *Arch Dis. Childhood.* 67:724-727, 1992.

[xxxvi] Al-Bayaty, HF., et al. Salivary and serum antibody to gliadin in the diagnosis of celiac disease. *J oral-Pathol Med.* 18:578-581, 1989.

[xxxvii] Riordan, SM., et al. Luminal antigliadin antibodies in small intestineal bacteria overgrowth. *Am J Gastroenterol,* 92(8). 1335-8, 1997.

[xxxviii] LoPresti, JS and Nicoloff, JT. Thyroid response to critical illness. Endocrinology of Critical Disease. Human Press. Totowa. NJ. 1997. pp 157-173.

[xxxix] Strakis, CA and Chrousos, GP. Neuroendocrinology and Pathophysiology of the Stress System, *Ann Ny Acad Sci,* Vol. 771, pp. 1-18,1995.

[xl] Stockigt, JR. Update on the Sick Euthyroid Syndrome, in Baverman, LE ed., Diseases of the Thyroid, Humana Press, Totowa, NJ, 1997, pp. 49-68.

[xli] Van Der Pomp G., et al. Elevated basal cortisol levels and attenuated ACTH and cortisol resposes to a behavioral challenge in women with metastatic breast cancer. *Psychoneuroendocrinology,* 21(4). 361-374,1996.

[xlii] Guhad, FA, et al. Salivary IgA as a marker of social stress in rats. *Neurosci Lett,* 216(2). 137-140,1996.

[xliii] Cunningham-Rundies, C., et al. *Proc. Nat Acada. Sci USA.* 75:3387,1978.

[xliv] Cunningham-Rundies, C., et al. *J Clin Invest.* 64:270, 1979.

[xlv] Takahashi, I and Kiyona, H. Gut as the largest immunolofic tissue. *J Parenter Enteral Nutr,*23(5 Suppl). S7-S12,1999.

[xlvi] Scott, H., et al. *Scan. J. Gastroenterol.* 15:81, 1980.

[xlvii] Romero-Piffigner, M, et al. *Mycopathologia.* 91:165, 1985

[xlviii] Schonheyder,H, et al. IgA and IgG serum antibodies to candida albicans in woen of child-bearing age. *Sabouraudia,* 21(3). 223-231, 1883.

[xlix] Harebakidis, S.T. *Acta Tropica.* 41:303,1984.

[l] Daynes, R., et al. *Eur J Immunol.* 20:793,1990.

[li] Manolagas SC, Anderson DC, Lindsay R. Adrenal steroids and the development of osteoporosis in oopherectomized women. *Lancet* 1979;2:597.

[lii] Catargi B, et al. Development of bone mineral density after cure of Cushing's syndrome. *Ann Endocrinol* 1996;57(3):203-208.

[liii] Bockman RS, Weinerman SA. Steroid-induced osteoporosis. *Ortho Clin North Am* 1990;21(1):97-107.

[liv] Guechot J, et al. Simple laboratory tests of neuroendocrine disturbance in deprrsion. *Bio Psychiat* 1982;16: 357-364.

[lv] Goddyer I, et al. Cortisol hypersecretion and in depressed school-aged children and andolescents. *Psychiat Res* 1991;37:237-244.

[lvi] Von Zesseen D, et al. Diurinal variation of mood and the cortisol rhythm in depression and normal states of mind. *Eur Arch Psychiat Neurol Sci* 1987;237:36-45.

[lvii] Gold P, Goodwin F, Chrousos G. Clinical and biochemical manifestations of depression. *NEJM* 1988;319:348.

[lviii] Turner R, Bieser M. Depression and predisposing influence of stress. *Behavioral and Brain Science* 1990;5:89.

[lix] J. Bolufer, "Salivary corticosteroids in the study of adrenal function," *Clinica Chimica Acta* 183(1989):217-226.

[lx] B. Kahn et al., "Salivary Cortisol: A practical method for evaluation of adrenal function," *Biological Psychiatry* 23(1988): 335-349.

[lxi] Davis SN, Tate D, "Effects of morning hypoglycemia on neuroendocrine and metabolic responses to subsequent afternoon hypoglycemia in normal man," *J Clin Endocrinol Metab* 2001 May;86(5): 2043-50.

[lxii] Davis SN et al., "Effects of differing antecedent hypoglycemia on subsequent counterregulation in normal humans," *Diabetes* 1997 Aug;46(8):1328-35.

[lxiii] Matyka, Crowen, et al., "Conterregulation during spontaneseuos nocturnal hypoglycemia in prepubertal children with type I diabetes," *Diabetes Care* 1999 Jul;22(7):114-50.

[lxiv] Bendston et al., "Nocturnal versus diurnal hormonal counterregulation to hypoglycemia in type I diabetic patients," *Acta Endocrinol* 1993 Feb;128(2):109-15.

[lxv] Garrel et al., "Decreased hypoglycemia effect of insulin at night in insulin-dependent diabetes mellitus and healthy subjects," *J Clin Endocrinol Metab* 1992 Jul;75(1):106-9.

[lxvi] Burge et al., "Effect of short-term glucose control on glycemic thresholds for epinephrine and hypoglycemic symptoms," *J Clin Endocrinol Metab* 2001 Nov;86 (11):5471-8.

[lxvii] Scott, Scandart, "Nocturnal cortisol release during hypoglycemia in diabetes," *Diabetes Care* 1981 Sept-Oct;4(5):514-8.

[lxviii] McGregor et al., "Elevated endogenous cortisol reduces autonomic neuroendocrine and symptom response to subsequent hypoglycemia," *Am J Physiol Endocrinol Metab* 2002 Apr; 282(4):E770-7.

[lxix] Davis et al., "Role of cortisol in pathogenesis of deficient counterregulation after antecedent hypoglycemia in normal humans," *J Clin Invest* 1996 Aug 1;98(3):680-91.

[lxx] Davis SN, Shavers C et al., "Prevention of an increase in plasma cortisol during hypoglycemia preserves subsequent counteregulatory responses," *J Clin Invevist* 1997 Jul 15;100(2):429-38.

[lxxi] Van Couter,E., et al. Age-relalted changes in slow wave sleep and REM sleep and relationship with growth hormone and cortisol levels in healthy men. *JAMA.* 284:861-888, 2000.

[lxxii] Kobayashi,T., et al. *Exp Brian Res Suppl.* 1:260-268,1985.

[lxxiii] Bom, et al. *Bio Psychiatry.* 2:1415,1986.

[lxxiv] Bergh, FT, et al., Dysregulation of the hypothalmo-pituitary-adrenal axis is related to the clinical course of MS. *Neurobiology.* 53;772-777,1999.

[lxxv] Sunderland T, et al. Reduced plasma dehydroepiandrosterone concentrations in Alzheimer;s disease. *Lancet* 1989;2:570.

[lxxvi] Nowaczynski WF, Further evidence of altered adrenocortical functions in hypertension:dehydroepiandrosterone secretion rate. *Can J Biochem* 1968;46:1031-1038.

[lxxvii] Fredrickson M, Tuomisto M, Bergman-losman B, Neuroendocrine and cardiovascular stress reactivity in middle-aged normotensive adults with parental history of cardiovascular disease. *Psychophysiol* 1991;28(6):656-664.

[lxxviii] Walker BR, Abnormal glucocorticoid activity in subjects with risk for cardiovascular disease. *Endocr Res* 1996;22(4):708-710.

[lxxix] Tsuda K, et al, Electrocardiographic abnormalities in patients with Cushing's syndrome. *Jpn Heart J* 1995;36(3):333-339.

[lxxx] Tedde R, Sechi LA, Marigliao A, Palo A, Scano L. Antihypertensive effect of insulin reduction in diabetic hypertensive patients. *Am J Hypertens* 1989;2:163-170.

[lxxxi] Randeree H, Omar MA, Motala AA, Seedet MA. Effect of insulin therapy on blood pressure in NIDDM patients with secondary failire. *Diabetes Care* 1992;15:1258-1263.

[lxxxii] Zavaroni I, Mazza S, Dall'aglio E, et al. Prevelance of hyperinsulinemia in patients with high blood pressure. *J int Med* 1992;231:235-240.

[lxxxiii] Potter van Loon, et al. The cardiovascular risk factor plasminogen activator inhibitor type I is related to insulin resistance. *Metabolism* 1993;42:945-949.

[lxxxiv] Juhan-Vague, et al. Fibrinolytic factors and the risk of myocardial infarction or sudden death in patients with angina pectoris. *Circulation* 1996;94(9):2057-2063.

[lxxxv] Dietschy JM, Brown MS. Effect of alterations of the specific activity of the intracellular acetyl CoA pool apparent rates of hepatic cholesterogenesis. *J Lipid Res* 1974;15:508-516.

[lxxxvi] Swensen TL. The role of cholesteryl ester transfer protein in lipoprotein metabolism. *Diabetes Metab Res* 1991;7:139-153.

[lxxxvii] Sozen I, Arici A. Hyperinsulinism and its interaction with hyperandrogenism in polycystic ovary syndrome. *Obest Gynecol Surv* 2000;55:321-328,

[lxxxviii] Nestler J. Insulin resistance effects on sex hormones and ovulation in the polycystic ovary syndrome. In:Reaven G, Laws A, eds. *Contemporary Endocrinology: Insulin Resistance.* Totowa NJ: Humana Press; 1999.347-365.

[lxxxix] Dahlgreen E, et al. Women with polycystic ovary syndrome wedge resected in 1956 to 1965: a long-term follow-up focusing on natural history and circulating hormones. *Fertil Steril* 1992;57:505-513.

[xc] Oberfiled S. Metabolic lessons from the study of young adolescents with polycystic ovary syndrome, is insulin indeed the culprit. *J Clin Endocrinol Metab* 2000;85:3520-3525.

[xci] Wild RA. Obesity, lipids, cardiovascular risk and androgen excess. *Am J Med* 1995;98:27S-32S.

[xcii] Wu XK, et al. Ovarian-adrenal cross-talk in polycystic ovary syndrome: evidence of wedge resection. *Euro J Endocrinol* 2000;143:383-388.

[xciii] Singh A, et al. Effect of insulin-like growth factor-type I (IFG-1) and insulin on the secretion of sex-hormone binding globulin and IGF-1 binding protein (IBP-1) by human hepatoma cells. *J Endocrinol* 1990;124:R1-3.

[xciv] Nestle J, et al. A direct effect of hyperinsulinemia on serum sex-hormone binding globulin levels in obese women with polycystic ovary syndrome. *J Clin Endocrinol Metab* 1991;72:83-89.

[xcv] Ciaraldi TP, et al. Cellular mechanisms of insulin resistance in polycystic ovarian syndrome. *J Clin Endocrinol Metab* 1992;75:577-583.

[xcvi] Peiris AN, Mueller RA, et al. Relationship of androgenic activity to splanchnic insulin metabolism and peripheral glucose utilization in premenopausal women. *J Clin Endocrinol Metab* 1987;64:162-169.

[xcvii] Pasquali R, Fabbri R, et al. Effect of weight loss and antiandrogenic therapy on sex hormone blood levels and insulin resistance in obese patients with polycystic ovaries. *Am J Obstet Gynecol* 1986;154:139-144.

[xcviii] Conway GS, Honour JW, Jacobs HS. Heterogeneity of polycystic ovary syndrome: clinical endocrine and ultrasound features in 566 patients. *Clin Endocrinol* 1989;30:459-470.

[xcix] Yen SS. The polycystic ovary syndrome. *Clin Endocrinol* 1980;12:177-207.

[c] Wildt L, Sir-Petermann T, et al. Opiate antagonist treatment of ovarian failure. *Hum Reprod* 1993;8:168-174.

[ci] Jenkins PJ, Grossman A. The control of gonadotrophin releasing hormone pulse generator in relation to opiod and nutritional cues. *Hum Reprod* 1993;8:154-161.

[cii] Carmina E, Ditkoff EC, et al. Increased circulating levels of immunoreactive beta-endorphin in polycystic ovary syndrome is not caused by pituitary secretion. *Am J Obstet Gynecol* 1992;167:1819-1824.

[ciii] Velazquez E, et al. Menstrual cyclicity after metaformin therapy in polycystic ovary syndrome. *Obstet Gynecol* 1997;90:392-395.

[civ] Venturoli S, et al. Ketoxonazole therapy for women with acne and/or hirstutism. *J Clin Endocrinol Metab* 1990;71:335-337.

[cv] Fuenmayor NM, et al. Relations between fasting serum insulin, glucose, and dehydroepiandrosterone-sulfate concentrations in obese patients with hypertension: short term effects of antihypertensive drugs. *J Card Pharm* 1997;30:523-527.

[cvi] Nestler JE, Clore JN, Blackard WG. Dehydroepiandrosterone: the missing link between hyperinsulinemia and atherosclerosis. *FASEB* 1992;9:3073-3074.

[cvii] Zakrzewska KE, et al. Glucocorticoids as counterregulatory hormones of leptin: toward an understanding of leptin resistance. *Diabetes* 1997;46: 717-719.

[cviii] Zakrzewska, et al. Induction of obesity and hyperleptinemia by central glucocorticoid infusion in the rat. *Diabetes* 1999;48:365-370.

[cix] Bermeis K, Vosmeer S, Kelly U. Effects of gluccorticoids and of growth hormone on serum leptin concentrations in man. *Eur J Endocrinol* 1996;663-665.

[cx] Weiderpass E, et al. Diabetes mellitus and risk of large bowel cancer. *J Natl Cancer Inst* 1997;89:660-661.

[cxi] Corpet D, et al. Insulin injections promote the growth of aberrant crypt foci in the colon of rats. *Nutr Cancer* 1997;27:316-320.

[cxii] Storlien LH, et al. Fatty acids, triglycerides and syndromes of insulin resistance. *Prostaglandin Leukot Essent Fatty Acids* 1997;57(4-5):379385.

[cxiii] Brenner R. Nutrition and hormonal factors influencing desaturation of essential fatty acids. *Prog Lipid Res* 1982;20;41-48.

[cxiv] Shibita S, et al. Chemistry and pharmacology of Panax. *Economic and Medicinal Plant Research.* 1985; 1:217-284.

[cxv] Avakia EV, Evonuk E. Effects of Pannax ginseng extract on tissue glycogen and adrenal cholesterol depletion during prolonged exercise. *Planta Medica.* 1979; 36: 43-48.

[cxvi] Shibata S, Tanaka O, Shoji, Saito H, Chemistry and pharmacology of Panax. In: Economic and Medicinal Plant Research vol. 1. London: Acadmic Pr;1985.

[cxvii] Huong NT, Matusomoto K, Watanabe H. The antistress effect of majoniside-R2, a major saponin component of Vietnemase ginseng: neuronal mechanisms of action. *Methods Find Exp Clin Pharmacol* 1998;20(1):65-76.

[cxviii] Gaffny BT, Hugel HM, Rich PA. Pannax ginseng in Eleutherococcus senticosus may exaggerate an already existing biphasic response to stress via inhibition of enzymes which limit the bindin of stress hormones to their receptors. *Med Hypotheses* 2001;56(5):56-72.

[cxix] Fulder SJ. Ginseng and the hypothalamic-pituitary control of stress. *Am J Chinese Med* 1981 ;9(2):112-118.

[cxx] Nishibe S, et al. Phenolic compounds from stem bark of Acanthopanax senticosus and their pharmacological effect in chronic swimming stressed rats. *Chem Pharm Bull.* 1990;38:1763-1765.

[cxxi] Golotin VG, et al. Effect of ionol and eleutherococcus on changes of the hypophysea-adrenal system in rats under extreme conditions. *Vopr Med Khim.* 1989;35:35-37.

[cxxii] Filaretov AA, et al. Effects of adaptagens on the activity of the pituitary-adrenocortical system in rats. *Biull Eksp Biol Med.* 1986;101:573-574.

[cxxiii] Fransworth NR, et al. Siberian ginseng (Eleutherococcus senticosus): Current status of an adaptagen. *Econ Med Plan Res.* 1985;156-215.

[cxxiv] Eleskka M, et al. Withania somnifera, a plant with a great therapeutical future. *Rev Med Chir Soc Med Nat Iasi.* 1989;93:349-350.

[cxxv] Grandhi A, et al. A comparative pharmacological investigation of Ashwagandha and Ginseng. *J Ethnopharmacol.* 1994;44:131-135.

[cxxvi] Kapoor LDL: Handbook of Ayuvedic Medicinal Plants, CRC Press: NY, 1990 (337-338)

[cxxvii] Sembulingam K, et al. *Indian J Physiol Phamacol.* 1997; 41(2);139-143.

[cxxviii] Wichtl MW. Herbal Drugs and *Phytopharmaceuticals.* Ed. N.M. Bisset. Stuttgart: Medpharm Scientific Publishers

[cxxix]Wagner H, et al. Plant Adaptagens, Phytomedicne Vol 1 1994, pp. 63-76.

[cxxx] Azizova AP, Seifulla RD. The effect of elton, leveton, fitoton and adapton on the work capacity of experimental animals. *Eksp Klin Farmakol* 1998;61:61-63.

cxxxi Maslova LV, Kondrat'ev Blu, Maslov LN, Lishmanov IuB. The cardioprotective and antiadrenergic activity of an extract of Rhodiola rosea in stress. *Eksp Klin Farmakol* 1994;57:61-63.

cxxxii Maimeskulova LA, Maslov LN. The anti-arrhyhmia action of an extract of Rhodiola rosea and of n-tyrosol in models of experimental arrhythmias. *Eksp Klin Farmakol* 1998;61:37-40.

cxxxiii Afanas'ev SA., Alekseeva ED, Bardamova IB, et al. Cardiac contractile function following acute cooling of the body and the adaptagenic corrections of its disorders. *Biull Eksp Biol Med* 1993;116:480-483.

cxxxiv Lishmanov IuB, Trifonova ZhV, Tsibin AN, et al. Plasma beta-endorphin and stress hormones in stress and adaptation. *Buil Eksp Biol Med* 1987;103:422-424.

cxxxv Spasov AA, Wikman GK, Mandrikov VB, et al. A double-blind placebo-controlled pilot study of the stimulating and adapatogenic effect of Rhodiola rosea SHR-5 extract on the fatigue of students caused by stress during an examination period with repeated low-dose regimen. *Phytomedicine* 2000;7(2):85-89.

cxxxvi Darbinyan V, Kteyn A, Panossian A, et al. Rhodiola rosea in stress induced fatique – a double blind cross-over study of a standardized extract SHR-5 with a repeated low-dose regimen on the mental performance of healthy physicians during night duty. *Phtomedicine* 2000;7(5):365-71.

cxxxvii Mungantiwar AA, Nair AM, Shinde UA, Saraf MN. Effect of stress on plasma and adrenal cortisol levels and immune responsiveness in rats: modulation by alkaoidal fraction of Boerhaavia difuasa. *Fitoterapia* 1997;6:498-500.

cxxxviii Kosaka C, Okdio M, Keneyuki, et al. Action of panathine on the adrenal cortex of hypophysectomized rats. *Horumon to Rinsho* 1973;21:517-525.

cxxxix Onuki M, Hoshino H. Effects of pantehine on the adrenocortical fuction. Experimental results using rabbits. *Horumon To Rinsho* 197018:601-605.

cxl Koska M, Kikui S, Fujiwara T. Kimoto T. Aciton of panathine on the adrenal cortex. *Horumn ToRhinsho* 1966;14:843-847.

cxli Onuki M, Suzawa A. Effect of panthine on the function of the adrenal cortex. Clinical experience using panatethine in cases under steroid hormone treatment. *Horumon To Rhinsho* 1970;18:937-940.

cxlii Bhatt HR. Antagonistic effectsof pentagastrin and cortisol on plasma level, urinary excretion and hepatic and renal uptake of vitamin B12 after intravenous injection in the cat. *J Physiol* 296, 1979.

cxliii Crook TH, Petrie W, Wells C, Massari DC, Effects of phosphatidylserine in Alzheimer's Disease. *Psychopharmacol Bull* 1992;28:61-66.

cxliv Ameducci L, Crook TH, Lippi A, et al. Use of phosphatidylserine in Alzheimer's Disease. *Ann Ny Acad Sci* 1991;640:245-249.

cxlv Cenacchi T, Bertoldin T, Farina C, et al. Cognitive decline in the elderly: A double-blind, placebo controlled multicenter study on efficacy of phosphatidylserine administration. *Aging Clin Ecp Res* 1993; 5:123-133.

cxlvi Latorraca S, Piersanti P, Tesco G, et al. Effects of phosphatidylserine on free radical susceptibility in human diploid fibroblasts. *Neurol Transm Park Dis Dement Sect* 1993;6:73-77.

cxlvii Maggioni M, Picotti GB, Bondiolotti GP, et al. Effects of phosphatidylserine therapy in geriatric patients with depressive disorders. *Acta Psychiatr Scand* 1990;81:265-270.

cxlviii Brambilli F, Magioni M. Blood levels of cytokines in elderly patients with major depressive disorder. *Acta Psychiatr Scand* 1998;97:309-313.

cxlix Brambilla F, Maggioni M, Panerai AE, et al. Beta-endorphin concentration in peripheral blood mononuclear cells of elderly depressed patients effects on phosphatidylserine therapy. *Neuropsychobiology* 1996;34:18-21.

cl Palmieri G. Double-blind controlled trial of phosphatidylserine in patients with senile mental deterioration. *Clin Trials J* 1987;24:73-83.

cli Kidd PM, Phosphatidylserine; membrane nutrient for memory. A clinical and mechanistic assessment. *Altern Med Rev* 1996;1:70-84.

clii Cenacchi T, Betoldin R, Farina C, et al. Cognitive decline in the elderly. A double blind, placebo-controlled multicenter study on efficacy of phosphatidylserine administration. *Aging* 1993;5:123-133.

cliii Crook TH, Tinklenberg J, Yesavage J, et al. Effects of phosphatidylserine in age-associated memory impairment. *Neurology* 1991;41:644-649.

cliv Monteleone P, Mag M, Beinat, et al. Blunting of chronic phosphatidylserine administration of the stress-induced activation of the hypothalamic-pituitary-adrenal axis in healthy men. *Eur J Clin Pharmacol* 1992;41:385-388.

clv Monteleone P, Beintat L, Tanzillo C, et al. Effects of phosphatidylserine on the neuroendocrine response to physical stress in humans. *Neuroendocrinology* 1990;52:243-248.

clvi Nerozzi D, Aceti F, Melia E, et al. Early cortisol escape phenomenon reversed by phosphatidylserine in elderly normal subjects. *Clinical Trial J* 1989;26:33-38.

clvii Fahey TD, Pearl MS. The hormonal and perceptive effects of phosphatidylserine administration during two weeks of resistive exercise-induced overtraining. *Biol Sport* 1998;15:135-144.

clviii LoPresti, JS and Nicoloff, JT. Thyroid response to critical illness. Endocrinology of Critical Disease. Human Press. Totowa. NJ. 1997. pp 157-173.

clix Strakis, CA and Chrousos, GP. Neuroendocrinology and Pathophysiology of the Stress System, *Ann Ny Acad Sci*, Vol. 771, pp. 1-18,1995.

clx Stockigt, JR. Update on the Sick Euthyroid Syndrome, in Baverman, LE ed., Diseases of the Thyroid, Humana Press, Totowa, NJ, 1997, pp. 49-68.

clxi Van Der Pomp G., et al. Elevated basal cortisol levels and attenuated ACTH and cortisol resposes to a behavioral challenge in women with metastatic breast cancer. *Psychoneuroendocrinology,* 21(4). 361-374,1996.

clxii Guhad, FA, et al. Salivary IgA as a marker of social stress in rats. *Neurosci Lett,* 216(2). 137-140,1996.

clxiii Cunningham-Rundies, C., et al. *Proc. Nat Acada. Sci USA.* 75:3387,1978.

clxiv Scott, H., et al. *Scan. J. Gastroenterol.* 15:81, 1980.

clxv Daynes, R., et al. *Eur J Immunol.* 20:793,1990.

[clxvi] Maggioni M, Picotti GM, Bondiolotti GP, et al. Effects of phosphatidylserine therapy in geriatric patients with depressive disorders. *Acta Psychiatr Scan* 1990;81:265-270.

[clxvii] Havel PJ. Leptin production and action: relevance to energy balance in humans. *Am J Clin Nutr.* 1998;67(3):355-358.

[clxviii] Freidman JM. Leptin, leptin receptors, and the control of body weight. *Nutr Rev.* 1998;56(2):S38-S46.

[clxix] Brindly DN. Role of glucocorticoids and fatty acids in the impairment of lipid metabolism observed in metabolic syndrome. *Int J Obes Relat Metab Disord* 1995;19(Suppl 1):S69-75.

[clxx] Bergh, F.T., et al. Dysregulation of the hypothalamo-pituitary-adrenal axis is related to the clinical course of MS. *Neurology* 53; 772-777,1999.

[clxxi] Shanmugasundaram ERB, et al. Use of Gynema sylvestre leaf extract in the control of blood glucose in insulin-dependent diabetes mellitus. *J Ethanopharmacol* 1990; 30: 281-294.

[clxxii] Persaud SJ, Al-Majed H, Raman A, Jones PM. Gymnema sylvestre stimulates insulin release in vitro by increased membrane permeability. *J Endocrinol* 1999; 163(2):207-12.

[clxxiii] Shanmugasundaram ERB, et al. Possible regeneration of the islet of Langerhans in streptozoticin-diabetic rats given Gynema sylvestre leaf extracts. *J Ethnopharmacol* 1990 30; 265-279.

[clxxiv] Shanmugasundaram KR, et al. Enzyme changes and glucose utilization in diabetic rabbits: the effect of Gymnema sylvestre, R.Br. *J Ethnopharmacol* 1983;7(2):205-34.

[clxxv] Kaskaran K, et al. Antidiabetic effect of a leaf extract from Gymnema sylvestre in non-insulin dependent diabetes mellitus patients. *J Ethnopharmacol* 1990; 30: 295-305.

[clxxvi] Khare AK, Tondon RN, Tewari JP. Hypoglycemic activity of an indigenous drug (Gymnema sylvestre, 'Gurmar') in normal and diabetic persons. *Indian J Physiol Pharmacol* 1983;27(3):257-8.

[clxxvii] Striffler JS, Polansky MM, Anderson RA. Overproduction of insulin in the chromium-deficient rat. *Metabolism* 1999;48:1063-1068.

[clxxviii] Striffler JS, Polansky MM, et al. Chromium improves insulin response to glucose in rats. *Metabolism* 1995;44:1314-1320.

[clxxix] Anderson RA, Polansky MM, Bryden NA, Canary JJ. Supplemental-chromium effects on glucose, insulin, glucagon, and urinary chromium losses in subjects consuming controlled low-chromium diets. *Am J Clin Nutr* 1991:54:909-916.

[clxxx] Offenbacher EG, Pi-Sunyer FX. Beneficial effect of chromium-rich yeast on glucose tolerance and blood lipids in elderly subjects. *Diabetes* 1980;29:919-925.

[clxxxi] Riales R, Albrink MJ. Effect of chromium chloride supplementation on glucose tolerance and serum lipids including high-density lipoprotein of adult men. *Am J Clin Nutr* 1981;34:2670-2678.

[clxxxii] Evans GW, Browman TD. Chromium picolinate increases membrane and rate of insulin internalization. *J Inorg Bio* 1992;46:243-250.

[clxxxiii] Roeback, Jr. JR, Hla KM, Chambless, et al. Effects of chromium supplementation on serum high density lipoprotein cholesterol levels in men taking beta-blockers. *Ann Intern Med.* 1991:115:917-924.

[clxxxiv] Anderson R, Cheng N, Bryden, et al. Beneficial effects of chromium for people with type II diabetes. *Diabetes.* 1996;45:124A.

[clxxxv] Anderson RA, Cheng N, Bryden NA, et al. Elevated intakes of supplemental chromium improve glucose and insulin variables in individuals with type 2 diabetes. *Diabetes* 1997;46:1786-1791.

[clxxxvi] Levine R, Streeten D, Doisy R. Effect of oral chromium supplementation on the glucose tolerance of elderly human subjects. *Metabolism* 1968;17:114-125.

[clxxxvii] French RJ, Jones PJ. Role of vanadium in nutrition: metabolism, essential and dietary considerations. *Life Sciences.* 1992;52:339-346.

[clxxxviii] Shechter, Li Meyerovitch, et al. Insulin-like actions of vanadate are mediated in an insulin receptor independent manner via non-receptor protein tyrosine kinases and protein phosphotyrisine phosphates. *Mol Cell Biochem.* 1995;153(1-2):39-47.

[clxxxix] Halberstm M, Cohen N, Shlimovish P, et al. Oral vanadyl sulfate improves insulin sensitivity NIDDM but not in obese nondiabetic subjects. *Diabetes.* 1996;45:659-665.

[cxc] Boden G, Chen X, Ruiz J, et al. Effects of vanadyl sulfate on carbohydrate and lipid metabolism in patients with non-insulin dependent diabetes mellitus. *Metabolism.* 1996;45:1130-1135.

[cxci] Cohen N,Halberstam M, Shlimovih P, et al. Oral vanadyl sulfate improves hepatic and peripheral insulin insensitivity in patients with non-insulin dependent diabetes mellitus. *J Clin Invest. 1995;95:2501-2509.*

[cxcii] Goldfine AB, Simonson DC, Folli F, et al. Metabolic effects of sodium metavandate in humans with insulin-dependent and noninsulin-dependent diabetes mellitus in vivo and vitro studies. *J Clin Enco and Meta*. 1995;80(11);3311-3319.

[cxciii] Shang H, Osada K, Maebashi M, et al. A high biotin diet improves the impaired glucose tolerance of long-term spontaneously hyperglycemic rats with non-insulin dependent diabetes mellitus. *J Nutr Sci Vitamin* 1996;42:517-526.

[cxciv] Jacob S, Henriksen EJ, Schiemann AL, et al. Enhancment of glucose disposal in patients with Type 2 diabetes by alpha-lipoic acid. *Arzneim-Rosch Drug Res* 1995;45(2):872-874.

[cxcv] Jacob S, Streeper RS, Fogt DL, et al. The antioxidant alpha-lipoic acid enhances insulin-stimulated glucose metabolism in insulin resistant rate skeletal muscle. *Diabetes* 1996;45:1024-1029.

[cxcvi] Streeper RS, Henriksen EJ, Jacob S, et al. Differential effects of lipoic acid stereoisomers on glucose metabolism in insulin resistant skeletal muscle. *Am J Physiol* 1997;273:E185-E191.

[cxcvii] Konard T, Vicini P, Kuster K, et al. Alpha-lipoic acid treatment decreases serum lactate and pyruvate concentrations and improves glucose effectiveness in lean and obese patients with type 2 diabetes. *Diabetes Care* 1999;22(2):280-287.

[cxcviii] Rudich A, Tirosh A, Potashinik R, Khamaisi M, Bashan N. Lipoic acid protects against oxidative stress induced impairment in insulin stimulation of protein kinase B and glucose transport in 3T3-L1 adipocytes. *Diabeologia* 1999;42:949-957.

[cxcix] Kawabata T and Packer L. Alpha-lipoate can protect against glycation of serum albumin, bu nto low density lipoprotein. *Biochem Biophys Res Comm* 1994;203:99-104.

[cc] Jacob S, Ruus P, Hermann R, et al. Improvement of insulin-stimulated glucose-disposal in type 2 diabets after repeated patenteral administration of thioctic acid. *Exp Clin Endocrinol Diabetes* 1996;104:284-288.

[cci] Jacob S, Ruus, Herman R, et al. Oral administration of RAC-alpha-lipoic acid modulates insulin sensitivity in patients with type-2 diabetes: a placebo-controlled pilot trial. *Free Radic Biol Med* 1999;27:309-314.

[ccii] Nagamstsu M, et al. Lipoic acid improves nerve blood flow, reduces oxidative stress, and improves distal nerve conduction in experimental diabetic neuropathy. *Diabetes Care* 1995;18:1160-1167.

[cciii] Paolisso G, D'Amore A, Galzenano D. Daily vitamin E supplementations improve metabolic control but not insulin secretion in elderly type II diabetic patients. *Diabetes Care* 1993;16:1433-1437.

[cciv] Salonen JT, Jyyssonen K, Tuomainen TP. Increased risk of non-insulin diabetes mellitus at low plasma vitamin E concentrations. A four year follow-up study in men. *Br Med J* 1995;311:1124-1127.

[ccv] Barbagallo M, Dominquez LJ, Tagliamonte MR, et al. Effects of vitamin E and glutathione on glucose metabolism: role of magnesium. *Hypertension* 1999;34:1002-1006.

[ccvi] Paolisso G, D'Amore A, Guigliano D, et al. Pharmacologic doses of vitamin E improves insulin action in healthy subjectsand in non-insulin-dependent diabetic patients. *Am J Clin Nutr* 1993;57:650-656.

[ccvii] Paolisso G, Gambardella A, Giugilano D, et al. Chronic intake of pharmacological doses of vitamin E might be useful in the therapy of elderly patients with coronary heart disease. *Am J Clin Nutr* 1995;81:848-852.

[ccviii] Jain SK, McVie R, Jaramillo JJ, et al. Effect of modest vitamin E supplementation on blood glycated hemoglobin and triglyceride levels and red cell indices in type I diabetic patients. *J Amer Col Nutr* 1996;15(5);458-461.

[ccix] Paolisso G, Ravussin E. Intercellular magnesium and insulin resistance; results in Pima Indians and Caucasians. *J Endocrinol Metab* 1995;80:1382-1385.

[ccx] Paolisso G, Sgambato S, Pizza G, et al. Improved insulin response and action by chronic mangnesium administration in aged NIDDM subjects. *Diabetes Care* 1989:12;265-269.

[ccxi] Paolisso G, Sgambato S, Gambardell A, et al. Daily magnesium supplements improve glucose handeling in elderly subjects. *Am J Clin Nutr* 1992;55:1161-1167.

[ccxii] Nadler JL, Buchanan T, Natarajan R, et al. Magnesium deficiency produces insulin resistance and increased thromboxne synthesis. *Hypertension* 1993;21:1024-1029.

[ccxiii] Humphries S, Kushner H, Falkner B. Low dietary magnesium is associated with insulin resistance in a sampling of young, nondiabetic Black Americans. *Am J Hypertens* 1999;12:747-756.

[ccxiv] Dominguez LJ, Barbagallo M, Sowers JR, Resnick LM. Magnesium responsiveness to insulin and insulin-like growth factor I in erthrocytes from normotensive and hypertensive subjects. *J Clin Endocrinol Metab* 1998;83:4402-4407.

[ccxv] Reddi A, DeAngelis B, Frank O, et al. Biotin Supplementation improves glucose and insulin tolerances in genetically diabetic KK mice. *Life Science* 1998;42:1323-1330.

[ccxvi] Kutosikos D, Fourtounas C, Kepetanaki A, et al. Oral glucose tolerance test after high-dose i.v. bioitin administration in normoglucemic hemodialysis paitents. *Ren Fail* 1996;18:131-137.

[ccxvii] Shang H, Osada K, Maebashi M, et al. A high biotin diet improves the impaired glucose tolerance of long-term spontaneously hyperglycemic rats with non-insulin-dependent diabetes mellitus. *J Nutr Sci Vitamin* 1996;42:517-526.

[ccxviii] Maebashi M, Makino Y, Furukawa Y, et al. Therapeutic evaluation of the effect of biotin on hyperglycemia in patients with non-insulin dependent diabetes mellitus. *J Clin Biochem Nutr* 1993;14:211-218.

[ccxix] Hegazi SM. Effects of zinc supplementation on serum glucose, insulin, glucagons, glucose-6-phosphate, and mineral levels in diabetics. *J Clin Biochem Nutr* 1992;12:209-215.

[ccxx] Mooradian AD, Morley JE. Micronutrient status in diabetes mellitus. *Am J Clin Nutr* 1987;45:877-895.

[ccxxi] Chen MD, Lin PY, Lin Wh. Investigation of relationship between zinc and obesity. *Kao Hsiung I Hsueh Tsa Chih* 1991;7:628-634.

[ccxxii] Singh RB, Niaz MA, Rastogi SS, et al. Current zinc intake and risk of diabetes and coronary artery diseasse and factors associated with insulin resistance in rural and urban populations of North India. *J Am Col Nutr* 1998;17:564-570.

[ccxxiii] Leslie RDG, Elliot RB. Early environmental events as a cause of IDDM. *Diabetes* 1994;43:843-850.

[ccxxiv] Urberg M, Zemel MB. Evidence of synergism between chromium and nicotinic acid in the control of glucose tolerance in elderly humans. *Metabolism* 1987;36:574-576.

[ccxxv] Pocoit F, Reimers JI, Anderson HU. Nicotimaminde – biological action and therapeutic potential in diabetes prevention. *Diabetologia* 1993;36:574-576.

[ccxxvi] Clearly JP. Vitamin B3 in the treatment of diabetes mellitus: case reports and review of the literature. *J Nutr Med* 1990;1:217-225.

[ccxxvii] Pozzilli P, Andreani D. The potential role of nicotinimide in the secondary prevention of IDDM. *Diabetes Metabol Rev* 1993;9:219-230.

[ccxxviii] Anderson HU, Jorgensen KH, Egeberg J. Nicotinamide prevents interleukin-1 effects on accumulated insulin release and nitric oxide production in rat islets of langerhans. *Diabetes* 1994;43:770-777.

[ccxxix] Heller W, Mushi HE, Gaebel G, et al. Effect of L-carnitine on post-stress metabolism in surgical paitents. Infusiosther Klin Ernahr 1986;13:268-276.

[ccxxx] Gunal AI, Celiker H, Donder E, Gunal SY. The effect of L-carnitine on insulin resistance in hemodialysed patients with chronic renal failure. *J Nephrol* 1999;12:38-40.

[ccxxxi] Mingrone G, Greco AV, Capristo E, et al. L-carnitine improves glucose disposal in type 2 diabetic patients. *J Am Coll Nutr* 1999;18:77-82.

[ccxxxii] Greco AV, et al. Effect of propionyl-L-carnitine in the treatment of angiopathy: Controlled double blind trial versus placebo. *Drugs Exp Clin Res* 1992;18:69-80.

[ccxxxiii] Abdel-Aziz MT, Abdou MS, Soliman K, et al. Effects of L-carnitine on blood lipid patterns in diabetic patients. *Nutr Rep Int* 1984;29:1071-1079.

[ccxxxiv] Regelson W, Lori R, Kalimi M, Dehydroepiandrosterone (DHEA) the "Mother Steroid" I: Immunologic Action. *Ann NY Acad Sci* 1994; 719: 553-563.

[ccxxxv] Nestler JE, Clore JN, Blackard WG. Dehydroepiandrsterone: the "missing link" between hyperinsulinemia and atherosclerosis. *FASEB J* 1992; 6: 3073-3075.

[ccxxxvi] Leiter E, Beamer W, Coleman D, Longcope C. Androgenic and estrogenic metabolitesin serum mice fed dehydroepiandrosterone: relationship to antihyperglycemic effects. *Metabolism* 1987; 36:863-869.

[ccxxxvii] Loria R Inge T, Cook S, et al. Androstenediol regulates systemic resistance against lethal infections in mice. *Arch of Virology* 1992; 127:103-115.

[ccxxxviii] Haffner SM, Valdez R, et al. Decreased testosterone and DHEA-S concentrations are associated with increased insulin and glucose concentrations in non-diabetic men. *Metabolism* 1994; 43:599-603.

An Interesting Interlude – A Case Study

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Abstract

A 67-year-old man in a coma three weeks and one day following severe head trauma is tracked chronologically from October 23, 2002 to February 15, 2003 and progress reported.

Key Words: Therapy Localization, Surrogate Testing.

Introduction

I have been a chiropractor for 54 years and involved in applied kinesiology since its introduction in 1964. During my career I have had my share of "incurable cases" and have been able to help most of these. Thanks to my conviction that the body heals itself, given the opportunity, and with the armamentarium provided by Dr. George Goodheart and the ICAK.

I had never had the opportunity to treat a person in a coma until an old friend of more than 40 years was involved in an automobile/bicycle accident on October 23, 2002.

Discussion

This is the chronology:

During the latter part of October, 2002, I received a phone message in Michigan that my old friend, Mr. S.C., was in a neurological center in Florida. He was reported to be in a coma and not expected to live. He had been thrown onto the hood of a car when struck while riding his bicycle. He wore no helmet and his head hit the windshield.

Mr. S.C. was a unique individual; a mechanical genius. He was a very energetic, alert person who could do most any repair job. Unfortunately, he was addicted to alcohol, averaging an incredible six half-pints per day. His favorite drink was Southern Comfort, hence the designation, Mr. S.C.

He had undergone coronary by-pass surgery (six arteries), twelve years before and his cardiologist reported extensive heart muscle damage and diminished cardiac output. He showed few clinical signs of this and rode his bicycle approximately ten miles per day about five days a week. (Incidentally, I put him on an extensive nutrition program after his surgery and he rehabilitated himself with incredible walking distances, before he moved to Florida and switched to the bicycle.)

I received frequent reports of his condition. He was not expected to live. I arrived in Florida November 13, 2002 and went to the hospital November 14. Though his eyes were open, there was no response to the therapist's attempts at signaling. My attempts to elicit a sign of recognition were futile. The physicians discussed his condition with Mr. S.C.'s wife. Little hope that he would live; massive brain damage. If he lived he probably would be institutionalized for life. I listened.

He had pneumonia. A tracheal tube had been inserted with a device for suctioning mucous attached. He was fed through a stomach tube. I observed the monitors for an hour. Heart rate was 128 to 136 per minute with spiking breaks in the graph every 6 to 10 beats. Respiration was 26 to 33 per minute.

He had random movements of the left arm and leg but flaccid paralysis of the right arm and leg.

Treatment Process:

(I) Using his wife as a surrogate, I had her place two fingers on the frontal bone looking for emotional involvement. No reaction.

Placed one finger on the right frontal bone reflex. No reaction.

Finger on left frontal bone. Surrogates muscle weakened.

Held left N.V. reflex two minutes after getting the pulse. NOTE: Since the aforementioned N.V. reflexes are associated with the stomach, urinary bladder, and/or emotional stress points, my practice assumption is that a two finger contact, one on each of vascular points indicates emotion and a single finger contact indicates organ or individual muscle association. Later I found some confirming evidence leading me to believe it was stomach. More about that later.

(II) The next procedure was to therapy localize all reflexes right and left for the supraspinatus, i.e. – N.L., N.V., and CSR. A stress center was found to T.L. on the left side and was tested by rubbing in the direction that abolished the T.L. Again, the muscle changes were the surrogate's.

(III) Noting a difference in the height of each eye socket, I did a hemispheric cranial adjustment.

(IV) I finished that day with a left sided temporal tap. No affirmation. I might not have thought much about any possible improvement from these simple and minimal incursions had I not been able to observe the changes in the monitors. Within two minutes of completing this routine, respiratory fell to 19, heart rate dropped to 89 to 93 and the rhythm became regular. I watched it for 15 minutes. The graph had no breaks in pattern! This was Thursday, November 14, 2002.

Second Visit, Friday, November 15, 2002.
The patient was improved and could recognize me and signaled by squeezing my fingers with his left hand. He also signaled recognition of his wife and my wife. Heartbeat was 95 to 98, regular pattern; respiration, 20 to 23.

Surrogate testing of the points treated the day before indicated no involvement. I repeated left sided temporal tap, this time with suggestions of rapid and complete recovery and left it at that.

Saturday, November 16, 2002.
Mr. S.C.'s wife was told that he had made "so much progress" he was to be moved to a rehabilitation hospital eighty-five miles away that day. Nothing happened Saturday or Sunday. I did not visit since I was expecting the move at any time. I phoned Dr. Goodheart and discussed what I had accomplished and procedures used. The patient was still completely paralyzed on the right side – arm and leg. Dr. Goodheart told me there was a reflex near the junction of sphenoid/temporal and parietal bones, i.e., neurovascular. This should be contacted and held for twenty minutes or more. The side to treat is opposite the side of dysfunction. This was consistent with everything else I had found.

Monday, November 18, 2002
Mr. S.C. was transferred to the new facility. The procedures for rehabilitation were extensive, and the hospital requested that his wife stay there and learn the routines of placing him in wheelchair and back to bed; how to suction mucous from the collecting tube at the site of the tracheal tube. He was still coughing up quantities of thick material from his lungs. She stayed at the hospital night and day for a week then came home for a day and repeated the procedure. Logistics at this time were difficult and I did not see Mr. S.C. until November 28, 2002.

At this time I used his wife to T.L. the previously treated points. There was no recidivism. I then T.L.ed the point Dr. Goodheart recommended. It was a positive T.L. I put a slight stretch on it and held it for 21 minutes. I was standing above head level. He made no movement except to roll his eyes up and to the right to look at me. On the 21st minute he reached up and removed my hand with his left hand. I asked him if I was bothering his head and he nodded slightly. Ten to fifteen minutes later, he suddenly bent his right elbow and laced his fingers together with his left hand. Before I left that day, he crossed his right leg over his left at the ankle. It was a seminal experience indeed. A medical doctor of my acquaintance once said to me, "I don't know how Goodheart comes up with the things he does. I believe he has a direct pipeline to God." That day I agreed.

November 30, 2002
I went back on this Saturday as I wanted to see what progress was made. I also wanted to be there when the professional staff was minimal. They had removed the tracheal tube and he was eating pudding and soft things of that sort. They were still feeding him via the stomach tube every four hours, also. He was sitting in a wheelchair and was able now to talk. His speech was very low and hard to understand, but not unintelligible. While I was there he placed his right foot on the floor and back on the footrest several times.

This time using surrogate testing with cervical motion: (1) I isolated an atlas/axis fixation and gave it a respiratory adjustment, (2) Using the same diagnostic model, I did a left temporal bulge adjustment and finished with a left temporal tap. We then got him into bed whereupon we T.L.ed the stomach meridian with a reaction at the Luo point ST40. I tapped it for 60 seconds abolishing the reaction.

Conclusion

NOTE (1) Some experts in kinesiology have questioned surrogate testing. Even Dr. Goodheart, who did most of the initial investigation, has said it has many variables. I have used it at times with satisfactory results. I believe this paper is one more validation of the technique.1

NOTE (2) In differentiating emotional stress from stomach or urinary bladder after the first treatment, I believe the stomach as primary organ response was confirmed by the results of tapping the stomach Luo point.

December 14, 2002
I next visited on this day. The patient's stomach tube had been removed and he was on semi-solid foods. I did little other than visit that day.

Christmas Day
My wife, myself, sister-in-law, and brother-in-law spent this day at the hospital with Mr. and Mrs. S. C. He had to eat his special diet but joined us later in the cafeteria. His strength was improving.

Mr. S.C. returned home January 11, 2003.
He now goes to a local hospital for physical therapy 2 or 3 times per week. I have a portable table and give him adjustments once per week. He seems to hold the corrections well and is gradually getting stronger, but he does not have balance to walk unaided. He has made little progress on the right arm. He has spastic flexion paralysis and does not respond well to what I have done. I am currently using ice and stretch as well as percussion to keep the shoulder and elbow joints from degenerating, but this is the one area which may not make much more improvement.

References

1. ICAK-U.S.A. Board Vote: policy and response to questions regarding nutrition and surrogate testing, MOTION, May 25, 1995.

"X" & "II" Tool for IRT, Chapman & Bennett´s Points

Jose Palomar Lever, M.D.

Abstract

This paper presents a new technique that can be used to treat injury memory, similar to the IRT of Dr. Walter H. Schmitt, Jr. This technique also helps to understand the neurological underlying causes.

Introduction

It is well known that muscle spindle mediated stretch reflexes are commonly modified by descending suprasegmental pathways arising from cerebellar and cerebral adaptations. These concepts are reviewed and a model is presented for understanding the effects of injury recall technique (IRT) in light of suprasegmental, primarily cerebellar adaptation.

Discussion

The three major sensory inputs for postural control are the eyes, the inner ears (the vestibular mechanism), and mechanoreceptors from the ankle joint. Postural adaptation depends on cerebellar integration of all three areas and its efferent supply to brainstem centers for descending pathways to motor neuron pools. The clinical findings of IRT are associated with cerebellar adaptation and habituation to areas of injury and trauma.

Following injury, there are various and numerous alterations in stretch reflexes as they adapt to flexor reflex afferent pathways arising from nociceptors. The secondary effects of nociception become the new normal as the person adapts to the trauma, just as the person adapts to the rolling boat. It is proposed that, if the trauma is significant enough, an adaptation of cerebellar modulation of stretch reflexes, which must take place, becomes the new norm as nociceptors continue to fire and change stretch reflex responses while the injury heals.

One can see that IRT is associated at least in part with cerebellar adaptation by applying functional neurological assessment of cerebellar function in parallel with IRT.

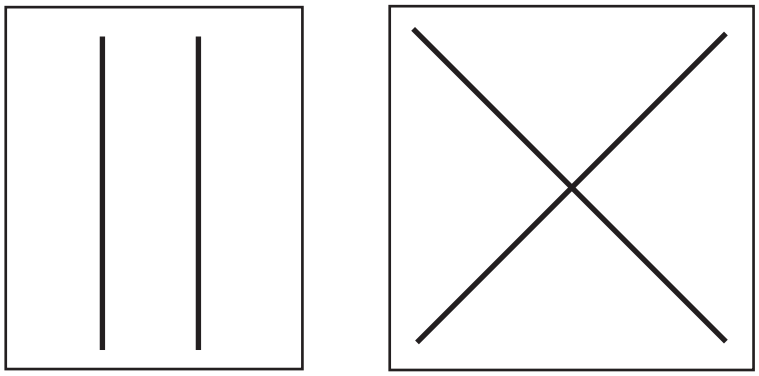
There are always inputs to the cerebellum from the vestibular complex in the inner ear. The three bilateral semicircular canals are arranged such that any head movement will be associated with activation of at least one, and usually two or three of them on each side. The semicircular canals cause reflex changes in postural muscle activity. Putting the head in a distinct position to activate primarily one of the canals will elicit reflex responses which are characteristic of that canal.

The six head positions, patient supine, which will activate specific semicircular canal activity, are:
1) rotating the head to the right (right lateral canal)
2) rotating the head to the left (left lateral canal)
3) tilting the head anterior and left (left anterior canal)
4) tilting the head posterior and right (right posterior canal)
5) tilting the head anterior and right (right anterior canal)
6) tilting the head posterior and left (left posterior canal)

As mentioned before, one of the three major sensory inputs for postural control are the eyes. The positive challenge for IRT is when a conditionally facilitated (strong) muscle becomes conditionally inhibited (weak) when the area of previous injury is activated by patient touch or doctor stimulus (usually pinching) and the talus is challenged cephalward. To demonstrate the cerebellar relationship to a positive IRT pattern, simply activate the area of injury by patient touch or doctor pinching, but rather than add the talus challenge, place the head in one of the six positions to activate one of the semicircular canals as above. This will result in a strong muscle weakening similarly as the cephalward talus challenge.

Correction of Visual IRT (VIRT) is accomplished by having the patient look at an "X" image while the area of injury is activated. Following this correction, activation of the injury with the challenge is now negative. This suggests that the IRT problem was associated with plastic adaptation in the cerebellum which is no longer present following VIRT correction.

My initial investigation of the **CROSSED LINES** and of the **VERTICAL PARALLEL LINES**, and their possible use in applied kinesiology, was previously reported (See: **NEW TOOL FOR DX & TX OF SWITCHING)** in the Proceedings of 2002, ICAK. I am now conducting a new investigation using these drawings in concert with IRT and Chapman points.



Procedure

METHOD No. 1

I studied 150 random patients, 40% of which were males and 60% were females, who seeked consultation for various reasons, and *who showed positive IRT challenge with Dr.* Schmitt's test using talus compression and /or neck extension.

1. As the initial test, I ruled out the possibility of switching and/or ligament stretch reaction.
2. I found a positive challenge for IRT.
3. I corrected the IRT challenge by activating the area of injury (rubbing) while the patient looked at the "**X**" drawing.
4. Following this correction, activation of the injury with Dr. Schmitt's challenge, as described before, was negative.
5. Subsequently, activation of the injury with the positive semicircular canal related head position was also negative.

METHOD No. 2
I studied 180 random patients, 45% of which were males and 55% were females, who seeked consultation for various reasons, and who showed positive T.L. and Challenge to Neurolymphatic (Chapman) points whose related muscle was weak.

1. Initially, I ruled out the possibility of switching and /or ligament stretch reaction.
2. I then found a weak muscle whose Chapman's point showed a positive challenge or T.L.
3. Next, I corrected the weak muscle by having the patient touch the Chapman point, while the patient looked at the "**X**" drawing for 30 to 45 seconds.
4. Following this correction, the challenge and T.L. of the Chapman point was negative and the related muscle was strong.

METHOD No. 3
I studied 60 random patients, 55% of which were males and 45% were females, who seeked consultation for various reasons, and who showed positive T.L. and Challenge to NeuroVascular (Bennett) points whose related muscle was weak.

1. Initially, I ruled out the possibility of switching and/or ligament stretch reaction.
2. I then found a weak muscle whose Bennett's point showed a positive challenge or T.L.
3. Next, I corrected the weak muscle by having the patient touch the Bennett point, while the patient looked at the "**X**" drawing for 30 to 45 seconds.
4. Following this correction, the challenge and T.L. of the Bennett point was negative and the related muscle was strong.

Results

The following results were found:

150 patients with IRT challenge positive, 100% correction was achieved using the visual "**X**" treatment.

180 patients with Chapman point, T.L. and challenge positive, 100% correction using the visual "**X**" treatment was attained.

60 patients with Bennett's point, T.L. and challenge positive, 100% correction using the visual "**X**" treatment was attained.

The 100% results using visual injury recall technique (VIRT) indicates that changes in sensory and motor functions, autonomic concomitants, and improved cognitive function can be obtained. The cerebellar adaptation theory of IRT also opens the door to understanding how to integrate IRT with other procedures.

The cerebellum is the integrator of all motor functions. We can influence the cerebellum in many ways, and a very powerful one, is with visual input.

Conclusion

The use of visual input has great potential within AK, and needs much more investigation. I believe that Visual inputs have the potential to expand the repertoire of valuable diagnosis and treatment tools.

Since this investigation demonstrated that visual inputs may be used to treat different problems like Switching, IRT, Chapman and Bennett reflexes and who knows what else, we need to discover the neurological links existing between them.

References

1. Schmitt, Walter H., Jr. Injury recall technique. Chiropractic journal of NC Vol. VI, 1990. pp. 25-30.
2. Palomar, José., Proceedings ICAK 2002.
3. Bronston, Gordon J. The M.C.R. test. Journal of current podiatric medicine 1987 36:24.
4. Burt, Alvin M.; Textbook of neuroanatomy. Philadelphia: W.B. Saunders, 1993.
5. Kandel, ER, Schwartz, JH: Principles of neural science. New York: Elsevier, 1985.
6. Walther, David S., Applied Kinesiology Synopsis, Pueblo, Colorado, Systems D.C. 2000, 2nd Ed.
7. Walther, David S., Applied Kinesiology Volume I, Pueblo, Colorado, Systems D.C. 1981.
8. Walther, David S., Applied Kinesiology Volume II, Pueblo, Colorado, Systems D.C. 1983.
9. Dennison Paul E., Ph.D., & Dennison Gail, Brain Gym Handbook, Ventura, California, Edu-Kinesthetics Publications, 1989.

Neurotoxicity and Elevated Homocysteine: The Roles Played by Homocysteic Acid, Aspartate and Glutamate and Activated Forms of Folic Acid, Vitamin B-12, and Vitamin B-6

Walter H. Schmitt, D.C., DIBAK, D.A.B.C.N.

Abstract

Homocysteic acid is a neurotoxin which is produced when homocysteine (homoCYS) is elevated due to decreased levels of the activated nutrients necessary for its conversion. Patients with elevated homoCYS and neurological symptoms become sensitive to the ingested excitatory neurotransmitters aspartate (ASP) and glutamate (GLU) which are found in various dietary sources. Correction of nutritional imbalances, avoidance of ASP and GLU, and treatment of those muscle-related neural pathways which are stressed by this altered chemistry using visceral challenge technique (VCT) are necessary to return many patients to normal function.

Introduction

Elevated homocysteine (homoCYS) levels have been implicated as risk factors for heart disease, cancer, and various neurological diseases including Alzheimer's disease (AD), Parkinson's disease (PD) and schizophrenia.[1] [2] The metabolism of homoCYS requires activated, coenzyme forms of vitamins B-6, B-12, and folic acid as well as other nutrients. The relationship of elevated homoCYS to neurological disease is due its conversion to homocysteic acid, which has excitatory neurotransmitter activity and acts as a neurotoxin. The lack of normal homoCYS metabolism in these patients can lead to neurological damage and neurological symptomatology, both named and unnamed.

Aspartate (ASP) and glutamate (GLU) are excitatory neurotransmitters which stimulate the N-methyl-D-aspartate (NMDA) receptor in neuron cell membranes. In already compromised neurons, activation of the NMDA receptor can drive the internal environment of these cells further into metabolic dysfunction and even death by apoptosis. Patients in this category become extremely sensitive to even low levels of dietary ASP and GLU such as are found in the artificial sweetener, aspartame and the flavor enhancer, monosodium glutamate, respectively.

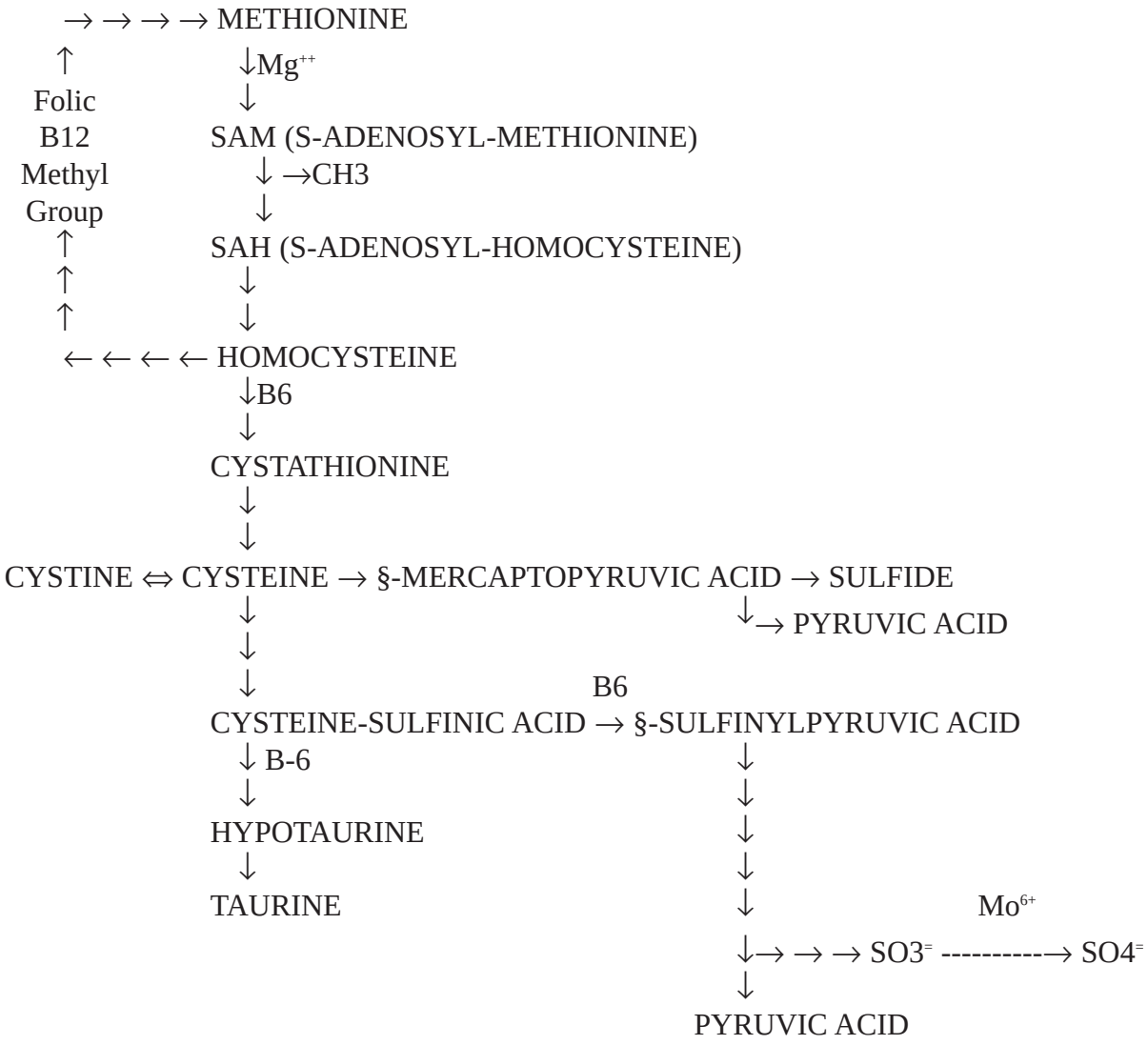
Decreasing the levels of homocysteic acid by decreasing homoCYS levels through nutritional means must be accompanied by avoidance of sources of ASP and GLU. All three substances, homoCYS, ASP, and GLU can be seen to cause a positive oral neuromuscular challenge in susceptible patients. The involved neurological circuits, usually Chapman's reflexes, may be identified by muscle testing and restored to normal activity by visceral challenge technique (VCT).

Discussion

The Role of Activated Nutrients in the Metabolism of Homocysteine

Figure 1 shows the metabolism of sulfur amino acids. Note that homoCYS can be recycled back into methionine (MET) requiring folic acid, vitamin B-12 and a methyl donor. HomoCYS can also be converted on towards cysteine (CYS) requiring vitamin B-6. The nutrients mentioned must be available in their activated,

Figure 1: **SULFUR AMINO ACID METABOLISM**



coenzyme forms. That is, folic acid must be in its 5-methyl tetrahydrofolate (5-MTHF) form, B-12 is in the form of methylcobalamin (methyl B-12), and B-6 must be in its pyridoxal-5-phosphate (P5P) form. Much of this work has been discussed by Chris Astill-Smith.[3] The conversion of B-6 to P5P is the topic of an earlier paper by this author and requires the nutrients zinc, magnesium, riboflavin, and phosphorus.[4] Dietary (and injected) vitamin B-12 is usually in the cyanocobalamin form. Cyanocobalamin is converted to hydroxycobalamin which then may become either methyl B-12 or adenosylcobalamin. Methyl B-12 may be more frequently indicated in patients with carbohydrate metabolism problems. This was first reported to me by Gordon Bronston, DPM.[5] and we have observed this pattern repeatedly since we started looking for it.

Dietary folic acid conversion to the 5-MTHF form involves a little more complex than the activation of B-12 or B-6. There is an enzyme called methylene tetrahydrofolate reductase (MTHFR) which converts methylene-THF to 5-MTHF, the active form for conversion of homocysteine back into methionine. (5-MTHF works

hand-in-hand with methylcobalamin.) Some studies show up to 30-35% of the population have only one gene or no genes for this 5-MTHFR enzyme, and 10-15% of the population is homozygous negative for the MTHFR gene (i.e., they didn't get this gene from either parent.) This genetic defect in the activation of folic acid is called a C-667T polymorphism and has been also shown to relate to neural tube defects in newborns.[6] In patients with this genetic polymorphism, supplementation with a form of 5-MTHF is essential. This is available from a few of our distributors.

Folinic acid is a form of formyl MTHF. So if a person takes folinic acid and has sluggish or lacking MTHFR gene function, then there is still a problem with conversion to 5-MTHF, hence a problem converting homoCYS. In the past, it has occasionally been possible to drive a sluggish chemical reaction forward by giving extremely high doses of the precursor, but since we have 5-MTHF available now, supplementing with this is preferable.

The methylation of homoCYS back into MET also requires the presence of methyl donors. Often these must be supplemented. Choline, betaine, and dimethylglycine are common supplementary sources of methyl groups.

In addition to the above, when homoCYS can not be converted on to CYS (requiring P5P), then CYS is not available for sulfoxidation on into sulfate or for the synthesis of glutathione, one of the primary antioxidant defense systems. Sulfate is an important liver phase 2 detoxification substance and inadequate detoxification can contribute to free radical formation and increased toxicity throughout the body. In Parkinson's disease, one of the early changes is the loss of glutathione from the neurons of the striate system, especially in the substantia nigra. Intravenous glutathione has been used successfully in some cases of Parkinson's disease by some forward thinking neurologists such as David Perlmutter, MD in Florida. There are many implications of decreased glutathione as discussed another earlier paper by this author.[7]

Many patients with neurological disorders, both named and unnamed, present with common muscle testing and applied kinesiology (AK) findings associated with elevated homoCYS and presumably elevated homocysteic acid. A good clue to elevated homoCYS is seen when a patient demonstrates a strengthening response of an inhibited muscle on insalivation of CYS. Although a CYS strengthening response could be due to faulty liver detoxification or other metabolic problem, many of these patients are low in CYS due to lack of normal metabolism from homoCYS. In this case, the patient will also demonstrate an inhibitory effect of oral homoCYS.

This inhibitory effect may be global in nature, or it may apply to only a few selected muscles and their respective Chapman's reflexes. If it is global in nature, therapy localization (TL) to one or more Chapman's reflexes will negate the general inhibition. If it is selective in nature, oral homoCYS may cause no general effects, but a strong indicator muscle may become inhibited with oral homoCYS combined with TL to one or more Chapman's reflexes.

This procedure of oral challenge and TLing Chapman's reflexes is called Visceral Challenge Technique (VCT) and is reviewed in a previous paper.[8] To normalize the neuromuscular effects of the stressor, injury recall technique (IRT) is performed with the stressor in the mouth while the patient TLs to the positive Chapman's reflex(es).

In patients with neurological patterns, homoCYS will usually be found to show a positive VCT pattern, either by global inhibition or selective inhibition on TL to one or more Chapman's reflexes. Most commonly, the supraspinatus / brain Chapman's reflex will be involved, usually unilaterally. The pectoralis major, sternal division (PMS) / liver Chapman's reflex is also commonly involved. Any other Chapman's reflex may TL as positive. Any positive Chapman's reflex should be treated with IRT while the patient holds the homoCYS in the mouth.

In my clinical experience, when people have neurological symptoms related to elevated homoCYS / homocysteic acid, they are also very sensitive to ASP and often to GLU. ASP and GLU are excitatory neurotransmitters which stimulate the NMDA receptors to open calcium channels. When the NMDA channels open, there is an uncontrolled influx of calcium into the neuronal cytoplasm. Excess intracellular cytoplasm can invoke programmed cell death by apoptosis. In addition, the influx of calcium can cause release of nitric oxide and other free radicals, and it further increased the release of GLU. The GLU further excites the cell membranes in the region allowing excess calcium influx in them, and the process can lead to a vicious cycle resulting in cell death of adjacent cells.

The oral consumption of ASP and GLU can be disastrous for these patients. Common sources are the artificial sweetener aspartame which is a combination of ASP and phenylalanine and the flavor enhancer, monosodium glutamate (MSG) which, of course, supplies GLU. In addition a number of nutritional companies add ASP (usually as magnesium aspartate) as a filler in supplements. Although this is a hypoallergenic filler, in patients with these types of neurological stress, the ASP can make the neurons sicker. It appears that this is the reason why some companies' products do not test well on some patients while they show excellent responses on others. My observations indicate that the small amounts of ASP found naturally occurring in food do not appear to create a problem.

Conclusion

Any patient with any named or unnamed neurological disorder should be screened with AK procedures to determine if there is an excess of homoCYS. A good starting place is to test for a strengthening reaction to CYS. But any suspicion of neurological involvement or other risk factors related to increased homoCYS (heart disease, cancer) should provoke the thought of challenging the patient with homoCYS. Since homocysteic acid is an excitatory neurotoxin related to elevated homoCYS, patients should be tested for the both the dietary forms as well as the activated forms of the nutrients which are necessary for homoCYS conversion. Many patients (as many as one-third considering the large percentage of people who have genetic polymorphisms of MTHFR) may only show a response to the activated form of the nutrient such as 5-MTHF, methylcobalamin, or P5P.

Patients with elevated homocysteine and neurological symptoms usually also become sensitive to the dietary sources of excitatory ASP and GLU. Avoidance of aspartame, MSG, and supplemental sources of ASP are an essential part of the regime. This is probably why some patients respond poorly, both clinically and with muscle testing procedures, to supplements containing ASP.

It is likely that much of the controversy regarding the dangers of aspartame and MSG is due to the fact that only certain patients are susceptible to the damage from these excitatory neurotransmitters. It would be interesting to see if patients who had trouble with aspartame or MSG are patients who also show elevated homoCYS.

Treatment of those muscle-related pathways and Chapman's reflexes which are stressed by homoCYS, ASP, and/or GLU using VCT is the final piece of the approach to these patients. In neurological cases, one supraspinatus / brain Chapman's reflex is usually involved. The PMS / liver Chapman's reflex is involved in most cases of elevated homoCYS whether or not there is neurological involvement. Any other Chapman's reflex which is associated with the patient's symptoms should also be evaluated with an oral challenge of homoCYS, ASP, and/or GLU. Any positive Chapman's reflexes should be corrected with IRT treatment.

This three-pronged approach of appropriate nutritional supplementation, offender elimination, and VCT with the offenders is necessary to return most of these otherwise difficult patients to normal function.

Procedure

1. Inhibited muscle may strengthen with oral CYS.
2. HomoCYS causes
   a. global inhibition which is negated by TL to specific Chapman's reflexes
   b. positive TL to specific Chapman's reflexes using a strong indicator muscle
3. Aspartic acid causes
   a. global inhibition which is negated by TL to specific Chapman's reflexes
   b. positive TL to specific Chapman's reflexes using a strong indicator muscle
4. Glutamic acid causes
   a. global inhibition which is negated by TL to specific Chapman's reflexes
   b. positive TL to specific Chapman's reflexes using a strong indicator muscle
5. Test for homoCYS metabolizing nutrients
   a. Folic acid / 5-methyltetrahydrofolate
   b. Vitamin B-12 / methylcobalamin
   c. Vitamin B-6 / pyridoxal-5-phosphate
      i. Zinc, magnesium, riboflavin, phosphorus
   d. Methyl donor (e.g., choline, betaine, dimethylglycine)
   e. Magnesium
6. Perform IRT to positive Chapman's reflex(es) with oral homoCYS, ASP, and/or GLU
   a. Supraspinatus / brain
   b. PMS / liver
   c. Any other area of concern
7. Supplement appropriate nutrients
8. Patient avoids offending substances

References

1. Regland, B., Germgärd, T., Koch-Schmidt, AC., and Gottfries, CG., 1997, Homozygous thermolabile Methylenetetra- hydrofolate Reductase in Schizophrenia like psychosis, in J. Neural Transm., 104, 931-41.
2. Regland, R., Blennow, K., Germgård, T., Koch-Schmidt, AC., and Gottfries, CG., 1999, The Role of the Polymorphic Genes Apolipoprotein E and Methylenetetra- hydrofolate Reductase in the Development of the Alzheimer Type, in Dementia, 10, 245-51.
3. Astill-Smith, Chris. New York Seminar Notes, March 24-25, 2001.
4. Schmitt, Walter H., Jr. Making B-6 work. ICAK collected papers, Winter, 1987.
5. Bronston, Gordon, Personal communication.
6. Bland, Jeffrey, Functional Medicine Update, Vol 22, No. 3, March, 2002.
7. Schmitt, Walter H., Jr. Glutathione-the most important molecule in the cell. ICAK collected papers, 1988-89, volume II.
8. Schmitt, Walter H., Jr. The somatic window on neurological function, part 1. Proceedings of the ICAK - U.S.A. Volume 1, 1999-2000. p. 141-148.

The Somatic Window on Neurological Function – Part 2. Inducing Patterns of Over Facilitation to Evaluate Cortical Hemispheric Dominance Patterns

Walter H. Schmitt, D.C., DIBAK, D.A.B.C.N.

Abstract

Right and left cerebral hemispheric activity may be monitored by combining neurological principles with manual muscle testing procedures. Specifically, activating a right or left cerebral cortex which is increased in its activity results in the presence of increased ipsilateral muscle tone and the presence of an open ileocecal valve (right) or open Houston valve (left.) This cortical hemispheric imbalance may be normalized and may require different procedures depending on the source of the over activity which include over facilitation and transneural degeneration. Procedures for assessment and treatment of cerebral cortical hemispheric imbalances are outlined.

Introduction

In a previous paper by this author entitled The Somatic Window on Neurological Function – Part 1,[1] the use of manual muscle testing for evaluation of the function of several central nervous system neuron pools was discussed. These included primarily effects mediated through the cerebellum following sensory receptor challenges using slow stretch of muscles to activate muscle spindle cells (MSCs).

The premise of that paper was that when a weak (inhibited) muscle strengthens (becomes facilitated) following a sensory receptor challenge, it suggests that a pathway between the sensory receptor and the muscle is inhibited somewhere along its course, and that the challenge creates enough excitatory activity in the pathway to temporarily overcome that inhibition.

In this paper, we add to this method of evaluation and introduce a new concept in using muscle testing as functional neurological evaluation. That is, it is possible to produce challenges which induce over facilitation of a neuromuscular pathway and use the observation of the over facilitation to identify sources of neurological imbalance. When activation of a neurological pathway causes an over facilitation of a muscle, or group of muscles, this may represent a problem in neurological function. The problem may arise from several sources including: 1) an over facilitation of a pathway arising from either an excessive facilitation or a deficient inhibition to the cells of origin, and 2) from a metabolic imbalance of the neuron pool itself associated with transneural degeneration (TND.) These issues will be further addressed later in this paper.

There are predictable neuromuscular and autonomic responses associated with activation of pathways which arise in the cerebral cortex and descend to the brainstem and spinal cord. There are also predictable cortical responses which may be evoked by activating specific sensory receptors, hence specific afferent pathways which eventually reach the cortex.

First we will discuss the normal activity of cortical descending pathways, and then we will discuss findings associated with problems in neuron pools associated with the cortical origins of these pathways. Then we will discuss methods of evoking cortical responses and evaluating these responses. Finally, we will suggest therapeutic and rehabilitative approaches which may be used to restore more optimal cortical function.

Discussion

Descending Cortical Pathways

Contrary to the popularly held notion, the vast majority of cortical neurons gives rise to pathways which descend ipsilaterally. Only about 10% of fiber tracts which originate in the cerebral cortex decussate to end up on the contralateral side. These crossed pathways are associated with fine motor movement and meaningful distal extremity activity and motor activity to the proximal musculature which supports these more distal activities. The most significant of these pathways are listed below.

CROSSED CORTICAL DESCENDING PATHWAYS

- CORTICOSPINAL TRACT & SUPPORTIVE PATHWAYS
  - CORTICORUBROSPINAL TRACT
  - CORTICORETICULOSPINAL TRACTS
- MAKE UP LESS THAN 10 % OF CORTICAL DESCENDING FIBERS

The effects of the ipsilateral cortical descending pathways which make up 90% of the corticofugal pathways are both somatic and autonomic. Activation of one hemisphere results in increased ipsilateral muscle tone in two ways: 1) a general increase in all ipsilateral muscle tone and 2) an increase in posterior (extensor) muscle tone above T-6 and an increase in anterior (flexor) muscle tone below T-6. The increase in tone is via changes in gamma motorneuron activity which increases the firing rates of muscle spindles. This in turn tends to cause a shortening of muscles at rest increasing the muscles' responses to stretch which is increased tone, or hypertonicity. This is true for any activity in the hemisphere, regardless of where or how or why it arises. In other words, cortical hemispheric activity may arise from peripheral sensory activity or other afferent activity, or from thoughts, emotions, or other cognitive activity which may arise within the hemisphere itself.

Cortical hemispheric activity has autonomic effects via corticoreticular pathways which impact the neuron pools in the mesencephalic reticular formation and the pontomedullary reticular formation (PMRF.) The PMRF areas are primarily parasympathetic (PS) in nature such as the dorsal motor nucleus of the vagus nerve, the nucleus of the tractus solitarius, and the superior and inferior salivatory nuclei.

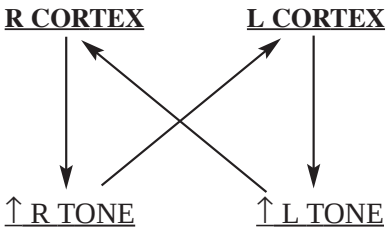
The PMRF projects caudally and ipsilaterally to the intermediolateral cell column (IML) of the entire spinal cord with an inhibitory effect. The IML from T-1 to L-2 is the source of primary sympathetic (SYM) neurons for the entire body. These SYM neurons are inhibited by the PMRF. Hence, activation of a hemisphere results in an increase in PS activity originating in the PMRF and an inhibition of SYM activity throughout the body, or an overall PS effect on the same side of the body. The ipsilateral somatic and autonomic effects of cortical activation are summarized below.

CORTICAL DESCENDING EFFECTS - IPSILATERAL

- ↑ ALL MUSCLE TONE
- ↑ EXTENSORS TONE ABOVE T-6
- ↑ FLEXORS TONE BELOW T-6
- ↑ PONTOMEDULLARY RETICULAR FORMATION (PS)→
- INHIBITION OF IML (SYM) OUTFLOW

There is wisdom in this arrangement of 90% of cortical descending fibers being ipsilateral. The increase in gamma motorneuron activity results in increase firing of muscle spindles (which increases ipsilateral muscle tone.) Increased muscle spindle activity increases ipsilateral cerebellar activity via the spinocerebellar tract afferents. The ipsilateral cerebellar activity projects to the opposite cortex, increasing contralateral cortical hemisphere firing. This contralateral cortical firing will increase muscle spindle cell activity and muscle tone on its side. This will then cause increased afferent spinocerebellar activity on that side which will project to the original cortical hemisphere increasing its function. So there is a "figure-of-8" type pattern of this neurological activity.

"FIGURE OF 8" PATTERN



Any activation anywhere along this pathway will tend to activate the entire figure-of-8 pathways, which will "wind up" the neurological system. Since neurons require receptor activation to maintain metabolic health, this built-in wind-up pattern keeps the nervous system healthy as long as normal functions of all afferent and efferent pathways are in tact. An example of wind-up is shown below.

"FIGURE OF 8" WIND-UP PATTERNS

↑ RIGHT CORTEX ACTIVITY
↓
↑ RIGHT MUSCLE TONE
↓
↑ RIGHT CEREBELLUM
↓
↑ LEFT CORTEX
↓
↑ LEFT MUSCLE TONE
↓
↑ LEFT CEREBELLUM
↓
↑ RIGHT CORTEX

Autonomic Effects of Cortical Hemispheric Activity
Autonomic effects of cortical function are mediated through the PMRF (as well as through the mesencephalic reticular formation.). The PMRF activity includes all types of autonomic functions which originate or are modulated ipsilaterally. These are summarized below.

PONTOMEDULLARY RETICULAR FORMATION

- LOCATION OF VITAL CENTERS
  - NUCLEUS OF TRACTUS SOLITARIUS, VAGUS (PS)
  - CARDIOVASCULAR, RESPIRATORY, DIGESTION
- STIMULATED BY IPSILATERAL CORTEX
- STIMULATED BY IPSILATERAL CEREBELLUM
- INHIBITS IPSILATERAL IML →
- (INHIBITS SYM)

There is a sidedness to autonomic function which has important clinical implications. This includes effects on the heart and on the digestion. The right SYM outflow affects the sinoatrial node (SA node) and left-sided SYM activity affects the atrioventricular node (AV node.) Therefore, a differential in right and left cortical outflow can result in alterations in heart activity related to the asymmetrical stimulation of the right and the left sides of the heart.

Cortical hemispheric function (like the function of any motor neuron pool) may be increased or decreased, relatively speaking. We will talk of increased or decreased cortical function, although these terms are a bit misleading.

Increasing right hemispheric activity will result in a net decrease of right SYM activity tending to decreased firing of the SA node which will slow the heart rate. Therefore, decreased right sided cortical function will impact the heart with an increase in SYM activity to the right side of the heart and will cause a tendency towards tachycardia.

DECREASED RIGHT CORTEX – CARDIAC EFFECTS

- ↓ RIGHT PONTOMEDULLARY →
- INCREASED RIGHT IML →
- ↑ RIGHT SYMPATHETIC
- (TACHYCARDIA)

Increased left-sided cortical activity will result in a decreased left-sided SYM activity. Decreased left hemispheric function results in an increased left SYM activity with increased AV node firing and a tendency towards arrhythmias.

DECREASED LEFT CORTEX - CARDIAC EFFECTS

- ↓ LEFT PONTOMEDULLARY →
- INCREASED LEFT IML →
- ↑ LEFT SYMPATHETIC
- (ARRYTHMIAS)

The digestive effects of cortical hemispheric activity also reflect right and left hemispheric patterns. While we just discussed the effects of decreased cortical activity on cardiac function, we will discuss the effects of increased cortical hemispheric activity on the digestion. Right cortical activity will be reflected in the functions of the ileocecal valve (ICV) on the right and left cortical activity will be reflected in the function of the Houston valve (HV) or rectosigmoid folds on the left. The HV is the neurological mirror-image of the ICV. Increased right cortical function will create an increased PS – decreased SYM effect on the right which includes a tendency towards an open ICV syndrome.

INCREASED RIGHT CORTEX – DIGESTIVE EFFECTS

- ↑ RIGHT PMRF / ↓ RIGHT IML →
- OPEN ILEOCECAL VALVE (↑ PS)

Similarly, increased left cortical function will create an increased PS – decreased SYM on the left including a tendency towards an open HV.

INCREASED LEFT CORTEX – DIGESTIVE EFFECTS

- ↑ LEFT PMRF / ↓ LEFT IML →
- "OPEN" HOUSTON VALVE (↑ PS)

Assessment of Cortical Functional Status

Cortical function may be monitored by a variety of neurologic assessment tools. A thorough discussion of these tools is the stuff of neurology textbooks and courses (such as those taught by the Carrick Institute) and is beyond the scope of this paper. One useful tool that has been documented is blind spot mapping.[2] Another useful tool is the Weber's test. Although this test is considered a hearing pathway test, it will frequently been seen to favor one side when no hearing problem can be identified. If a problem with the auditory pathway can be ruled out, Weber's test may be used as an indicator for cortical activity suggesting an increase in cortical firing on the side of increased perception or a decrease in cortical firing on the side of decreased perception. Blind spot mapping and Weber's test are readily performed in any office requiring no special equipment and little time.

The relative functions of the right and left cortical hemispheres may also be monitored by observing peripheral somatic and autonomic indicators. For example, testing for muscle tone by passive range of motions and comparing right to left is a simple somatic indicator. Likewise, comparing right to left pupillary light responses is a good indicator for assessing autonomic activity. There are numerous other somatic and autonomic indicators which may be monitored.

Manual muscle testing and applied kinesiology procedures may be used with great effectiveness and correlated with the traditional somatic and autonomic indicators listed above. The "somatic windows" on neurological function created by muscle testing and AK techniques supplies an enhanced ability to observe the manifestations of these fundamental neurological dysfunctions. Autonomic function may also be assessed by AK challenging procedures for ICV and HV.

Combining testing for these somatic windows with the more traditional assessment procedures can give the clinician a better picture of the neurological status of the patient. Adding various neurological challenge procedures greatly amplifies the ability to understand the patient's neurological status and focus on the area of primary involvement, thereby enhancing the response to therapies. However, we must expand traditional AK muscle testing approaches to get a full view of cortical neurological status.

AK muscle testing has always been used to test for inhibited muscle activity, often following a variety of neurological challenge procedures using sensory receptor activation. That is, AK procedures typically test an inhibited muscle and observe for changes toward normal facilitation following sensory receptor challenges, or test a normally facilitated muscle and observing for inhibition following challenges.

It is equally important and valuable to observe for changes of increased facilitation following sensory receptor and other neurological challenges. This opens the door to evaluation of many neurological activities not available by testing focused only on inhibited muscles. Using MSC activity or Golgi tendon organ (GTO) activity to inhibit has been called autogenic inhibition (AI) by Belli.[3] Any normally functioning muscle should demonstrate a "strong" response to the demands of a manual muscle test, and should become "weak" to this same testing procedure immediately following an AI challenge procedure. An over facilitated muscle will test as "strong" and will continue the "strong" response to the manual muscle test immediately following an AI challenge. The addition of observing for over facilitated muscles (as well as for inhibited muscles) is the key to thoroughly assessing the nervous system through the somatic window.

There are dozens of ways to activate each cortical hemisphere, some of which are very specific for different parts of the brain. Only a very few of these other methods will be discussed below. For example, you may induce right cortical activity by a number of means such as having the patient hum a tune. The response of muscles to testing before and during such right cortical activity should not be expected to change. However, when there is increased right cortical activity, right-sided muscles will be seen to assume a pattern of over facilitation during the cortical activity. That is, in an increased right cortex state, during humming, all right-sided muscles will show strong responses to testing which are not inhibited by AI; they become over facilitated during the right brain activity. Similarly, in an increased right cortex, there will be increased right-sided PS activity which will be seen as an open ICV during standard ICV challenging procedures.

The ICV and HV may be monitored by traditional AK procedures.[4] We may challenge right and left cortical function and observe for patterns of inhibition, facilitation, over facilitation, and ICV / HV to determine the status of cortical function. On the right, if we invoke a challenge to stimulate right cortical activity, we can monitor right-sided muscle testing patterns and an open ICV. On the left, we can invoke a challenge of left cortical activity and monitor left-sided muscle activity and the HV.

The somatic - muscle testing factors for right and left cortical function are summarized below.

IF INCREASED RIGHT CORTEX – RIGHT CORTICAL CHALLENGE WILL CAUSE:

- ↑ RIGHT MUSCLE TONE / AI DOES NOT INHIBIT ANY RIGHT-SIDED MUSCLES
- OPEN ILEOCECAL VALVE (↑ PS ON RIGHT)

IF INCREASED LEFT CORTEX – LEFT CORTICAL CHALLENGE WILL CAUSE:

- ↑ LEFT MUSCLE TONE / AI DOES NOT INHIBIT ANY LEFT-SIDED MUSCLES
- "OPEN" HOUSTON VALVE (↑ PS ON LEFT)

It is important to recognize that all sensory inputs pass through the thalamus and affect the contralateral cerebral cortex except olfaction. Olfactory receptors send their afferents directly into the ipsilateral midline (phylogenetically older) cerebral cortex structures and bypass the thalamus. This allows for separate challenge procedures for different parts of the cerebral cortex. In the presence of an increased cortical hemispheric activity (whether from over facilitation, under inhibition, or TND) usually all of the cortical challenges listed below will result in ipsilateral muscle over facilitation and the ICV open or HV open challenges, respectively. Occasionally the challenge procedure will be positive for only one area of cortical function or another. However, no further effort will be made in this paper to differentiate between the effects of challenge procedures for higher, newer cortical functions (neocortex) and lower, older cortical functions (paleocortex.)

It is imperative to fix injuries prior to cerebral cortex challenging. Nociception will drive the contralateral cortex and create the illusion of a cortical hemispheric problem. Therefore, all injuries requiring injury recall technique (IRT), nociceptor stimulation-blocking technique (NSB), or set point technique (SP) must be corrected prior to attempting assessment of cortical hemispheric activity. The presence of any uncorrected injuries will completely negate the reliability of the procedures in this paper. All active injuries and nociceptor firing must be treated appropriately with IRT, NSB, of SP techniques prior to proceeding.

Following correction of injuries and active nociception, you may proceed with the right and left challenge procedures outlined below. Immediately following (or during) the challenge, evaluate for changes in ipsilateral muscle strength, especially AI (MSC to weaken) to ascertain if there is normal or increased muscle tone in response to the challenge. Also, challenge the ICV (for right cortical challenges) or the HV (for left cortical challenges.)

Ask the patient to activate the right cortex by initiating activity there. This can be achieved by having the patient hum, or perform left-sided distal meaningful motor activity such as "playing the piano" movements with the left fingers and/or toes, or alternately touching the four left fingers to the left thumb. You may also activate the right cortex by appropriate sensory receptor stimulation. This includes sniffing through the right nostril, left hemifield visual inputs, or other left-sided sensory receptor stimulation.

Then ask the patient to activate the left cortex by initiating activity there. This can be achieved by having the patient perform math (orally count or multiply,) perform right-sided distal meaningful motor activity such as "playing the piano" movements with the right fingers and/or toes, or alternately touching the four right fingers to the right thumb. You may also activate the left cortex by appropriate sensory receptor stimulation. This includes sniffing through the left nostril, right hemifield visual inputs, or other right-sided sensory receptor stimulation.

Right and left cortical challenge procedures are summarized below.

RIGHT CORTEX CHALLENGES

- RIGHT BRAIN ACTIVITY (MUSIC / HUMMING)
- RIGHT OLFACTION
- MEANINGFUL LEFT DISTAL EXTREMITY
  - LEFT FINGER AND TOE PIANO PLAYING
  - LEFT FINGERS TO THUMB
- LEFT VISUAL FIELD STIMULATION
  - LEFT TEMPORAL LIGHT
  - LEFT HEMIFIELD STIMULATION (EYELIGHTS)

LEFT CORTEX CHALLENGES

- LEFT BRAIN ACTIVITY (MATH)
- LEFT OLFACTION
  - MEANINGFUL RIGHT DISTAL EXTREMITY
  - RIGHT FINGER AND TOE PIANO PLAYING
- RIGHT FINGERS TO THUMB
  - RIGHT VISUAL FIELD STIMULATION
  - RIGHT TEMPORAL LIGHT
- RIGHT HEMIFIELD STIMULATION (EYELIGHTS)

An increase in one cerebral hemispheric activity compared with the other is known as hemisphericity. The side of hemisphericity will demonstrate increased function on evaluation compared to the opposite side.

When there is increased right hemispheric activity (a right hemisphericity,) right cortical challenges will be seen to induce an increase in muscle tone on the entire right side (as seen by failure of AI to inhibit any muscle) and a positive challenge for an open ICV. In the presence of increased left hemispheric activity (a left hemisphericity,) left cortical challenges will be seen to induce an increase in muscle tone on the entire left side (as seen by failure of AI to inhibit any muscle) and a positive challenge for an open HV. There may be exceptions to these rules, and bilateral effects of facilitation or inhibition may be seen. But these are the general patterns.

Interpretation of these findings of increased hemispheric function should be correlated with other somatic and autonomic signs as mentioned previously. There are many sources of neurological imbalances, and although this paper specifically focuses on those of the right and left cerebral hemispheres, the presence of these cortical imbalances may be secondary to other primary problems in the central and/or peripheral nervous systems. In clinical practice, the evaluation of cortical hemispheric balance should be done in the context of evaluation of the entire nervous system, but for the purposes of this paper, we will discuss these diagnostic and treatment entities as if they are the sites of primary involvement.

Therapeutic Options

There are a number of therapeutic possibilities when assessment reveals increased cortical hemispheric activity. One possibility is that there is transneural degeneration present in that hemisphere, and that activation of the hemisphere results in a barrage of increased activity. In TND, this increase will be followed by a fatigue of function on repetitive activity. The rate of onset of fatigue may be rapid or slow, which makes determination of the state of cortical TND rather difficult in many patients.

Restoration of TND neurons to normal metabolism depends on supplying the neurons with the three things that they need for normal metabolism: 1) oxygen, 2) fuel (glucose and substances necessary for its oxidative phosphorylation,) and 3) stimulation. Those chemical factors associated with oxygen and fuel (both excesses and deficiencies) which may interfere with normal neuron metabolism, have been discussed in an earlier paper by this author.[5]

Stimulation is essential for normal activation of second messenger-dependent intracellular metabolic activity as well as for switching on nuclear genetic expression for synthesis of proteins. These proteins serve at least two purposes in the maintenance of normal intracellular metabolic activity: 1) they are the enzyme substrates which are necessary for metabolic activity, and 2) their anionic nature contributes a significant negativity to the intracellular environment helping to maintain the normal negative resting potential of the neuron. (Many of these proteins contribute to structural components of the cell as well.)

In the presence of adequate oxygen and fuel, the restoration of normal metabolic activity in a TND neuron depends on stimulation of cell membrane receptors, both excitatory and inhibitory. Excitatory stimulation may drive the TND neuron to over stimulation and even to the point of metabolic exhaustion and programmed cell death by apoptosis. THIS POTENTIAL FOR OVER STIMULATION IS A SERIOUS DANGER TO THE PATIENT'S WELL BEING! For this reason, one must proceed slowly during stimulation of TND neurons with excitatory inputs and closely monitor autonomic activity during stimulation to assure that the patient is not being injured by the activity.

Starting with minimal stimulation, and then slowly increasing the intensity, frequency, and duration to the patient's tolerance will allow the neuron's metabolism to improve with each subsequent stimulation, and slowly build up its tolerance to stimulation as the therapeutic inputs increase. Prior to initiating excitatory stimulation to TND neurons, ascertaining that the general body chemistry is adequate for supplying glucose and cofactors is recommended. Supplying oxygen during such stimulation is also a valuable adjunct in the normalization of TND neuron pools.

Observation of the patient's pulse during excitatory stimulation to a pool of TND neurons also provides a safety net. Over stimulation which threatens to fatigue the metabolic state of TND neurons will result in the body's attempt to increase oxygen and fuel to the area, hence an increase in cardiac output as evidenced by increased heart rate. Conversely, treatment which reduces stress in the nervous system will result in a decrease in pulse rate.

Another possibility for increased hemispheric activity is a very strong, highly integrated pool of neurons due to overuse such as the left brain of an accountant, or the right brain of a recreational drug user. Another scenario is increased function due to prolonged stimulation from afferent activity such as contralateral pain or asymmetrical gait. In these latter cases, one would not want to further increase the activity of this neuron pool. This is one reason why it is so important to clear any nociceptive activity prior to testing for cortical balance.

When finding differences in cerebral hemispheric balance, the question becomes: "Is one side over active, or the other side under active, or both?" Since activity in one hemisphere can be inhibitory to the other hemisphere, stimulation of a hemisphere with decreased activity will tend to bring both sides into balance. There are other scenarios which can complicate the assessment process and mislead the clinician.

When it is clear that one hemisphere is increased in function, we must then question whether this is due to TND or to over facilitation. If it is TND, increasing activity of that hemisphere, starting slowly, etc. will immediately improve the assessment parameters. If it is over facilitation, therapies directed to this hemisphere will aggravate the assessment parameters.

Fortunately, first clearing nociception and then following the procedures outlined above and below, and then reassessing the patient during and at the end of the treatment procedure regularly yields gratifying responses.

In the case of right cortical increased function from TND, the findings and treatment procedures would be as follows:

RIGHT CORTICAL BALANCING TECHNIQUES - ASSESSMENT

- R BRAIN ACTIVITY CAUSES:
- OVERFACILITATION OF ALL RIGHT-SIDED MUSCLES
- ICV OPEN

RIGHT CORTEX TND - TREATMENT

SLOWLY INCREASE STIMULATION WHILE MONITORING PULSE, BILATERAL ROM

- RIGHT BRAIN ACTIVITY (MUSIC / HUMMING)
- MEANINGFUL LEFT DISTAL EXTREMITY
  - LEFT FINGER AND TOE PIANO PLAYING
  - LEFT FINGERS TO THUMB
- LEFT VISUAL FIELD STIM
  - LEFT TEMPORAL LIGHT
  - EYELIGHTS – LEFT HEMIFIELD

In the case of left cortical increased function from TND, one would find and follow the following patterns:

LEFT CORTICAL BALANCING TECHNIQUES – ASSESSMENT

- L BRAIN ACTIVITY CAUSES:
- OVERFACILITATION OF ALL LEFT-SIDED MUSCLES
- HV OPEN

LEFT CORTEX TND - TREATMENT

SLOWLY INCREASE STIMULATION WHILE MONITORING PULSE, BILATERAL ROM

- LEFT BRAIN ACTIVITY (MATH)
- MEANINGFUL RIGHT DISTAL EXTREMITY
  - RIGHT FINGER AND TOE PIANO PLAYING
  - RIGHT FINGERS TO THUMB
- RIGHT VISUAL FIELD STIM
  - RIGHT TEMPORAL LIGHT
  - EYELIGHTS – RIGHT HEMIFIELD

In the case of an overactive cortical hemisphere which is not from TND, but rather from the hemisphere being over facilitated or from decreased function of the opposite hemisphere, treatment should be directed away from the over facilitated side and toward the contralateral, decreased cortical hemisphere. Stimulation of the under functioning hemisphere will help to bring both hemispheres into balance. Monitoring of somatic and autonomic indicators as discussed previously is important for before and after assessment to ascertain that the appropriate therapies have been administered.

Following appropriate treatment, the original assessment parameters will be absent. That is, after proper therapy to the right cortex is achieved, activating the right cortex will result in no change in right-sided muscle tone (AI will cause normal inhibition,) and a challenge to the ICV will be negative. Likewise, left cortical activation will not change left-sided muscle tone and challenging the HV will be negative.

Similarly, other somatic (e.g., ranges of motion) and autonomic findings (pupil responses, Weber's test, etc.) should have returned to or towards normal. Usually the patient will also report a sense of well-being or possibly feeling more alert.

Conclusion

These procedures are valuable in many different types of patients and are best used in the context of thorough neurological evaluation of the patient. It is best if the patient has first been adequately evaluated and treated to normalize the nutritional biochemistry. This will provide the necessary delivery of oxygen and fuel for the neurological therapies to achieve their optimal improvement in neuron metabolic function. It is also essential that all injury activity, both recent and ancient, be cleared with the appropriate techniques. The integration of applied kinesiology principles with principles of chiropractic neurology allows for these objectives to be achieved in the most efficient manner. The patient benefits, and the doctor has the opportunity to observe neurological function come to life before his or her eyes.

References

1. Schmitt, Walter H., Jr. The somatic window on neurological function, part 1. Proceedings of the ICAK - U.S.A. Volume 1, 1999-2000. p. 141-148.
2. Carrick, FR Changes in brain function after manipulation of the cervical spine. J Manipulative Physiol Ther 1997; 20:529-545.
3. Belli, Richard. Autogenic Inhibition. Proceedings of the ICAK-USA, volume I, 1995-1996. pp. 15-18.
4. Walther, David S. Applied Kinesiology synopsis. 2nd edition. Pueblo, Colorado: Systems D.C., 2000.
5. Schmitt, Walter H., Jr. Transneural degeneration and the links between the nervous system and the body chemistry. Proceedings of the ICAK - U.S.A. Volume 1, 1999 2000. p. 149-156

The Somatic Window on Neurological Function – Part 3. Encephalic Transneural Degeneration: The Cause of Many TMJ Problems and Bilateral Joint Problems

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Abstract

Concepts of transneural degeneration (TND) are reviewed. Patterns of mesencephalon activity in the presence of TND are discussed, particularly those of TMJ problems and bilateral extremity problems. Assessment procedures to identify the presence of a TND mesencephalon and treatment – rehabilitation procedures for normalizing mesencephalon function are presented.

Introduction

Transneural degeneration (TND) of neuron pools is an established metabolic phenomenon[1] which affects many motor functions which are commonly monitored by applied kinesiology (AK) manual muscle testing procedures. The TND concept as it relates to muscle testing responses is that there can be entire neuronal pools (that were related in their embryological development and hence, permanently interconnected) that become metabolically stressed or metabolically challenged.

Neuronal pools in this context are gross areas of neural tissue such as the left cortex, or the right cerebellum, or the left medulla, or the right mesencephalon, or the right intermediolateral column of cells which reaches from the mesencephalon to the end of the spinal cord. That is, big chunks of brain or brainstem or spinal cord are involved and all pathways associated with them are affected. Because each cell in a neuron pool has connections with each other cell in the pool (from original embryological connections), they are metabolically linked throughout life. Alterations in the firing rates, hence the metabolism, of one group of cells impacts the metabolic rates, and the firing rates of all other cells in that pool.

The mesencephalon can be considered one of these neuron pools, and more specifically, the right mesencephalon and the left mesencephalon can each be thought of individually as separate neuron pools. This upper brainstem area contains many nuclei and pathways which are listed below in no particular order of importance. This paper will focus on clinical effects which arise primarily from the mesencephalic nucleus of the trigeminal nerve (mesV) and the parabrachial nucleus (PBN).

Discussion

Neuronal degeneration occurs in three ways: anterograde degeneration (anterograde chromatolysis, Wallerian degeneration,) retrograde degeneration (retrograde chromatolysis,) and transneural degeneration. The first two types are associated with axonal interruption. TND is associated with the metabolic effects on neurons when they no longer receive adequate stimulation of their cell membrane receptors to keep the neurons metabolically healthy such as occurs in the presence of deafferentation. Deafferentation can be pathological (arising from nerve damage such as a cut nerve or from an area of neuropathy which no longer sends messages along its axons) or functional (usually due to a lack of normal joint range of motion and decreased firing rates of sensory nerves on those cells on which they synapse.)

Stimulation of membrane receptors is essential for normal activation of second messenger-dependent intracellular metabolic activity as well as for switching on nuclear genetic expression for synthesis of proteins. These proteins serve at least two purposes in the maintenance of normal intracellular metabolic activity: 1) they are the enzyme substrates which are necessary for metabolic activity, and 2) their anionic nature contributes a significant negativity to the intracellular environment which helps to maintain the normal negative resting potential of the neuron.

TND neuron pools are metabolically "sick." The cells may be exhausted and yet they may over fire with bursts of short-lived activity. This is like the proverbial whipping of a tired horse. Or another analogy is what it feels like when you are sick in bed with the flu. You may feel like you are going to die. You are exhausted and can barely move, but at the same time, highly irritable. If someone comes into your room and flips on the light switch, you burst into a barrage of telling them to turn off the lights and leave you alone. Then you again collapse into your misery. TND cells act the same way. They are too close to their firing threshold (resting membrane potential is more positive than normal) so they fire easily. But their metabolic state is also near exhaustion, and they fatigue rapidly once activated.

Restoration of TND neurons to normal metabolism depends on supplying the neurons with the three things that they need for normal metabolism: 1) oxygen, 2) fuel (glucose and substances necessary for its oxidative phosphorylation), and 3) stimulation. Those chemical factors associated with oxygen and fuel (both excesses and deficiencies) which may interfere with normal neuron metabolism, have been discussed in an earlier paper by this author.[2]

In the presence of adequate oxygen and fuel, the restoration of normal metabolic activity in a TND neuron depends on stimulation of cell membrane receptors, both excitatory and inhibitory. Both excitatory and inhibitory activation of membrane receptors influence intracellular metabolism and genetic expression. However, excitation and inhibition have opposite effects on the resting potential of the cell, and in a TND cell which is very close to firing threshold, too much excitation can drive the cell to excessive anaerobic metabolic activity and even to programmed cell death by apoptosis. For this reason, inhibitory inputs to TND cells may be preferable to excitatory inputs, at least at first until the cell has rehabilitated to the point where it can respond to excitation without endangering its metabolic state.

When excitatory inputs to a TND cell are used, one must proceed slowly. Start with minimal stimulation, and then slowly increase the intensity, frequency, and duration to the patient's tolerance. This will allow the neuron's metabolism to improve with each subsequent stimulation, and slowly build up its metabolic tolerance as the therapeutic inputs increase. Prior to initiating excitatory stimulation to TND neurons, it is important to make sure that the general body chemistry is able to adequately supply glucose and cofactors. Supplying oxygen during such stimulation is also a valuable adjunct in the normalization of TND neuron pools.

We usually observe the patient's pulse during excitatory stimulation to a pool of TND neurons. Over stimulation which threatens to fatigue of the TND neurons will result in the body's attempt to increase oxygen and fuel to the area via an increase in cardiac output as can be observed by an increase in heart rate. Conversely, treatment which reduces stress in the nervous system will often result in a decrease in pulse rate.

Mesencephalic TND Patterns

It appears that there are somatic patterns of mesencephalic TND which may be identified by muscle testing procedures. This paper will discuss how to identify the presence of metabolic stress in the mesencephalon as well as treatment procedures designed to restore normal stimulation and metabolic health to this upper brainstem area.

Below are listed some of the important neuron pools in the mesencephalon.

Some Important Nuclei in the Mesencephalon

• Mesencephalic nucleus of the trigeminal nerve (mesV)

- Parabrachial Nucleus (pattern generator nuclei)
- Oculomotor Nucleus (Cranial nerve III) including Edinger-Westphall nucleus
- Trochlear nucleus (Cranial Nerve IV)
- Red nucleus
- Substantia nigra
- Extrathalamic Cortical Modulatory Nuclei
  - Ventral tegmental area - DA
  - Raphe nuclei (partial) - 5-HT
  - Locus ceruleus (partial) - NE
- Superior and inferior colliculi
- Periaqueductal gray

The PBN has been discussed in an earlier paper by this author.[3] In this paper the PBN was proposed as the integrating area for structural, chemical, and mental/emotional inputs whose output generated specific patterns of neuromuscular activity including the basis for concepts of "centering the spine."[4]

In fact, the PBN represents areas of nervous tissue that, when activated, can cause descending activation bilaterally of groups of flexor muscles, groups of extensor muscles, and other patterns of neuromuscular activity. This appears to be the basis for many bilateral extremity problems – in both upper and lower limbs, both proximally and distally.

The mesV nucleus contains the cell bodies for mechanoreceptors (MRs) associated with the temporomandibular joint and muscles. It is the only area in the body where the sensory cell bodies are within the central nervous system. (All other sensory cell bodies reside in the dorsal root ganglia, outside the CNS.) The localization of the mesV cell bodies in the mesencephalon means that the metabolic activity of these cells makes a major contribution to the metabolism of the mesencephalon. Likewise, the mesV cells are significantly impacted by the metabolic activity of the neurons of the remainder of the mesencephalon.

Each mesencephalon half (as is each other grouped neuron pool) is not only subject to the influences of all of the neurons in its pool, it is also subject to metabolic changes based on the firing rates of afferent neural pathways into it. The following list contains a number of pathways which carry afferent information into the mesencephalon, and hence can affect the firing rates and metabolism of the mesencephalon as a whole.

Some afferents pathways to the mesencephalon
- Visual light reflexes (CN III and superior colliculus)
- Auditory pathways (inferior colliculus)
- Slow stretch distal extremities (Dentatorubro…thalamocortical tracts)
- TMJ muscles (mesV)
- Ipsilateral cortical function – directly (Corticorubro and corticoreticular tracts)
- Ipsilateral cortical function - indirectly (through basal ganglia and thalamus - Corticostriatothalamoreticular…)
- Hypothalamic (autonomic or endocrine)
- The amygdala and limbic system afferents (both directly and through the basal ganglia)

These afferents can be used for challenging and treating mesencephalon metabolic activity. Although these afferent pathways have specific connections, their firing into these connections will influence the metabolism of all mesencephalon cells due to the interconnections of all of the neurons residing in this neuron pool area.

Below are listed some of the descending and distally directed efferent pathways leading out of the mesencephalon.

Descending and distally directed efferent pathways from the mesencephalon

- Oculomotor nerve - Cranial nerve III including Edinger-Westphal nucleus (pupil constriction)
- Trochlear nerve - Cranial nerve IV (eye movement down and in)
- PBN - Centering the spine patterns (via reticulospinal tracts through pons and medulla)
- Contralateral distal flexor activity (rubrospinal tract)
- TMJ functions via connections to the motor nucleus of V in the pons
- Descending pain control (via reticulospinal pathways from PAG)

Efferents which originate in the mesencephalon may be used to monitor the metabolic status of the area. A most popular tool is observation of the pupillary reflexes. One can observe for the speed and strength of onset, the time to fatigue, and the amount of fatigability, if any, and compare the right and left sides. This information must then be compared and correlated with other functional neurological findings. All of this may be correlated with muscle testing (motor) findings to give a complete picture of the nature and extent of mesencephalic involvement in the patient's overall status.

Because the mesencephalic nucleus of the trigeminal nerve (mesV) is the location of the sensory neurons related to TMJ proprioception, the TMJ significantly contributes to and is significantly affected by the metabolic state of the mesencephalon. This suggests that alterations in the metabolism of the mesencephalon will affect the TMJ. It also suggests that changes in stomatognathic proprioception may affect the metabolism of the mesencephalon. Due to the large size of the mesV, the effect in either direction can be great.

Metabolic stress in the mesencephalon neuron pool is the basis for many TMJ related symptoms, and is probably the basis for many of the more difficult TMJ problems. Mesencephalic TND with neuron pools close to threshold and firing at the least provocation, or even spontaneously, explains unusual and difficult symptoms such as the involuntary movements like tooth chattering and grinding of the teeth (i.e., spontaneous firing in the mesencephalon causing spontaneous firing of the mesV to the motor nucleus of the trigeminal nerve in the pons.) Likewise, it explains part of the recurrence of many other TMJ related problems, and how it is so difficult to get many TMJ patients stable, even with proper dental intervention and occlusal balancing. It also explains why the TMJ can be associated with so many other bodily functions, including neuromuscular, mental/emotional, and autonomic symptoms. That is, any change in TMJ proprioception may be met with an undesirable, spontaneous, and unpredictable mesencephalic response just like the person with the flu in the simple explanation of the TND concept given above. And depending on which pathways are most affected, mesencephalic projections cephalward and caudalward can impact a plethora of structural, chemical, and mental functions in an adverse manner.

Assessment of Mesencephalon Using Sensory Receptor Challenges and Muscle Testing Outcomes

Therapy localization (TL) to the TMJ during ranges of motion has been the AK standard assessment of TMJ function since it was first introduced by Goodheart in the 1970s. In the case of a TMJ associated with a mesencephalic metabolic problem such as TND, we have found that TMJ TL is often positive in any and every TMJ range of motion (ROM). This pattern will be negated if the patient inhales oxygen, and many times, if the patient is given an oral challenge with homeopathic mesencephalon. Other afferent activity to the mesencephalon will also change the muscle testing inhibitory response. This includes activation of light reflexes, slow stretching of the contralateral distal flexor muscles which fire through the spinocerebellar tracts to the cerebellum and then on to the mesencephalon via the dentatorubro tract. Ipsilateral brain activity (right brain – humming, left brain – math) will also impact the mesencephalon.

In these patients activation of the TMJ in any and all ROMs will also cause the evidence of a pattern of bilateral extremity muscle weakness. If there are bilateral symptoms in the upper and/or lower limbs, the bilateral weakness which shows up with any TMJ ROM will be obviously related to the patient's symptoms. If the patient does not complain of bilateral limb symptoms, there will be some (seemingly random) bilateral weakness pattern occur on TMJ ROMs such as bilateral posterior tibialis weakness, bilateral wrist/finger extensor weakness, bilateral rectus femoris weakness, and so on. More often the bilateral weakness(es) will be present in distal muscles, but any pattern may be present.

As mentioned above, sensory activity which drives the mesencephalon will also negate the positive TMJ TL in any ROM as well as the TMJ ROM-induced bilateral extremity weakness. This includes slow stretch of the distal flexors in the opposite extremity, activation of the light reflexes on the side of involvement (by direct light stimulation to the eye, or by contralateral temporal field stimulation), cortical activity on the side of involvement (right cortex – humming, left cortex – math), and so on. Those activities which negate the TMJ TL will be used therapeutically to restore adequate afferentation, hence adequate metabolic activity to the mesencephalon.

Therapeutic Afferentation of the Mesencephalon

Correction of TND in a metabolic pool depends on the availability of oxygen, glucose, and energy (ATP- producing) nutrients.[2] TND correction also requires adequate stimulation of the cell membrane receptors from afferent sources. This afferent stimulation turns on membrane receptors which subsequently stimulate cytoplasm metabolic activity and nuclear DNA-mediated protein synthesis. The protein synthesis increases the negativity of the cytoplasm, making the cell move away from firing threshold resulting in a healthier (metabolically speaking) neuron.

The type of afferent stimulation to the mesencephalon should be the same form as that which negated the positive challenge (i.e., what negated the TMJ TL during ROMs.) This usually includes ipsilateral cortical activity (right cortex – humming, left cortex – math), contralateral stretching of distal limb flexors (i.e., stretching the fingers and toes in the direction of extension – stretching the flexors), and light directed towards the left mesencephalon Edinger-Westphall nucleus. This can be by direct light stimulation into the ipsilateral eye, or more preferably, by temporal field stimulation in one eye and nasal stimulation in the other such as provided by Eyelights hemifield stimulation. Also, having the patient move the TMJ through the various ranges of motion that caused the weakness will stimulate the mesencephalon in a positive metabolic direction, as long as the stimulation is not overdone.

Supplying the mesencephalon with adequate, but not too much, afferentation is essential to restoring the metabolic health of the area. Excess afferent stimulation of the cellular membrane receptors can drive a near-threshold neuron past its metabolic tolerance into anaerobic metabolism with the production of lactic acid, loss of cell membrane integrity and death by apoptosis. Exceeding the metabolic threshold of neurons will have negative, if not disastrous effects. This can be seen in the difference of the effects of visual light stimulation on the mesencephalon depending the length of the stimulus. Light stimulation held past the point of pupil constrictor fatigue is different than a stimulus which is released prior to fatigue. Short, intermittent stimulation which is released prior to fatigue will improve the metabolism of the mesencephalic neuron pool, building up the resistance of those neurons to fatigue. Light stimulus held past the point of fatigue will only contribute to the TND state of these same neurons by driving them closer to firing thresholds and fatiguing them further.

As mentioned above, monitoring of autonomic parameters during afferent stimulation can help to ascertain that the stimuli are beneficial rather than exceeding the metabolic threshold of the cell. The most efficient method of autonomic observation is by monitoring the patient's pulse rate during afferent stimulation. Excess stimulation of neurons will cause the body to attempt to increase circulation to the area (to supply glucose, oxygen, and other nutrients) and this will result in an increase in pulse rate. Maintenance of or decrease in pulse rate during afferent therapy reflects reduction of metabolic stress in the nervous system, and in an indi-

cation that treatment can proceed with no danger. Afferent stimulation simultaneous with supplying oxygen can help protect against over stimulation.

Following adequate therapeutic afferentation, TMJ TL will be negative in any ROM as will TMJ ROM-induced bilateral extremity muscular inhibition (if it was previously present.) Peripheral ranges of motion will be more equal bilaterally, often with increased ROMs. Patients usually report a decrease in symptoms in various areas of their bodies, especially bilateral extremity symptoms which were associated with the locations of bilateral muscle weaknesses which were induced by the TMJ ROMs challenge. Normalization of TMJ ROMs and other TMJ related functions such as joint clicking will often be seen.

Conclusion

Normalization of TMJ sensory activity via AK and dental intervention is the fundamental step in restoring TMJ activity toward optimal function. Rehabilitation of the metabolically stressed (TND) mesencephalon is essential to attain long term stability in this area of the body. A "volatile" mesencephalon explains a number of TMJ symptoms including those which are far distant, often bilateral in nature, mental/emotional in nature, autonomic in nature, and/or, those which are related to so many and varied difficult stomatognathic symptoms.

Procedure

1. Diagnosis by Mesencephalic Challenge

- Does TL to the Right TMJ with the teeth clenched (and other TMJ ROMs) weaken a strong indicator muscle? And/or create a bilateral limb muscle weakness?
  - Is the positive TMJ TL negated by:
    Slow stretch of the left distal flexors?
    Right cortical activity?
    Visual left hemifield stimulation?
    Oxygen
    Mesencephalon homeopathic?
- Does TL to the Left TMJ with the teeth clenched (and other TMJ ROMs) weaken a strong indicator muscle? And/or create a bilateral limb muscle weakness?
  - Is the positive TMJ TL negated by:
    Slow stretch of the Right distal flexors?
    Left cortical activity?
    Visual right hemifield stimulation?
    Oxygen?
    Mesencephalon homeopathic?
- If so: Right or Left Mesencephalon treatment

2. Treatment

Perform simultaneously with nasal oxygen if available (1-2 liters flow rate):

- Doctor slowly stretches patients contralateral distal flexors (toes and ankle, fingers and wrist)
- Patient performs ipsilateral cortex activity (humming, math)
- Perform contralateral hemifield stimulation (Eyelights)
- Patient is instructed to move the TMJ through all ROMs

If oxygen is not available: Slowly increase intensity, frequency, and duration of stimulation so as to not exceed the metabolic limits of the TND cells. Monitoring pulse rate is useful to assess this.

Following Mesencephal Rehabilitation Correction:
Recheck challenge procedures as above.

References

1. Kandel, ER, Schwartz, JH: Principles of neural science. New York:Elsevier,1985. p. 193.
2. Schmitt, Walter H., Jr. Transneural degeneration and the links between the nervous system and the body chemistry. Proceedings of the ICAK - U.S.A. Volume 1, 1999-2000. p. 149-156.
3. Schmitt, Walter H., Jr. Where is the triad of health? Proceedings of the ICAK - U.S.A. Volume 1, 1996-97. p. 83.
4. Schmitt, Walter H., Jr. Centering the spine functional neurological and biochemical considerations. in Collected papers of ICAK - summer 1987. Shawnee Mission, KS:International College of Applied Kinesiology, 1987

Reoccurring Pitch Pattern and the Frontal Fault

Paul T. Sprieser, D.C., DIBAK

Abstract

An examination of the relationship of reoccurring pitch pattern of the PRY-T technique to the frontal bone cranial faults.

Introduction

The PRY-T technique has been part of applied kinesiology syllabus since 1980 and has become a mainstay of practice ever since. Over the years, it has been my observation that the correction of Pitch had always been very simple and long lasting. By long lasting I mean that a single correction would remain corrected for at least six month or longer. However, recently I started having cases that the Pitch pattern would be back on many subsequent visits and I had to repeat the correction two or three times.

So, I started wondering what I have overlooked that allows this pattern to return so quickly. To my surprise, I found a definite relationship in this recurrent pattern to a hidden frontal fault that was not picked up in my initial examination or apparent to visual observation or from patient's case history.

Discussion

The PRY-T technique in one of the first observations I make during my examination and also one of the structural corrections I make. This is because of the connection of modular distortion patterns to the dura and its effects on switching.[1] And also because of the effects correction of the Pitch pattern and its improvement in range of motion seen at the hip joint.[2] This fact is readily observable by both patient and doctor and it is something that I always bring to the patient's attention.

I want the patient to know the importance of having good structural alignment and how it improves not only the appearance but also affects their health. I explain that their bodies use a certain percentage of it metabolic energies to produce joint lubricants and that this type of distortion causes an additional burden on them and could lead to many types of chronic health problems.

Pitch is described as the position of an airplane in reference to its lateral axis, a line that parallels the wings. It refers to climbing or diving in reference to an airplane. In structural terms this relates to anterior or posterior positioning of the pelvis, head, or some other body module.[3]

Method

When I found a Pitch pattern had returned, on a subsequent visit, I started to re-examine what muscle weakness could allow this pattern to return. What I found was a hidden cranial fault and in this case, a frontal fault. What I used to uncover the fault was eye into distortion or E.I.D.[4]

As we all know one of the characteristic weakness of this fault is bilateral neck flexor weakness and this could cause the loss of the normal A to P relationship seen in the Pitch pattern.

I challenged the Pitch pattern with the patient's feet on the table with hip in flexion and then put the neck into flexion and had a weakness occur in the PMC indicator muscle. I then had the patient place one hand on the involved side of frontal fault, with thumb on maxilla just under the malar surface of the zygomatic bone and the remaining finger of the same hand on the frontal bone.[5] I then repeated the test using the free arm that was not therapy localizing the frontal bone with hip and head and cervical spine in flexion. This negated the positive weakness of the PMC confirming the connection to the frontal bone cranial fault.

I have seen this presently in about twenty-five different cases and occurrence was with both internal and external rotation of the frontal fault. I don't believe that the frontal fault is necessarily the primary cause of the frontal faults, because I find almost every new patient has a Pitch pattern in more then 90% of case but I don't find frontal faults present in that high a percentage.

Conclusion

I feel that the frontal fault play a role in reoccurring Pitch patterns rather then the main causative factor. As far a causative factors for the frontal fault whiplash type of injuries seem to be one of the main factors especially when the force if A to P or a P to A type of injury.[6]

References

1. Sprieser, Paul, T., Collected Papers of I.C.A.K.-The Formation of the Frontal Bone Fault, Winter 1981, p. 355.
2. Sprieser, Paul, T., Collected Paper of I.C.A.K.-A New Slant on Switching and the PRYT Technique, 2002-2003, p.p. 197-202.
3. Walther, David, S., Applied Kinesiology, Synopsis, 2nd Edition, Systems DC, Pueblo CO, 2000, p. 213.
4. Walther, David, S., Applied Kinesiology, Synopsis, 2nd Edition, Systems DC, Pueblo CO, 2000, p.p. 43-45.
5. Walther, David, S., Applied Kinesiology-The Advanced Approach in Chiropractic, Systems DC, Pueblo CO, 1976, p.p. 144-145.
6. Sprieser, Paul, T., Collected Papers of I.C.A.K.-The Formation of the Frontal Bone Fault, Winter 1981, p. 355.

Supraspinatus Muscle as an Indicator of Brain Serotonin Levels

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Abstract

In applied kinesiology, the supraspinatus muscle had it organ relationship to the brain, but to any reasonable knowledge the brain controls all muscles. I propose that the relationship of the supraspinatus muscle is actually an indicator of the brain's neurotransmitter serotonin level.

Introduction

For the past two years I have been working with Thought Field Therapy (TFT) and I have received many referrals from psychotherapists and psychiatrists. Many of these patients had been suffering from depression, general anxiety disorders (GAS), and obsessive-compulsive disorders (OCD) and phobias.

The standard medical therapy has been to prescribe anti-depressants or serotonin reuptake inhibitor (S.S.R.I.) drugs. I don't believe you can pick up a newspaper, magazine or see a television or hear radio commercial every day of the week that doesn't tout the benefits of treatment with these drugs for the above mentioned conditions.

As a chiropractor I don't necessarily believe in the use of drugs although sometime it may be necessary to get a patient through a crisis. The one common finding I have noted with all patients who were taking anti-depressants was a weakness of the supraspinatus muscle. This seems to be constantly present even when the drugs have ameliorated the patient's symptoms.

The other noted factor is the use of TFT as a therapeutic treatment for depression, OCD, GAS, and phobias would strengthen the weak supraspinatus when the SUD level reached zero,[1] this would seem to indicate change in the brain chemistry that cause the muscle to strengthen because no other five IVF factors had been treated. This could also mean when no weakness of this indicator muscle is present for a given period of time that the body had corrected the serotonin level and drug therapy could be discontinued.

Discussion

Over the past two years a number of article had been published in the New York Times Science section about depression and research into identifying methods that help patients with depression. Two particularly interesting studies that were conducted at U.C.L.A. by two different researchers show changes in the prefrontal cortex both on PET scans and EEG.

Ian A. Cook, M.D., showed electrical changes on EEG in the prefrontal cortex 48 hours and one week after taking Prozac (fluoxetine) and Effexor (venlafaxine). In patients that responded to drug therapy compared to the group that received a placebo did not show this electrical changes. One important finding from this study was the drug therapy only worked 50% of the time and the placebo worked 38% of the time in patients that showed clinical improvements in their symptoms. This does not seem to me to be a very good batting average.[2]

The other study was conducted by Arthur Brody, M.D. demonstrated PET Scan decrease in high activity in the prefrontal cortex with talk therapy. This demonstrated chemical changes in serotonin level that psychotherapy (talk therapy) produced without the use of drugs.

Both studies show the importance of the prefrontal cortex in depression and also the effects of serotonin on this region of the brain. Guyton points out this region of the brain just behind the eye serves as executive function, integrating information and inhibition of emotional impulses from deep brain center of the limbic system.[3] Other articles show that drugs such as Prozac and Zoloft have an effective as anti-aggressive on emotions rage.

Muscle testing is a functional neurological tool that is controlled by the brain through the spinal chord. Dr. George Goodheart had demonstrated the connection of the supraspinatus muscle to the brain as it primary organ association. In a study conducted at the New York Chiropractic College in 1989 showing somatosensory evoked potential changes during muscle testing confirming the electrical changes noted between a weak or strong muscle found by manual muscle testing.[4] This is similar to both electrical changes noted by Dr. Cook on EEG and the metabolic activity changes noted by Dr. Brody on PET Scan in research studies at U.C.L.A.

We know that serotonin or 5-Hydroxytryptophan (5-HT) is derived from the essential amino acid tryptophane that the body converts to serotonin by metabolizing by an oxidative deamination to 5-Hydroxyindoleacetate by an enzyme that catalyzes this reaction is monoamine oxidase (MAO).[5]

So a patient suffering with depression could have a metabolic problem with not having enough tryptophane in their diet or perhaps an absorptive problem with this amino acid. Or there may be a problem in the conversion of 5-HT to serotonin.

The one common denominator that I kept finding was the weakness of the supraspinatus muscle bilaterally in patients with depression. This would immediately strengthen when the right drug or nutritional factors was placed in the patient mouth. The importance of this is that we now had a method of finding the correct treatment method be it nutritional such as Saint John's Wart, S.A.M.E., or with drug therapy because only the products that strengthen the bilaterally weak supraspinatus muscle will give clinical improvement in the patient's symptoms. This will also allow the medical doctor to know what drug is most likely to work and when to stop drug therapy.

Methods

This study consisted of a total of 226 patients, 132 females and 94 males. Age range of females from 16 to 85 and males from 18 to 78. This sampling was from the same group that data was collected for the paper Though Field Therapy and its Associated Effects on the Autonomic Nervous System, with an additional 120 more patient collect over the next year.

Patients were requested to bring in their medication that the medical doctor had prescribed. The drugs that were tested were Zoloft, Wellbutrin, Paxil, Prozac, Effexor, Remeron, Elavil, Norpramin, Depakote and Tofranil. I also tested non-prescriptive treatment of St. John's Wart and S.A.M.E.

I use the supraspinatus muscle that was found to be bilaterally weak to test both the drugs and non-prescriptive alternatives. What I found was a strengthening of the supraspinatus muscle when the proper medication or alternative therapy was put in the patient's mouth.

I would use TFT methods to treat the patient emotional problems what I noted was the supraspinatus weakness would clear when the Subjective Units of Discomfort (SUD) reached 1 or zero. The patient usually noted an improvement in their symptoms especially as the level dropped below three. Most would have an immediate response but some did feel any change till the following day.

Conclusion

The most significant finding is the consistency of the presence of the bilateral supraspinatus in patients with low serotonin levels diagnosed with depression, general anxiety syndrome, obsessive-compulsive disorders, and phobias. I find this weakness always present in any patient that has been taking serotonin reuptake inhibitors for these conditions. This weakness remains present even though the patient symptoms have been eliminated due to the medication.

When this weakness remains corrected without further intervention on the doctors' part it appears to mean that the brain has corrected the serotonin imbalance and medication can be slowly stopped.

I believe this simple test can give every mental health care practitioner a more objective value of their methods and should be included in their patient evaluation.

References

1. Gallo, Fred, P. Ph.D.-Energy Psychology, CRC Press, Boca Raton, FL. 1999.
2. Cook, Ian, A. M.D.-Early Changes in Prefrontal Activity Characterize Clinical Responders to Antidepressants, Neuropsychopharmacology 2002-Vol 27, No1, Published by Elsevier Science Inc.
3. Guyton, Arthur, C., M.D.-Structure and Function of the Nervous System, W. B. Saunders Co., Phil., 1976 p.p. 194-195.
4. Leishman, Gerald, M.D., Shambaugh, Philip, D.C., Ferentz, Avery, D.C., Somatosensory Evoked Potential Changes During Muscle Testing-International Journal Neuroscience, 1989. Vol. 45, p.p 143-151.
5. Murray, R.K., M.D., Granner, D.K., M.D., Mayer P.A.,Ph.D., Rodwell V.W., Ph.D., Harper's Biochemistry, 21st Edition, Appleton & Lange, Norwalk CT, 1988.

Division III

Comments

The Relationship of Switching to the Yaw #2 of the PRY-T Technique

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Abstract

The Yaw #2 pattern is the causative factor of neurological disorganization in 99% of the population. A new connection to the myofacial system is examined as a reason for the persistence and reoccurrence of this pattern.

Introduction

During the past November seminar "Body-Heart-Mind," Dr. Goodheart introduced the idea of facial meridians. This idea came from his reading a book called Anatomy Trains. This book, contain the idea that the muscle system linkage to the facial tissues follow a specific pattern that could be likened to railroad tracts.

These tracts were either local or express systems that carried the forces of gravity throughout the skeletal system and allow us to maintain and move in the upright position. Since the force of gravity is always pulling us downward. This pull is counter by the facial meridian tract system that exists in all of our bodies.[1]

When I saw Dr. Goodheart demonstrate the presence of these patterns in 90% of the population. I felt this was the answer to the persistence of the yaw #2 pattern return that I have been observing in-patients over the past four years.

Discussion

Switching or Neurological Disorganization has been a part of AK as long as I can remember. I have been using AK in my practice for the past 34 years. In the early years we started our examination and treatment of our patients by first stimulating (rubbing) in a circular fashion K27 and CV8 umbilicus simultaneously for 30 seconds. This would assure us that the patient's switching was corrected.[2]

As applied kinesiology became more organized so it could be taught in a more uniform fashion. Dr. David Walther first book *Applied Kinesiology-The Advanced Approach In Chiropractic*, was published in 1976. Page 54 showed three forms, Ocular Lock, Conception Vessel and Governing Vessel CV 24 and GV27 and finally K27 and Umbilicus CV8.[3]

As described in the text Ocular Lock was caused by loss of the level head causing a tilt and gimballing of the eye to compensate for this distortion. This can be challenged by having the patient turn his eyes as far as he can to one side without moving the head. This would cause a strong indicator muscle to weaken. Having the patient read a text in the normal fashion from left to the right, which would cause a strong muscle to weaken, could also challenge this. But if the patient read the same text from the right to the left there would be no weakness. This was treated by simultaneous stimulation of K27 and CV8 umbilicus for 30 seconds.[4]

This meant that K27 and CV8 switching had to be present for Ocular Lock to be present, but it also meant that K27/CV8 switching pattern was basic to this phenomena. In other words you had to have K27/CV8 switch in order for Ocular Lock to be present. But a K27/CV8 could be present without Ocular Lock. It also seems logical if head level and eye gimballing was the cause if you level the head this should also correct Ocular Lock.

The CV24/GV27 pattern of switching was described as a weakness when one point is therapy localized, or both CV24/GV27 are TL simultaneously causing a strong indicator muscle to weaken. The treatment is described as contact to CV24 and CV2 (upper symphysis pubis) simultaneously with solid pressure for 24 seconds. Next contact to GV1 (luo point at the tip of coccyx) and hold for 20 seconds. Then challenge the vertebra that is the associated point for GV, which is B16 located between T6-T7.[5]

The above methods are again reiterated in Dr. Walther's next book *Applied Kinesiology-Volume #1* – "Basic Procedures and Muscle Testing," published 1981, pages 134-140. When Dr. Walther published his third book in 1988 Applied Kinesiology-"Synopsis", many more factors had been added such as foot and gait mechanism, nasal tap, auxiliary K27 and hidden switching, pages 148-154. *0* edition published 2000 pages 170-181 remaining relative unchanged from the 1988-1st edition. The most important factor that is stressed is not to treat switching until you find the cause and that brings us to the start of this topic.

The other factors are mentioned in the Synopsis. I will start with Nasal tap. The Nasal tap is associated with a positive ocular lock if corrected by stimulation of K27/CV8 have the patient lateralize the eye and take to nasal sniffs if a weakness occurs tapping is done rapidly for sixty second on both sides of bridge of nose.6

The Auxiliary K27/CV8 is located adjacent to transverse process of T11 vertebra bilaterally this is B19. If this point TL one at a time they are stimulated simultaneously with CV8 (umbilicus).[7]

Hidden switching refers to patterns that do not TL to K27/CV8 without doing special procedures to bring out the problem. This could require having the patient TL K27 in standing or gait positions before a muscle weakness becomes apparent.[8]

Finally there are two other TL switching patterns worth mentioning. First it a switching seen in-patient's with schizophrenia. They will TL by crossing their contacts to the opposite K27. Patient must avoid have the hand touch one and other when TL K27. The second switching is called ionic switching this is usually due to some type of nasal airflow obstruction. The patient with this form usually only shows therapy localization with palms up with the (dorsal hand surface) rather then the palm surface. Rubbing CV8 umbilicus and GV1 coccyx treats this.[9]

Over the past five years I have been collecting data on switching and its association to the PRY-T; those finding were published in the Collected Paper of ICAK-2002 pages 197-202 titled A New Slant on Switching and the PRY-T Technique. If you do a postural evaluation of your patient standing on a plumb line you will note that the pelvic girdle is carried forward in the transverse or horizontal plane on the right and the shoulder girdle is forward on the left. The patient is stuck in a walking or stride position with right leg forward and left arm forward even though there two feet are together in the same line. This pattern is called the Yaw #2.[10]

This distortion can also be seen if you sight up on the pelvic region of the patient whether supine or prone when you stand at the head or foot of the table. I would see this pattern return constantly no matter how I corrected it. The other parts of the PRY-T patterns, which stand for Pitch, Roll, Yaw #1 and Tilt when corrected during our first visit usually remain corrected for many months sometimes up to a year. So I asked myself the famous question that Dr. Goodheart always asks, and that is, Why is that? Meaning, why is it the way it appears? In this case why will the Yaw #2 not stay corrected?

In searching for an answer, I found the muscle that is involved in this pattern is the psoas on shoulder forward side and latissimus dorsi on the pelvic forward side. The nutritional support was found to be RNA, Ionic Calcium (Miera) Carried by Biotics Research and SOD superoxide dismutase. What I also discovered that the Yaw # 2 pattern was responsible for switching at least 99% of the time with the remaining 1% divided into the rest of the PRY-T patterns.[11]

The correction is simple and can be done in an adaptive side posture with the patient pushing their knee region (upper leg) against the doctor. The shoulder that is not resting on the table is straight up and the pelvis being pushed forward near the PSIS. Or it can be corrected prone or supine using the SOT block. Supine the block is

placed below the side of the pelvis near the femoral head that is closer to the table and the side that is off the table is gently thrust back towards the floor. In the prone position the block is placed under the area near the femoral head closet to the pelvic part of the table and the side that is most (superior) away from the table is gently thrust floor-ward. This correction should correct the Yaw #2 pattern and also correct the switching. This can be rechecked to TL to K27.[12]

The importance of correcting switching should be apparent and should be the first thing that is checked for on every patient and on every visit. Since the principles of AK are based on muscle testing the information we obtain from these test are only as good as the accuracy of the muscle test. Switching or neurological disorganization will cause misinformation that is always random. By this I mean it could produce a muscle weakness that postural analysis tell you is on the left but the muscle test weak on the right. It could cause a challenge for a structural correction to be the opposite of what it should be and thereby cause an iatrogenic problem for the patient. It could give you the wrong respiratory assist pattern for cranial corrections. So start each patient examination and treatment with a level playing field by correcting switching.

As I described in my paper A New Slant on Switching and the PRY-T Technique the yaw#2 pattern was back every time I saw a patient. This was also confirmed while teaching the 100 Hour AK course. We would correct the students on Saturday and the majority of doctors would have it back on Sunday. I knew that some factor had not been discovered that allow this pattern to return.

At this point I know that the number of patients that I have observed has been over 8,000 during the past four years of research on the pattern, so I feel I can speak with some certainty, and that is every patient is switched every time you see them. The other fact is that the yaw#2 pattern is the cause of switching at least 99% of the time.

In searching for a more lasting correction I had found the muscles that were weak that allow the pattern to occur. The correction of this weakness for example, a yaw#2 on the left would be a weak psoas on the left and a weak latissimus dorsi on the right with the five I.V.F. factors brought about only a short lived correction. These finding were described in the Collected Paper of ICAK, "Yaw#2 Muscular Pattern, 1982.[13]

I had also tried increasing the stride length on the short step side, Isogai squatting exercise, Cranial Sacral method of blocking under the short stride side and opposite shoulder and the tennis balls under the occiput. These methods helped but still the correction was short lived in comparison to the other factors of the PRY-T such as pitch or yaw#1. Usually one correction would last six months to a year.

I came upon the nutritional support that related to this modular distortion patterns that I described in my paper "A New Slant on Switching and the PRY-T Technique", Collected Paper of ICAK, 2002. These nutrients were RNA, Mire ionic calcium and super oxide dismutase (SOD). The inclusion of these still did bring about any lasting correction of the yaw #2 pattern.[14]

If the facial meridians were the answer for the persistence of the yaw #2 pattern, then Folic Acid/B12 should also be associated to switching.

Method

To test this premises I that patients TL K27 and chew the Folic Acid/B12 tablet and the retest a positive indicator muscle. The Folic Acid/B12 negated the weakness cause by the TL to K27.

The next step was to block challenge (BID) for yaw #2 with the patient supine this produced a weakness to a strong indictor muscle, which was negated by the Folic Acid/B12. This seems to me to confirm the connection of the facial meridians to the yaw #2 pattern and to switching.

While reading the book Anatomy Trains I came upon a drawing of an ape hanging from a branch showing the line of facial meridians that carried from the forearms wrist flexors through the biceps and coracobrachialis to

the pectoralis minor to the rib cage and into the rectus abdominis to the pubic bone. By using pincer palpation I was able to confirm this pathway related to the yaw #2 pattern. It seem reasonable to me that this line carried beyond the pelvis into the lower extremity on the opposite side this went through the psoas and adductor muscle down tibia where it cross over and continued down and lateral head of the gastrocnemius and peroneus longus, tertius, and brevis on the right.[15]

When I found the chapter called Functional Lines in Anatomy Trains I believed that I found to the anterior and posterior facial meridians that caused the yaw #2 pattern to return. The posterior line extended further then shown in the text. If the patient had a yaw #2 on the left pincer palpation would show the following positive weakness. The right forearm extensor through the triceps into the latissimus dorsi down to the sacral fascia crossing the sacrum into the gluteus maximus, vastus lateralis. This line then cross medially popliteal fossa and muscle and down the medial head of gastronemius.[16]

I have test this pattern on approximately 200 patient to date and it seems to hold up as the cause of yaw #2 pattern. Since I added these functional lines correction by percussion and included folic acid/B12 the yaw #2 correction have lasted as long as 5 weeks with most patients.

Conclusion

The cause of persistence yaw #2 pattern is the facial meridian pull along the functional lines and the body's attempt to counter the pull of gravity, along with right handedness and brain dominance in 85% of greater of the population. The inclusion of Folford's method of percussion and nutritional support of folic acid/B12 has made a great improvement in maintenance of this correction.

References

1. Myers, Thomas, W. Anatomy Trains-Myofascial Meridians for Manual and Movement Therapists, Churchill Livingstone, NY, 2001.
2. Walther, David S. Applied Kinesiology-The Advanced Approach In Chiropractic, System D.C., Pueblo, CO. 1977.
3. Ibid.,Walther, 1976 p. 54.
4. Ibid.,Walther, 1976.
5. Walther, David S. Applied Kinesiology-Vol. #1-Balsic Procedures and Muscle Testing, System D.C., Pueblo, CO, 1981, pp 134-140.
6. Walther, David S. Applied Kinesiology-"Synopsis", System D.C., Pueblo CO, 1988, pp. 148-154.
7. Ibid., Walther, 1988, pp. 148-154.
8. Walther, David S., Synopsis –2nd Edition, 2000, System D.C., Pueblo CO, 2000, pp 170-181.
9. Ibid., Walther, 2000, pp. 170-181.
10. Sprieser, Paul T.-A New Slant on Switching and the PRY-T Technique, Collected Papers of ICAK-2002 pp. 197-202.
11. Ibid., Sprieser-Collected Papers of ICAK 2002 pp. 197-202.
12. Ibid., Sprieser-Collected Papers of ICAK 2002 pp. 197-202.

13. Sprieser, Paul T.-Yaw #2 Muscular Pattern, Collected Papers of ICAK-W 1982, p 255.

14. Ibid., Sprieser-Collected Papers of ICAK 2002 pp. 197-202.

15. Ibid., Myers-Anatomy Trains, 2001, p 52.

16. Ibid., Myers-Anatomy Trains, 2001, pp. 183-190.