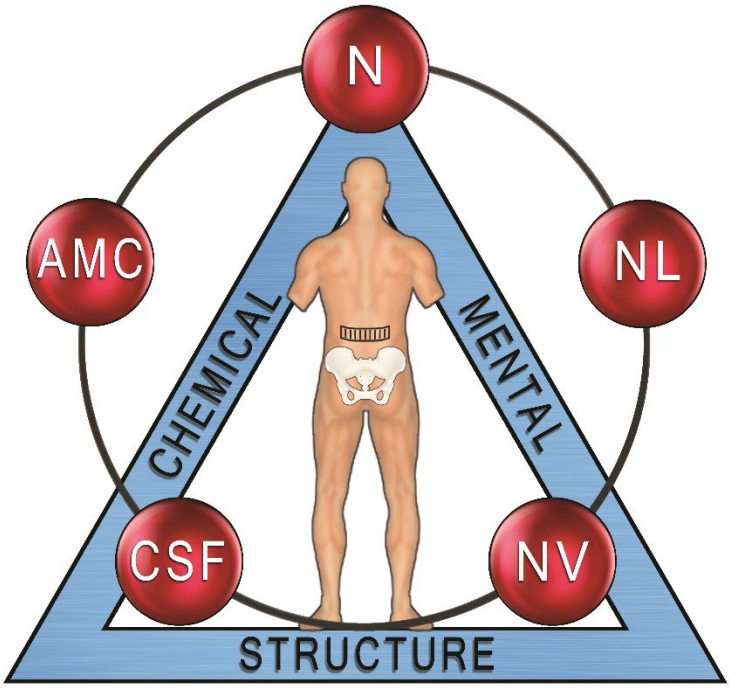

INTERNATIONAL COLLEGE OF APPLIED KINESIOLOGY U.S.A.

Experimental Observations of Members of the ICAK

Volume 1, 2022-2023

Seventy-Third Collection of the Proceedings of the Annual Meeting

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

Experimental Observations of the Members of the ICAK

Volume I, 2022-2023

Proceedings of the Annual Meeting

Professional Applied Kinesiology™

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

Experimental Observations of the Members of the ICAK

Volume I, 2022-2023

Proceedings of the Annual Meeting

Presented:

July 28 – 31, 2022
Atlanta, Georgia

Publications Staff:

Angela Capra, Executive Director
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Message from the Chairman

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

As we continue to move past the pandemic it has become more important than ever for members of the International College of Applied Kinesiology®-U.S.A. to share their insights, outcomes, case histories and research through the papers presented in these Proceedings.

This year we continue our in-person Homecoming. We welcome the opportunity to be together and share what we have learned over the last year. As we all know, there is as much learned in the breakrooms and hallways as there is in the conference.

It continues to be our hope that the Homecoming, along with these published works, document the first steps toward furthering the application of applied kinesiology in diagnosis and clinical skills ultimately becoming the part of the accepted body of knowledge we embrace. We invite and encourage all members to participate in contributing to and expanding upon the basis of neuro-functional muscle testing we call applied kinesiology. Your clinic is your laboratory, your patients the source of unlimited observation and input, and whether a case or double-blind study, they all add to the knowledge base.

We are pleased to have the opportunity share with the members of ICAK-U.S.A. the advances and successes of this year. It is truly a gathering of academic eagles and clinical genius. Thank you and congratulations to all of you who have taken the time to contribute.

A special thanks to Drs. David Engel, John Podlaski and Corey Osbourne for your time and knowledge in helping to produce this publication.

With great excitement we look forward to seeing you and sharing at this year's ICAK-USA Homecoming.

Introduction

This seventy-third collection of papers from members of the International College of Applied Kinesiology®-U.S.A. contains fourteen original papers written by eight 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.

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

Instructions to Authors

Proceedings of the ICAK-U.S.A.

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

The ICAK-U.S.A. recognizes that the usual procedure for selection of papers in the scientific community is a blind review. However, the purpose of *The Proceedings of the ICAK-U.S.A.* is to stimulate dialogue, creative thinking and critical review among its members; thus, review in this instance is not blinded. These papers are distributed only to the members of the ICAK-U.S.A. for general comment and evaluation, and for the members to put into perspective the validity of the described approaches. The purpose is to put before the membership primary observations that may lead to more in-depth study and scientific investigations, as well as spawn new areas of research. Such is to inspire progress in the field of applied kinesiology.

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

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

Manuscripts are accepted by the ICAK-U.S.A. for publication with the understanding that they represent original unpublished work. Delivery of a manuscript to the ICAK-U.S.A. Central Office does not imply it will be published in the Proceedings. Manuscripts are reviewed by the Proceedings Review Committee and authors will be notified in a timely manner of their manuscripts acceptance or rejection. The author may appeal any paper rejected to a separate committee composed of members of the Publications and Research Advisory Committees. The decision of this committee on publishing the paper will be final.

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

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

Continuing call for papers includes:

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

Hypotheses—projections from previous observations that may establish a solid basis for further in-depth investigations.

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

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

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

Case reviews—a retrospective comparative assessment of the diagnosis and treatment of several cases of a similar condition i.e., the comparative evaluation of two or more case reports.

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

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

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

The following editorial policies will apply:

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

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

Patient anonymity—Ethical and legal considerations require careful attention to the protection of the patient's anonymity in case reports and elsewhere. Identifying information such as names, initials, actual case numbers, and specific dates must be avoided; other identifying information about a patient's personal history and characteristics should be disguised.

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

Conflict of interest—in recognition that it may at times be difficult to judge material from authors where proprietary interests are concerned, authors should be prepared to answer requests from the editor regarding potential conflicts of interest. The editor makes the final determination concerning the extent of information released to the public.

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

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

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Manuscript Preparation

Authors are requested to submit final manuscripts via email to icak@dci-kansascity.com. Each manuscript file should be titled with the author’s last name and the manuscript tile. All manuscripts must be submitted in Microsoft Word.

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

Approved Manuscript Style

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

Summary of Requirements

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

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

- Title page (page 1)
- Abstract and key word page (page 2)
- Text pages (starting on page 3)
- Acknowledgment page
- Reference page(s)
- Table page(s)
- Legends for illustrations pages(s).

Detailed Preparation Procedure

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

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

the ICAK-U.S.A. should begin to adopt SI as their primary system of measurement as quickly as it is feasible.

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

Title Page

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

Abstract and Key Word Page

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

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

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

Text Pages

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

Please visit the following link online for helpful information on writing patient case reports:

www.soto-usa.org/Newsletter/DCInternetEdition/dc_internet_ed_vol_3_no3Abstrak/Green%20Johnson%20Case%20Reports.pdf

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

Introduction

Clearly state the purpose of the article. Summarize the rationale for the study or observation. Give only strictly pertinent references and do not review the subject extensively; the introduction should serve only to introduce what was done, why it was done and what could be done to address shortcomings or gaps in what we have learned from what was done.

Materials and Methods

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

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

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

Results

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

Discussion

Emphasize the new and important aspects of the study and conclusions that follow from them. Do not repeat in detail the data given in the Results section. Include in the Discussion the implications of the findings and their limitations and relate the observations to other relevant studies. Conclusions that may be drawn from the study may be alluded in this section; however, they are more formally presented in the section to follow.

Conclusions

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

Acknowledgments

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

Reference Pages

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

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

References must be verified by the author(s) against the original document. Abstracts, “unpublished observations” and “personal communications” may not be used as references, although reference to written (not verbal) communications may be inserted in parentheses in the text. Information from manuscripts submitted but not yet accepted may be referred to in parentheses in the text. Manuscripts accepted but not yet published may

be included in the references with the designation “In press.” When a previously cited reference is used again, it is designated in the text in parentheses by the number originally assigned to it by its first use: do not assign it another number or use the notation “op cit.”

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

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

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

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

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

Journals

1. Standard article You CH, Lee KY, Chey RY, Menguy R. Electrogastrographic study of patients with unexplained nausea, bloating and vomiting. Gastroenterology 1980; 79:311-4.

2. Corporate author The Royal Marsden Hospital Bone-Marrow Transplantation Team. Failure of synergeneic bone-marrow graft without preconditioning in post-hepatitis marrow aplasia. Lancet 1977;2:242-4.

3. No author given Coffee drinking and cancer of the pancreas [editorial]. Br Med J 1981;283:628.

4. Journal supplement Magni F. Rossoni G, Berti F. BN-52021 protects guinea-pig from heart anaphylaxis. Pharmacol Res Commun 1988;20 Suppl 5:75-8.

5. Journal paginated by issue rather than volume Seaman WB. The case of pancreatic pseudocyst. Hosp Pract 1981;16:24-5.

Books and other monographs

6. Personal author(s) Eisen HN. Immunology: an introduction to molecular and cellular principles of the immune response. 5th ed. New York: Harper and Row; 1974. p. 406.

7. Editor, compiler, chairman as author Dausset J, Colombani J, editors. Histocompatibility testing 1972. Copenhagen: Munksgaard; 1973. p. 12-8.

8. Chapter in a book Weinstein L, Swartz MN. Pathogenic properties of invading microorganisms. In: Sodeman WA Jr, Sodeman WA, editors. Pathologic physiology: mechanisms of disease. Philadelphia: WB Saunders; 1974. p. 457-72.

9. Published proceedings paper DuPont B. Bone marrow transplantation in severe combined immunodeficiency with unrelated MLC compatible donor. In: White HJ, Smith R, editors. Proceedings of the 3rd Annual Meeting of the International Society for Experimental Hematology. Houston: International Society for Experimental Hematology; 1974. p. 44-6.

10. Agency publication Ranofsky AL. Surgical operations in short-stay hospitals: United States—1975. Hyattsville (MD): National Center for Health Statistics; 1978. DHEW publication no (PHS) 78-1785. (Vital and health statistics; series 13; no 34).

11. Dissertation or thesis Cairns RB. Infrared spectroscopic studies of solid oxygen [dissertation]. Berkeley (CA): University of California; 1965.

Other articles

12. Newspaper article Lee G. Hospitalizations tied to ozone pollution: study estimates 50,000 admissions annually. The Washington Post 1996 Jun 21; Sect. A:3 (col. 5).

13. Magazine article Roueche B. Annals of medicine: the Santa Claus culture. The New Yorker 1971 Sep 4:66-81.

Table Pages

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

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

Illustration Legend Pages

Type legends for illustrations double-spaced, starting on a separate page, following the table pages. Identify each legend with Arabic numerals in the same manner and sequence

as they were indicated in the text in parentheses (e.g., Figure 1). Do not type legends on artwork copy or on pages to which illustrations may have been mounted; they must be typed on separate pages from the illustrations themselves.

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

Illustration Preparation

Illustrations (including lettering, numbering and/or symbols) must be of professional quality and of sufficient size so that when they are reproduced for publication all details will be clearly discernible; rough sketches with freehand or typed lettering are not encouraged. All illustrations should be submitted embedded in the manuscript document in the appropriate place.

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

Cite each figure in the text (generally in parentheses) in consecutive order. If a figure has been published, acknowledge the original source and submit a written permission letter from the copyright holder to reproduce the material. Permission is required, regardless of authorship or publisher, except for documents in the public domain*.

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Manuscript Submission Summary

Manuscript components

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

- Manuscript electronically via email of CD (The author should be sure to retain the original file in case of loss of the submission copies in transit.)
- Release form (signed by all authors, and by employer if study was a work for hire).
- Permission letter(s) of permission to use previously published material in all forms and media (if applicable).
- Consent form(s) to publish photographs in which subjects may be identifiable (if applicable).
- Cover letter from principal author (or author specified as correspondent) providing any

special information regarding the submission which may be helpful in its consideration for publication.

Submission Instructions

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

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4919 Lamar Ave.
Mission, KS 66202

Applied Kinesiology
Status Statement

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

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

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

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

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

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

- Myofascial dysfunction (microavulsion and proprioceptive dysfunction)
- Peripheral nerve entrapment
- Spinal segmental facilitation and deafferentation
- Neurologic disorganization
- Viscerosomatic relationships (aberrant autonomic reflexes)
- Nutritional inadequacy
- Toxic chemical influences
- Dysfunction in the production and circulation of cerebrospinal fluid
- Adverse mechanical tension in the meningeal membranes
- Meridian system imbalance
- Lymphatic and vascular impairment

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

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

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

- Transient directional force applied to the spine, pelvis, cranium, and extremities.
- Stretching muscle, joint, ligament, and tendon

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

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

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

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

An applied kinesiology-based examination and therapy are of great value in the management of common functional health problems when used in conjunction with information obtained from a functional interpretation of the clinical history, physical and

laboratory examinations, and from instrumentation. Applied kinesiology helps the physician understand functional symptomatic complexes. In assessing a patient's status, it is important to understand any pathologic states or processes that may be present prior to instituting a form of therapy for what appears to be a functional health problem.

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

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

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

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

Approved by the Executive Board of the International College of Applied Kinesiology–U.S.A., June 16, 1992. Updated May, 2001.

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Experimental Observations of Members of the ICAK

[redacted]

Volume 1, 2022-2023

An Evolution and Progression of Using Applied Kinesiology for Accessing Negative Reactions to Foods

Michael Lebowitz, D.C.

Abstract

Using manual muscle testing for negative reactions to foods reaches back to the beginnings of Applied Kinesiology (AK). Refinements, methods, improvements, etc. have occurred over the past four decades with the work of Goodheart, Schmitt, Lebowitz, Lang, and many others. We will examine the work primarily of Drs. Walter Schmitt and Michael Lebowitz and the progression of testing methods to eliminate false negatives, increase efficiency and efficacy etc.

Key Indexing Terms

Muscle Testing, Food Reactions, Hypertonic Reactions, Biomagnetic Testing

Introduction

Testing various foods for muscle inhibition has been a part of AK going back as far as 1969 (1). Goodheart observed that on specific patients stimulating either the gustatory or olfactory reflexes would cause inhibition of an indicator muscle. Testing for foods via AK became more common in 1982 with the advent of a test kit of powdered food put out by Dr Walter Schmitt in 1982 of 28 foods. According to Dr. John Schmitt[2] by 1982 patients that didn't respond to traditional AK were tested on these foods and elimination often brought symptom resolution along with stimulating the Chapmans reflex that negated the positive muscle test.

Discussion

Beginnings: 1969-1981

Goodheart observed that on specific patients stimulating either the gustatory or olfactory with various foods, pollens and other noxious substances would cause inhibition of an indicator muscle. He found a correlation to hypochlorhydria (as shown with a bilateral pectoralis major weakness when tested simultaneously) as well as a deficiency in calcium. Walther shortly afterwards found a temporal bulge cranial fault also correlated with these reactions.[3] As research progressed, hypoadrenia was also found as a possible cause.[3] Hydrochloric acid and calcium were often supplemented as well as doing the necessary structural corrections

Antronex/Histidine 1982-3

In 1983 Dr Walter Schmitt wrote the first ICAK collected paper[4] on food testing. He would take an inhibited muscle and see if it strengthened (became temporarily facilitated) on insalivation of Antronex or weakened on insalivation of histidine. If either test were positive, the food kit above was then used to discern specific foods. This was a positive step in treating but at the same time many false negatives were occurring (patients negative to the screening procedure but positive on some of the foods in the kit) and each progression below minimized these more and brought us better results.

Kinin Mediated: 1986

On a personal level by 1986 I (ML) was suffering from chronic fatigue and attended a Goodheart/Schmitt seminar in the Detroit area. Dr Schmitt presented his new research on "Kinin Mediated Allergies" with an accompanying paper of the same name.[5] At the seminar I was asked to insalivate a sample of cholecystokinin (CCK). Inhibition (weakening) of an indicator muscle was a positive test and represented another indicator of food sensitivity that eluded the histidine/Antronex screening. This was very useful especially with patients experiencing brain fog and fatigue. It was often negated by zinc supplementation as well as Chapmans reflex for the pancreas.

Other Patterns: 1987-89

By 1987 Drs. Schmitt and Lebowitz started teaching together and delving deeply into fungal dysbiosis, food reactions etc. During that time Lebowitz wrote three papers for the International College of Applied Kinesiology (ICAK) that identified three new types of food findings. One[6] was a pattern of positive foods being negated by therapy localization (TL) to the thymus gland and typically also negated by the mineral copper. He also observed another type negated by niacin (often found when epinephrine negated a weak indicator muscle – one step allergy screening paper).[7] On these patients a muscle facilitation response on an inhibited muscle was found on insalivation of epinephrine. Up to this point, most foods observed were causing universal muscle inhibition. Lebowitz observed in 1989 that certain foods would only weaken a specific muscle but not cause universal weakness.[8,9] Even with these 5 methods, it was observed that there were positive foods eluding these screening tests. Food test kits had expanded to approximately 100 foods. Set point technique[10] was added as a technique to "desensitize" the patient to the positive foods along with certain nutrients and correcting dysbiosis to help resolve the issues. As time went on other desensitization techniques such as IRT,[11] master set points[12] etc. were also utilized. Simultaneously Lang was developing an AK technique utilizing the same method of diagnosis but using a form of neurolinguistic programming to desensitize by gently pushing the eyes (4-8 oz. pressure) in the direction that abolished the weakening response of the food as well as either inspiration or expiration (whichever negated the food reaction).[13] Using the gustatory reflex in the above techniques limited the number of items that could be tested each visit plus the actual insalivation could potentially alter the reactions of foods tested after a positive test.

Biomagnetic Testing: 1990-1

Until 1991 only gustatory or olfactory challenge was generally accepted as a valid applied kinesiology method.[14]

In 1991 based on the research of William Philpott MD,[15] Lebowitz experimented with and introduced Biomagnetic testing to the ICAK which is still used today in many AK practices. Philpott had an instrument similar to an ohm meter and he observed that placing a food the patient was sensitive to under the south pole against the patient's body would cause the meter to register while if they were not sensitive to the food, then no change would occur. Lebowitz changed this into an AK procedure.1[6] This involved placing the suspect substance under the south pole of a strong 2x5 magnet up against the person's body and seeing if a strong indicator muscle weakened. The first patient Lebowitz observed this on was a disabled DC with a severe case of depression. Upon testing foods with oral insalivation all foods were negative but when placing the foods under the south pole against the patients head over 30 foods showed a positive reaction. Avoidance of those foods resolved the patient's severe depression, and he was able to return to work after a seven-year hiatus. Besides having less false negatives, other benefits of biomagnetic testing included time savings as well as ease (testing many substances on the tongue was cumbersome and limited in how many you could test in one visit). It was also not addressed how actual insalivation of the substance could alter physiology and subsequent items tested in that visit. As a result, testing 100 foods in one visit only took a few minutes and clinical results would be faster. This technique was shared with various physicians who were able to reproduce the results[17,18] and over time the technique gained much popularity within applied kinesiology.

There were still some variables to consider- one sample of a food may test positive and another negative due to differences in growing conditions, pesticides, herbicides (organic or synthetic), hybridization, etc. It might also change if the food was fresh vs. dried, vs cooked etc. It was also observed that if a patient had been avoiding a food for an undetermined time period often a false negative would occur. It also appeared that testing the substance under the magnet over GV21 or GV27 in general showed the most positive responses and became the initial area to place the food in the Lebowitz protocol. This was realized after much trial and error with experimentation over various reflexes, organs, etc.

Vial Testing: throughout the 1990s

Lebowitz was testing various powdered food vials and seeing if the test results correlated well with homeopathics and various energetic vials of the same substance. For many years he was unable to find a strong correlation. Around the year 2000 he was able to find a company whose methods of making energetic vials was different and they correlated strongly with the actual substance. As a result, he and many others switch to energetic vial testing. This made for faster exam time without losing accuracy. This company no longer exists but there are other reputable companies though it is encouraged to cross test with actual foods to in a sense vet the company you are purchasing from for reliability.

Food Toxins: 2011

Despite the advances above there were still many times where either a) a patient knew they were reactive to a food and we would not get a positive muscle test or 2) a patient would eliminate a certain food or foods and experience a resolution of symptoms despite the fact that the food when muscle tested had never exhibited a positive response. Pondering this, Lebowitz decided to purchase a vial of alpha solanine[19] - a neurotoxin found in the

nightshade family of foods. Using himself as a test subject, another physician found him to weaken on it despite not weakening on any nightshade food vials (tomatoes, potatoes, etc.). On eliminating nightshades from his diet his chronic hip that only had approximately 20 degrees of external rotation now had 90 degrees plus. Using that vial on patients he found 56% of his patients showed an inhibitory response but only 10-15% of individual nightshade foods. He then procured vials of albumin, zein, caffeine, theobromine, paraxanthine, theophylline,[20] lactose, casein and alpha gliadin[21] and found similar findings and clinical improvement when removing the positive substances. Interestingly the alpha-gliadin vial was much more clinically relevant than a vial of gluten. This discovery helped many chronic patients who had plateaued to reach new levels of wellness and is still a major part of what has been come to be known as the Lebowitz Protocol.[22] Over time oxalic acid[23] vials as well as a lectin[24] vial and a histamine vial were added to "food toxin screening. As mentioned earlier, some of these vials tested positive much more frequently when specific muscles were used. This was first observed a few years later. Most important was using the peroneus muscle for oxalates and the psoas for histamine. Many of the other food toxins were more sensitive to using a pectoralis (could be sternal and or clavicular division) as opposed to a regular indicator muscle. This is similar to findings of Schmitt and Lebowitz back in the late 1980's where they would put patients into either forward or backward "C" curves to elicit more findings[25] in the sense that we needed to "stress" the patient to bring out findings.

Hypertonic Reactions: 2011

For some time Lebowitz was looking into hypertonic muscle reactions[26] where exposure to a substance (food, nutrient, etc.) would cause an indicator muscle to have a hypertonic reaction (muscle would no longer weaken with approximating of its spindle cells). It was observed that some patients (especially younger ones) instead of showing inhibition of an indicator muscle, the muscle would become hypertonic as a positive food reaction. This was more common with foods containing caffeine and other stimulants but in some patients most of their reactions were hypertonic so we began testing for both inhibition and hypertonic reactions.[22] It was also observed that when testing foods that hypertonic reactions would turn into inhibition reactions if you continued to place the food under the magnet and leave it there for approximately 5-10 seconds.

Present day

At this stage we find there are a number of ways a food may exhibit a positive reaction a) weakening a facilitated indicator muscle b) causing a facilitated indicator muscle to exhibit hypertonicity, c) an indicator muscle will only show a or b when the substance is placed under the magnet over an area of complaint or dysfunction d) Placing the substance over an area you just "normalized" via an adjustment, manual stimulation, acupuncture etc., will cause recidivism of the finding.

Testing may still bring some false negatives:

If the patient hasn't eaten the food for a while oftentimes a false negative will occur on testing

In very complex patients, vials may be negative while if the patient brings in the actual food it will test positive. As stated before, this could be due to sprays, harvesting and soil conditions, hybridization, etc.

Some energetic vials did not use the real substance as a starting material which could affect the test.

Vials in a base with alcohol, sugar or charcoal could yield aberrant results if the patient is either sensitive to the substance or needs it (charcoal can be therapeutic and a sugar base can give a false negative if the patient is hypoglycemic at the time of testing, alcohol may give a false positive in patients with “live stress” or inflammation).

Once the foods are discerned there are many ways to negate the positive tests and many fine AK physicians have developed techniques to help these but there are a few things to remember:

You can’t “fix” what you can’t find so using some of the enhanced techniques above can increase the number of findings

Food reactions are often secondary to dysbiosis, deficiencies, toxicities, genetics or inherited factors, emotional factors, etc. If this is the case, results will be either short lived or findings could change with different foods found on subsequent visits.

As the world becomes more toxic in multiple ways and all types of stress increase, we are finding that elimination of the foods is needed more commonly and the patients, as a generalization aren’t as responsive to the “desensitization” techniques as was common in the past.

If a patient avoids a food for a period of time and then comes back in to see if it is time to reintroduce it, if they have been 100% avoiding it a false negative is common. You can have to patient reintroduce a portion of the food for 2 days in row before coming in. They might, if it is still an issue, have an observable recurrence of a symptom or you will find it on your retest. If many foods are involved this can be a little more complicated.

Conclusion

Using AK to access negative food reactions is an invaluable tool. Just simply testing a food for inhibition of an indicator muscle probably brings a false negative reaction over 50% of the time. Enhanced techniques as stated above can greatly increase findings and results. A history of the evolution is presented for the physician to understand how we got to where we are today.

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Applied Kinesiology and the Factors and Biomechanics of Androgenic Alopecia: A Corrigible Look

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Abstract

Baldness and balding are epidemic problems that affect both sexes at varying ages on almost all levels of being. The subject is a large one. Understanding and correction require the identification of balding types by category, among many things. These include categories related to stress, improper nutrition, hormonal imbalances, and, of late, gravity and COVID-19-a type of stress related hair loss. The types of baldness by common diagnostic name include Androgenic alopecia which includes male pattern balding (MPB), female pattern balding (FPB), and receding hairline. Then other types such as alopecia universalis, alopecia areata, traction alopecia, and central centrifugal cicatricial alopecia (CCCA), In the last few years, we have identified causes of hair loss related to dysfunction of the stress system by way of adrenal, thyroid, and pituitary dysfunction, nutrient deficiencies, chemical toxicity and poisoning of the scalp and hair follicles, emotional trauma, and the focus of this paper, - gradual biomechanical aberrations. It appears that when gradual biomechanical aberrations are identified and addressed, and if stem cells in the hair follicle are properly stimulated and supported, hair regrowth, and the prevention of androgenic alopecia may be possible. Applied Kinesiology tools applied to the cranium and hair follicle allow the identification of causes and the creation of a care plan with a foundation in the restoration of sound cranial biomechanics and function. This means that for the management of improved scalp and hair health to be successful and long lasting, it must include correction and basic maintenance of the biomechanical structures of the cranial vault, osteo-myofascial system, and remaining stomatognathic system.

Key Indexing Terms

Chiropractic, Applied Kinesiology, Male Pattern Balding, Androgenic Alopecia, Hair Follicle Stem Cells, Cranial Fault, Subluxation, Manual Muscle Text, MMT, Myofascial System, Functional Medicine, Galea, Occipitofrontalis, Molding

Introduction

Unanticipated hair loss affects untold numbers of the population of the world and the USA. There are many causes and types of hair loss. The causes include the more obvious gross nutrient deficiencies, traumatic stress, and then, more ambiguous and incomplete, are the various explanations for male pattern and female pattern baldness. This paper will

orient, arrange perspective, as well as validate work already performed on these two subjects and advance them to a new level not seen before, using tools we already own.

Jargon relating to hair loss

Bone remodeling is the process of bones changing size, shape and sometimes growth, holographic subluxation – is a shape based subluxation that does not match the nervous systems expectation of shape; molding – the process of changing shape of the skull intentionally or not; vellus hair – fine hair; lanugo hair – baby hair; anagen – first stage of hair shaft development and the growth period; catagen – the disruption of the growth process and end of hair shaft growth and nourishment; telogen- the resting phase of a hair follicle; telogen eflluvium – the expulsion of a hair shaft from the follicle; Androgenic alopecia – hair loss based on elevated androgens; miniaturization - thinning of hair so that a weak and fine hair shaft grows; dihydrotestosterone – reduced testosterone; Superficial Musculoaponeurotic system (SMAS) – fascial system in the head and neck that includes the platysma muscle superior to the clavicle rising to the superficial temporal fascia..

Background and Preparation

A web search on the topic of hair loss provides millions of results. From gels to lotions to surgery and implants to wigs, hair is often on many people's minds.

Hair loss affects 80 million Americans, with approximately 60% of men and 40% of women suffering from it. The most prevalent type among the 80 million is androgenic alopecia (AA) (American Academy).

This type includes male and female pattern balding (*Skin Conditions by the Numbers*, n.d.) . Current explanations for male pattern balding follow. In 1987, a seminal study was done on scalp Dihydrotestosterone (DHT) elevation on bald areas of the scalp of men suffering from male pattern balding (MPB). We know that testosterone irreversibly converts to dihydrotestosterone (Caballero).

Testosterone + Alpha 5 Reducatase ---------> DHT

Male pattern balding by convention since 1987 was stated to be related to an elevated level of scalp DHT and scalp 5 alpha reductase, the enzyme that converts testosterone to DHT, which causes "susceptible hair follicles" to produce weak hair shafts and then produce none at all. Blood tests for DHT in one study showed no difference between control and studied subjects. To date, there are no blood tests that accurately correlate blood androgen levels with balding (Donovan 2014).

<u>Steroids</u>

The common agreement and background information is that androgens have been determined to target specific hair follicles that have more receptors for 5-alpha reductase and DHT, and more of these are located in the frontal lobe than the occipital (Price 2003). This is the area of male pattern baldness. These androgens are well documented to also be responsible for secondary sex characteristics such as the following in men:

- Pronounced body hair characteristics (beard, chest, etc.) and is usually coarser.

- Heavy musculature
- Angular features (i.e. square jaw, triangular mid region)
- Narrow hips
- Muscular pectorals (chest)
- Overall, there is less fat tissue.
- Deeper voice

In women, these secondary sex characteristics include:

- Less pronounced body hair characteristics (mostly in pubic region, hair all over is usually finer)
- Lighter musculature
- Rounded features (i.e. softer facial features, hourglass mid-region)
- Wider hips (for child bearing)
- More pronounced breasts with more fatty tissue
- More fat tissue overall
- Higher voice

The following androgens also play a role in hair growth:

Testosterone creates hair growth and secondary sex characteristics that differentiate men from women.

Cortisol is a stress and everyday adrenal hormone that is elevated during stressful times and is catabolic to all tissues. Chronic exposure is also catabolic and gene expression in many cells can change from an acute exposure as an anti-inflammatory to a chronic exposure that stimulates nuclear genomic increases in levels of NfKb, making the inflammation epigenetic or a chronic product of genetic expression instead of acute local irritation.

Cortisol may also work in a feedback loop with DHT. Elevated cortisol levels cause local increases in DHT levels as these are meant to lower the cortisol (Toufexis).

Gravity

Another hypothesis proposed in a 2008 paper is that hair loss is a direct product of the forces of gravity. According to this new theory, the pressure created by the weight of the scalp is the cause of male pattern baldness (USTUMER 2008).

Genetics

Genes are thought to play a role in AA. The problem in each person is thought to be polygenic and a perfect storm between endocrine imbalances and polygenetic factors. Two genes have been identified. We do know that epigentic factors can modify genetic expression. Improper nutrition has been put forth as a major factor in some types of baldness; not solely in the case of AA.

Phases of hair growth

At birth, or in an anorexic adult, we see lanugo hair. This hair eventually falls out after birth and is replaced by fine hair called vellus hair. Vellus hair is present until puberty. After that, a terminal or adult hair replaces it.

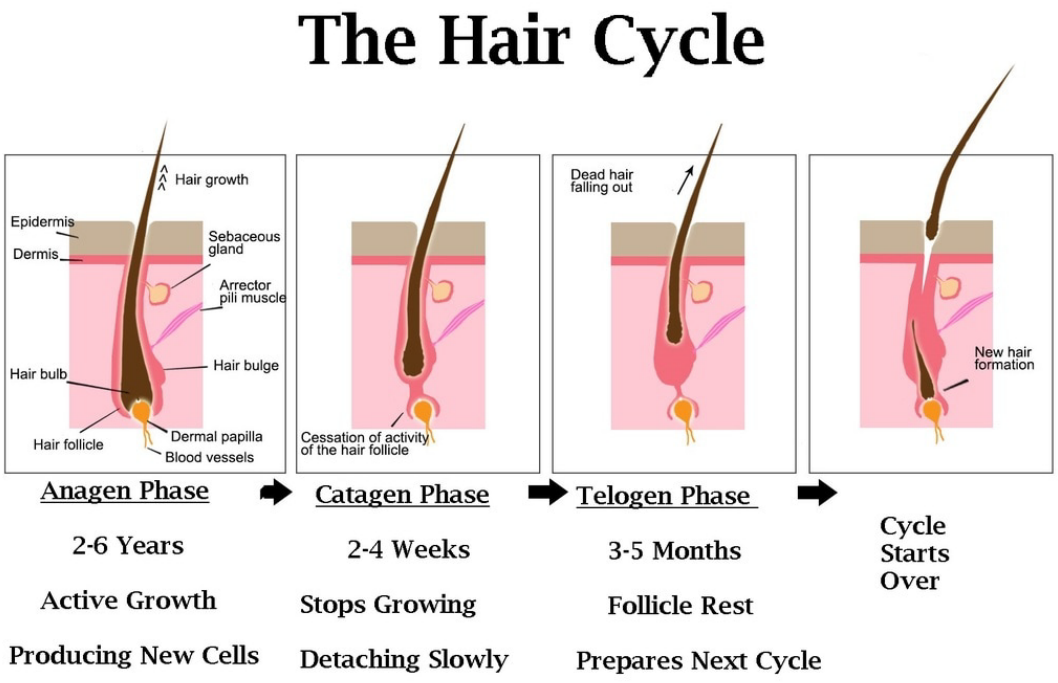
Parts of the scalp hair unit

Hair is composed of a shaft – "the hair," which grows from a follicle that has germ cells at the bottom of the follicle that receive blood from blood vessels in the underlying tissue.

Phases of terminal hair growth

Terminal hair growth occurs in stages of follicle and shaft activity. Four of the most basic stages are listed below (Kiesewetter 1990);

- Anagen–lasts 2-7 years and represents the growth of the hair shaft and how long it can become.
- Catagen -the hair shaft shrinks and detaches from its nutrient source – it has a 10-day duration and can be gradual or acute and is impacted by cortisol.
- Telogen - This is the unit's resting phase. The shaft of hair is now detached but still in the follicle. The stem cells have made or are resting but can make a new hair shaft. At any given time, 10-15% of the hair is in this stage. This can last three months or longer.
- Exogen: This is still in the resting phase, but new hair will begin to grow, and the detached hair will leave the follicle and be shed.



(*The Hair Growth Cycle*, n.d.)

Current treatments for androgenic alopecia

The current treatments for hair loss include:

1. Wigs/ barber's synthetic creations.
2. Surgery –via various implant methods with an unpublished success rates.
3. Minoxidil, Finesteride or alpha-5 reductase blocker medications.
4. Low level laser therapy to increase circulation.

A review of the skull, scalp, cranial blood supply, myology

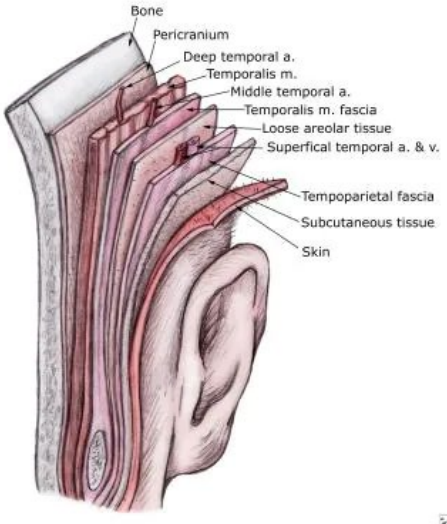
The human cranium is composed of 29 separate flexible membranous bones, postulated and objectively measured to have movement at the joints or sutures between them. These movements are governed by factors such as muscular contraction, attachments and reciprocal tension membrane forces from inside the skull as well as fascial forces from local and remote locations of the human muscle and skeletal system.

Depending on how they are counted, there are about 30 muscles on each side of the human skull. There are 20 muscles in the neck that directly attach to the skull and the cranio-cervical, superficial and deep fascia, as well as the superficial muscular aponeurotic system (SMAS) (Whitney 2021). The fascial ties are common to the head, neck, and the entire body as one connected unit. Another fascial connection is the aponeurosis that covers the calvarium called the galea aporneurotica; this flat tendon is the insertion of two opposing muscles—the frontalis and occipitalis, also known as the occipitofrontalis. On either side of the skull is a location for the temporalis muscle.

Traveling through this vast network of muscles, bones, and fascia are the arteries that supply these structures. The scalp's blood supply arrives via the superficial temporal artery, a branch of the external and internal carotid arteries. The carotid arteries were named by Galen based on the Greek word for stupor, as he found when you compressed them, one could go into a stupor.

The scalp, which tops the calvarium, is a network of adipose tissue, skin, and hair follicles and sits on top of the galea aponeurotica.

The scalp layers from bone calvarium to the skin and hair progress as illustrated below;



Discussion

A novel pathogenisis model for male pattern baldness

The male pattern balding process from start to finish takes approximately 5-25 years to complete. The process is not an acute problem but chronic and progressive for most; stress chronically causes disease, but it is also episodic for most. The hair loss pathogenesis is from terminal hair to vellus to lanugo to bald and then dormancy.

Working backwards from a bald scalp to a healthy scalp with a full understanding of wholism, we see that the end result of hair loss is a dormant or weakly producing hair shaft from a hair follicle that still has stem cells in it.

The "swan song" of a dying hair follicle

The dormant hair follicle once had a miniaturized hair shaft; we called this vellus hair. The hair follicle reacted to elevations of cortisol, and the cortisol caused the hair follicle to be weaker and smaller. This in turn caused a reactive elevation of DHT, which is used by the body to lower cortisol. Lower cortisol levels allow the hair follicle to strengthen or start a new but this does not happen and so elevation of DHT is the swan song of catabolized hair follicles, not the root cause of hair loss-the small hair, now in catagen and doomed, will shed eventually in the exogen phase. Perhaps more rapidly if the levels of cortisol do not decrease. Now the question is, what increased the cortisol?

What triggered catagen – cortisol

We know that elevations of cortisol, either acute or chronic, can throw a hair follicle into catagen leading to hair loss. Stress, shock or trauma do this acutely but usually that happens with the loss of patches of the scalp.

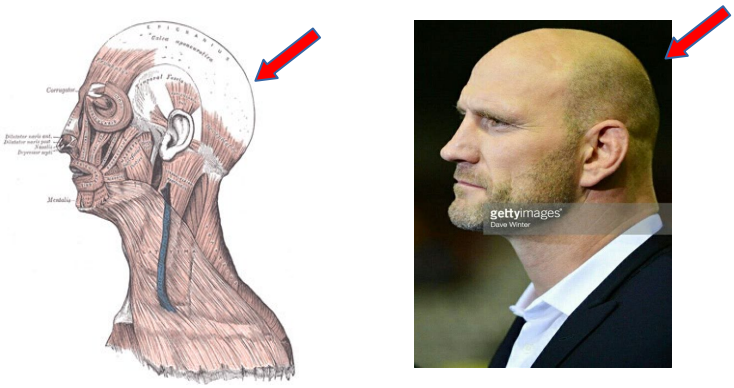
Chronic stress causes the adrenal glands to increase their cortisol output to deal with various types of stress. One such type is structural stress. Structural stresses may present as abnormal cranial bone movement, myofascial dysfunctions and adhesions. Then changes in the shape of bones from years of cranial bone remodeling create a holographic mismatch, leading to increased stress and insidious increases in cortisol, decreases in blood supply and functional hypoxia for the scalp via the superior temporal artery. As this process insidiously happens, silently and slowly, so too does the loss of the terminal hair, propelling it into catagen status (S 2020).

The causes of cortisol excesses are best reserved for future works but includes non-optimal interactions with, structural, chemical, thermal, emotional and geopathic and electromagnetic stressors. Chronic exposure may increase cortisol levels.

Hair follicle miniaturization is complicatedly the product of chronic excess cortisol exposure and slow deprivation of oxygen and nutrients in the case of androgenic alopecia (AA) (Toufexis 2012).

The years of hypoxia also cause muscle dysfunction, which creates fascial distortions that lead to chronic but low force hypertonicity and imbalance; and as Dr. George Goodheart used to say, "Muscles do move bones." And so, Wolf's law takes effect to make the problem chronic. According to this law in part, a bone in a healthy animal will adapt to the load under which it is placed. Hence, we see actual molding of the skull (Wolff 1892).

So, we start to get growth and shape changes in the cranial bones as a result of persistence and consistent myofascial tension, imbalance, and muscle dysfunction. These osseous changes cause changes in the tension of the galea which cause further fascial distortions and further decreases in blood supply and resultant diminished nutrition to the scalp. As a result, cortisol secretion is stimulated, miniaturization of more follicles occurs, and DHT increases to neutralize the cortisol, and we start over again, year after year; thinner and thinner. The "gravity theory" too has been in play all the while. Except the pulling is the myofascial system, not the gravitational forces of the planet. The end product is loss of hair, male and female pattern balding, and distortion of the shape of the cranial bones (Graham Kennedy, 2013). Oddly and overlooked is the fact that the balding of male pattern balding is almost in pattern with galea aponeurotica in many individuals.



The illustration compared to the photo demonstrates galeaic hairloss in the male pattern balding fashion.

Well before the above scenario was probable, cranial imbalances and deviations from normal cranial respiratory movement were an issue. This caused a decrease in blood support through increases in tension and myofascial dysfunction from chronic and acute stress.

These structural stresses that affect cranial movement and scalp mobility and nourishment may occur anywhere in the human body, but most commonly in the neck and shoulders.

Correction

Now that we have identified the pathogenesis. Where do we start on the correction? At the end of course! In any situation where damage is being done you need to:

1. Get to a safe place – in this case we need to identify areas of cortisol stimulation and start addressing them. This may be very individual. (i.e. overwork, overtraining, emotional situations unhandled, etc.)
2. Stop the damage – The hair follicle has been deprived of vital blood, oxygen and nutrients. It may be using anaerobic glycolysis to survive. The end product is lactic acid. Some people notice this because of soreness, itching and burning. That means that NAD is being used for this process so testing for Niacin not niacinamide is a first step to stopping damage.
3. Next to control damage we must discourage the local testosterone conversion by 5 alpha reductases. We can use frequently applied essential oils and topicals for this.
4. Check each of the 29 bones of the cranium for holographic subluxations.
5. Check for cranial faults and sutural faults.
6. Release the frontooccipitalis muscles.
7. Evaluate all muscles of the skull for proprioceptive dysfunction.
8. Evaluate the entire somatagnathic system.
9. Evaluate fascial balance and for adhesions using the midline scalp pull test.
10. General fascial release per fulford may be achieved by using percussion at the following locations:
    - left and right deltoid tuborosities
    - left fibular head, C2, L2 and S2
11. Evaluate and release the sacrum and coccyx.

Now that you have addressed the various structural concerns all of the following nutrient areas need to be assessed and corrected:

1. Scalp cortisol challenge
2. Zinc status
3. B3
4. Methylation status – homocysteine protocol (Schmidt)
5. Essential fatty acid blood spot profile
6. Vital amino acids needs must be assessed and met.
7. Cranial remolding exercises must be performed three times daily. Cranial molding exercise may cause cranial faults so the cranium should be re-evaluated to ensure no harm has been done. These exercises must be prescribed based on the holographic imbalance in each cranial bone identified (e.g. parietal bulge is most common)
8. Calvarial muscles must be evaluated for any types of dysfunction
9. Each muscle of the facial skull muscle be evaluated for dysfunction
10. Topical and internal use herbs that improve blood supply are adjunctive and needed.
11. Adrenal function must be assessed and addressed per the AK Synopsis.

Conclusion

Thirty-five years prior to this paper, a discovery was made that described, not the cause but the end result of one process of hair loss. Dihydrotesterone elevations are the end

result and hair unit “swan song” from years of blood deprivation and all of the life-embodying factors that it carries to the tissues and cells of the scalp. Applied kinesiology allows the pieces of this puzzle to be found and arranged in a fashion that allows us to see the big picture of the puzzle completed. To restore full normal function to the hair unit requires a panopoly of factors for restoration and stabilization of a weak and ailing hair units and the scalp they reside in. But this can be done by those willing to put forth a little work and effort.

Acknowledgments are made to Harmonic Energetics, Integrated Healthcare of Montclair LLC, and the ICAK.

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Applied Kinesiology Management of Dysmenorrhea: Chiropractic Case Report

Karly Abel Kantarevic, D.C.

Abstract

Dysmenorrhea is a common gynecological health issue with symptoms impacting more than half of all menstruating peoples, causing severe pain and interfering with activities of daily living. This case report demonstrates the value and effectiveness of Applied Kinesiology assessment and procedures for noninvasive, simple, nonsurgical management of primary dysmenorrhea. Utilizing a specific technique called Pituitary Pump technique, along with chiropractic adjustments and targeted herbs/supplements a young patient was able to resolve severe primary dysmenorrhea quickly and long term address the underlying hormone imbalances. To date the patient reports regular cycles without menstrual cramping, radiation of pain, hip or back pain and is not using any supplements or medications.

Key Indexing Terms

Dysmenorrhea, Hormone Imbalance, Pituitary Pump Technique, Applied Kinesiology, Chiropractic

Introduction

Dysmenorrhea is defined as painful menstruation usually occurring just prior to or during the first 24-72 hours of a menstrual period.[1] More than half of all menstruating women have mild pain for 1 to 2 days each month. The prevalence of more severe pain is highly variable (15.8-89.5%) potentially due to the use of oral contraceptives or nonsteroidal anti-inflammatory drugs (NSAIDs).[2] Dysmenorrhea is one of the most common gynecological health issues and is known to interfere with daily activities including absenteeism from school or work due to severity.[3]

There are two types of dysmenorrhea. Primary dysmenorrhea is not associated with pelvic pathology. It is most common in adolescence near menarche and less common with increasing age. Secondary dysmenorrhea is caused by another medical condition such as endometriosis or pelvic inflammatory disease. It usually begins later in life and tends to last the duration of menstruation.[4] Either type of dysmenorrhea may experience low back pain, weakness, fainting, pain radiating down the legs, dizziness, nausea, diarrhea, headache cramping, and/or vomiting. While any woman can develop dysmenorrhea those who smoke cigarettes, drink alcohol (especially during their period), are overweight, have a heavy menstrual flow, a positive family history, or had an earlier age of menarche are at an increased risk.[2,4]

Treatment is aimed at providing symptomatic relief and/or assessing the underlying pathology. The first line of treatment in traditional approaches is in reducing pain through NSAIDs or birth control (pills, IUD, patch, etc.). Other therapies are not well studied however, diet changes, exercise, acupuncture, applying heat, and relaxation techniques have been recommended. In severe cases surgical options are considered such as uterine ablation or hysterectomy.[2,4] Due to worldwide prevalence and significant impact on daily life other therapies for dysmenorrhea should be studied and noted.

The purpose of this study is to demonstrate nonsurgical noninvasive management of primary dysmenorrhea with Applied Kinesiology (AK) procedures.

Clinical Features

A 17-year-old female presented with severe primary dysmenorrhea, cystic acne covering most of the face, chest and back, as well as menstrual irregularity. Menstruation was heavy occurring infrequently averaging 2-3 months in between cycles and lasting anywhere from 5-11 days. The patient noted that a day or two before menstruation occurred stabbing low back and hip pain would radiate down the legs causing numbness into the upper thighs. These symptoms would continue several days into menstruation and disrupted daily life especially hobbies, sports, and leisure which the patient rated 4/10 (with 10 being totally unable to function) on the General Pain Disability Index Questionnaire. The patient had not found relief, medical help, or assistance through 4 plus years of symptoms. She was not regularly using any medications or supplements, she did try NSAIDs and a heating pad as needed for symptoms without much relief.

Upon initial examination, using AK Manual Muscle Testing (MMT), bilateral gluteus maximus, gluteus medius, piriformis, sartorius & gracilis muscles were inhibited. The lateral shear cranial fault, upper cervical fixation as well as spinal subluxations- C2, C6, T4, T7, T10, L3, L5- were noted. Touching the circulation sex meridian (CX 9) inhibited any previously strong indicator muscle for the patient. Energetic testing of separate vials estradiol also inhibited the patient. Using any of the inhibited endocrine related muscle listed above and touching the pituitary neurolymphatic (NL) (located at the glabella) strengthened the muscle.

Management and Outcomes

The goal for the treatment plan was to alleviate primary dysmenorrhea symptoms as quickly as possible and longer term address the underlying hormone imbalance. This means addressing issues found during the initial examination that could improve many areas of the body at a time. The first treatment began with the Pituitary Pump technique.[5]

<u>Pituitary Pump Technique:</u>

A. Diagnosis
    a. Find a weak endocrine related muscle
    b. TL to pituitary NL at glabella
    c. If weak endocrine muscle goes strong (or a strong muscle goes weak) pituitary involvement
B. Treatment

a. Challenge mastoids bilaterally and simultaneously posterior to anterior (P-A) test previously strong muscle, if muscle goes weak positive need for Pituitary Pump
b. Phase positive challenge to respiration (should be inspiration)
c. Step 1- as patient inhales they will dorsiflex their feet as they exhale, they will plantarflex their feet bilaterally for 60-90 seconds as the doctor presses on the patient's mastoid processes simultaneously and bilaterally with about 8 oz pressure
d. Step 2 - rub glabella NL for 30-60 seconds
e. Step 3 - repeat step 1 for 30-60 seconds as needed
f. Retest to verify correction

This technique completely changed how the patient's body was testing through AK MMT. Immediately after verifying correction, bilateral gluteus maximus, gluteus medius, piriformis, sartorius & gracilis muscles were all very strong. The lateral shear cranial fault, upper cervical fixation as well as spinal subluxations- C6, T4, T7, T10- all cleared and no longer therapy localized (TL) or inhibited the patient. TL to the circulation sex meridian (CX 9) did not inhibit any muscle for the patient. C2, L3 & L5 were adjusted via Arthrostim. Recommendations for herb and supplements were given, Endo Supreme by Supreme Nutrition (2 per day) and Estrovite by Apex Energetics (1 per day), until patient was next seen.

Within a week patient reported no hip or low back pain and that skin was noticeably improving. Treatment continued seeing the patient 1 time per week for 4 weeks- the Pituitary Pump technique was done at the third and fifth visit, L3 was adjusted at each visit, gluteus medius was the only muscle continually inhibited throughout this time.

Patient had first menstrual cycle during our care, 8 weeks from initial exam, with cramping that did not radiate into the legs, no hip pain or low backstabbing, however, skin breakouts continued. Iron levels were checked due to the heavy bleeding and the patient was diagnosed with mild anemia, for this an iron supplement, Hemevite by Apex Energetics, was added and Endo Supreme was increased to 3 per day 1 week before and the week during menstruation- otherwise only 1 Endo Supreme per day throughout the rest of the entire cycle length. The patient's second menstrual cycle during our care was 28 days long, menstruation was 5 days in length, cramping occurred but was mild and did not radiate, skin break outs flared. Cycles were monthly from this menstrual period on and within 3.5 months using Pituitary Pump technique one more time the patient no longer reported radiation of stabbing low back and hip pain with menstruation and mild to no cramping.

Since menstrual cycles became regular about 3 months into care, treatments were scheduled a few days before expected menstruation. The Pituitary Pump technique was checked at each treatment and used a total of 6 more times within the past 4 years. Estrovite was stopped within the first 6 months. Hemevite was stopped when no longer testing as anemic which was within the first year of care. The patient did 3 months of Accutane within the first 6 months of our care together- for her widespread cystic acne. After stopping Accutane, the patient's skin has remained clear except for occasional blackheads.

Patient did continue Endo Supreme 1 per day daily, increasing to 2 per day 1 week before and after her cycle for 18 months in total.

For the past year to present the patient is not using any medications or supplements. She reports 28–31-day cycles with 3-5 days menstruation that is light to regular without menstrual cramping, no radiation of pain, no stabbing low back or hip pain, and her skin is clear with occasional blackheads.

Discussion

With dysmenorrhea being one of the most common gynecological health issues impacting more than half of all menstruating peoples, causing absenteeism, severe pain and interfering with activities of daily living, it is important to further study therapies to improve or resolve these health complaints. Therapies like AK should be noted as being noninvasive, nonsurgical, and cost effective. AK procedures and methods are used to treat and manage a wide range of conditions and biomechanical concerns. In this case, utilizing the Pituitary Pump technique helped a young patient resolve severe primary dysmenorrhea and even after several years continue to have regular, non-painful, healthy menstrual cycles without any medications, supplements, or NSAIDs.

Presently, conventional treatment is aimed at providing symptomatic relief and/or assessing the underlying pathology. After potential pathology has been assessed for and addressed, use of NSAIDs and or birth control (pills, IUD, patch, etc.) are routinely used to address symptoms. Neither of which help resolve the underlying hormonal imbalance and neuroendocrine dysfunction causing the complaints, and both have been linked to severe long term health issues. Chronic NSAID use has been linked with cardiovascular, cerebrovascular, gastrointestinal, and renal adverse effects.[6] Although they may reduce pain symptoms for this specific case report NSAIDs were not addressing the patients' complaints, and definitely not addressing the underlying neuroendocrine issues causing her symptoms. Birth control pills are commonly linked to bleeding between periods, headaches, sore breasts, and nausea- nearly identical to dysmenorrhea symptoms and do not resolve the underlying root cause for the symptoms.[7]

The herbs and supplements recommended were low to no adverse risk and short to moderate term use, aiming to resolve the underlying health issues. There is growing medical literature for the awareness of DIM supplementation to support health estrogen levels (Estrovite by Apex Energetics is a DIM supplement used). Typically, a minimum of 30 days is needed for DIM use to notice results, most use it for 6-12 months, there are no significant adverse side effects.

With painful menstruation it is common to also have heavy menstruation, with heavy menstruation anemia could be a factor. Interesting to note, anemia is a risk factor for dysmenorrhea and another reason this patient had a blood draw and iron supplementation recommended. The Endo Supreme from Supreme Nutrition is pure Pfaffia paniculate or suma. Suma is traditionally used for PMS, hypoadrenia, anemia, and an overall endocrine

adaptogen. It typically takes about 1 month for results and there are no known contraindications or side effects.

The Pituitary Pump technique offers a noninvasive approach to address the underlying neuroendocrine imbalances. The technique needed to be done a few times before seeing lasting and significant results. The need for this technique was checked at each appointment until cycles were regular and then assessed for monthly around expected menstruation. This checking in and assessing was a vital part of long-term success. With the above measures not only did patient symptoms resolve, but the underlying cause of irregular menstrual cycles was also addressed in noninvasive, nonsurgical, cost effective, and long-term safety methods. Adding clinical value and importance to the area of other therapies not well studied for primary dysmenorrhea.

Conclusion

With the prevalence and severity of dysmenorrhea it is important to further study therapies to improve or resolve these health concerns. Therapies like AK should be noted as being noninvasive, nonsurgical, and cost effective. This case report demonstrates how utilizing the Pituitary Pump technique, chiropractic adjusting, and targeted herbs/supplements helped a young patient resolve severe primary dysmenorrhea long term.

The treatment plan goal was to alleviate primary dysmenorrhea symptoms as quickly as possible and longer term address the underlying hormone imbalance. Within the first 3.5 months of care utilizing the Pituitary Pump technique a few times and targeted herbs/supplements the patient had regular cycles, with mild to no menstrual cramping, and without hip or low back stabbing. She has remained under care for the past 4 years. For the past year to present the patient is not using any medications or supplements with continued resolution of symptoms. She reports 28–31-day cycles with 3-5 days menstruation that is light to regular without menstrual cramping, no radiation of pain, no stabbing low back or hip pain, and her skin is clear with occasional blackheads.

This case demonstrates the value and effectiveness of Applied Kinesiology methods and procedure for noninvasive, simple, nonsurgical management of primary dysmenorrhea.

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Ergogenic and Specialty Nutrients and Technical Protocols for Athletes

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Abstract

The author discusses specific nutrients to consider for athletic training and performance. Professional Applied Kinesiology (PAK) methods for nutrient selection are presented based on the authors experience.

Key Indexing Terms
Professional Applied Kinesiology, Sports Nutrition. Applied Kinesiology

Introduction
Frank nutrient deficiencies are common in the athletic population. Certain specialty nutrients can also be used regardless of deficiency to improve performance, conditioning, and recovery. The potential benefit can be screened with very simple Manual Muscle testing (MMT) methods.

Discussion

The author prefers to compartmentalize sports nutrients into relevant categories:

- General Category Nutrients
- Tissue strength factors
- Recovery and ATP support
- Hydration dynamics
- Pre-performance nutrients
- Muscle/strength Gain factors
- Endurance nutrients
- Concussion/ TBI support
- Sprain/strain injury support
- Hormone and Stress support

General Category (GC) Nutrients

- **Apply to all other categories** and provide broad nutrient support
- High-grade, activated Multi-vitamin-mineral daily
- Low-moderate dose whole food multi-mineral daily
    - preferably mineral-amino acid complex form such as mineral-glycinates or threonates

- Perfect Aminos approximately ~10 grams daily[1]
- Vitamin D
    - 2500-8,000 IUs/day depending on age
    - ~10,000 IUs/day for flu prevention
    - Blood levels 25-oh cholecalciferol optimally 50-100 ng/ml[2]
- Coenzyme Q10 - 200mg/day accelerates recovery, ATP support
- Omega 3 fish oil - 3g/day or more
    - Increase and add Black Currant Seed oil if sore day after training, competition, or injury
- Quality sleep
    - Avoid back-lit devices like cellphones an hour before bedtime
    - GC nutrients important here

Tissue Strength Factors

- Liposomal Phosphatidyl Choline:
    - Welds the lipid bilayer of human cells together to make the cell membranes
    - Cell membrane strength prolongs cell lifespan and preserves raw materials the body would otherwise use to replace an injured or worn cell
    - Heart, Liver, and cognitive support[3]
- Iodine
    - Raises cellular voltage for normal cell respiration, growth, division
    - Endocrine and exocrine essential secretory nutrient
    - Facilitates fat burning for max aerobic function[4]
- Omega 3 fish oil
    - High dose, phospholipid bound like high grade krill oil
    - Incorporated into cell membrane lipid bilayer
    - Improves membrane health and fluidity and, thus, mitochondrial energy production and cell lifespan
    - Anti-inflammatory, circulation, immune, cognitive, and neuron health
- Glucosamine and Chondroitan Sulfate (GS/CS), whole food Vit C, organic copper
    - Building blocks for connective tissue repair and regeneration

Recovery and ATP Support[5]

- Post event:
    - One teaspoon sodium bicarb or tri-salts in liter of water immediately after
    - Walking, cycling cool down 20 minutes
    - Cold water immersion
    - High sodium foods like Himalayan or Celtic sea salt with continual hydration
    - Balanced meal of good fats, low-moderate protein and complex carbs
    - Spleen 21 and Pericardium 6 massage 3x/day for at least 30 seconds to accelerate recovery of metabolic/organ balance

- Alpha lipoic acid, acetyl L-carnitine, Mg, Mn, COQ10, D-Ribose with GC nutrients for mitochondria support/ATP production
- Female endurance athletes: whole food Iron daily[6]
- NRF2 stimulation daily for anti-oxidant support[7]
- Mild hyperbaric chamber
    - 30 minutes to an hour/day

Hydration Dynamics

- Ph balanced, negatively charged filtered water like Penta
    - Can add healthy food-based electrolyte powder[1]
    - 4-6 ounces numerous times through the day
    - Berry teas, bone broth and high sodium soups
    - L-glutamine[5]
    - Avoid hyper-alkaline water fad[8]
- Deep tissue work – hydration is partly about exchanging water reserves in the fascia for new water[9]
- Kidney Yin reserves[10, 11]
    - Physical indicators of Yin depletion:
        - Left psoas chronic inhibition
        - Red tongue with centerline crack and/or coating completely or partially peeled off (geographic tongue)
        - Dryness – lungs, throat, mouth, lips, eyes, urogenitals
        - Decreased tolerance of heat and mental stress
- Kidney tissue, organotherapy, TCM KI Yin tonics may be needed
- Whole food Vitamin A or E
- Acupoints – muscle test and treat points which strengthen any muscle inhibition, have patient massage throughout the week
- Kidney 1 – Wood point, “Gushing Spring/ Bubbling Well”
    - Important grounding point gathers healthy negative electrons from Earth when in contact with natural surfaces
- KI 3 - Source point, “Supreme Stream”
- KI 7 – Tonification point, “Returning Current”
- BL 23- “Kidney Transporter”, CV 4- “Origin Pass”, GV 4- “Life Gate”, LI 4- “Joining Valley”

Pre-performance nutrients[5]

- GC nutrients
- Buffered creatine, medium chain triglycerides 30 min pre-competition
- Individual ATP nutrients listed above that strengthen any AK MMT indicators
- Electrolyte/Energy drink as needed
    - No more than 6-8% carbohydrate
- Deep breathing techniques for anxiety, jitters, stress
    - take twice as long to exhale as it takes to inhale

    - example: inhale for 3-4 secs, then exhale for 6-8 secs, 5-10 min total

Muscle/strength Gain

- GC nutrients
- Perfect Aminos ~10g/day[1]
- Buffered Creatine ~5g/day[5]
- Hydroxymethylbutyrate (HMB), RNA - if AK MMT indicated

Endurance essentials[5]

- GC
- Complete vitamin E: tocopherols and tocotrienols
- Female athletes: whole food Iron ~10 mg daily
    - Keep serum ferritin optimally above 60[6]
- NRF2 stimulation- strong anti-oxidant action[7]
- Extra vit B2, D-ribose - if AK MMT indicated
- Broad spectrum medium chain triglycerides[12]
    - Can use before or during competition in cranberry juice or coconut water, start low-dose initially
    - Add buffered creatine if event involves multiple high intensity efforts

Concussion/Traumatic brain injury support[5, 7, 13]

Patient should be tested for B-vitamin genetic poly-morphisms and supplement any deficiencies, or if AK MMT indicated

- Especially P5P, Pantethine, 5-MTHF
    - Unburdens mesencephalon function to prevent brain neuron threshold overstimulation
- B1 facilitates blood-brain barrier
    - Consider thiamine cocarboxylase, thiamine pyrophosphate, thiamine triphosphate, or the fat-soluble Benfotiamine
- PB125 – strong NRF2 stimulation, anti-inflammatory, adaptogenic
- Omega 3 fish oil – anti-inflammatory, cools overloaded neurons
- Vit D (25-OH cholecalciferol) blood levels 50-100 ng/ml, high grade CBD oil
    - Help to prevent chronic auto-immune encephalitis
- Intestinal permeability support like L-Glutamine
    - Fortifies gut-brain axis
    - Urine Indican test for gut bacteria excess[14]
        - If positive test SI, LI Alarm points for oregano oil, caprylic acid, artemisia, olive leaf extract and probiotics
- Decrease stress
    - Including radio-frequency pollution from cell phones and smart meters[15]
        - Cell-phone neutralizer, RF-shielding paint or Faraday fabric shielding, especially for sleeping areas

    - Music, esp classical is encouraged
    - Use specific Adaptogens that strengthen AK MMT

Sprain /Strain injury support

Acute Phase:

- Proteolytic enzymes 3-4x/day between meals to digest swelling
- Anti-inlammatories such as Omega 3 fish oil minimum 3g/day for pain
- Magnesium as needed for muscle spasm
- Magnet, laser therapy, and/or fascial flush with gravity over injured area to promote circulation and local fluid exchange
- Continue GC

Subacute phase:

- Same but discontinue enzymes once swelling is reduced
- Manganese (glycerophosphate if possible) and GS/CS for connective tissue healing support
- Iodine ~12mg/day, laser, and magnets to support cell respiration and voltage for new cell growth[4]
- Treat any muscle inhibition with AK, Chiropractic, MFT

Repair and Remodeling phase:

- Continue nutrients, AK, Chiropractic, and MFT for optimum healing and return to play

Hormone/Stress support:

- Complete vitamin E, B3 for cholesterol conversion to testosterone, estrogen, progesterone, and adrenal cortex hormones as needed[16]
    - Low testosterone with elevated cortisol common in male athletes with overtraining syndrome[5]
- P5P, Vit C, Molybdenum, and protein necessary for catecholamine hormones from adrenal medulla as well as thyroid hormone[17]
- Choline bitartrate[5]
    - esp if any breathing issues during or after exercise
    - strengthen lungs, liver, digestion, adrenals
- Good fats from coconut, olive, avocado oils, European-style butter, eggs, and liver function to create healthy cholesterol level ~170-210 mg/dL
    - Total cholesterol below ~150 mg/dL is no longer adequate for normal steroid-based hormone production[6]
        - Causes amenorrhea common in female athletes
        - Low testosterone levels in male athletes
- Test AK hormonal, reproductive, and brain indicators for above nutrients and adaptogenic herbs
    - Ashwaganda, Holy Basil, Rhodiola, astragalus, eleuthero, cordyceps, reishi

    - Thousands of tested Olympic and pro athletes use adaptogens without testing problems
    - Balanced training with adequate aerobic base, the GC nutrient support, and quality sleep can often eliminate or improve stress, hormone deficiency/imbalance, and competition anxiety without need for adaptogens or common stimulants[5]

Things to generally avoid:

- For NCAA, USOC, and Professional athletes
    - Nutrient formulas and suppliers who do not show NSF certification
    - Others may be appropriate and high grade and test well using AK MMT but the athlete may be non-compliant due to fear of testing for a banned substance
- The following 8 factors that tend to create AK/MMT findings of neurologic disorganization (NDO), and abnormal muscle facilitation and inhibition
    - For MMT purposes abnormal Gait patterns can be utilized as NDO indications that will improve when productive nutrients and training methods are discovered or when unhealthy "nutrients" are identified and eliminated
        - Example: right gait pattern – with right foot forward the right latissimus should be facilitated and test with normal strength, if not then patient is NDO
    - HSAIDS – High Dose Synthetic and Isolated Dietary Supplements[5]
        - Often cause positive banned substances test
        - Very low grade and often toxic, pro-oxidation, and carcinogenic
        - May weaken or sometimes strengthen MMT indicators
        - Examples
            - Protein isolate powders
            - "Vitamins" E, A, C, K, B6
            - Iron – common deficiency in female athletes but should be verified in serum ferritin test and only whole food source
            - Copper, Zinc, Selenium
            - Beta-carotene
            - Intravenous "IV nutrients"
    - Junk fast food and soda pop
        - Laden with toxic, inflammatory, hormone-disrupting additives, excitotoxins, corn and soy oil, high fructose corn syrup or other refined carbs, or calcium disruptors like phosphoric acid
    - Hydrogenated or trans-fats
        - Crisco, margarine, fried fast food
        - Very inflammatory
        - Deranging to hormones, cell wall structure, brain, and metabolism
    - Refined carbohydrates
        - Promote inflammation, hormone, and metabolism disruption by over-stimulating insulin

        - Intermittent fasting, Paleo, low carb, and exercise will help restore insulin balance and sensitivity
        - May require zinc, vanadium, chromium support
    - Most "electrolyte" and "Energy" drinks contain:
        - Acesulfame potassium (carcinogen)
        - Glutamate based excitotoxin artificial sweeteners
            - Use stevia, honey, maple syrup instead
        - Stimulants such as high levels of isolated caffeine
    - Stimulants like guarana, caffeine
        - Organic cold brew coffee is rich in healthy polyphenols
            - Brewed half or all decaf without creamer and sugar can be enjoyed within reason
    - Growth Hormone promoting "nutrient" formulas
        - Often cause hyper-adrenia and insulin disturbance
        - Sleep, exercise, real nutrients, and Kidney Chi promote growth naturally
    - Unbalanced training[5,18]
        - Excessive high-intensity or strength training is anaerobic, pro-oxidation, acidic; nutrient, hormone, immune, and Yang depleting; joint surface destroying over time
        - Balance anaerobic training with aerobic activities at least 50/50 based on time and intensity over the course of each week, month, year
            - Use Heart Rate monitor when training (180 minus your age formula for max aerobic training heartrate)
            - Use Heart Rate Variability (Ithlete app) and Maximum Aerobic Function test (Maffetone) to assess for overtraining syndrome
        - Use above to successfully train skeletal muscle, cardio-vascular system, brain, and metabolism
        - Incorporate chiropractic, MFT, and jumping/bouncing activities to train the fascial system
            - Encourages functional elasticity of body based on Tensegrity model of movement

For functional AK testing of potentially beneficial, safe, and legal nutrients the author has observed excellent results using various "entry points" for muscle testing. Back Shu (Associated points), Front Mu (Alarm Points), and Source Points which create improved MMT of any previous inhibited muscle are excellent entry points for testing. TCM Tongue and pulse diagnosis, or SOT Occipital fiber analysis are also superb indicators of possible diagnostic organ/meridian points to use with AK MMT. For example, an athlete with a pale tongue and teeth impressions on the sides of the tongue will display general weakening of a previously intact muscle, and/or strengthening of muscle inhibition, upon TL to the SP Alarm point. The indicated entry point can then be therapy-localized by touch or using a magnetic wand together with any indicator muscle (such as shoulder flexor group). The

resulting muscle inhibition can then be exploited for screening nutrients on the body under the south pole of a flat bar magnet.[11]

The magnetic wand method maintains muscle inhibition by preventing the body from adapting and causing the entry point indicator to eventually disappear. Nutrients which strengthen should then be confirmed by standard AK intra-oral insalivation of the nutrient, if possible. Other AK testing methods may also be reliable and fruitful for the athletic patient.[11]

With certain exceptions like Vitamin D and Ferritin, it is recommended to consider standard blood nutrient testing with strong skepticism. Most nutrient reserves are held in tissues and organs, not the blood circulation. Many blood analysis software programs do not adequately account for such nutrient reserve tissue dynamics. Blood analysis software must be based on expert, non-linear, sophisticated algorithms to be useful and accurate. Other Urine lab findings are very helpful such as Koenisberg, Indican, Sulkowich, Vitamin C, and urine dipstick screening.[14, 19]

Conclusion

We have seen this approach to sports nutrition testing provide significant improvement in longevity, health, and fitness during training and competition. Therefore, it is an opportunity for the PAK practitioner to functionally test nutrients and help the athlete improve and perform better by legal, healthy, and individualized clinical nutrition.

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Keys To the AK Castle: Building A Foundation

Dylan B. Miller, D.C.

Abstract

Why is the ICAK, after 60 years, struggling to gain physicians? How can the International College of Applied Kinesiology (ICAK) and Applied Kinesiology (AK) begin to thrive and not slowly fade away? Why does the 100-hour certification course remain unchanged in the past 35 years? Where is the standardized foundation and protocol to draw in students and practitioners, giving their studies purpose and function? (Professional Applied Kinesiology (PAK) is the official term for AK. However, I will simply be using the term AK for much of this paper).

Key Indexing Terms
Professional Applied Kinesiology, Education, Certification, Protocol, Foundation

Discussion
Times have changed quite rapidly with the "discovery" of AK in 1964 and advancement of science during the past 60 years. Dr. George Goodheart Jr.'s observations and knowledge jump-started the biggest healing revolution in human history. Dr. Goodheart discovered a way to connect with the body's healing centers through functional physiology, muscle testing, and the nervous system.

So why is the ICAK not growing in numbers large enough to sell out football stadiums? Why is AK still a healing secret to humankind? How can AK maintain the integrity of a "private club" yet grow and help to heal the world? In my experience and observations, PAK lacks a form of diagnosis and treatment protocol for beginning students.

Dr. Goodheart said in his 1984 Metabolic seminar (tape 1 37:25):

"There is nothing better than to try to present something to your peers, and try and do as well as you can to advance your profession. When you try and do a good job, you are putting forth your best effort and be a credit to our profession….. When something is right it's simple. The body is simply intricate and intricately simple!"

A simple truth I have observed first-hand while trying to learn AK during chiropractic school and beyond; a truth which comes from unconditional love, and has weighed heavily on my mind for over 13 years. The unchanged number of active members in the ICAK speaks for itself. Why isn't AK growing as an organization and cornerstone of knowledge for the healing arts? Because students lack a solid, consistent, unified foundation and protocol to lean on from the beginning. The student crop is scared away from AK due to lack of consistency and direction for use.

The common theme as a professional student while in chiropractic college was to see large numbers of students interested in AK, students having many questions about AK, and wanting to be treated by AK, then never commit to learning it. AK was “neat information, but how do you use it?” (This is still the case with fellow practitioners years later) Instead, students gravitate towards simpler techniques, with simplified protocols, big promises, and little knowledge compared to the entirety of what AK encompasses. It was disappointing to see the sharpest minds in school end up chasing other techniques, doing their own thing, and in many instances, wasting time and energy chasing knowledge AK has had mastered for decades.

No student should ever have to find their own path when trying to acquire the basics of AK. It is the job of the institution to set groundwork for students to succeed, not just give information without direction or purpose. I have found an obvious need for a workable protocol as well as foundational consistency in the basic PAK courses for students to cohere to. Not only are students scared away from PAK due to the lack of any sort of standard protocol to follow, they are then hit with foundational course inconsistency between teachers. The confusion becomes overwhelmingly frustrating for students thirsting for answers and faced with hard decisions of where to spend their time, money, and efforts to guide the rest of their careers.

This confusion then carries over to all the active members of the ICAK down the road. Patients have no idea what to expect from any PAK practitioner as everyone’s foundations are so diverse depending on who they studied under and what time period. How wonderful would it be to send a patient to a PAK certified physician and know there will be a basic care standard of which that PAK practitioner will uphold? How wonderful will it be for students from across the world to be able to expand from the same basic protocol and set of standardized classes which will, at last, set a new standard of care throughout all humankind?

Below is my journey in trying to navigate PAK as a student.

During my training, which started after Dr. Goodheart’s passing, I was very fortunate to meet Dr. Evan Mladenoff (Dr. M) as my introductory teacher into PAK. Dr. M always refers to PAK as the greatest “private club” on the planet. From day one, he instilled in me the thirst for knowledge and loving philosophy, upon which AK was founded. If AK and Dr. M hadn’t found me while in college, I would have given up and would not have become a chiropractic physician. AK held the knowledge no other source in school took the time to address, from a group of the greatest healers and minds in the world.

While taking the 100-hour PAK course 5-6 times with 4 different teachers, it started to become clear that each class had its own take on AK and the healing art. After being exposed to all this wonderful knowledge, I still had no idea how to fit all the pieces together to actually use. I thirsted to find the source of this knowledge base which founded AK. My search through many books, classes, and stories left me to wonder how someone who was such a great healer could leave this healing art so elusive for others to replicate. Every

source of information had a little different version of what Dr. Goodheart meant to them and their interpretation of his work. Each teaching was full of wonderful information, from wonderful educators; most of whom were able to grow alongside Dr. Goodheart, and the ICAK as it developed. Each AK source filled me with more knowledge, but at the same time, with more questions of how to actually use AK. After struggling to connect all the dots, I finally decided to go back to the source in order to meet and learn from Dr. Goodheart himself, via VHS-recorded seminars.

Each VHS tape would build my confidence in AK and its potential for healing. However, I still did not know how to put all this great information into practice. At last, I discovered Dr. David Leaf's, "Mo-Jo Enterprise" seminar series, featuring Dr. Goodheart. This organized all the AK information in extensive detail and simply broke all the procedures down into a usable form. This series solidified the majority of AK procedures the Applied Kinesiology Synopsis 2nd Edition, only briefly addressed. I finally came away with confidence to treat a patient with purpose, direction, and applied kinesiology.

After studying Dr. Leaf's series, I was ready to dig into, what I saw at the time, as "advanced" material from the Chiropractic Neurologist, Dr. Wally Schmitt and the Quintessential Applications A(K) Clinical Protocol (QA Protocol). I knew of the QA Protocol (hereinafter "QA") series for quite some time. However, in my mind, I thought the information would be too advanced for me until I had a better background built up in AK. How could a student like me be ready to take a class from a chiropractic neurologist and get anything out of it? Little did I know, nothing could have been further from the truth. This class taught me a timeless, scientifically sound, up-to-date, basic neurological foundation from which to build all other AK skills upon.

As a student of AK, my biggest disappointment came when I least expected it. I started QA, and realized much of the AK information I had worked so hard to understand, had been superseded by updated science. Dr. Walter H. Schmitt, Jr. put his entire life's work into organizing (with Dr. Kerry McCord) and scientifically proving Dr. Goodheart's observations, along with everyone else's knowledge involved in creating AK across the globe. QA is more than a protocol, it was the foundation I had been looking for all along. After completing the 16-session course, suddenly all techniques made more sense, and knowledge had a home to grow in. AK was no longer a barrage of fascinating, elusive, individual pieces, but a well understood, open-ended system to use on patients with reason and reproducibility.

QA is the basic foundation from which all PAK should be based: namely to heal humankind. Dr. Schmitt's life's work is ready for the ICAK to adopt as a foundation from which to grow. The PAK basic 100-hour certification course is not obsolete, but rather outdated. The information base is too impractical for students, has grown and changed too much, and the ICAK has grown almost 0% in the past 30 years.

If the ICAK wants to thrive instead of slowly fade away with time, QA gives everyone a scientifically sound, timeless, 16-course foundation, with a protocol, online presence, and

book from which to work. Online learning is here to stay, and the roughly 160-hour course will not suffice all on its own. The course still needs lab time and personal touch from the 100-hour teachers to ensure the most fundamental aspects of AK. The teachers are the cornerstone for proper manual muscle testing technique and personal perspective on AK. The PAK basic course may need to be 260-hours or more. Watch the ICAK and all associated techniques and classes flourish and grow once the QA Protocol and updated basic foundation is put in place to draw students in to develop from.

As we all know, students in general will strive towards meeting the minimum standard required by any organization. As a Professional Applied Kinesiologist, this standard needs to be set higher than a 100-hour certification. Is 100 hours enough to represent PAK and what it stands for and encompasses? Not to run-down any other certifications, but isn't AK much bigger than a single 100-hour meridian therapy certification, a specific muscle technique such as ART or Graston? Is AK not an open-ended diagnostic system meant to be interchangeable with all techniques and ever evolving information?

100 hours is simply not enough in its current, outdated form. Students are not given a comprehensive set of AK tools to ensure their success. The ICAK standard needs to be set much higher to attract not only the most driven healers, but the brightest minds in the profession. We need to maintain the integrity of the ICAK as a "private club" while encouraging growth. This is accomplished by upping the basic foundation "ante" (260+ Class hours +Labs) required to enter the club, and giving students a foundational protocol to follow.

Let us pause while I explain the importance of my thoughts for the basic foundation course changes, and the very real adversity the ICAK will face in this new future. Long-term, we need to think about how to preserve the integrity of the ICAK as we start to set a new standard in all healthcare by implementing this foundation. This is much bigger than just a change to a 100-hour certification program. After implementing this updated basic foundation, PAK will quickly expand into the undeniable force within healthcare it was always meant to be. This kind of change is sure to shake the current trillion-dollar healthcare system and has the very real possibility of large corporations and powerful people trying to shut us down by any means necessary. Ensuring all PAK certified members are held to a high standard from this point forward, and given all the information and direction to succeed will make it much more challenging to ever bury the truth that all PAK practitioners carry and hold from their basic PAK certification. A 260+ hour foundation full of Dr. Schmitt's truths is the most certain way to soften even the hardest of healthcare hearts. Who better to safely represent the basic foundation of ICAK than our very own, world renown, medically respected on every level, unchallengeable, and unshakable Dr. Walter H. Schmitt Jr.? I have considered every aspect imaginable over the past 13 years and these are my final conclusions and solutions to a big problem that needs to be addressed within our ICAK family. Last, but not least, I believe the "the hiStories of A.K. "Film"" should also be a requirement for the basic PAK certification to ensure the history of AK and Dr. Goodheart's wisdom are never lost.

The ICAK can be the unstoppable force it was meant to be once students are finally given a set of keys to the AK castle with the QA Protocol as the basic foundation from which to build. ICAK will grow and all subset classes associated with AK will expand as a result.

Conclusion

It is time to overhaul the basic PAK certification requirements and class standards to set a new foundation for the ICAK. The basic PAK certification coursework needs to be updated and unified. There must be a standard protocol for beginning students and practitioners to lean on and utilize. Students are too confused with every 100-hour certification course being different from the next as well as driven away without a protocol to guide the information into fruition.

There are AK protocols throughout this community, however, they seem to be based around advanced AK concepts. I know many teachers within the ICAK that have their own protocols and systems, and there is no way to know or say who has the best program or can put the best class together. Dr. Michael and Dr. Noah Lebowitz have an entire protocol system, Dr. David Leaf and Dr. Tom Roselle have Leadership to Legacy-Basic Fundamentals of AK system in place, Dr. Sheldon Deal has more protocols and ways to prioritize healing the body than any 10 practitioners put together, Dr. Evan Mladenoff has his meridian therapy and concussion protocols, Dr. Richard Belli and Dr. Michael Allen have chiropractic neurology protocols in place, Dr. George Gonzalez has many rehab protocols, Dr. John Bandy has trauma clearing protocols, Dr. J Dunn has genetic variant methylation protocols, Dr. Hank Tolhurst, Dr. Katharine Conable, and Dr. James Hogg have taught the AK courses for decades and have their own systems in place along with so many others that go unmentioned here, as the list would never end. We have many wonderful members and teachers from which to build a new foundation. Why reinvent the wheel and put another class together to do what has already been done? The QA protocol is complete and ready to put into place today.

Together the ICAK will stand the test of time, divided the ICAK will fall. The basic PAK certification course and foundation is currently divided and without direction to draw members in. What better honor than to put Dr. Walter H. Schmitt Jr.'s lifetime of work, aka QA as the new basic foundation for Professional Applied Kinesiology (PAK) and the ICAK?

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New Methods of Reducing Spinal Fixation Recurrence

James D.W. Hogg, D.C.

Abstract

Spinal fixations are a common cause in many areas of muscular neuron-inhibition often resulting in severe pain and functional issues. Classic corrective approaches[1] are effective in clearing these problems but for some patients' fixations tend to recur. This paper will explore effective methods of reducing recurrence using treatment of coccygeal locking and injury reflexes.

Key Indexing Terms
Spinal Fixations, Coccygeal Locking, Injury Reflex, Disc Degeneration, Low Back Pain, Mid-Back Pain, Shoulder Pain, Neck Pain, Knee Pain

Introduction
As we all have learned, spinal fixations can cause a number of neuromuscular inhibitions causing severe pain and dysfunction for our patients.[1] This is a problem I check on pretty much all of my patients on a regular basis. It's an extremely effective technique and often the only way to clear some structural problems and pain. Unfortunately for some patients these structural problems become recurrent. I've had some patients who needed fixation work on every visit despite checking them for the need of vitamin E and giving instructions to avoid certain positions and activities that may cause fixations to recur.

Over the years I've developed the use of some techniques that have been very helpful in decreasing fixation recurrence which I'll share below along with a review of fixations and the problems they produce for our patients.

Discussion

Here's a short review of common fixations and their effects. Pain across the low back, especially with bending or lifting as a result of bilateral gluteus maximus inhibition caused by upper cervical fixations. Sometimes low back and hip problems from poas inhibition can be caused by occipital fixations. Posterior knee pain with a tendency for hyperextension because of bilateral popliteal inhibition caused by mid cervical fixations. Severe shoulder pain and dysfunction along with numbness on the arms and hands because of bilateral deltoid inhibition caused by cervico-thoracic subluxations. Mid-back/scapular pain along with posture problems because of bilateral lower trapezius inhibition caused by thoraco-lumbar fixations. Neck pain and restricted range of motion because of inhibition of the posterior muscular group caused by lumbar and sacroiliac fications. Of course, there are more fixation results that have been presented in papers over the years but these are the classics.

While all of these fixation results are important, those that I see most common with patient complaints are upper cervical fixations resulting in anywhere from mild to incapacitating low back pain and dysfunction as well as cervico-thoracic fixations resulting in mild to incapacitating shoulder problems as well as arm and hand numbness, numbness being less common. The other fixations seem all in the middle frequency range except for mild cervical fixations which I tend to see the least frequently.

Although the above results of spinal fixations are serious enough, we all know that there's another important effect for long-term fixations. Spinal discs, as we know, don't have much blood supply. They require movement of the spinal segments that they live between to help pump out toxins and pump in nutrients. With the reduction in movement between spinal segments caused by fixations that remain for prolonged periods, discs can start to degenerate.

The meninges run from cranial and upper cervical areas down to the dorsum of the first segment of the coccyx.[2] They line the intervertebral canal and have been known to alter flexion and extension of the spine when under tension from coccygeal locking.[3] Because of the cranial attachments meningeal tension from coccygeal locking can contribute to cranial subluxations in the temporal and saccital areas.

While the above are classic AK findings with meningeal tension, a variation I've been finding the last few years is that meningeal tension can be a contributor to recurring spinal fixations. When I've adjusted the same fixation several times and educating the patient about activity and life habits doesn't provide a reason for recurrence, I check for meningeal tension related to coccygeal locking. If the coccygeal challenge shows positive, I perform the classic simultaneous adjustment of the coccyx and upper cervical area.[3] In this situation, once the coccyx is clear, the spinal fixation also becomes clear and the associated muscles become facilitated.

Often when patients have this problem, there is not just one recurring fixation. There are often four or five fixations. What I loved was discovering that, in this situation, clearing the coccyx will often clear ALL the fixations and facilitate all the associated muscles! Sometimes there will be one fixation left that needs to be adjusted.

Materials and Methods

To release meningeal tension related to coccygeal locking:

1. Testing
    a. Walther suggests that the test needs to be the patient touching and pushing inferior on their coccyx.[3]
    b. Since this is more a challenge than a therapy localization, I've found that it also works for the doctor to push inferiority on the patient's coccyx.
        i. The challenge needs to be maintained during the muscle test.
        ii. If the patient performing the challenge, they need to lay their finger along the surface of the coccyx and push inferiorly without much anterior pressure

iii. If the doctor is performing the challenge, they should use three finger tips along the surface of the coccyx and push inferiorly without much anterior pressure.

c. A key to both of the above is to cover most of the coccyx to effectively push inferiorly without having to push much anteriorly.

2. Treatment
   a. Lay your middle finger of one hand along the surface of the patient coccyx
   b. With your other hand, contact the occiput and upper cervical area
   c. Gently pull both hands together until you feel a subtle increase in motion in both areas
      i. Usually, I find the upper cervical area will loosen a few seconds before the coccyx but sometimes there will be a different sequence
      ii. If you don't notice the subtle loosening/movement, just hold the points for 30-40 seconds
3. When done, simply retest as above to ensure correction
4. Once meningeal tension has been released you will find that most if not all fixations have released themselves. You may also find that some cranial subluxations like sagittal suture and temporal bulge have been released.

If patients have recurrent meningeal tension via coccygeal locking, it is often because the way they tend to sit puts pressure on the coccyx. In that case I instruct them to make sure to sit up rather than slouching so "your sitting weight is on your buttocks rather than your tailbone".

While I find that meningeal tension from coccygeal locking is the most common cause of recurring fixations, I sometimes find that Injury Recall Technique (IRT), as introduced to ICAK by Dr. Walter Schmitt,[4] is needed instead. As you probably remember, injury recall can be a common cause of many recurring problems as the body maintains an injury reflex resulting from a trauma that may be days or years old. Fortunately, it's easy to reset the reflex that was useful at the instant of trauma but has "forgotten" to get turned off.

In the case of the fixation related extended injury reflex:

1. Pinch lightly over the segments that are locking together
   a. pull superior on the talus bone in the foot and test a previously facilitated muscle.
      i. If this results in inhibition of the test muscle, it tells you that there is a fixation in the pinched area.
      ii. Of course, there is a small chance that the talus is subluxated and that's why the the facilitated muscle became inhibited. If you suspect this, just pull superior on the talus without touching the fixation area and see if it causes muscular inhibition. 99.9% of the time you will probably find that a simple superior talus challenge has no effect.
2. Once you've established that there is an injury reflex in the area of the fixated segments

a. simply repeat the pinch challenge in the fixation area and pull inferior on the talus.

3. Another classic way to challenge for fixations is to have the patient TL over the fixated segments instead of pinching and repeat the above testing.
   a. In this case, treatment will be with TL instead of pinching.
4. One more method I presented in 2004 is to stimulate the suspected injury reflex area with a low level laser therapy (LLLT) (5).
   a. Swipe the light back and forth over the suspect area 2-3 times, challenge the talus superior
      i. if positive, repeat the laser swipe and pull inferior on the talus to clear the injury reflex.
      ii. Basically, swiping with the laser light is the same as pinching the suspect area.
   b. The LLLT approach is especially useful when treating IRT for surgeries or other deep seated injury areas.

Conclusion

Clearing fixations and keeping them clear is important to enhance function and pain relief for our patients. My patients and I have been extremely pleased by the impressive reduction in spinal fixation recreance using coccygeal release and injury recall techniques. While treating spinal fixations is extremely important, it's even more important to find and treat the underlying cause.

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Shoulder Dysfunction Patterns

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

Abstract

The author discusses common specific patterns of shoulder dysfunction. A review of Professional Applied Kinesiology (PAK) aspects of diagnosis and treatment of shoulder problems is presented and expanded based on the author's clinical experience. Correlations to typical Professional Applied Kinesiology (PAK) findings and structural factors are clarified. Specific PAK diagnostic and treatment protocols are recommended.

Key Indexing Terms

Professional Applied Kinesiology, Shoulder, scapular dyskinesia, suprascapular nerve entrapment

Introduction

Abnormal scapular biomechanics, or scapular dyskinesia, is a common finding in most symptomatic shoulder cases as a result of trauma or chronic functional issues. **Scapular dyskinesia** and **suprascapular nerve entrapment** are discussed as part of the clinical puzzle with shoulder symptoms.[1,2] Most of these cases can be rapidly diagnosed based on static and dynamic shoulder observation as well as manual muscle testing (MMT).[3,4,6] This author has observed this problem in many athletic patients involved in repetitious throwing, punching, shooting, and swinging motions, many of whom had been treated with limited or no success in other ways.

Discussion

The shoulder is one of the most sophisticated movement systems of human anatomy. Four bony articulations, many muscles, fascia, and other related tissues comprise the shoulder complex and its remarkable constellation of movement.[2]

The motion of the scapula, however, should be considered of primary importance in shoulder function and health. It is normally capable of elevation, depression, protraction, retraction, and rotation.[2]

Scapular depression and scapular protraction are common dysfunctional postures of the scapula. These or other aberrant postures of the scapula will jeopardize the critical **alignment of the glenohumeral joint** as well as the **suprascapular nerve**. Therefore, there are many muscles attached around every margin and surface of the scapula. In other words, proper gleno-humeral alignment, or "rhythm", is the primary purpose of normal muscular control of scapula posture and movement. As the arm is abducted, adducted, flexed, or extended the muscular and fascial controls are attempting to position the shallow glenoid fossa in perfect alignment to the larger head of the humerus in every possible position. When this mechanism fails there is abnormal scapula biomechanics, or *scapular dyskinesia*.[2]

An example of scapular dyskinesia can be seen in an NFL quarterback when his physical limitations force him to release the ball lower than desired. By contrast the quarterbacks with superb shoulder motion can release close to the top of the throwing arc. To accomplish this the upward rotation of scapula is essential to maintain optimum gleno-humeral alignment, and to prevent acromion interference with humeral abduction.

As a result of scapular dyskinesia, a prevalent observation in the throwing, punching, shooting, and swinging athlete is **entrapment of the Suprascapular nerve**. This nerve originates from C5 or C6 spinal nerves and brachial plexus. It navigates anterior to posterior through the suprascapular fossa then through the spino-glenoid notch to innervate **supraspinatus**, **infraspinatus**, and various shoulder structures.[1,4]

Leaf points out that scapular dyskinesia creates excessive stretch against the nerve. As a result, the infraspinatus and supraspinatus can become inhibited and even atrophied. The muscle weakness can be quite profound, even 2 or 3 of 5 on Wexler muscle grading. **Teres minor** muscle test can also appear weakened due to the overlap in function of the teres minor and infraspinatus in external rotation. Obviously concomitant nerve impingement of C6 or C7 nerve roots will make the suprascapular nerve more vulnerable to distal impingement in the scapula.[3,4,6]

AK Infraspinatus test:

Provocative infraspinatus test with shoulder oriented anteriorly (Leaf):

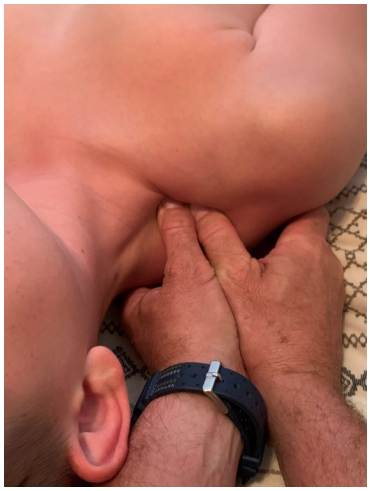
The author has observed excellent recovery of suprascapular nerve function by also performing deep transverse friction massage at the two bony tunnels on the scapula where the nerve passes.

Treatment for suprascapular nerve:

1. With patient supine **suprascapular notch** is located by *deep caudal pressure medial to the coracoid process and posterior to the clavicle*[1]

    - Using supported thumb contact doctor slowly presses into area
    - allows overlying muscles to accommodate and depth needed to reach scapula surface underneath
    - Tranverse friction massage (TFM) can be engaged for approximately 30 seconds or at least until a vasomotor response of heat and moisture is felt
        - ✓ goal is to break up fibrotic adhesions and lengthen local fascia to give relief to the nerve

TFM at Supra-scapular notch



TFM at Infra-glenoid notch

2. **Spino-glenoid notch** requires similar treatment with patient prone[1]
    - *At inferior-lateral spine of scapula just medial to junction of acromion process and lateral angle of spine of scapula*
    - much easier to reach but requires determined TFM
3. nutrients can help speed nerve healing
    - Methylcobalamin B12, Vitamin D, Benfotiamine, Alpha Lipoic acid, GLA, Omega 3 fish oil

General shoulder treatment tips:[3,4,6]

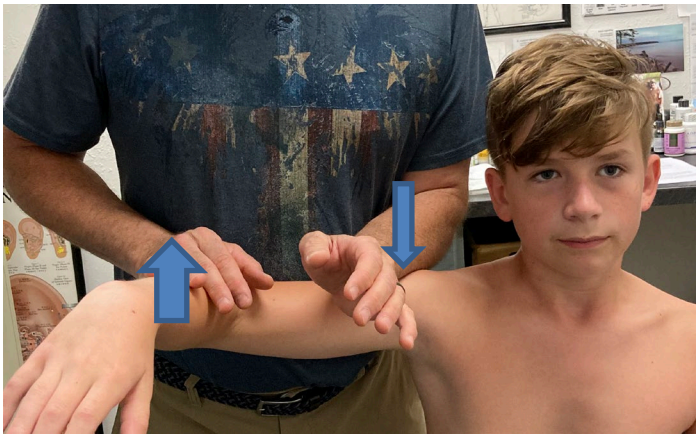
4. Challenge and manipulate all shoulder articulations (see below)
5. Upward scapula rotation requirements for throwing motion
    - Facilitate upper and lower trapezius, serratus anterior, teres major
    - Lengthen levator scapulae, rhomboid, pectoralis minor, coracobrachialis, upper serratus anterior, deltoids
        -
    - Fatty planes between serratus anterior and ribcage, and between subscapularis and serratus anterior allow scapular glide.[8]

- Improved nerve supply and fascial release around shoulder encourages flow of necessary trophic factors to nourish fatty planes

6. Generally, the scapula and clavicle posture need to move superiorly more freely (chiropractic, MFT) and humerus needs to be stabilized more inferiorly (subscapularis and latissimus)
7. Treat thoracic outlet issues if needed – see Leaf Flowchart manual[3]
   - Pec Minor, Scalenus anticus, Costoclavicular syndromes, cervical rib
8. Shoulder muscle origins and insertions that are tender are usually trauma or repetitive stress injuries
   - treat with deep pressure with clove oil
   - muscles with spinal origins use spearmint oil[7]
9. Treat scars in area from previous surgery or trauma
   - Laser, neural therapy injections, fascial flush, acupuncture, Essential oils

PNF stretch of Pec muscles with lateral to medial mobilization of scapula:

Superior humerus adjustment



Inf clavicle adjustment

Sterno-clavicular mobilization

Shoulder-Knee relationship observations

There also seems to be an important biomechanical relationship between the scapula and contra-lateral patella. During the highest period of scapular load, as the arm is beginning to be flung forward, the scapular stabilization forces, especially **serratus anterior, mid trapezius,** and **rhomboid**, are attempting to maintain the scapula flat against the ribcage. At the same instant the opposite patella is being pulled into the patella groove of the distal femur with very great force by **quadriceps**. In other words, these two articulations, scapula and patella, are under high load at the same time during throwing.

Therefore, it appears that common patellar maladies such as bursitis, supra- and infra-patellar tendon adhesions or myofascial derangement, or excessive lateral pressure syndrome associated with chondromalacia, can affect the quality of overall afferent sensory bombardment during the throwing action, thereby affecting shoulder coordination as well. Symptomatically, many shoulder patients will also complain of knee problems. Similar to a ligament interlink scenario, both the scapular dysfunction and the patella must be addressed for optimal performance.

Knee Treatment considerations

For bursitis around the patella:

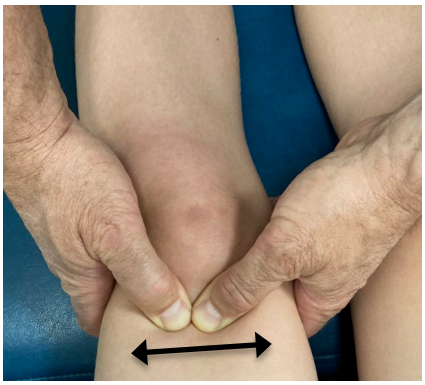
- acid calcium supplementation is given along with anti-inflammatory formulas

Patellar tendon adhesions and chondromalacia:

- will usually present with inhibition of vastus medialis divisions and shortened vastus lateralis and I-T band
    - use MFT to lateral thigh
    - patient can use thigh roller to self-treat or maintain
- TFM is typically helpful across the supra-patellar tendon from myotendinous junction down the entire tendon to the enthesis

- A disturbed, ropey, or inflamed enthesis may also benefit from zinc supplementation
- Cycling in a light-moderate gear at an aerobic heart rate for at least 20-30 minutes is recommended as excellent rehabilitation and reconditioning for almost any knee problem

TFM at supra-patellar tendon



<u>Global structural considerations relevant to shoulder correction</u>

- *Diagnose and treat C5-6-7*
  - *fixations, misalignment, disc syndromes*[3,4,7]
  - *Accessory nerve*
    - *Assess for occipital fixation, often right lateral misalignment*
    - *" upper cervical fixation and subluxation*
    - *" cranial faults, esp. spheno-basilar*
- *Address pelvic SOT Categories*[3,4,8]
  - *DeJarnette explained adaptations to pelvic misalignment include latissimus pulling shoulder down and upper trapezius pulling it up in order to help stabilize the mis-aligned pelvic structure*
  - *Dural health effects everything*
- *Correct ribcage mis-alignment*
  - *Right upper ribcage usually subluxated inferiorly*
    - *Adjust superiorly with inspiration*
  - *Left lower ribcage often fixated in extension*
    - *Adjust ant to post, and sup to inf with expiration*
- *Consider organ relations to persistent muscle imbalance*[3,4,8]
  - *Treat via CMRT visceral manipulation*
  - *Organ nutrient support*
  - *AK Five factors*
- *Past emotional/physical trauma may require*[3,4]
  - *Psychosomatic techniques*
  - *Support for Adrenals*
  - *Miasmatic or constitutional homeopathy*

Rt upper ribcage inspiration assist

Lt lower ribcage expiration assist

Conclusion

We have seen these related injuries and biomechanical factors effect power, accuracy, timing, and comfort in athletes from little league to professional levels of sport.

Thus, in addition to thorough AK shoulder examination and treatment, we suggest testing and treating all muscle divisions of the lower extremity, especially knee muscles, as part of our global mindset when addressing shoulder problems. In fact, this clinical frame of mind is an essential part of the DNA of any skilled PAK practitioner.

We treat the problem where the problem is…
not just where the symptoms manifest.

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Sural Nerve Neuritis in Athletes

Barton Stark, D.C., DIBAK, DIAMA

Abstract

The author discusses sural nerve dysfunction seen in athletes. Professional Applied Kinesiology (PAK) methods for diagnoses of sural nerve problems are presented based on the authors experience.

Key Indexing Terms
Professional Applied Kinesiology, Sural Nerve Neuritis, Sural Nerve Entrapment

Introduction
Lateral sural cutaneous nerve (LSCN) neuritis can occur in athletes from shoe pressure on the nerve or from direct physical impact trauma during contact sports. The nerve can be treated with conservative chiropractic and myofascial therapy (MFT) aided with PAK diagnosis.[1,2]

Discussion

This condition can result in significant numbness, tingling, and/or pain along the lower lateral leg and foot. Sensitivity to touch and pressure over the nerve can be very intense. *Tinel's sign* may be positive over the nerve, which is commonly entrapped by scar tissue and adhesions. Achilles tendon, foot, and ankle problems can be confused with the sural nerve symptoms or concurrent. It can develop over time after an athlete has been kicked or stepped on numerous times or from tight footwear. Thus, it is often undiagnosed.[1,2]

The LSCN provides only sensory and has no motor component. However, it does contribute to the overall afferent bombardment to cord and brain from lower lateral leg, ankle, and foot. If the extremity involved is chronically symptomatic, then it is one more opportunity for the PAK practitioner to help the athlete improve and perform better by cleaning up the related nociception and sensory signals from the area.

The author has used numerous methods to help the nerve heal including chiropractic, MFT, acupuncture, low-level laser, ice, and anti-inflammatory remedies. If there is no improvement from conservative treatment the patient may need referral to rule out other potential medical issues.

Chiropractic treatment recommendations

- Challenge and treat nerve origin and other areas that can affect the nerve[3]
  - L5 and S1 subluxation
  - SOT Pelvic Categories

- Piriformis syndrome
- Fibular head subluxation
    - Usually postero-lateral
    - May need taping below fibula head to stabilize fibula and knee (see fig 1)
    - Assess lateral and medial hamstrings and popliteus
- Talus subluxation (see fig 2)
    - Usually medial, often lateral after inversion sprain, can also be anterior
- 5th metatarsal subluxation (see fig 3)
    - Adjust for dropped 5th met head

Figure 1 – fibula taping and adjustment

Figure 2 – medial talus adjustment

Figure 3 – met head adjustment

Myofascial Therapy considerations

- Directionally challenge the skin over the following locations
  - Distal Biceps Femoris, popliteal fossa, and the area from posterior middle calf to postero-lateral malleolus, lateral foot
  - Treat fascia in direction of strength to patient tolerance with slow, deep passes (see figs 4, 5)
- Additional treatments especially if local scars in the area from previous surgery or trauma
  - Laser, neural therapy injections, fascial flush, acupuncture, Essential Oils of helichrysum, peppermint, lavender
  - Nerve flossing (see fig 6)

9 le,
then plantar and dorsi-flex ankle approximately 10-20 times

Figure 4 - releasing surface fascia

figure 5- deeper fascia layers

Figure 6 – sural n. flossing

Conclusion

We have seen this approach to sural nerve symptoms provide significant improvement in comfort during training and competition. Therefore, it is an opportunity for the PAK practitioner to help the athlete improve and perform better by cleaning up the related nociception and sensory signals related to this often excruciating and undiagnosed condition.

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The Watch Effect

Paul T. Sprieser, BS, D.C., DIBAK

Abstract

An interesting phenomenon that I learned about while studying Applied Kinesiology in the early 1970's that was observed in the class being taught in New York and New Jersey. Someone in the class said a muscle weakness would occur if a watch was placed over SP21 on the left side only.

Key Indexing Terms
Pectoralis Major Sternal (PMS), Pectoralis Major Clavicular (PMC), Tensor Fascia Lata (TFL), Gluteus Medius

Introduction
I was being exposed to muscle AK testing during my senior year at the Chiropractic Institute of New York in 1968. A few of the instructors spoke about Dr. Goodheart and demonstrate muscle testing.

I would be graduating in August 1968. That year the American Chiropractic Association was having it national meeting at the Americana Hotel in New York City. My whole graduating class was able to attend the meeting. This was my first exposure to Dr. Goodheart, one of the keynote speakers at the convention.

When I started studying AK in 1969, the class were being taught by Dr. Sal Cordaro, Dr. Jerry Deutsch, and Dr. Jose Rodríguez. I know this was demonstrated in the class with the use of muscle testing. Everyone in the class stopped wearing the watch on the left wrist, which was usually right-hand individuals. My wife and I still wear our watches on the right wrist even today.

Discussion

On March 30, 2021, I was discussing subtle energy patterns of the acupuncture system with a patient and showed her the "Watch Effect". So I decided to do a research study and collected data on 108 patients from 3/30/21 to 7/29/21. The group was made up of 57 female and 51 males. The watches being used were mechanical (wind up and self-winding), battery powered mechanical and digital, and must be running to have this effect. This effect also happens with a stop watch mechanical or digital. On a mechanical or digital watch it must be running and keeping time, if it is stopped it does not cause a weakness. Spleen 21 is located in the mid axillary line, at the 7th intercostal space.[1,2]

The indicator muscles being used in supine position were the Pectoralis Major Sternal (PMS), Pectoralis Major Clavicular (PMC), Tensor Fascia Lata (TFL) and the Gluteus Medius, If tested in the prone Hamstring would be used.[1] If a stronger muscle was to be

used to prove a point the Quadriceps was used. All muscles showed a profound weakness on all 108 patients in this study.
I have tested this phenomena on and off for 47 years. The number of test done would be at least 5000 or more times with the same outcome. I wondered the significance of this weakness?

I believe a weakening response is usually a sign that a incompatibility to the body is usually a negative sign. This is seen in allergy testing on food or drug reactions. Benefits usually would not weaken an indicator muscle. I would expect it to be neutral, causing no weakness.

I spoke to other AK practitioners in particular was Jonathan Curzon, DC, of Perkasie PA. He mentioned that the spleen meridian was the Great Lower Channel connecting the stomach and heart.[3] Over stimulating Sp21 would cause profound weakness of the bodies muscle in general. This Channel goes around the abdominal wall below the diaphragm in the hypochondria gathering the blood around the circumference of the body.[3,4] The spleen channel also is known as the leg greater Yin. I have also noted searching the internet a book mentioned Kosho Ryu Natural Laws of Motion, that Sp21 is located at the 6th intercostal space.

Conclusion

I know this watch effect phenomena is a fact and Dr. Curzon had been checking this in his practice for a week on 20 patients and confirmed my finding[6] I still don't know how to explain this subtle energy change with the watch, but I do know it exists. I also know that the effects of wearing a watch on the left wrist does not cause any weakening unless the watch is placed directly over spleen 21 on the left.

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Experimental Observations of Members of the ICAK

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Informal Proceedings

Keys To the AK Castle: Healing Love

Dylan B. Miller, D.C.

Abstract

The purpose of this paper is to expand on the elusive subject of healing. How can healing be achieved according to the greatest healers throughout history? How can the International College of Applied Kinesiology (ICAK) and Applied Kinesiology (AK) continue to expand and heal the world?

Key Indexing Terms
Professional Applied Kinesiology, Healing, Doctor, Teacher, Physician, Love

Discussion

The word "Doctor" holds many different definitions as well as meanings to each individual. Doctor could simply mean someone who holds a certain degree of education. Doctor is also defined as a physician or qualified practitioner. To this author, one cannot fulfill the word doctor without being a "Teacher." What good is knowledge if it is not shared with others? What is knowledge? How do the messages of some of the greatest doctors/teachers and healers throughout history all tie together? Can one individual really make a difference?

Dr. Walter Schmitt, Jr. references his previous remarks at the end of the 15th session Quintessential Application A(K) Clinical Protocol (QA) course by saying, "When we restore normal afferent activity, supply the appropriate nutrients, and administer to the mental and emotional environment, we pave the way for efficient cellular function and allow the optimal expression of our genetic potentials as individual humans. We have the opportunity to change the genetic potential to manifest ourselves and our patients at a higher level of our genetic potential; to achieve our human potential and then have those people we treat; have them interact with people at a more appropriate level, and a more efficient level, and then have them interact with patients; possibly sending us more patients to help improve those peoples' genetic potentials, and as people interact who are working more efficiently, they slowly, but surely the community starts to improve its function, and slowly but surely maybe the world starts to improve its function."

Once again, this brings forth the question: Can one person really make a difference in an entire population? The journey of this paper begins with this burning question and countless hours pondering how one can best serve humankind. The aforementioned words of Dr. Schmitt, one of the greatest doctors in history, continued to play in my head, bringing

forth many sleepless nights thinking about AK. The thought of Dr. George Goodheart, Jr. inevitably weighs in when thinking of AK. If anyone made an impact on a population surely Dr. Goodheart did, but how could we prove his impact?

Below (Fig. 1-3), is what I believe to be the effect of this one man and his healing love on an entire community. Detroit, Michigan was the home of Dr. Goodheart, one of the greatest healers ever known in the history of humankind. It is well known Detroit has been on a steady decline since the 1950's, so I wanted to see if my hypothesis held up according to the data. This is the declining population of Detroit, Michigan broken down per 10 years from two closely matching internet data sources.

1950-1960 population declined by 179,424

1960-1970 population declined by 158,662

1970-1980 population declined by 308,143

1980-1990 population declined by 175,393

1990-2000 population declined by 82,649

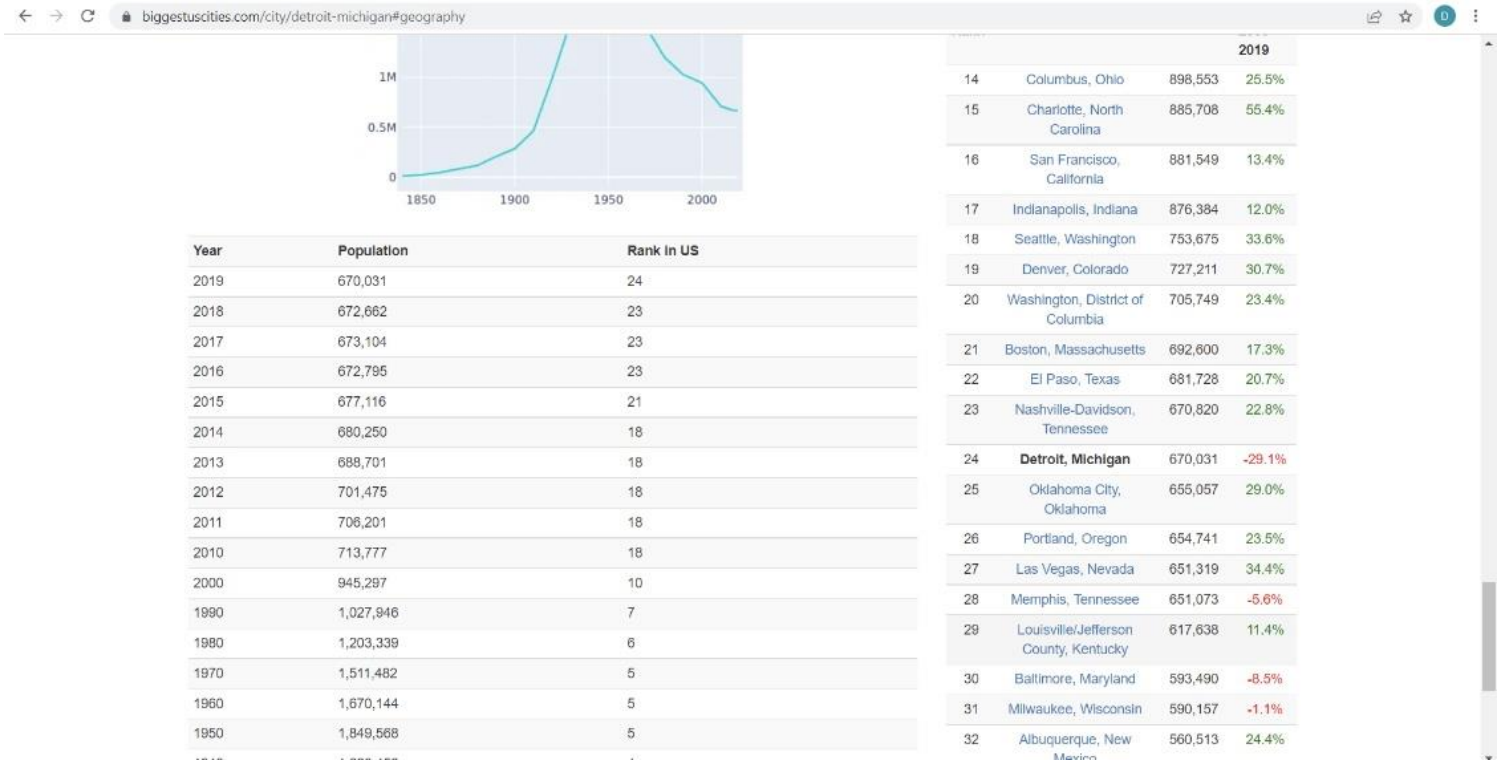
2000-2010 population declined by 231,520

2008-2010 population declined by 198,855

What does this mean? Dr. George Goodheart, Jr. passed away in 2008. One of the largest population declines in the history of Detroit happened in a short 1.5-year span after Dr. Goodheart's passing. This data confirms my hypothesis that Dr. Goodheart's loving energy held over 100,000 people together over this large region. His healing love was pulled from that region in mid-2008. Realistically, it would take a little time for a family to decide to move, then find a place to live in order to fully move from the area.

The data we have here is slightly incomplete as we do not have year-by-year breakdown of all years involved, however, this proves my hypothesis without digging into 2008 economics and all the details. Obviously, there was a large economic downturn that happened in 2008, however, as a mind-blowing thought, who's to say Dr. Goodheart's loss didn't contribute on even a bigger than imaginable scale? How amazing is it, that one person can potentially affect an entire community, which does impact the world? How can

we as individuals become exceptional healers and have as large or even larger impact than Dr. Goodheart?



Year	Population	Rank in US
2019	670,031	24
2018	672,662	23
2017	673,104	23
2016	672,795	23
2015	677,116	21
2014	680,250	18
2013	688,701	18
2012	701,475	18
2011	706,201	18
2010	713,777	18
2000	945,297	10
1990	1,027,946	7
1980	1,203,339	6
1970	1,511,482	5
1960	1,670,144	5
1950	1,849,568	5

		2019	
14	Columbus, Ohio	898,553	25.5%
15	Charlotte, North Carolina	885,708	55.4%
16	San Francisco, California	881,549	13.4%
17	Indianapolis, Indiana	876,384	12.0%
18	Seattle, Washington	753,675	33.6%
19	Denver, Colorado	727,211	30.7%
20	Washington, District of Columbia	705,749	23.4%
21	Boston, Massachusetts	692,600	17.3%
22	El Paso, Texas	681,728	20.7%
23	Nashville-Davidson, Tennessee	670,820	22.8%
24	**Detroit, Michigan**	670,031	-29.1%
25	Oklahoma City, Oklahoma	655,057	29.0%
26	Portland, Oregon	654,741	23.5%
27	Las Vegas, Nevada	651,319	34.4%
28	Memphis, Tennessee	651,073	-5.6%
29	Louisville/Jefferson County, Kentucky	617,638	11.4%
30	Baltimore, Maryland	593,490	-8.5%
31	Milwaukee, Wisconsin	590,157	-1.1%
32	Albuquerque, New Mexico	560,513	24.4%

(FIGURE 1)

biggestuscities.com

[redacted]

Year	Population	Rank in US
2019	670,031	24
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2012	701,475	18
2011	706,201	18
2010	713,777	18
2009	910,921	11
2008	912,632	11
2007	917,234	11
2006	918,849	10
2005	921,149	10
2004	924,016	10
2003	926,903	10
2002	928,870	10
2001	936,905	10
2000	945,297	10
1990	1,027,940	7

(FIGURE 2)

worldpopulationreview.com

Detroit Population by Year Show Sourcess

Year	Population	Growth	Annual Growth Rate
2021	664,139	-2,946	-0.44%
2020	667,085	-2,946	-0.44%
2019	670,031	-2,946	-0.44%
2018	672,977	-1,654	-0.25%
2017	674,631	-2,512	-0.37%
2016	677,143	-2,267	-0.33%
2015	679,410	-3,199	-0.47%
2014	682,609	-9,259	-1.34%
2013	691,868	-8,315	-1.19%
2012	700,183	-4,935	-0.70%
2011	705,118	-6,013	-0.85%
2010	711,131	-240,139	-2.87%
2000	951,270	-76,700	-0.77%
1990	1,027,970	-175,400	-1.56%
1980	1,203,370	-310,690	-2.27%
1970	1,514,060	-156,080	-0.98%
1960	1,670,140	-179,430	-1.02%
1950	1,849,570	226,120	1.31%
1940	1,623,450	54,790	0.34%
1930	1,568,660	574,982	4.67%
1920	993,678	527,912	7.87%

(FIGURE 3)

At Dr. Schmitt's last ICAK presentation he shared the words of the late, Dr. Goodheart, which gave us his final teachings on healing he privately shared with Dr. Schmitt, "All people are the same. Man is created in God's image. The doctor is no different from any of his patients. The work that a doctor does in figuring out what is wrong with a patient, especially with an AK doctor, is not for the benefit of the patient, but for the benefit of the doctor. For the benefit of the doctor in the sense to align the doctor with the patient or tune into his energy or whatever you want to say along those lines. So that the doctor may now, act as an intermediary between God and patient. He said, a true healer may simply touch a person and that person will get better, but most of us have to find a way to tune into the patient, so we may act as a conduit to the healing energy to the patient, and that's what AK helps us do."

Dr. Schmitt, "So, if that doesn't create humility, I don't know what will. Perhaps the goal of being any kind of doctor might be better to have the goal to become a healer!"

When Dr. Schmitt shared these words at the 2021 ICAK meeting, I was very excited, as they helped tie the concepts of healing together which I had been working on for many years. I had planned to share this presentation with Dr. Schmitt, instead of in his honor. Dr. Goodheart says, as healers, our job is simply to be a vessel for healing energy (God) and the patient. In order for us to better understand what this means we must dig much deeper and call upon more historical sources.

Healing is a difficult subject, and one into which I have put a great deal of study. I make no claims of being a great healer, however, who better to help teach a subject than someone who is a student of that subject? Everyone has special gifts granted to them at birth. Being a healer is not a natural gift for me, so I have invested a great deal towards the subject of healing since entering the chiropractic profession. This paper isn't to push any certain religious belief or practice on anyone. This is simply to share from the resources from which I am most familiar, and hopefully expand on what Dr. Goodheart had found to be true throughout his lifetime as a healer.

It would be difficult to discuss healing without thinking of the stories from the Bible about Jesus of Nazareth. It is a commonly accepted and historically documented fact that Jesus did exist and must have been some sort of magnificent healer. Again, this is not to get into religion, but rather to learn from purportedly the greatest healer of all time. After studying the Bible most of my life, I found that one of the greatest values to the Bible is held in the New Testament quotes of Jesus. Many books of the bible are written by different individuals on the same accounts of Jesus from a different first-hand perspective of each event. This adds validity to the accounts of Jesus's healings and teachings as we will see, and helps give even more depth to Dr. Goodheart's words.

From Genesis 1:27 The Bible says "So God created mankind in his own image, in the image of God he created them; male and female he created them.

1 John 4:16-17 The Bible says “God is Love.”

In Matthew 22:37-39 Jesus is quoted saying the greatest commandments we as humans can do is “Love the Lord your God with all your heart and with all your soul and with all your mind. This is the first and greatest commandment. And the second is like it: Love your neighbor as yourself.”

To summarize, the Bible says God equals love, it also says humans are made in God’s image. Therefore, all humankind equals love because God equals love. Jesus, the greatest healer in human history, says the most important two commandments we can do as humans is be in love with love (God), and be in love with giving love to others. Healing is reconnecting the patient back to the love of which we all are made.

Dr. Goodheart said many times “You do not have to do it; you have to let it! Find the need, supply the need, then observe the results.” One aspect of healing I believe even Dr. Goodheart sold short on was that a true healer may simply touch a person and that person will get better. This is a great goal for all of us, however, I believe most of us get in the way of healing because we think we actually have to physically do something in order for healing to take place. Jesus simply commands healing in many instances without touching anyone.

Luke 7:11-15 (Jesus Raises a Widow’s Son) “Soon afterward, Jesus went to a town called Nain, his disciples and a large crowd went along with him. As he approached the town gate, a dead person was being carried out-the only son of his mother, and she was a widow. And a large crowd from the town was with her. When Jesus saw her, his heart went out to her and he said, “Don’t cry.” Then he went up and touched the bier they were carrying him on, and the bearers stood still. He said, “Young man, I say to you, get up!” The dead man sat up and began to talk, and Jesus gave him back to his mother.

John 5:6-8 (Jesus Heals an Invalid Man) “One who was there had been an invalid for thirty-eight years. When Jesus saw him lying there and learned that he had been in this condition for a long time, he asked him, “Do you want to get well?” “Sir,” the invalid replied, “I have no one to help me into the pool when the water is stirred. While I am trying to get in, someone else goes down ahead of me.” Then Jesus said to him, “Get up! Pick up your mat and walk.” At once the man was cured; he picked up his mat and walked.

Luke 6:10 (Jesus Heals a Man’s Crippled Hand) “Jesus looked around at them all, and then said to the man, “Stretch out your hand.” He did so, and his hand was completely restored.”

Mark 3:5 (Jesus Heals a Man’s Crippled Hand) “Jesus looked around at them in anger and, deeply distressed at their stubborn hearts, said to the man, “Stretch out your hand.” He stretched it out, and his hand was completely restored.”

Matthew 12:13 (Jesus Heals a Man's Crippled Hand) "Then Jesus said to the man, "Stretch out your hand." So he stretched it out and it was completely restored, just as sound as the other."

Luke 17:11-14 (Jesus Heals Ten Men With Leprosy) "Now on his way to Jerusalem, Jesus traveled along the border between Samaria and Galilee. As he was going into a village, ten men who had leprosy met him. They stood at a distance and called out in a loud voice, "Jesus, Master, have pity on us!" When Jesus saw them, he said "Go, show yourselves to the priest." And as they went, they were cleansed."

Luke 5:12-13 (Jesus Heals a Man With Leprosy) "While Jesus was in one of the towns, a man came along who was covered with leprosy. When he saw Jesus, he fell with his face to the ground and begged him, "if you are willing, you can make me clean." Jesus reached out his hand and touched the man. "I am willing," he said. "Be clean!" And immediately the leprosy left him."

Mark 1:40-42 (Jesus Heals a Man With Leprosy) "A man with leprosy came to Jesus and begged him on his knees, "If you are willing, you can make me clean." Jesus was indignant. He reached out his hand and touched the man. "I am willing," he said. "Be clean!" Immediately the leprosy left him and he was cleaned."

Matthew 8:1-3 (Jesus Heals a Man With Leprosy) "When Jesus came down from the mountainside, large crowds followed him. A man with leprosy came and knelt before him and said, "if you are willing, you can make me clean." Jesus reached out his hand and touched the man. "I am willing," he said. "Be clean!" Immediately he was cleansed of his leprosy."

Luke 4:38-39 (Jesus Heals a Woman With Fever) "Jesus left the synagogue and went to the home of Simon Peter. Now Simon's mother-in-law was suffering from a high fever, and they asked Jesus to help her. So he bent over her and rebuked the fever, and it left her. She got up at once and began to wait on them."

Mark 1:29-31 (Jesus Heals a Woman With Fever) "As soon as they left the synagogue, they went with James and John to the home of Simon and Andrew. Simon's mother-in-law was in bed with a fever, and they immediately told Jesus about her. So he went to her, took her hand and helped her up. The fever left her and she began to wait on them."

Matthew 8:14-15 (Jesus Heals a Woman With Fever) "When Jesus came into Peter's house, he saw Peter's mother-in-law lying in bed with a fever. He touched her hand and the fever left her, and she got up and began to wait on them."

John 4:49-50 (Jesus Heals a Boy From Afar) "The royal official said, "Sir, come down before my child dies." "Go," Jesus replied, "your son will live." The man took Jesus at his

word and departed. While he was still on the way, his servants met him with the news that his boy was living. When he inquired as to the time when his son got better, they said to him, "Yesterday, at one in the afternoon, the fever left him." Then the father realized that this was the exact time at which Jesus had said to him, "Your son will live."

Matthew 10:5-8 "Jesus sent out with the following instruction: "Go, as you go, proclaim this message: 'The Kingdom of God (Love) has come near.' Heal the sick, raise the dead, cleanse those who have leprosy, drive out demons. Freely you have received, freely you shall give."

John 14:12,14 Jesus said, "Very truly I tell you, whoever believes in me will do the works I have been doing, and they will do even greater things than these, because I am going to God…You may ask me for anything in my name, and I will do it."

From these examples of healing, we can see multiple accounts of Jesus merely commanding the person to be well with his healing love and healing takes place. In Jesus's teachings of healing, he says we should all be able to do anything he has done and even greater works if we follow his instruction. So freely we have received, so freely shall we give. Healing love is ours to command. Could the words we speak to our patients be more important and powerful than we know? Maybe Dr. Goodheart was the greatest healer according to Dr. Schmitt, because he also stated, Dr. Goodheart had the greatest bedside manner of anyone he had ever known. I believe that the more love with which we fill ourselves, the more powerful healers we can become.

The question thus becomes: How can we wield love to become better healers?

Dr. David Hawkins, psychiatrist, authored "Power VS Force: An Anatomy of Consciousness (The Hidden Determinants of Human Behavior)". Much of Dr. Hawkins life's work was inspired from Dr. Goodheart's muscle testing observations. Dr. Hawkins believed muscle testing could be used not only to ask the patient questions, but also be utilized on a more energetic level, to ask for universal truths.

According to Dr. Hawkins each level of human consciousness rates on a scale from 0-1000. Each emotional state can be rated on this scale, and emotions that calibrate below 200 are considered as having a negative impact on society (Fig. 4).

MAP OF CONSCIOUSNESS

God-view	Life-view	Level	Log	Emotion	Process
Self	Is	**Enlightenment**	700-1000	Ineffable	Pure Consciousness
All-Being	Perfect	**Peace**	600	Bliss	Illumination
One	Complete	**Joy**	540	Serenity	Transfiguration
Loving	Benign	**Love** (LWP) ↑ Appreciation	500	Reverence	Revelation
Wise	Meaningful	**Reason**	400	Understanding	Abstraction
Merciful	Harmonious	**Acceptance**	350	Forgiveness	Transcendence
Inspiring	Hopeful	**Willingness**	310	Optimism	Intention
Enabling	Satisfactory	**Neutrality**	250	Trust	Release
Permitting	Feasible	**Courage**	200	Affirmation	Empowerment
Indifferent	Demanding	**Pride**	175	Scorn	Inflation
Vengeful	Antagonistic	**Anger**	150	Hate	Aggression
Denying	Disappointing	**Desire** Lust / (LWP) ↓ passionate	125	Craving	Enslavement
Punitive	Frightening	**Fear**	100	Anxiety	Withdrawal
Disdainful	Tragic	**Grief**	75	Regret	Despondency
Condemning	Hopeless	**Apathy**	50	Despair	Abdication
Vindictive	Evil	**Guilt**	30	Blame	Destruction
Despising	Miserable	**Shame**	20	Humiliation	Elimination

(FIGURE 4)

From Dr. Hawkins's work, we can see that when a person has the "courage" to go outside of themselves and change their focus towards producing something for others instead of only on themselves, they can become a positive influence on society. He states that

unconditional “love” is a very powerful level of consciousness; one which the vast majority of people will never reach. However, we can obviously feel and use “love” quite easily as a common emotion.

From Jane Thurnell-Read’s (Life-Work Potential (Now Ergopathics Canada) “Emotional State Composites” electromagnetic signature kit, we can see similar correlations to Dr. Hawkins’s work (Fig. 5-8). From (Fig. 4&7) we see Jane Thurnell-Read places “appreciation” on the same level as “love”. According to Jane’s work, the word “appreciation” should be interchangeable with “love”. Jane groups “desire” and “craving” together under the heading of “lust” as well as “passionate”, which we will touch on a bit later.

Dr. John Schmitt developed the “Heart-Focused Technique(s)” quoted in the QA protocol. With the Heart-Focused Technique(s), the patient is asked to hold all their attention on their heart and fill their heart with a feeling of “appreciation.” This is based on research from the Institute of HeartMath® and other studies, which show healers should place their healing thoughts from their own hearts, which can and will amplify that healing energy.

Focusing appreciation through the heart of the practitioner and into the patient is a proven healer. This is such a powerful tool. The QA protocol recommends not having the patient do this technique too early during the course of treatment or it will mask many other findings. Given that the feeling of appreciation is equal to love, it is yet another example of love being a healer.

EMOTIONAL STATE COMPOSITES TEST KIT (10 vials)

Product Code 8084

This is a kit of ten primary or broad spectrum emotions or states of being. There are thousands of different emotions, so making an emotions test kit was always going to be a challenge. The way in which these countless emotions have been distilled and categorised has been established through kinesiology testing and the study of anthropological and psychological research in this field.

Each of these ten emotional states encompasses many other emotions of varying intensity. The other emotions are listed under the primary emotion.

Positive emotions can be as problematic as negative emotions. People can feel they do not deserve to be happy or that happiness is boring. Some people will have problems with two opposing emotions, for example, happiness and sadness, and vacillate between them, sabotaging themselves when they are happy and when they are unhappy.

It could be argued that there are no completely positive or negative emotional states; emotions are appropriate or inappropriate depending on circumstances and in order to be full human beings we need to be able to experience all the emotions at different times.

EC 01 Anger

Abrasive	Enraged	Irate	Rough
Abhorrent	Exasperated	Irritable	Rude
Abusive	Explosive	Irritated	Ruthless
Acrimonious	Fed-up	Jealous	Sabotaging
Aggressive	Ferocious	Livid	Sadistic
Aggrieved	Fierce	Mad	Sarcastic
Angry	Fiery	Malevolent	Savage
Annoyed	Forceful	Mean	Seething
Antagonistic	Frustrated	Merciless	Severe
Antagonized	Fuming	Murderous	Sharp
Argumentative	Furious	Offended	Simmering
Belligerent	Gruff	On fire	Smouldering
Bitchy	Grumpy	Out of sorts	Spiteful
Bitter	Hard	Outraged	Steely
Boisterous	Harsh	Peevish	Stern
Brutal	Hated	Petulant	Stubborn
Bugged	Heartless	Piqued	Sulky
Bullying	Hostile	Pissed off	Sullen
Burning	Hot-headed	Pissed (American	Unkind
Caustic	Hot-tempered	usage)	Unrelenting
Chaotic	Impatient	Pushy	Up in arms
Choleric	Ill humoured	Upset	Resentful
Cranky	Ill tempered	Pitiless	Vehement
Cross	Incandescent	Provoked	Vengeful
Cruel	Incensed	Quarrelsome	Vicious
Cynical	Indignant	Raging	Violent
Defensive	Inflamed	Raving	Wicked
Defiant	Infuriated	Rebellious	Wild
Demanding	Inhuman	Resentful	Wilful
Destructive	Insensitive	Resistant	Wrathful
Disgusted	Insulted	Revolted	
Displeased	Irascible	Riled	

EC 02 Apathy

Alienated	Dead	Doomed	Impassive
Alone	Defeated	Drained	Inattentive
Bored	Depressed	Emotionless	Indifferent
Cold	Demoralised	Empty	Lazy
Cool	Discouraged	Helpless	Listless
Cut off	Disillusioned	Hopeless	Lost

(FIGURE 5)

Low	Pointless	Stuck	Unresponsive
Negative	Powerless	Tired	Useless
Numb	Resigned	Unemotional	Vague
Overwhelmed	Self-defeating	Unfeeling	Worthless
Passive	Spaced out	Unfocussed	
Pessimistic	Spacey	Uninterested	
Phlegmatic	Stoned	Unmoved	

EC 03 Bravery

Adventurous	Creative	Focussed	Resolute
Alert	Courageous	Gallant	Secure
Alive	Daring	Heroic	Self-sufficient
Anticipating	Decisive	Honourable	Strong
Assured	Determined	Independent	Sure
Aware	Dynamic	Intrepid	Tireless
Bold	Eager	Invincible	Undaunted
Brave	Enthusiastic	Motivated	Valiant
Capable	Excited	Optimistic	Vigorous
Centred	Exhilarated	Plucky	Visionary
Certain	Exuberant	Positive	Willing
Competent	Fearless	Purposeful	Zealous
Confident	Flexible	Resilient	

EC 04 Fear

Afraid	Distraught	Intimidated	Soft
Alarmed	Distressed	Irrational	Stressed
Anxious	Distrustful	Miserable	Sullen
Apprehensive	Disturbed	Mixed-up	Superstitious
Ashamed	Doubtful	Nervous	Suspicious
Awed	Dread	Neurotic	Tense
Baffled	Edgy	On edge	Terrified
Bashful	Embarrassed	On tenterhooks	Threatened
Bewildered	Evasive	Over-wrought	Timid
Bothered	Faint-hearted	Panicky	Tormented
Careful	Fearful	Panic-stricken	Torn
Cautious	Foreboding	Paralysed	Trapped
Clammy	Frantic	Paranoid	Trembling
Concerned	Frightened	Perplexed	Troubled
Conflicted	Guilty	Perturbed	Uncertain
Confused	Hesitant	Petrified	Uncomfortable
Cowardly	Horrified	Phobic	Uneasy
Cowed	Hot and bothered	Puzzled	Upset
Cowering	Humiliated	Reluctant	Vulnerable
Cringing	Hysterical	Restless	Wary
Daunted	Ill at ease	Scared	Weak
Defensive	In suspense	Shaky	Worried
Degraded	In turmoil	Shocked	Yellow
Dismayed	Inhibited	Shy	
Dissatisfied	Insecure	Skulking	

EC 05 Happiness

Affable	Alert	Amiable	Animated
Agreeable	Alive	Amused	Anticipating

(FIGURE 6)

Appreciated	Enthusiastic	Joyful	Pleasant
Blessed	Excited	Joyous	Pleased
Blissful	Exhilarated	Jubilant	Radiant
Blithe	Exuberant	Kind	Rapt
Carefree	Floating on air	Kindly	Relieved
Charming	Full of life	Laughing	Satisfied
Cheerful	Gay	Light-hearted	Sparkling
Comfortable	Glad	Lively	Sunny
Congenial	Glowing	Merry	Sure
Content	Good-humoured	Nonchalant	Sweet
Contented	Good-natured	On cloud nine	Sweet-tempered
Delighted	Gratified	Optimistic	Thrilled
Eager	Happy	Overjoyed	Vibrant
Ecstatic	Happy-go-lucky	Over the moon	Vital
Elated	Hopeful	Patient	Vivacious
Encouraged	Jolly	Playful	Worthy

EC 06 Love

Accepted	Cherished	Gentle	Nurtured
Accepting	Cherishing	Helpful	Nurturing
Adored	Comforted	Included	Obsessed
Adoring	Compassionate	Including	Respected
Affectionate	Connected	Intimate	Savoured
Appreciated	Consoled	Light-hearted	Smitten
Aroused	Consoling	Liked	Sociable
Blessed	Doting	Lovable	Tender
Blissful	Embraced	Loved	Warm
Cared for	Enchanted	Lovely	Welcoming
Caressed	Esteemed	Loving	
Caring	Friendly	Needed	

EC 07 Lust

Ardent	Gluttonous	Miserly	Scheming
Carnal	Greedy	Obsessed	Selfish
Compulsive	Hungry	Over indulgent	Sensual
Craving	Impatient	Passionate	Sexy
Demanding	Lascivious	Possessive	Voracious
Desiring	Lecherous	Predatory	Wanton
Driven	Libidinous	Pushy	Wicked
Envious	Licentious	Randy	
Fixated	Lustful	Raunchy	
Frustrated	Manipulative	Reckless	

EC 08 Peace

Abundant	Beautiful	Easy	Placid
Accepted	Benevolent	Easy-going	Quiet
Amicable	Benign	Harmonious	Relaxed
Appreciative	Blissful	Intuitive	Restful
At ease	Blithe	Light	Serene
At peace	Calm	Mellow	Still
Balanced	Carefree	Peaceful	Tranquil

(FIGURE 7)

Undisturbed	Unruffled	Untroubled

EC 09 Pride

Aloof	Egotistical	Patronising	Snooty
Arrogant	Gloating	Pious	Special
Bigoted	Haughty	Prejudiced	Stoical
Boastful	Hypocritical	Proud	Stubborn
Clever	Icy	Righteous	Stuck up
Closed	Imperious	Rigid	Supercilious
Complacent	Inflexible	Self-important	Superior
Conceited	Isolated	Self-respecting	Uncompromising
Contemptuous	Judgemental	Self-satisfied	Unfeeling
Critical	Narrow-minded	Selfish	Unforgiving
Disdainful	Opinionated	Smug	Unyielding
Dogmatic	Overbearing	Snobbish	Vain

EC 10 Sadness

Abandoned	Doleful	Needy
Abject	Down	Oppressed
Abused	Downcast	Pathetic
Anguished	Downhearted	Pensive
Upset	Forlorn	Pessimistic
Apologetic	Gloomy	Pitiable
Bad	Glum	Pitiful
Bereaved	Grey	Regret
Betrayed	Grief-stricken	Rejected
Bleak	Grieving	Remorseful
Blue	Grim	Sad
Broken hearted	Guilty	Sombre
Burdened	Heart broken	Sorrowful
Cheated	Heavy	Sorry
Dark	Heavy-hearted	Stuck
Defeated	Hurt	Suicidal
Dejected	In despair	Tearful
Desolate	In the dumps	Tormented
Despairing	Inadequate	Tortured
Desperate	Inconsolable	Undeserving
Despondent	Inept	Unfortunate
Disappointed	Insignificant	Unhappy
Disconsolate	Introverted	Unloved
Discontented	Joyless	Unpleasant
Discounted	Left out	Unsmiling
Discouraged	Lonely	Unwanted
Disgruntled	Longing	Upset
Disheartened	Martyred	Vulnerable
Distraught	Regretful	Wistful
Dismal	Melancholic	Woebegone
Dispirited	Miserable	Wounded
Displeased	Morose	Wretched
Dissatisfied	Mournful	

(FIGURE 8)

According to the 1989 book “Healers on Healing,” the common denominator in healing is beyond any doubt, Love. This book is written in small sections by over 30 different healers throughout the world, including Dr. Goodheart.

Dr. Richard Belli often describes becoming a better doctor and healer as a perpetual cycle of learning. He says as the doctor gains more knowledge and is able to help more people, then the patients seeking care increase in difficulty, the doctor then needs to increase their knowledge to help those people, and as the doctor gains more knowledge to help those people their spectrum of healing grows wider and wider. Dr. Goodheart used to similarly summarize by saying, a doctor needs to catch on fire as a knowledgeable healer and people will come from miles around to watch you burn.

The purpose of giving these examples is to help us find common denominator truths across many resources and lifetimes of work. How can we become better healers? A healer needs to be filled with so much healing love that they can radiate love to a patient, and restore that person to love, from which humankind is created. Many healers need to touch patients or have techniques in place to allow that healing love transfer to take place. However, healing may also be accomplished by simply commanding it to be so.

How can we fill ourselves with love and become more powerful healers? From this author’s study and observation, I believe knowledge is love. How could knowledge not be considered a labor of love? How many hours of condensed lifetimes of knowledge go into each book, paper, seminar etc.? Even if someone contributes to a knowledge base in an unloving way, the truth that persists will still be love.

I used to think, as many of us do, the information we learn will be what heals the patient. However, why is it that the more we learn, the more patients come to us already healed? This is because we have filled ourselves with labors of love (aka knowledge). The healing is allowed to take place because we have filled ourselves with that love.

As a chiropractic graduate of 2014, this I can clearly see, as I attain more knowledge, I have more patients walk into the office saying some variance of the below examples:

-I was going to come a while back, but whenever I decided to finally call, I didn’t hurt anymore so I put off coming to see you.

-This has been bothering me for a few weeks, but today it doesn’t hurt.

-Of course, this has been hurting, but today it’s fine since I’m here.

This isn’t knowledge itself making patients feel better, this is healing love being transferred to the patient. The knowledge has yet to be applied at that point. However, knowledge, which is love, is able to act as an intermediary between patient and doctor as Drs. Goodheart and Belli both describe.

To become better healers, we need to fill ourselves with knowledge (labors of love) and project that love through our hearts. This is why the ICAK is so important. The ICAK is the world's leading source of loving knowledge dedicated to the betterment of humankind. Referring to (Fig.4 (see above)) from Dr. Hawkins's chart and combined sketched figures from Jane Thurnell-Read's (LWP) work, we can see "Hate" (calibration equals 150) is a more positive emotional state, according to Dr. Hawkins's calculation, than "Passionate" (calibration equals 125). These are both negative levels of consciousness (below 200), however, we can clearly see someone can do more harm being passionate about something than hating that same thing. It is better to hate AK than be passionate about AK, how powerful is this thought? In today's society, passion is many times confused with love. These terms are often used interchangeably with devastational difference. As healers, we need to ensure we are full of love and not passion, knowledge not lust, and above all, love for ourselves and our fellow humankind.

Now that we see how to exercise healing love, how can we, as individuals, not only impact entire regions, but help heal the world? We accomplish this together, as an organization, through the ICAK. We are a family, and the world needs our healing. As Dr. Schmitt said at his last ICAK meeting presentation, "Perhaps the goal of being any kind of doctor might be better to have the goal to become a healer!"

"You don't have to tell how you live each day; You don't have to say if you work or you play; A tried, true barometer serves in the place—However you live, it will show in your face. The false, the deceit that you bear in your heart, Will not stay inside where it first got a start; For sinew and blood are a thin veil of lace—What you wear in your heart, you wear in your face. If your life is selfless, if for others you live, For not what you get, but how much you can give: If you live close to God in his infinite grace—You don't have to tell it, it shows in your face." - Author unknown

Conclusion

Dr. Walter Schmitt, Jr. who dedicated his entire being towards the healing love of humankind. The best student anyone has ever spoken to, the greatest doctor/teacher to lecture, the kindest ear anyone has ever spoken to, the gentlest titan to ever live, and true healer to humankind. God/Love- “Well Done, My Good and Faithful Chiropractic Servant.”

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Lymphatic System: The Forgotten System Specialty

Paul T. Sprieser, BS, D.C., DIBAK

Abstract

This system is studied by everyone in the health care professions, yet it does not appear that anyone takes it on as a specialty. It is closely related both anatomically and functionally to the cardiovascular system. But more importantly it is connected to the body's immune system, along with the spleen and thymus gland. This data review paper examines why it is essential to correct any muscular weakness in the lower extremities that assist in the return of lymph to general circulation.

Key Indexing Terms
Lymphatic System, Brain, Brain Disease, Thoracic

Introduction

With a recent discovery the Lymphatic System (LS) has taken center stage in the health care, with the discovery that the brain has its own LS. This fact was just recently discovered and I came upon it in the health section, 5/25/17 of the Newark Star Ledger, reported by David Kohn, Washington Post.[1]

The original statements about the lymphatic system from Guyton's Physiology stated that all tissues of the body, with the exception of a very few, have lymph channels, that drain excess fluid directly from the interstitial spaces. The exceptions include the superficial portions of the skin, the central nervous system, deeper portions of the peripheral nerves, the endomysium of muscles and the bones.[2] University Of Virginia School Of Medicine have determined that the brain is directly connected to the immune system by vessels previously thought not to exist.[3] The significance of this discovery, which has been identified in human samples might explain the pathophysiology of many brain diseases such as multiple sclerosis (MS), autism and Alzheimer's disease as well as brain trauma from concussions.[4]

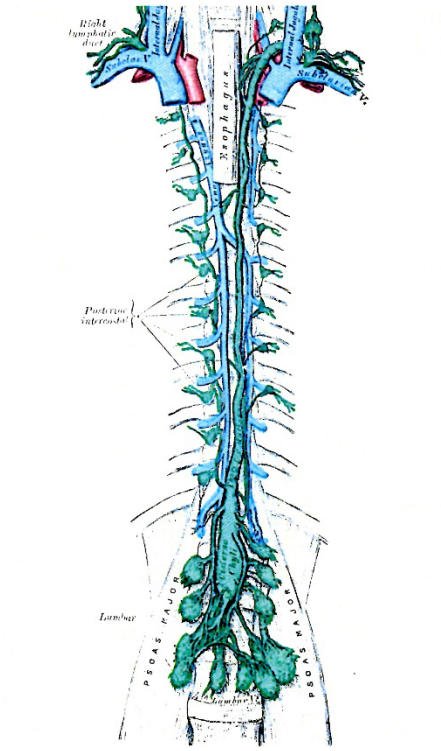
In the past 158 years there have only been a few new anatomical discoveries, a new division of a muscle of mastication with a suggested name of sphenomandibularis,[5] a patch of fibrous tissue near the top of the spinal column that linked a neck muscle to the dural matte.[6] And finally the discovery that the brain has its own lymphatic system.[7,8]

The lymphatic system has been well documented in Gray's Anatomy as far as location and Guyton's Physiology its function, and its workings are more a passive system on picking up interstitial fluids and mopping up waste and fats. Its immune function is to fight infections. The movement of lymph is due to muscular action on the channels and nodes in the extremities. The return flow is in one direction back to the venous return in the

subclavian vein at the base of the neck and shoulder. The movement in the torso and abdominal areas are also due to core muscle along with diaphragmatic respiratory actions and the skeletal movement that the produce the flow of lymph upwards. This internal cause motional changes of organ and tissue pressure applied to these vessels.[9,10]

Discussion

A brief review of this system is necessary to understand how this applied to the brain lymphatic system. The movement of lymph is by way of contraction of the intrinsic muscle in the presence of one-way valves exerts a pumping action on the lymph. This is supplied by the surrounding muscle masses in the extremities as well as the core musculature assist lymphatic pumping flows from the thoracic duct terminus is 50 to 100cc per hour at resting pressure of zero. The pressure rises with exercise, increase in body temperature and alimenation.[10]



The above Illustration appears in 1962 and again in the 1974 Gray's Anatomy fig. 337, page 624 titled THE THORACIC AND RIGHT LYMPHATIC DUCT. Permission granted from Running Press, Philadelphia Pennsylvania.

The makeup can be described in the following way: 1.) external capillary networks which collects lymph in varies organs and tissues, 2.) elaborate system of collecting vessels which carry lymph from lymphatic capillaries to blood stream, which empty into the great vein at the root of the neck, 3.) the rounded bodies called lymph nodes that act like filter in-the path of the collecting vessels, 4.) other organs such as tonsils, spleen and thymus.[9]

The lymphatic capillaries and collecting vessels line throughout the layer of endothelial cells form a closed system. Special attention should be made to the small intestine collection system of lacteals or chyliferous vessels as well as peyer's patches.

The function lymphatic system is to guard the body from infection form pathogens that may enter and be carried in the lymphatic vessels. The other very important function is to return the leakage of interstitial fluid to the blood vessels. This leakage is due to the difference of the osmotic pressure of electrolytes as well as proteins carried in the blood stream. The larger molecules are easily carried because of the loosely attachment of cells that overlap. This system of interstitial fluid pressure is greater outside these pressures (hydrostatic and colloidal osmotic), causing the cells to act as a one-way valve. Guyton's states "this is an essential function of the interstitial space and the lymphatic capillaries. If it ceases to function we would die within two hours.

The anatomical makeup of the lymphatic system in the trunk area of the body explains how this system operates. The thoracic duct which is on the left side is the largest, it arises from the cistern chyli, which is a saclike enlargement that lies in front of the upper lumbar portion of the vertebral column close to the celiac artery. This dilated sac is at the lower end of the thoracic duct and is located retroperitoneal and posterior to the abdominal aorta on the vertebral bodies of the first and second lumbar vertebrae, and passes through the aortic opening of the diaphragm up to the junction of the left subclavian and internal jugular veins.[9,10]

Location of the cisterna chyli is not by accident but rather by design. As Dr. Goodheart would say "Why Is That", meaning was is it the way it is being observed. It is located at the junction of the thoracic and abdominal region which creates the natural change in spinal curves with the apex at T12, which is at the region of the cisterna chyli located at the bodies of the L1 and L2 lumbar vertebra located just lateral to the lymphatic sacs of this system on the psoas muscles, and all located retroperitoneal which creates greater tension on this organ. You can see that the diaphragm attaches at the posterior region at T12 and it muscular/tendentious insertion attach just below by way of the right and left crus into the L1 and L2 vertebra. Why do you think this occurs? Simply stated that structure and function go hand in hand. This location allows the pumping action of breathing and effects of the diaphragm respiratory movement of breathing to pump and move the lymph back into the venous return at the subclavian veins at the junction of
the cervical/thoracic region. If the venous return blood flow is slowed which occurs when the rib cage is fixated and the pectoralis minor muscle allows the tissues that surround the subclavian vein this will slow or stop the lymphatic return and effect both the cranial (brain) and bodily returns. This leads to a buildup of toxin and waste products of metabolism of the brain in the cerebral tissues and in the CSF. These must be carried off by venous return and the brain lymphatic vessels.

The idea that many of the brain diseases could be connected to accumulation of toxin and biological waste has could account for many brain diseases that have been reported for the past eight or ten years. Dr. Paolo Zamboni, an Italian vascular surgeon from Ferrara, Italy who, has been studying and researching MS for twenty-five years, because his wife had

MS. He noted that she and 65 other MS patients he was treating all had a vascular abnormality that he called Chronic Cerebrospinal Venous Insufficiency (CCSVI). His study was published in the Journal of Vascular Surgery in 2009. Because of his observation another double-blind study was financed by the M.S. Society with the New York State University at Buffalo, led by Robert Zivandinov, MD. This study found a higher rate of CCSVI in the MS patients at a rate of 56% then in the health control group. Since 2009 122 studies have been published on this matter and appears in PubMed of the NIH.

This waste accumulation has been suggested to be connected to many types of neurological disease besides MS, such as Parkinson's, Alzheimer's, Amyotrophic Lateral Sclerosis (ALS), Huntington Chorea, and Traumatic Brain Injury (TBI). This has become more apparent since the discovery of the brain lymphatic system also known as the glymphatic system, the "g" stand for glia cells. This coupled with the venous return form the brain and the reabsorption of the Cerebral Spinal Fluid (CSF).

Another factor that is being researched is the genetic factor of the E4-allele of apolipoprotein E, (Apo E4 gene). This is being studied by Dr. Trumble showing in a Bolivia settlement of Tsimane, showing that parasitic infection of Leishmania is used to create protection from dementia. Dr. Francisco Lopera, a neurologists from Antioquia, Colombia, South America, who has been treating families from this region with a specific genetic mutation on chromosome 14, a tiny flaw in the DNA, that creates a 50/50 chance of early onset Alzheimer's.[13,14,15]

Drs. Lopera and Kosik have been with grants from the NIH, looking for drugs that might prevent the accumulation of the beta amyloid proteins that lead to the tangles that seem to cause Alzheimer's disease.[11,12]

The title of this paper "The Lymph System the Forgotten System", seems to make this system the orphan system. What I mean by the orphan system it has no (parents), there are no specialist for this system. Each organ system in medicine has it specialists, heart/cardiologist, lung/pulmonologist, nervous system/neurologist, but the lymphatic system no lymphologist. The medical doctors who deal with this system are treating diseases such as cancer or infectious diseases such a Lyme or parasitic infections. The only doctors who look at this system in its anatomical and structural function are Chiropractors and Osteopathic Physicians and complementary care systems of message to maintain and improve it functions.

Anatomical Components

What must be considered with the lymphatic system besides the specific lymphatic capillary channels are lymph nodes as well as the cisterna chyli and the right and left thoracic ducts. The skeletal structures are the lower thoracic and upper lumbar spine along with the ribcage. The musculoskeletal system that must be considered are psoas major, iliacus, quadratus lumborum, abdominal, diaphragm, and most important is the pectoralis minor.[16,17,18]

The lymphatic system is mainly a passive system that depends on the action of muscle, respiratory action of breathing, contraction of skeletal muscles especially below the neck region, as well as pumping action of the heart and arterial blood vessels, to pump the lymph to the cisterna chyli and up the left thoracic duct. This represents approximately 75% of the body lymphatic drainage.

The area of the head and brain drainage will occur for respiratory and vascular action on the dura mater which cause a pulling and contraction due to the osseous connection within the skull at the foramen magnum and connection to C1-C2 and C3 in the spinal canal. There is a loose connection from C4 down to L2 by way of the dentate ligaments, and a firm connection of the dura in the sacral canal at S2 on the anterior wall, the final connection is at the coccyx by way of the filum terminale.

Guyton's lists six extrinsic factors for the movement of lymph: 1) muscular activity, 2) passive movement, 3) pulsation of blood vessels, 4) motility of the intestinal tract, 5) venous pressure, and 6) gravity.[19] What appears to be overlooked is the respiratory excursion of the diaphragm and the lungs have pressure changes both in the thoracic and abdominal cavity. This action is carried over to the spine because the rib is attached to the twelve thoracic vertebra and cause movement during inspiration, which cause the ribs to expand lateral and upward. On expiration we see the ribs moving caudally (downwards) and the lateral border moving medially.[2]

The anatomical structures of the body that must be evaluated and stabilized are taken Guyton's list of six extrinsic factors.

1. Muscle activity which can be measured by Manual Muscle Testing of lower extremities (thigh and calf), pelvis and thoracic for core muscles, as well as upper extremities (arm and forearm), and neck region. The use of the Chapman reflexes known as Neurolymphatic (NL) reflexes, which improve lymphatic flow both skeletal muscles if they are found weak and the associated organ system.

2. Passive movement which consist of the breathing action and the movement of the diaphragm, which assist the lymphatic movement upward from the cisterna chyli in the abdominal cavity into the thoracic cavity. This would be assisted by expansion and contraction of the rib cage along with pressure exerted by the lung action on the left thoracic duct.

3. Pulsation of blood vessels consisting of muscular action of the arterial vessels for the pumping action of the heart will assist the valve system in the lymph vessels to move the lymph flow back to the subclavian vein in the junction of neck and shoulder.

4. Intestinal tract motility helps in the absorption of fats for the digestive system and return of lymph to the liver for processing.

5. Venous pressure which helps in the collection of the interstitial fluid and return them to the blood stream. The main flow at the thoracic duct terminus is 50 to 100 cc per

hour at arresting pressure of zero. This represents at least 75% of the lymphatic return of the body. This can increase with exercise, body temperature, and alimentation. This can be assisted with the use of an inversion table, as well as message and pressure fabric used in the arms and leg region. The use of a percussion instrument can be a useful tool.

Conclusion

The Importance of the lymphatic system cannot be over stated, Guyton Physiology quote the following "this is essential function of the interstitial space and the lymphatic capillaries, if it ceases to function we would die within two hours. Carrying this forward to the fact, the brain has a lymphatic system could connect this to many brain diseases is now being questioned.

It is essential that we correct any muscular weakness in the lower extremities that assist in the return of lymph to general circulation. Special evaluation of the diaphragm for fixation and hiatal hernia and the correction of the Yaw #2 modular distortion pattern is essential for the working of this system. Most importantly the pectoralis minor muscle should be evaluated for any involvement, such as weakness, facial shorting and the strain counter-strain patterns, which will cause a slowing down of the venous return at the subclavian vein. This in turn cause a backup of traffic in the entry point to the vascular system effecting the entire body.

Finally, all cranial fault patterns should be corrected to allow for proper CSF production, circulation and reabsorption. This will allow the removal of metabolic waste from the brain and to allow the effect of Dural respiratory action and its effect on the brain's lymphatic system.

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Observations on Some Individuals from the mRNA Covid Vaccine and Covid Infection

David Leaf, D.C., DIBAK

Abstract

The paper discusses negative reactions of some of the patients encountered in the office over the last 15 months. It describes a novel approach that helped to normalize musculoskeletal imbalances in these individuals.

Key Indexing Terms
Covid, mRNA, Side Effects, Muscle, Coordination

Discussion

In the last 15 months, over 150 patients have presented with neuromuscular defects related to the first, second, or booster dose of Moderna or Pfizer vaccine against Covid or having had Covid after having the vaccine or having the latest version. This paper describes the symptoms and a possible treatment that negates some of these deficits.

Last June, an 84-year-old was brought in for treatment by her daughter, a patient. The older woman had suddenly had trouble walking and could not stand up without assistance. Testing showed inhibition of all four extremities, coordination problems, and abysmal handwriting.

I have used brain activity to see if the symptoms could be reversed in the past. First, I ask the patient to count and hum to see if there is any change in muscle strength. The right extremities tested stronger while counting and the left extremities while humming. I then asked her to sing, but the only song she could think of was "you are my sunshine," which were the only words. Finally, I asked her to try and stand up while singing, and she immediately stood up, which she could not do without assistance.

I then asked the daughter when the decline in her ability occurred, and he related this was four days after the second Modena vaccine. She was then able to walk out of the treatment room while singing without help. She returned three weeks later and no longer needed to sing. I asked her about her experience, and she said the other people in the nursing home were glad not to have to hear her sing but amazed at the difference in her ability to stand and walk.

I then started testing patients with multiple extremity weakness or coordination symptoms that had come on suddenly.

If all extremities test inhibited, I then ask the patient to close their eyes and touch the part of their body being touched. Most of them miss the location from 1 inch to 3 inches and report noticing a decrease in their coordination. They are then asked to write a sentence like "today is Monday."

Next, I take my cellphone, play music with words, and repeat the tests. Amazingly, all increased in strength, improve their coordination test, and their handwriting improved. Turning off the music reverses the improvement. Depending on their weakness and age, I have them stand from a sitting position or walk in the hall without the music. Again, everyone has noticed the improvement when walking with the music stimulation.

Not all persons with the vaccine or have had Covid have these reactions. Others have come in with varying symptoms like eczema, headaches, reactivation of Lyme, and visceral symptoms. One 70-year-old female reported menstruating seven days after the second Moderna injection. One patient with Hashimoto's disease under control for ten years developed rheumatoid arthritis as her primary and rheumatoid doctor diagnosed it one week after the second injection.

Recently three patients came in with the loss of taste and smell following having the vaccine or with a case of Covid. One had lost the sensations for five months. I challenged his olfactory nerve against a substance, and he became globally inhibited and had hypertonicity of the temporalis muscle even though he could not smell the substance.

Suggested procedure
If a patient presents with inhibition in all extremities, then test for coordination. Observe them going from sitting to standing and their walking gait. Have the patient close their eyes and see if they can touch where you are touching them. You can do other tests like balance with eyes open and closed.

I then ask them if they have a preference for music with words. Then, if you have a cellphone with vocal music, play it and retest the patient for improvement.

I have found that if the patient plays music softly in the background during the day and while they are mobile for 2 – 4 weeks, they are functional without the stimulation.

One 73-year-old male came in with this pattern. He noticed extreme weakness in his arms four days after having the second vaccine. He went to his primary care, who told him the vaccine could not cause this, and to go to physical therapy, where he got worse. I found the above pattern and asked him his favorite music, and he said the "Stones." Then I played a song, and his muscles became normally functional. He told me he was to go to his primary care the next day and tell him about this. I asked him not to do it unless he had the doctor test his muscles and then notice the change in function when the music played. He returned two weeks later and reported that he had done that. The doctor was surprised and told him to return to physical therapy and make sure music was playing while doing the therapy. He no longer needed the music to activate the inhibited muscles on his return.

Conclusion

If encountered with a patient with bilateral inhibition of muscles, ask questions about their vaccine status or possible infection. Challenge the individual with music with words and retest for a positive effect.

Paraneoplastic Syndromes

Paul T. Sprieser, BS, D.C., DIBAK

Abstract

Clinical series of symptomology that we commonly see in our AK practices, which can easily be mistaken for conditions we could treat.

Introduction

This paper covers a subject that is not well known for most of the health care professions, as pointed out by Lisa Sanders, M.D., New York Times, medical writer column Diagnosis. On Sunday's NY Times Magazine 11/25/18 Diagnosis titled "Jitters". The female patient had manifested symptoms of feeling jittery, with excitement felt out of control, with a racing heart. She described it as the 'heart pounding in her throat and a spacy-headed jitters". This continued going for a number of weeks with frequent trips to the ER. Her symptoms of anxiety and depression, as well as other symptoms of speaking nonsense in a strange babble.

Eventually she was taken to a psychiatric hospital in Vancouver, Washington, and given a diagnosis of excited catatonia. In the hospital her doctor thought she seem confused; her speech was garbled; and she seemed to have seizure-like episodes of shaking and inattention. In addition, she did not respond to any medication. At another hospital she was transferred to she was examined by a neurologist who thought possibility of a paraneoplastic syndrome, trigger by antibodies that has been set off by and ovarian growth, known as a teratoma. This type of tumors can contain a mixture of cell types-skin, bone muscle or tissues from various organs and in rare occasions, the tumors will grow types of brain cells.

Discussion

This syndrome can affect the nervous system and can be a group of uncommon disorders that develop in some patient who have cancer. It can affect many other organs system including hormones (endocrine, skin (dermatologic), blood (hematologic and joints (rheumatologic).

When cancer fighting agents of the immune system can attack part of the brain, spinal cord and peripheral nerves or even muscles, this can lead to muscle movement or coordination problems and well as memory or thinking skills, or even sleep.

The signs and symptoms of paraneoplastic syndromes of the nervous system can develop quickly, often over days to weeks and may even develop even before a cancer is diagnosed. The following is a list of symptoms:

1. Difficulty walking
2. Difficulty maintaining balance
3. Loss of muscle coordination

4. Loss of muscle tone or weakness
5. Loss of fine motor skill, such as picking up objects
   Difficulty swallowing
6. Slurred speech or stuttering
7. Memory loss and other thinking (cognitive) impairment
8. Vision problems
9. Sleep disturbances
10. Seizures
11. Hallucinations
12. Unusual involuntary movements

This syndrome occurs in about 7 % of cancer patients, mostly those with carcinoma of the lung (usually oat cell) or ovary. The diagnosis is one of exclusion and should not be accepted until metastatic or none metastatic causes are ruled out. The etiology is unknown, although an autoimmune mechanism is involved because circulating antibodies are found.

Specific Types of Paraneoplastic syndrome of the nervous system include:

- Cerebella Degeneration. This is the loss of nerve cells in the area of the brain that controls muscle functions and balance. Other symptoms are unsteady or impaired walking, lack of muscle coordination, inability to maintain our trunk posture, dizziness, nausea, involuntary eye movement, double vision, difficulty speaking or swallowing,

- Limbic encephalitis. The inflammation affects the limbic system, which controls emotions, behaviors and certain memory functions. There can be personality changes or mood disturbances, memory loss, seizures, hallucinations, or drowsiness.

- Encephalomyelitis. The inflammation of the brain and spinal cord.

- Opsoclonus-myoclonus. This syndrome is due to dysfunction of the cerebellum or its connections. It can affect rapid, irregular eye movements (opsoclonus) and involuntary, chaotic muscle jerks (myoclonus) in the limbs and trunk.

- Stiff person syndrome. Originally called stiff man syndrome, is characterized by progressive, severe muscle stiffness or rigidity, mainly affecting your spine and legs.

- Myelopathy. Injury is to the spinal cord. The symptoms will depend on the levels effects bowel and ladder function, and severe weakness and numbness.

- Lambert-Eaton myasthenic syndrome. Disrupted communication between nerve and muscle. Symptoms pelvic and lower extremity muscle weakness, fatigue, trouble swallowing, speaking, irregular eye movements and double vision. Other symptoms can be dry mouth and impotence. This disorder when associated with paraneoplastic syndrome is typically associated with lung cancer.

- Myasthenia gravis. Characterized with nerve and muscle communication with rapid fatigue of any voluntary controlled muscle actions, which can include muscle of the face, eyes, arms and legs. Also included can be the action of chewing, swallowing, talking and breathing. This is usually associated with cancer of the thymus gland (thymoma).

- Neuromyotonia. Also known as Isaacs' syndrome-is characterized by abnormal impulses in nerve cells outside the brain and spinal cord (peripheral nerve hyperexcitability). This cause muscle movement characterized as twitching or muscle rippling that looks as a "bag of worms," progressive stiffness and slowed movements.

- Peripheral neuropathy. Referred pain patterns that effect the sensory nerves of the peripheral nervous system and can cause pain any part of the body.

- Dysautonomia. Is a wide range of signs and symptoms resulting from injury to the nerves that regulate nonvoluntary body functions (ANS), such as heart rate, blood pressure, sweating, and bowel and bladder control. Symptoms include low blood pressure, trouble breathing and irregular heartbeats.

Conclusion

With the past history and reputation that AK has gained for producing great results in all types of health care issues. It is quite possible that we will get some patient seeking help who will have their symptoms due to paraneoplastic syndrome. The knowledge of this condition should be a part of all of us who use AK in our daily practice.

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Shoulder Impingement Syndromes

David Leaf, D.C., DIBAK

Abstract

A commonly overlooked condition in the shoulder is impingement syndrome. Unfortunately, two common ones are found in the office. These are found with the arm in flexion and the other in abduction.

Key Indexing Terms
Shoulder, impingement, biceps long head, muscle testing

Introduction
This paper will describe common impingement syndromes encountered in the office. Corrective exercises will be discussed as well as the need for proper muscle testing to be performed.

Discussion

Impingement in the shoulder is a common condition that is often overlooked. It is widespread in sports like tennis, swimming, and throwing sports. It is also common for anyone using their arm repetitively like a painter. However, many people have it due to repetitive stress, falls, and poor sleeping positions. Home activities like scrubbing, window washing, hammering, cutting wood, or any repetitive activity causing the arm to move in flexion and extension or in rotation can cause this condition. In an injury to the shoulder or any joint, fluid comes with inflammation. Inflammation is the beginning of this condition. The fluid fills the space between the humerus's superior surface and the acromion's inferior surface. A common complication of this condition is bursitis.

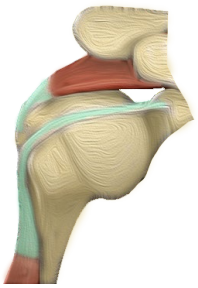

Figure 1 Normal humeral head position

A Medline literature search on this condition was published in 2017. The following quote is from that paper.

"The initial treatment is conservative, e.g., with nonsteroidal anti-inflammatory drugs, infiltrations, and patient exercises. Conservative treatment yields satisfactory results within two years in 60% of cases. "[1]

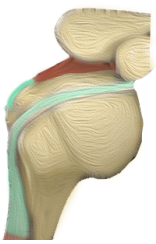

Figure 2 Elevated humeral head

The subacromial space lies between the head of the humerus and the acromion, the coracoacromial ligament, and the coracoid process. In addition, the subacromial space contains the subacromial bursa and the rotator cuff, especially the supraspinatus tendon.

Patients will report pain or severe ache elevating the arm above the horizontal or sleeping in a side-lying position. There may be no history that they will say caused the problem. This condition, except for those in certain sports, increases in incidence with the age of the patient

Figure 3 Normal supraspinatus

If there is a decreased range of motion in flexion or in abduction, impingement must be considered. Muscle testing can show the normal function of most of the muscles surrounding the shoulder. Impingement is the result of muscular imbalances and poor body mechanics.

If the decreased range of motion is found in the flexion of the arm, the next step is to examine the function of the long head of the biceps. The tendon of the long head biceps passes over the superior margin of the head of the humerus. When raising the arm in flexion, the function of the tendon of the biceps is to suppress the humeral head allowing it to clear under the acromion. Inhibition of the long head of biceps results in failure of this inferior pressure allowing superior migration of the head of the humerus, causing the tendon of the supraspinatus to be compressed.

The consequence of impingement syndrome is twofold. The first is that the supraspinatus tendon becomes injured and slowly becomes frayed. The second is arthritic changes due to the inflammation leading to calcifications in the shoulder.

This problem can lead to surgical intervention to reduce or remove these calcifications. However, surgeon sometimes will also say that the acromion is too long and needs to be reduced.

Figure 4 Impingement test

One of the problems in analyzing this condition is adequate testing. The usual method of examining the supraspinatus with the arm in a lower position will not uncover this problem.

However, a second test for the supraspinatus elevating the arm up to approximately 120 to 130 degrees with the humerus internally rotated so that the thumb is pointing downward can uncover this problem. In shoulder problems, both tests should be routinely performed.

O – 60 degrees
Supraspinatus, Deltoid
Infraspinatus, Subscapularis
Teres Minor

Figure 5 Arm flexion muscles 0 - 60

60 – 120 degrees
Anterior Serratus
Upper Trapezius
Subclavius

120 – 170 degrees
Spinal Motion
Supraspinatus

Figure 6 Arm flexion higher

When the supraspinatus is found inhibited in the elevated testing position, the most common cause is the failure of the long biceps tendon. Testing should then be performed for the biceps long head. Care must be taken in this test to observe what the patient does with their hand and wrist. The fingers cannot be flexed, and the hand must also be kept in a neutral position to prevent the facilitation of the long head of the biceps.

Figure 8 Biceps long head good test

Figure 7 Poor test recruitment

The most common cause of inhibition is micro adhesions restricting the tendon's movement. These protein cross-links cause adhesion of the tendon to the humerus. When palpated with cross fictional motion they will be tender and somewhat painful to the patient during the correction. Cross frictional massage is the easiest way to loosen the tendon and allow for the proper functioning of the long head of the biceps. Following correction of the long head of the biceps, a PNF type movement should be performed with the patient bringing their arm forward as if imitating the uppercut in boxing and then extending the arm back behind them, straightening the arm and contracting the triceps. This motion is repeated for 6 to 8 repetitions. Retesting the arm's flexion usually shows an increase in range of motion. The patient is instructed to do this daily for at least 7 to 10 days.

Figure 10 PNF motion - resist biceps activation

Figure 9 PNF motion resist arm extension

Restriction of the arm in the abduction can have three causes. The first is inhibition of the muscles active in this motion. These would first be the supraspinatus for the first 30 degrees, then the deltoid. Next, above 130 degrees, motion in the shoulder and the spine and then supraspinatus work in coordination.

It needs to be mentioned here that the humerus has to rotate externally to abduct the arm fully. If the arm is raised into abduction with the palm facing inferior, it is impossible to raise the arm above 100 to 110 degrees. The second condition to consider is where the deltoid muscle has shortened. Dr. Goodheart demonstrated that the deltoid muscle could become shortened, limiting the ability of the humerus to elevate properly by jamming the humeral head in the glenoid. His treatment was to traction the arm and percuss over the deltoid with the most significant amount of percussion superior to the deltoid tubercle. This is easily done in the office. The patient can be shown how to do this easily while tractioning their arm. They can use their opposite hand for percussion. Unfortunately, many of the percussion guns that patients buy can be misused. They can press too hard, causing muscle trauma. They should be instructed to bring the machine into the office for instruction on their use and misuse.

The third cause of this is a failure of the subscapularis to contract, rotating the humeral head and exerting slight inferior pressure. Testing will usually show a normal functioning subscapularis. However, the muscle becomes inhibited when the arm is raised and fails to function when needed to rotate the head of the humerus. Goodheart called this condition a reactive muscle. A better name for this condition is a proprioceptive neuromuscular inhibition problem. Treatment of this is more complicated. First, the scapula needs to be stable as well as the clavicle. Any muscle imbalance failing to stabilize these structures needs to be addressed. Then, the coordination of muscle activity between the deltoid heads, the supraspinatus, the long head of biceps, and rarely the coracobrachialis and the subscapularis needs to be tested. A quick test to start with is to percuss the shoulder while performing a PNF type of motion that may correct this problem. If this fails, testing these muscles and determining which causes the subscapularis inhibition must be performed. Next, correcting any spindle cell imbalance, followed by PNF movements to reinforce the coordination, should be performed. Finally, there will be a spinal problem at the level of the innervation of the abnormally functioning muscle that needs to be addressed.

Figure 11 Resist infraspinatus external rotation

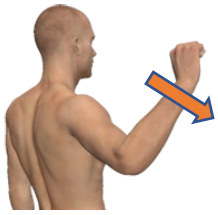

Figure 12 Resist subscapularis internal rotation

A third condition causing this can be adhesions between the infraspinatus and the subscapularis or adhesions of the shoulder capsule. The diagnostic key for this is decreased arm range of motion in internal and external rotation. Michael Leahy created a technique, the Active Release Technique, to correct these adhesions. A more straightforward way to help these conditions is to use percussion with motion. If the joint capsule is thought to be involved, percuss the shoulder joint anterior and posterior while resisting internal and external rotation. This is effective in many adhesive capsulitis patients. If the problem is between the subscapularis and the infraspinatus, do the same resisted arm rotation but place the percussion head in the axillary fold. This allows the percussion wave to vibrate the adhesions while you are resisting the motion.

Figure 13 Location for percussor

Conclusion

Impingement syndromes in the shoulder are mostly overlooked. However, the syndromes should be considered in anyone with shoulder ache, pain, or restricted motion. If found and treated correctly, it is a simple problem to address and correct if caught before arthritic changes occur.

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Figures all original by author:

1. Normal humeral head position
2. Elevated humeral head
3. Normal supraspinatus test
4. Impingement test
5. Arm flexion muscles 0 – 60
6. Arm flexion higher
7. Biceps long head good test
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10. PNF motion resist extension
11. Resist infraspinatus external rotation
12. Resist subscapularis internal rotation
13. Location for percussor

Experimental Observations of Members of the ICAK

Memorial Proceedings

Dr. Wally Schmitt

Dr. Wally Schmitt passed away unexpectedly on the afternoon of Saturday November 20, 2021 while enjoying his usual jog in the neighborhood in which he had lived for almost 40 years. Those who knew him, were mentored by him or attended his seminars, his colleagues and patients, family & friends all deeply mourn his loss. In honor of his memory please enjoy the following dedicated paper.

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Expanding the Neurological Examination Using Functional Neurologic Assessment: Part II Neurologic Basis of Applied Kinesiology

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Functional Neurologic Assessment and treatment methods common to the practice of applied kinesiology are presented. These methods are proposed to enhance neurological examination and treatment procedures toward more effective assessment and care of functional impairment. A neurologic model for these procedures is proposed. Manual assessment of muscular function is used to identify changes associated with facilitation and inhibition, in response to the introduction of sensory receptor-based stimuli. Muscle testing responses to sensory stimulation of known value are compared with usually predictable patterns based on known neuroanatomy and neurophysiology, guiding the clinician to an understanding of the functional status of the patient's nervous system. These assessment procedures are used in addition to other standard diagnostic measures to augment rather than replace the existing diagnostic armamentarium. The proper understanding of the neurophysiologic basis of muscle testing procedures will assist in the design of further investigations into applied kinesiology. Accordingly, the neurophysiologic basis and proposed mechanisms of these methods are reviewed.

Key words: functional neurology, functional neurologic assessment, neurologic examination, manual muscle testing, physiology, functional medicine, functional illness, neurologic assessment, kinesiology, muscle testing, muscular strength, muscle contraction, skeletal muscle, diagnosis, physical examination.

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INTRODUCTION

Goodheart (1964) introduced manual muscle testing for functional neurological assessment (Walther, 1988). He called his observations applied kinesiology (AK). AK is a functional neurologic assessment and treatment process that extends the neurological examination taught in medical and

chiropractic colleges to include the identification of subtle shifts away from optimal neurologic status. These shifts are associated with declines in function that may contribute significantly to patient morbidity (Fries). Changes in patterns of motor function that occur in response to the introduction of sensory stimuli of known value can be used to evaluate the functional status of central and peripheral neurologic pathways and guide the clinician to therapeutic measures to restore optimal neurologic function.

Much of the data gathering process unique to applied kinesiology relies on the manual assessment of muscular function as a method to evaluate changes in functional neurologic status reflected as changes in motor function. These observed changes in muscular function are assumed to be associated with changes in the central integrative state (CIS) of anterior horn motoneurons. The CIS is defined as the summation of all excitatory inputs (EPSPs) and inhibitory inputs (IPSPs) at a neuron. It is possible, therefore, to have a wide variation of central facilitated states and central inhibited states of neurons summating from many sources. The functional strength of a skeletal muscle is affected by the CIS (Goodheart, 1964; Walther, 1988; Guyton, 1991; Denslow, 1942) of the anterior horn motoneuron cells, (Feinstein, 1954) which in turn reflects changes elsewhere in the neuraxis.

The CIS may also be affected by the health of the neuron itself. The neuron's ability to maintain metabolic pathways, membrane receptors, and membrane pumps so that it is capable of responding to the demands placed on it by EPSPs and IPSPs is a critical factor, and one which may be identified by the AK assessment process as well. However, consideration of transneural degeneration and various axonopathies is beyond the scope of this paper. The clinical factors presented here will assume normal health of neurons. Although this may or may not be the case in clinical practice and must be considered by the clinician, the addition of this variable in this presentation would complicate and obscure the principles which are being presented. Similarly, although characteristics of individual neurological receptors and pathways are emphasized herein, the clinician must consider all of the interconnectedness of the nervous system, which is also within the realm of AK assessment but beyond the scope of this presentation.

Functional neurological assessment is performed by introducing sensory receptor-based stimuli, monitoring changes in the CIS through manual muscle testing, and interpreting the outcomes of manual assessment according to the knowledge of the relevant neuroanatomy. The introduction of sensory receptor-based stimuli of known value usually creates predictable changes in patterns of motor output. These motor changes are observed through muscle testing responses, and compared with the predicted responses, allowing the clinician to derive data about the state of the patient's neuraxis. Each step in the process of diagnosing and treating a patient using AK consists of creating a specific neurologic context, which is thought to be the sum of all sensory receptor-based afferent stimulation and all centrally generated effects at that moment, and observing changes in the patient's motor responses to that context.

AK clinical diagnostic procedures are focused on identifying functional neurological changes before they become end stage tissue disorders. Since the health of the nervous system is dependent on its ability to receive and respond to sensory information, treatment procedures are primarily sensory receptor-based therapies designed to normalize afferentation.

For example, the activation of touch, pressure, vibration, and other types of mechanoreceptors (MRs) is known to block afferent signals from nociceptors. (Sherrington, 1948; Feinstein, 1954) In the presence of adequate nociceptor activation, as when touching a hot stove with the hand, there is flexor reflex afferent (FRA) activity that creates muscle facilitation and inhibition patterns associated with the flexor withdrawal reflexes. There will typically be facilitation of limb flexors and inhibition of limb extensors with contralateral stabilization, creating withdrawal of the affected limb away from the painful stimulus. There will be patterns of facilitation and inhibition associated with these activated reflex pathways which can be identified using manual muscle testing. Introducing MR inputs (mechanically rubbing an area of tissue whose nociceptors are firing, for example) to block the pain will also result in a facilitation effect of muscles whose inhibition was caused by the FRA response. The effect of such an introduced stimulus may also be assessed through manual muscle testing. Treatment procedures are aimed at restoring a balanced level of neurologic function, with

appropriate levels of facilitation, which are observed clinically to be associated with restoration of other normal functions such as autonomic and neuroendocrine balance, proper neuro-immune function, and reduction of pain.

Muscular Facilitation and Inhibition

Clinicians using AK commonly refer to the result of a manual muscle test as a “strong” response or a “weak” response (Leisman, Zenhausern, Ferentz, Tesfera, Zemcov, 1995). A muscle that cannot meet the demands of testing pressure is termed “weak.” The weak testing outcome is hypothesized to be associated with an inhibitory CIS of the muscle’s alpha motoneuron (AMN) pool. If the motoneurons in the pool are inhibited (further away from depolarization threshold, or hyperpolarized), then the subject cannot adequately depolarize the pool on demand and adequate muscle contraction to meet the demands of the manual muscle test cannot take place. The result is a weakness in the muscle test outcome. In this paper, the terms “strong” and “weak” are used interchangeably with the terms “conditionally facilitated” and “conditionally inhibited.” These latter terms are intended to refer to the hypothesized conditional facilitation or inhibition of alpha motoneurons, reflecting changes in their CIS.

The functional status or CIS of the anterior horn motoneurons is maintained by convergence of multiple segmental and suprasegmental pathways. The segmental pathways are sensory pathways that are either of somatic or visceral origin and arise from a variety of sensory receptors in skin, joints, fasciae, viscera, and from various chemoreceptors. The suprasegmental pathways are descending pathways that can be of a conscious origin (cortical) or of a reflexogenic origin (brainstem, cerebellum) including postural and gait patterns. A conditionally inhibited muscle is thought to be associated with an inhibitory CIS summation of the converging pathways to the alpha motoneuron controlling that muscle (Leisman, 1989).

Leisman, et al (1995) have provided the first electrophysiologically based definition of what AK practitioners observe as conditionally facilitated and inhibited muscle responses to manual muscle testing procedures: The “ability or inability of a muscle to lengthen but to generate enough force to overcome resistance is what is qualified by the examiner and termed “Strong” or “Weak.”

Leisman and his colleagues found that there were significant differences in force/integrated EMG ratios in what is described as muscle testing strengths and weaknesses and that these differences could not be attributed to fatigue of the tested muscles. (Leisman *et al*., 1995)

In an earlier paper, Leisman et al (1989) also identified differences in somatosensory evoked potentials (SSEPs) recorded during the testing of conditionally facilitated versus conditionally inhibited muscles. SSEPs were recorded from the contralateral median nerves of fifteen subjects while three different, previously identified muscles were tested. In all subjects the baseline (no muscle test) and control “strong” muscle test recordings were comparable while recording from the “weak” muscle showed increased amplitudes in the contralateral layer components.

Subjective reports differentiating between pathological and functional weaknesses have been supported by objective evidence (Lawson, 1997; Leisman,1989; Leisman, 1995). If the peripheral nerve and the neuromuscular junction are intact, then a weak muscle test response is hypothesized to represent inadequate anterior horn motoneuron activity due either to excessive inhibition or to inadequate facilitation of these neurons. If a muscle is conditionally facilitated, it is presumed that the summation of inputs to the AMNs results in net excitation. With manual muscle testing, it is usually possible to subjectively localize which particular well-defined neurological pathways are overly excited or inhibited and to identify the source(s)of these deviations from normal function. Though one might typically assume that the normal state of a muscle involves adequate facilitation to meet the demands of manual testing, this is not always the case neurophysiologically. Some normal states are characterized by inhibition. For example, when a patient assumes a stance which resembles a gait pattern (standing with opposite arm and leg forward), neurophysiology would predict that the alpha motoneurons controlling arm flexors ipsilateral to the forward leg would be in a state of inhibition and alpha motoneurons controlling ipsilateral arm extensors would be in a state of facilitation, so that the ipsilateral arm could move into extension, in keeping with the normal

contralateral motion of walking. If one were to test the arm flexors in this gait position and find them adequately facilitated, the clinician would consider this result abnormal.

Accordingly, the interpretation of findings of conditional facilitation and inhibition are only relevant in the context of predictions based on known neurologic normals. When a variable in the form of sensory receptor stimulation is introduced, the clinician looks for the presence or the absence of a predictable change. If the stimulus creates a change in central integration that affects motor outputs, a change in muscle testing response will be observed, toward either facilitation or inhibition, according to the circumstances.

Receptors and Their Effects on Central Modulation

In addition to MR and nociceptors, receptor-based afferent stimulation can be introduced using retinal, olfactory, and taste receptors. These receptors introduce afferent stimuli that can impact on suprasegmental targets, affecting their central integrative states, and therefore affecting all motor pathways either directly or indirectly.

In addition to general sensory and special sensory receptor-based afferents, impact on neurological function may arise from alterations in biochemical status affecting neuronal membrane potentials, neurotransmitter levels, and hypothalamic monitoring of the blood milieu. Neuronal activity of cortical origin, whether from cognitive, emotional, or other processes, also impacts the neuraxis significantly.

Receptor activity is transmitted throughout the entire nervous system and interacts with other factors to affect the CIS of virtually all neuron pools. When a group of sensory receptors fire, the function of other non-adjacent portions of the nervous system may be affected. This may lead to changes in modulation of other neurological systems downstream from the functional changes. Any neural system which has motor connections, either locally or by ascending or descending pathways, may have its functional status evaluated using the AK system of functional neurologic assessment. This includes not only somatic sensory systems, but also central systems for visual motor activity (e.g., accommodation reflexes), vestibular reflexes (e.g., tonic labyrinthine reflexes), reticulospinal (autonomic) pathways, emotional (psychosomatic) pathways, and so on. By evaluating the effects of olfactory and gustatory stimuli, neurochemical evaluation may be performed as well.

Afferent Stimulus as Receptor-Based Challenge

In normal individuals, muscles function in a relatively predictable manner. Patterns of excitatory or inhibitory stimuli, introduced by the clinician through sensory receptor stimulation, are observed clinically to create predictable changes in the outcomes of manual muscle tests. These changes are hypothesized to be associated with changes in patterns of facilitation and inhibition of motoneurons. Applied kinesiologists commonly refer to the introduction of a known sensory stimulus as a “challenge.” Such sensory challenges constitute the first half of the process basic to applied kinesiology, in which afferent stimuli of known value are introduced. The second half of the process is the observation of changes in motor function through manual muscle testing. The clinician must then interpret the observed change according to the known neuroanatomy and neurophysiology associated with the stimulus and the resulting motor response, in much the same way that a clinician would interpret the outcome of a deep tendon reflex or the constriction of a pupil in response to a light stimulus.

Normal Gait Patterns Observed With Manual Muscle Testing

Normal gait involves alternate shoulder flexor and extensor facilitation and inhibition. In the normal individual testing the general shoulder flexors and extensors in the neutral standing position should reveal normal muscle strength testing outcomes. Placing a subject in a simulated gait position (for example, standing with the left leg and right arm forward and the right leg and left arm back) should result in a conditional inhibition (muscle testing weakness) of the shoulder flexors on the left side as

well as a weakness of the shoulder extensors on the right side. This is what would be predicted based on the normal arm swing of gait.

It is hypothesized that alterations from this normal pattern would be observed if there was improper afferent activity arising from sensory receptors that are a part of gait activity. This deviation from normal is thought to consist of alterations in patterns of facilitation and inhibition of muscles. This effect has been observed as muscle testing responses which vary from the predicted normal patterns (Leisman, 1989). Therapeutic interventions are aimed at normalizing afferent activity to restore normal neuromuscular function.

Functional Evaluation of Spinal Cord Interneuron Function

The following tests are used to evaluate spinal cord interneuron function. Muscle testing responses that diverge from predicted neurologic normals guide the AK practitioner to various therapeutic measures, aimed at restoring normal receptor afferentation. The therapeutic measures vary according to the specific clinical circumstance, and are beyond the scope of this paper.

Spinal interneurons affecting AMNs are facilitated by both segmental and suprasegmental pathways. Segmental influences include: 1) autogenic facilitation (such as by the 1a fiber mediated deep tendon reflex), 2) reflexogenic inhibition from a contracted muscle, or 3) flexor reflex afferent (FRA) activity. Excitation of each of the three pathways is predicted to be associated with facilitation of a primary muscle and inhibition of its antagonist. One may examine these pathways to identify possible sources of aberrant influence on the alpha motoneuron of the muscle being tested.

For example, tapping the right patellar tendon to elicit a deep tendon reflex should result in facilitation of the right quadriceps, inhibition of the right hamstring, inhibition of the left quadriceps, facilitation of the left pectoralis major, and inhibition of the right pectoralis major. A strong voluntary contraction of the right quadriceps is expected to create the same pattern just described.

AK practitioners could examine the functional status of these pathways by observing patterns of conditional facilitation and inhibition of muscles sub served by the pathways in question. Failure of the predicted response immediately following a patellar tendon tap or muscle contraction may indicate a functional neurological problem.

Pinching or squeezing a tendon to fire an inhibitory afferent from the Golgi tendon organ is predicted to be associated with conditional inhibition of the muscle of origin followed by an immediate return to normal facilitation on a subsequent muscle test. Failure of this response should lead the clinician to investigate the source of neurological interference resulting in the dysfunction.

Axial traction of a joint for eight to ten seconds activates Type III joint receptors, whose afferents are inhibitory to muscles which cross the joint being stretched. This traction is followed by testing a muscle that crosses the joint. The muscle should be inhibited and the muscle testing response would be predicted to reflect this conditional inhibition (Leisman et al, 1996). Failure of the inhibited response leads to further investigation.

In a similar fashion, the facilitatory effects of Type II joint receptors may be evaluated. A conditionally inhibited muscle is predicted to respond with a shift toward facilitation when its related joint is stretched quickly. This should occur even when the muscle spindles of the muscle are prevented from stretching by the nature of the joint stretch. Failure of this strengthening response is grounds for further investigation.

All of the above mentioned tests evaluate well known, well defined interneuron and reflex pathways. These pathways are also dependent on other segmental and suprasegmental influences for normal function. AK muscle testing combined with traditional neurological examination procedures greatly enhances the data gained from the neurological exam.

Using Mechanoreceptor and Nociceptor Stimulation To Guide Therapy to Injured Areas

The effects of pain are dependent on the interaction of two types of sensory receptors and their afferent pathways: nociceptors (NOCs) and MRs. Skin NOCs can be manually stimulated by pinching. Skin MRs can be manually stimulated by rubbing or stroking. These receptor-based stimuli constitute a mechanism by which inputs can be strategically introduced, as a challenge to the system's central modulatory mechanisms. The intent of such a "challenge" is to assess the extent to which changes in sensory input create alterations of motor function.

By observing muscle testing outcomes resulting from such sensory receptor diagnostic challenges, there are five possible outcomes: rubbing facilitates, rubbing inhibits, pinching facilitates, pinching inhibits, or no change in muscle status results. Each of the first four possibilities would direct the clinician to a different treatment procedure to normalize the patient's functional neurological status and reduce pain if present.

If an alteration in motor function is a consequence of nociceptive afferent stimulus, one would expect to observe a return to normal motor function if the nociceptive stimulus was blocked or terminated. Since the activation of mechanoreceptors blocks the input from adjacent nociceptors, one may selectively stimulate mechanoreceptors in tissues (such as rubbing or stroking the skin over the effected area) and observe motor control patterns in muscles to identify those areas of the body whose stimulation results in a normalization of motor function, presumably by achieving a blocking effect of nociception. Observed patterns are interpreted in light of the known neuroanatomy.

Nociception has extremely important effects on muscular and autonomic activity via reflex pathways both segmentally at the spinal cord level and suprasegmentally at the hypothalamus and brainstem levels, regardless of whether or not a person experiences conscious pain.

Suprasegmental Modulation of Pain

In the assessment of functional alterations related to pain, MR and NOC receptor stimuli are the basis for sensory receptor diagnostic challenges that may guide therapeutic choices. If rubbing or stroking the skin over an area of trauma results in neuromuscular facilitation, there will also be an abnormal response to autogenic facilitation of the muscle spindle as described above. (Lynn, 1985)

If pinching over an area of recent or old injury results in neuromuscular facilitation, the result suggests that the additional nociception causes the facilitation response via the descending medullary pathways through interneurons to the MNs in the anterior horn. This suggests that the weakness is present due to a net MN inhibition (due to a lack of excitation) which may be overcome by activating the CRN descending pathway.

If pinching (or adding any increased nociception from any stimulus) over an area of injury results in a general neuromuscular inhibition response, the result suggests excessive NOC activity, affecting all MNs.

By these sensory receptor challenges, the neurological pathways related to a specific patient's pain may be assessed and the most appropriate pain control techniques may be employed.

Tonic Labyrinthine Reflexes

The tonic labyrinthine reflexes (TLR) are present at birth and create patterns of excitation and inhibition of the limbs helping to maintain upright posture or to catch the body in the case of a fall. There are four distinct TLR response patterns: face up, face down, right ear down, and left ear down. In the face down position of the head, the flexors of all four limbs are facilitated. In the face up head position, the extensors of all limbs are facilitated.

The right ear down position is associated with excitation of the right limb extensors and an inhibition of the right limb flexors. Likewise, this head position is associated with excitation of the left sided limb flexors and an inhibition of the left limb extensors.

The effects of TLR on muscle testing are profound. The TLR are not strong enough to create muscle testing weakness by themselves, but they are powerful enough in their excitatory effects to overcome many other sources of inhibition hence hiding otherwise weak muscle patterns. Clinical application of TLR principles suggest that the supine (face up) would not be expected to demonstrate conditional inhibition of extensor muscles tested manually, due to the power of these lateral vestibulospinal tract mediated reflex pathways. Likewise, a prone (face down) patient would not be expected to demonstrate conditional inhibition of a flexor tested manually. In the supine position a weak flexor muscle should be strengthened while the head is in the opposite ear down position. These patterns of head position in relation to gravity must be considered when performing muscle testing for any purpose.

For example, when an orthopedist tests the muscles of the rotator cuff in a supine patient, the generalized facilitation of extensors by the TLR creates an artificially increased facilitation of supraspinatus, infraspinatus, and teres minor muscles, all of which are extensors. These muscles should either be tested in a prone position, or other measures should be taken to momentarily disable the facilitating effect of the TLR. Such measures are beyond the scope of this discussion.

TLR head position patterns may also be examined to ascertain the status of the TLR and the lateral vestibulospinal tracts. Variations from predicted facilitation and inhibition patterns guide the clinician's attempt restore the adequate function of this postural control mechanism. For example, if a right side extensor is identified as conditionally inhibited in a supine patient, turning the head to the right ear down position should increase the extent of facilitation of the extensor. Failure of right ear down position to yield this effect is suggestive of an inability of the TLR to create the expected efferent pattern of facilitation to right sided extensors. Further assessment and therapeutic measures are pursued to normalize this effect.

Functional Deafferentation

Patterns of conditional muscular inhibition have been often ascribed to "functional deafferentation" of the various afferent pathways affecting a muscle's motoneurons. Functional deafferentation may be simply defined as the loss of normal and expected sensory activity. A common source of functional deafferentation is the loss of normal joint range of motion resulting in decreased mechanoreceptor (MR) activation. AK therapeutic procedures are often directed at normalization of the sources of facilitation and inhibition, with the intent of restoring normal efferent motor control mechanisms for muscular activity. This effect restores normal muscular control of joint movement. Changes in the status of joint and muscle function can be both a cause and effect of changes in the extent of afferentation.

Stroke Antalgia-Like Patterns of Inhibition

Functional deafferentation of the cerebral cortex can affect not only its somesthetic sense but also its motor responses. If, due to a lack of normal afferentation, a motor area does not receive adequate stimuli, its inability to fire its usual motor pathways can result in a dysfunction in which the muscle imbalances, and sometimes the symptoms, are reminiscent of stroke antalgia, but without the typical tissue pathology. Autonomic concomitants will accompany this pattern via the impact of the reticulospinal pathway at the intermediolateral cell column (IML). The effects of altered reticulospinal activity include changes in autonomic modulation that influence among other things, dilation of the pupils, respiration, heart rate and rhythm, circulation, sweating, shivering, digestion, etc.

The hypothalamoreticulospinal tract inhibits ipsilateral anterior muscles above T6, and ipsilateral posterior muscles below T6. Decreased frequency of firing of the neurons in this pathway, as is the case in functional deafferentation, has been observed to move the CIS of involved alpha motoneurons toward facilitation, resulting in an increased tone of the anterior muscles above T6, and an increased tone in the posterior muscles below T6 on the ipsilateral side. This increased

facilitation yields increased inhibition of the antagonistic muscles. As a result, some or all of the ipsilateral upper limb extensors and abductors and ipsilateral lower limb flexors and adductors will be conditionally inhibited, and unable to meet the demands of manual testing. The observed pattern is very similar to stroke antalgia, and also resembles the normal gait pattern. It has been called a "pyramidal distribution of weakness." It is actually an inhibition of the extrapyramidal tract that has lost its ability to perform its requisite inhibitory functions that modulate pyramidal influences.

This stroke antalgia-like distribution of inhibition is primarily due to MR deafferentation from joints and muscle spindles via the thalamus and cerebellum. These muscle imbalances create symptoms that can lead to joint pathology and increased injury potential. Since joint MR afferents provide a significant portion of the total afferent stimulation to the contralateral thalamus, any reduction of MR stimulation would yield a reduced frequency of firing of thalamic interneurons, and therefore a reduced frequency of firing of descending efferents from the thalamus through the hypothalamoreticulospinal pathway.

Another area where the effects of asymmetrical thalamic firing can be observed is in vision. Primary visual afferents terminate in the lateral geniculate body of the thalamus and conveyed from the thalamus to the occipital cortex. Should the CIS of the thalamus (including the lateral geniculate body) be shifted toward inhibition, then the ability to convey primary afferents through the thalamus to the visual cortex where the visual inputs are perceived will be decreased. As a result, the physiologic blind spot will usually be larger in the visual field contralateral to the side of the thalamus receiving inadequate afferentation. The long literature on stabilized retinal image addresses this well (Riggs, 1953)(Leisman, 1974,1976). Blind spots may be mapped in the office setting and correlate with the muscle testing findings of a stroke antalgia-like distribution of neuromuscular inhibition. Significant changes in blind spot size have been demonstrated after manual manipulation of joints on the side ipsilateral to the enlarged blind spot. Such manipulation is presumed to have its effect via restoration of adequate afferent firing from joint MRs to the contralateral thalamus.

Viscerosomatic and Somatovisceral Interactions

The autonomic nervous system motoneuron cells in the IML column receive significant input from somatic factors. (Lynn, 1985; Willis, 1985) Nociceptive sensory fibers are flexor reflex afferents (FRAs) which synapse in the IML column. (See figure 1).

The significance of this fact neurologically is that clinicians cannot even touch their patients, much less manipulate them, without creating substantial effects on the IML column and the autonomic nervous system motoneurons. It is impossible to treat patients for neuromuscular or musculoskeletal problems without having meaningful effects on the motoneurons of the autonomic nervous system.

The body is constituted in such a way that somatic inputs into the nervous system cannot be made without affecting visceral function. Nor can visceral function be activated by any means (manipulative, nutritional, allopathic, homeopathic, etc.) without having significant effects on somatic motor function as well. Those who profess to treat musculoskeletal complaints without creating visceral effects are misinformed.

Autonomic Components of AK Evaluation

Autonomic signals which originate at the hypothalamus are transmitted to the brainstem reticular formation. This information descends to the spinal cord via reticulospinal tracts which affect both IML MNs and AMNs. Therefore, certain systemic and local changes in sympathetic (SYM) and parasympathetic (PS) function affect, in a predictable, specific fashion, the CIS of AMNs and hence, muscle strength and weakness patterns.

Nociceptors cause predictable FRA muscular activity previously discussed. An additional effect of many FRA afferents is stimulation of the SYM IML. (Burt, 1993)

Since changes in autonomic functions affect somatic motor pathways through established mechanisms, changes in muscle function will accompany changes in autonomic (SYM and PS) status in predictable ways. AK evaluation of autonomic functions employs observations of conditional muscular facilitation and inhibition associated with observed changes in autonomic function. Such patterns of facilitation and inhibition may be present in a static fashion when alterations of autonomic tone are also present in a static fashion, as can be observed in routine neurological examination. Subtle changes in autonomic tone may be identified through the observation of patterns of change in conditional facilitation or inhibition of muscles in response to sensory receptor challenges directed at altering autonomic tone. Altered autonomic tone in a visceral organ will be clinically associated with either increased or decreased SYM activity and/or increased or decreased PS activity to that organ. Each of these four states can be identified by evaluating muscle testing patterns and responses to sensory receptor stimuli.

Specific sensory receptor based diagnostic challenges may be used to identify segmental (local) or suprasegmental influences on the function of the primary autonomic motoneurons in the IML, hence on the AMNs and muscle function. Systemic influences affect the entire autonomic nervous system, whereas local influences may be used to evaluate the level of function of one organ at a time. An example of each of these is discussed below.

Local Autonomic Stimulation of Visceral Referred Pain Areas

Due to viscerosomatic convergence, (Swedlow, 1986) the body cannot tell the difference between nociception arising in the skin and that arising from an organ. Activation of the skin NOCs over the classical visceral referred pain (VRP) area for a given organ (see figure 2) will often result in a change in patterns of neuromuscular facilitation and inhibition, yielding changes in muscle testing outcomes. Similarly, activation of MRs over the same referred pain area may also bring about a change in muscle testing response by blocking visceral NOC activity.

Clinically, if increasing NOC activity by pinching an area result in a conditional facilitation of muscle, this can usually be interpreted as an indication that an increase in SYM stimulation in the area of the pinch has yielded a favorable effect. One might infer from this that the ambient SYM activity in the area is inadequate. This decreased SYM activity is due to an inhibited CIS of the IML arising from lack of facilitation or lack of inhibition of inhibition by the afferents to the IML. This interpretation is especially applicable when a pinching or other NOC stimulus is applied to the VRP areas (see figure 2). A conditional facilitation of muscles following pinching a VRP area would suggest that the related organ was relatively deficient in SYM activity, as reflected in the CIS of its IML MNs, whose state is identified by observing the state of the AMNs for the muscles tested. This could be due to either deficient SYM activity or increased PS activity or both.

The converse of this, pinching over a VRP causing a conditional inhibition of muscles, suggests excessive SYM activity of that organ.

Nociceptive reflexes, including those affecting the IML and SYM function, are blocked by mechanoreceptor (MR) activity. Hence, if rubbing over a VRP area to increase MR afferents creates a conditional facilitation of muscle, it suggests a need for less SYM activity, more PS activity, or both for that organ.

If rubbing over a VRP creates a conditional inhibition of muscle, this suggests that the associated organ has excess PS activity.

When many or all VRPs respond to the same pinching (SYM) or rubbing (PS) challenges, a systemic challenge is often positive, as we will discuss below.

Systemic Autonomic Stimulation – Visual

There are a number or systemic autonomic stimulation patterns associated with changes in patterns of neuromuscular facilitation or inhibition. . Only one of these is presented here.

It is established that the act of focusing from near to far and focusing on a distant object are events that involve activation of the SYM system. When conditionally inhibited muscles become facilitated with visual near-to-far activity or with focusing on a distant object, this is often evidence of a need for more systemic SYM function. A conditional inhibition response on near-to-far activity or distant focusing usually implies an excess of SYM activity. In either case, changes in patterns of facilitation or inhibition of muscle are interpreted in light of the impact of the shift of the system toward increased SYM function.

It is likewise established that the act of focusing from far to near is an event that involves activation of the PS system. If a conditionally inhibited muscle becomes facilitated with focusing on a near object such as the tip of the nose, it suggests a need for more PS function. A conditional inhibition response on far-to-near activity or near object focusing usually implies an excess of systemic PS activity. Normalization of these systemic autonomic imbalances may involve many procedures including dietary changes, manipulation, sensory receptor manipulation to normalize afferent activity (see next section), nutritional supplementation, stress reduction techniques, and so on.

A Model for So-Called Neurolymphatic Reflex Activity Increasing Parasympathetic Activity

The so-called neurolymphatic reflexes (NLs) are somatovisceral reflexes first described by Chapman. Most are located in the intercostal spaces. Chapman identified palpatory findings of nodular, indurated areas localized segmentally in intercostal and paraspinal areas, and associated them with disease in visceral organs neurologically associated with each segmental level. Chapman recommended manipulation of the tender areas until the tenderness or induration decreased.

Goodheart (1965) first described the use of the NL reflexes in relation to manual muscle testing. He found that specific patterns of conditional neuromuscular inhibition would respond with facilitation after manipulating one of the areas as described by Chapman. In this way, specific reflex areas described by Chapman became associated with specific observed patterns of conditional muscular inhibition. Since Chapman had identified relationships between these reflexes and specific organs, the associated patterns of conditional muscular inhibition associated with specific reflexes began to be likewise associated with the same specific organs. This was the beginning of clinical observations of a specific muscle-organ relationship. Areas of increased tissue irritability would be expected to send increased afferent signals to the spinal cord. This effect would be increased during weight bearing and during movement (including respiration), when the tissues are irritated by movement or pressure.

Increased afferentation in the intercostal spaces would be expected to reflexogenically increase SYM activity. This has been shown in laboratory animals by increasing both NOC and MR sensory input. (Coote, Dowman, and Webber, 1969)

Clinical and anatomical evidence suggests that the response achieved by manipulating the NL reflexes is due to a relative increase of PS activity due to a resolution of the pattern of ischemia and muscular spasm associated with the irritable NL area and a subsequent reduction of over stimulation of SYM activity at the IML. Although manipulation of the NLs often causes an increase of stimulation of local nociceptors during the manipulation, the net result following NL treatment is decreased irritability. This decreases the excessive afferent stimulation that is driving the local IML neurons to increased SYM activity. If PS outflow to those organs remains the same, the net result of treating an NL will be an increased relative PS activity of those organs that are affected. This is consistent with clinical observation. The need for the use of NL to increase PS activity is indicated clinically when the stimulation of MRs in a related organ’s VRP yields a conditional facilitation of tested muscles.

The changes in muscular facilitation from treating a NL reflex are likely due to the collateral connections from the IML axons that reach AMNs. It is reasonable to expect increased muscular facilitation of conditionally inhibited muscles and a restoration of normal inhibition of "tight" or "spasmed" antagonists as a result of normalizing feedback from an active NL reflex.

General Muscle Weakness Patterns Following Challenge Procedures

A common observation by those practicing manual muscle testing procedures is a pattern in which all muscles exhibit a pattern of conditional inhibition following a sensory receptor challenge. This pattern suggests that anterior horn motoneurons must be inhibited throughout the neuraxis.

An area known as the inhibitory center of Magoun and Rhines (or the medial reticular extensor inhibitory area) in the medulla gives rise to descending inhibitory pathways which are generally inhibitory to extensor muscle reflex activity throughout the body. (Kandel and Schwartz, 1985) Given the general inhibitory effect of certain sensory challenges on both flexor and extensor muscles, it is reasonable to speculate that there is a central site which creates a general inhibition simultaneously to both extensors and flexors. The exact location of the neurological center for general muscle weakness patterns remains a mystery, but one might speculate that it is related to the reticular formation and the descending reticulospinal tracts.

Dural Concepts

AK techniques include cranial bone manipulation, performed with a light pressure. These techniques are based on a model of dura mater tension, particularly cranial dural tension. A cornerstone of the cranial dural concept is that cranial sutures have motion, and that this motion has an effect on nervous system function. This has been a much debated issue, but there is support for the concept that human sutures do have motion based on anatomical and developmental analysis, (Pritchard, 1956) squirrel monkey studies demonstrating lack of sutural ossification, (Retzlaff, 1976) squirrel monkey studies demonstrating nerve endings in sutural articulations, (Retzlaff, 1978) and clinical results (Weiner, 1990).

Alteration of normal cranial motion results in what are called “cranial faults” or “cranial lesions.” The cranial bone motion is thought to affect and be affected by changes in dural tension, in both the cranium and the spinal canal. Since the dura mater has strong anatomical attachments to the sacrum and the coccyx, techniques involving cranial manipulation are often accompanied by sacral or other spinal manipulations. This has given rise to the terms like “craniosacral mechanism” and “craniosacral techniques.”

Cranial faults are treated by manipulative procedures directed to the cranial bone structures and often accompanied by sacral or other spinal manipulation. These are most often very specific, gentle manipulations, occasionally performed coincident with a phase of respiratory activity. They will affect MR activity in the tissues manipulated as well as in the MRs in the cranial sutures and intracranial dural receptors.

Observations of the clinical outcomes from cranial manipulation demonstrate a wide diversity of physiological and therapeutic response including resolution of everything from low back pain to tachycardia. Considering the vast neuronal network affected by the trigeminal nerve, the vagus nerve, and the vestibular mechanism which may be impacted by cranial manipulative techniques, these responses seem plausible.

As discussed by Walther (1988), Upledger (1983), and others, the cranium is believed to function such that manipulation of one cranial bone will affect the entire craniosacral mechanism. It is thought that craniosacral techniques generally restore normal juxtapositional relationships and motion to the craniosacral mechanism, hence they are associated with a change in dural relationships or dural tension.

The innervation of both spinal and cranial dura arises almost totally from nociceptors (Willis, 1985). The cranial dural innervation and its areas of referred pain are reviewed in a paper by Schmitt (1992). The dura is supratentorially innervated by the trigeminal nerve and infratentorially by sensory branches of the vagus nerve (Broadal, 1981). Many cranial faults are observed clinically to be accompanied by a pattern of bilateral conditional inhibition of the neck flexors, particularly the sternocleidomastoid (SCM) muscles. It is proposed that this bilateral neck flexor pattern should be

interpreted as a representation of an FRA withdrawal reflex pattern associated with neck extension. This pattern is most likely initiated by nociceptive activity somewhere in the head and neck mediated through the various cranial afferents and their respective pathways. As with other pain patterns, MR stimulation over an area of NOC activity is expected to block the NOC afferents, briefly restoring facilitation to muscles whose inhibition is a consequence of the NOC activity. In this way, the clinician can introduce MR activity selectively by rubbing over each of the cranial bones or sutures, and observing changes in the pattern of inhibition of neck flexors. For example, if introduction of MR stimulation over one temporal bone yields a restoration of normal facilitation of neck flexors, therapeutic measures would be directed at the restoration of proper position and motion of that temporal bone.

It is also reasonable to describe the observed clinical therapeutic responses of cranial manipulation as taking place via the afferent effects of these nerves.

Upledger (1983) describes a great deal of movement in the craniosacral respiratory mechanism. The constant motion of the craniosacral mechanism may be enough to maintain a base line level of mechanoreceptor barrage from the associated structures. Accentuation of this movement by cranial manipulation may be adequate to bring hyperpolarized cranial receptors to threshold, firing the involved pathways, and reestablishing a frequency of firing that is maintained beyond the time of treatment. A normal amount of craniosacral motion will maintain a normal amount of afferent input to vital centers. An abnormal amount of afferent activity will create abnormal afferentation to these centers. This is thought to be normalized by mechanical manipulation of cranial bones to restore normal relationships and motions.

Examining extracranial MRs which are stimulated by craniosacral manipulative techniques sheds some light on the clinical responses. For example, one technique designed to correct mechanical torquing lesions of the sacroiliac joints involves placing a prone patient on orthopedic wedges (DeJarnette blocks) and repeatedly pressing on the sacrum coincident with respiration. This, of course, bombards the system with MR input from the SI joints, the skin, muscles, and other tissues being contacted, intercostal and other respiratory activity

Three Types of Manual Muscle Testing

Manual muscle testing texts vary widely according to the type of contraction (e.g., eccentric or concentric) to be measured, the timing and force of the tester, the timing and force of the patient's resistance to the movement, and other factors. Most describe the testing of muscles chiefly in order to establish gross muscular strength levels.

Our clinical observations have led to the discernment of three distinct methods of performing a manual muscle test. Clinically speaking, these are: 1) clinician started testing, 2) patient started testing with maximal contraction, and 3) patient started testing with submaximum contraction. Each of the three types of muscle testing responses is measured by the patient's ability to resist an incremental increase in force applied by the tester in the direction of eccentric contraction. The tester attempts to lengthen the patient’s muscle out of a shortened pre-test position. Each type of test differs in the amount of pre-loading the patient is allowed prior to the addition of the eccentric testing force. The most significant factor in extracting clinical information from a muscle test response is who (tester or patient) starts the test, how much force is applied, and in which direction (i.e., eccentric, concentric, or isometric).

1) Type I – Clinician Started Testing

Clinician started testing is an eccentric test of the muscle with no pre-loading. The tester positions the patient for the test, makes contact with the patient, and tells the patient to resist his force in the appropriate direction. Then the tester pushes against the line of pull of the muscle while the patient attempts to eccentrically contract the muscle, resisting the tester’s attempt to further lengthen the muscle out of its shortened state.

2) Type II – Patient Started Testing to Maximum

In patient started testing with maximum contraction, the pre-loading allows a full isometric contraction of the muscle. The patient is instructed to push against the tester's hand while the tester holds the position as firmly as possible. When the tester observes that the patient has reached a maximum isometric force, the tester then pushes with increased force in the direction of eccentric contraction. This type of testing is what Leisman, et al, employed in their research efforts (1989, 1996).

3) Type III – Patient Started Submaximum Testing

Patient started testing with submaximal contraction is a pre-loading of submaximal concentric contraction followed by eccentric test. The patient is instructed to push against the tester's hand in the direction of the muscle's normal pull, creating concentric shortening of the muscle to be tested. Immediately upon sensing the initial few degrees of concentric contraction, the tester then pushes against the patient's force in the direction of eccentric lengthening. The patient then resists the tester's attempt to further lengthen the muscle out of its shortened state.

It is obvious that training and skill are necessary to perform these tests and discriminate between the outcomes. Muscle testing for functional neurological assessment testing is far more sophisticated that simply having the patient shrug the shoulders to ascertain if cranial nerve XI is intact. The procedures, however, have reported significant reliability (Lawson, 1997), as well as significant criterion validity (Leisman et al, 1996).

A conditional inhibition response to an AK muscle test suggests that the CIS of those AMNs reflects either excessive inhibition or inadequate facilitation, in spite of the conscious descending excitatory inputs created by the patient attempting to perform the test. These conscious effects are considered to be a constant from one test to another. Measuring the eccentric portion of the test initiates a stretching of muscle spindles that should excite the AMNs and tend to reinforce the descending conscious pre-loading inputs to the AMNs for that the muscle.

Neurophysiologic Basis of Differences in Patient Response to the Three Types of Test

Descending conscious effort by the patient in response to the eccentrically placed force creates inputs to the spinal cord that can be considered a constant in the manual muscle test. Any changes in muscle strength of the nature observed in AK are related to the eccentric portion of the test. Therefore, it is the effect primarily on the alpha motoneurons (AMNs) of that muscle that is reflected in the outcome of the muscle test. The gamma motoneurons (GMNs) by themselves are not able to produce enough force to affect the type of testing changes that are perceived.

When a Type I test reveals a conditional inhibition of a muscle, it suggests that the central integrative state (CIS) of those AMNs is inhibited in spite of the conscious descending excitatory inputs present which are considered a constant from one muscle test to the next. Performing a Type I test causes a stretching of muscle spindles which should excite the AMNs and tend to reinforce the descending conscious inputs to the AMNs for that the muscle. With the exception of the primary muscle spindle Ia monosynaptic pathway, we know that (MR) activity at the spinal cord level affects muscle function through interneurons (INs) which affect the gamma loop. MR reflexes do not affect the AMNs directly, but affect muscle function through the GMNs and the gamma loop. (This is in direct contrast to nociceptors which synapse on INs which affect the AMNs directly and bypass the gamma loop.)

When a muscle shows a Type I weakness only, we can assume that the muscle spindle (which is being stretched from the onset of the test) is not adequately firing through the monosynaptic Ia pathway to the AMNs to reinforce the constant conscious descending facilitation of the AMNs adequately to overcome the tester's force. In a Type I only conditional inhibition, this is because the local CIS of the GMNs is being inhibited from the segmental MR inputs (eg., joint fixation with inadequate joint mechanoreceptor firing, active intercostal trigger points, etc.) This GMN inhibition results in a laxity (lengthened position) of the intrafusal fibers and slack in the muscle spindle, hence a lack of responsiveness to the stretching of the muscle as the test is initiated. There is a lack of

reinforcement via the Ia reflex pathway to the AMNs which are thereby unable to keep the muscle firing up to the demands of the test. Thus, a Type I only pattern of conditional inhibition is present.

A Type II inhibition, then, is thought to be associated with inhibition of the AMNs even when the gamma loop is allowed to be fully facilitated by conscious descending pathways (both the constant pathways which always fire during any type of muscle test AND the additional pathways which are facilitated when the patient is allowed to recruit them during the isometric phase of the test.) In other words, many descending pathways (reticulospinal, rubrospinal, cortico-cerebello-vestibulospinal, etc.) are thought to be recruited and activated when the patient is requested to perform a Type II test. These pathways are all thought to work through the gamma loop (and some also affect AMNs directly.) Sources of inhibition (eg., autonomic, endocrine, cranial faults, TMJ, etc.) to one or more of these pathways to the gamma loop are thought to result in an inhibition of the the gamma loop, hence inadequate reinforcement of the Ia pathway during the Type II test and an inhibition response to the testers added eccentric force at the end of the test. This is thought to be similar to the mechanism of a Type I test. But in a Type I test, the GMNs are being inhibited by local MR reflexes whereas in the Type II test, the GMNs are being inhibited by a descending suprasegmental pathway.

Descending suprasegmental pathways mentioned above which make up part of the corticofugal muscle activation system and operate through the gamma loop are affected by interfering inputs from the hypothalamus, vestibular equilibrium system, etc. This interference prevents maximum cortical activation of these corticofugal pathways, yielding inadequate activation of the GMNs and the gamma loop. Hence the Ia pathway will not be able to respond adequately to the stretching demands at the eccentric part of the muscle test and the Type II test will demonstrate inhibition.

The Type III test is also different in its mechanism. It is observed clinically that the Type III pattern of conditional inhibition is associated with nociceptive activity. It is known that nociceptors affect the AMNs through INs directly and bypass the GMNs and the gamma loop. This pathway is known as the flexor reflex afferent (FRA) pathway and the FRA IN group is sensitive to other segmental and suprasegmental inputs besides nociceptors (in fact to A-beta as well as A-delta and C afferents.) The present discussion will focus on the nociceptors.

If a Type III pattern of inhibition is present, the FRA INs, (presumably in lamina VI and the intermediomedial group of lamina VII) are being activated for the FRA response by the nociceptors. These FRA INs both excite and inhibit both flexors and extensors by the flexor withdrawal reflex, the reciprocal extensor inhibition, and the crossed extensor reflexes which affect both flexors and extensors. This FRA IN pool is a major way station for various descending pathways on their route to effect affect AMNs. It is these FRA INs which are presumably impacted by immune and visual (autonomic) reflex imbalances transmitted via the medullary reticulospinal tracts (MRST.)

Type III inhibition patterns are a result of activation of inhibitory INs which are part of the FRA reflex pathway from nociceptors. These INs pass directly to AMNs and bypass the GMNs and the gamma loop. Nociception is also transmitted to the caudal reticular formation (CRN) where it reflexively, via the MRST, descends in the dorsolateral fasciculus to inhibit the effects of incoming nociception at several spinal cord areas throughout the length of the spinal cord.

One might show a Type I and Type II but not a Type III pattern of inhibition if there are no local FRAs active, and if the MRST is functioning normally (from normal brainstem, hypothalamic, and cortical inputs), so that this pathway will be recruited in the muscle test to fire the FRA IN pool for the muscle in question which fires directly to the AMNs bypassing the gamma loop. So this powerful input to the AMNs is thought to be adequate to override the lack of stimulation from the Ia reflex, at least at the level of force required to overcome the tester's pressure in the Type III test.

When a maximum amount of force is necessary as in the Type II test, all AMN activity must be recruited which requires the recruitment of the gamma loop. That would explain why the Type II test, in which the patient exerts more force, could demonstrate a pattern of inhibition while the Type III test, in which the patient exerts less force, would demonstrate a pattern of facilitation.

Responses of Three Muscle Test Types to Receptor-Based Challenge Stimuli

Rubbing or stroking the skin over an area of injury is thought to activate MRs that block nociceptive afferents and facilitate previously inhibited muscles, if the inhibition pattern is due to the nociception. In the presence of excessive nociceptive stimulation, the muscle will demonstrate all three types of weakness to testing, with clinical significance placed on the Type III pattern. The clinician can confirm the nociceptive source of neuromuscular inhibition by activating skin MRs (i.e., by gently rubbing the skin over the injured area) and observing a facilitation of the previously inhibited muscle.

In contrast, pinching over an area of injury, recent or ancient, may also create a facilitation of previously inhibited muscles observed with all three types of testing (or at least Type III if it was the only inhibition pattern present) due to excitation of medullary CRN reticular formation neurons that were previously being inhibited from some other source. This is the key to understanding the nature of Type III inhibition pattern.

Pinching activates the nociceptor - spinoreticular - MRST reflex pathway to descend and inhibit incoming nociception, in a similar but slightly different manner than the local MR effect. The MRST, affecting the same INs as the MRs, is also responsive to hypothalamic - reticulospinal influences which (presumably) include the effects of immune system related effects in the hypothalamus. Type III inhibition patterns have been noted clinically in conjunction with immune system problems. These immune system problems presumably can be separate from other hypothalamic - reticulospinal influences mediated through the mesencephalic parabrachial nucleus pattern generator neurons that affect pontine reticular formation neurons. These neurons are the origin of the pontine reticulospinal tract (PRST.) The PSRT neurons synapse on GMNs (and AMNs) but primarily rely on the gamma loop, hence the effect of inhibition in this pathway results only in Type I and Type II inhibition patterns.

Therefore, it is proposed that an immune system problem registers in the hypothalamus that affects descending hypothalamic - MRST pathways that in turn affect INs that directly affect AMNs (no GMNs) that will be associated with Type III inhibition patterns.

Clinical Correlations of the Three Types of Testing

Any combination of the three types of weakness may be present in the same muscle. Patterns of neuromuscular inhibition limited to only one of these three test types are often missed in traditionally performed neurological testing. Clinical observations on the significances of the three different types of tests are as follows.

Type I testing is the most commonly employed type of testing by applied kinesiologists. Procedures that normalize weaknesses of this type have been found to include segmental inputs such as restoring afferentation by correction of a fixated vertebral motion segment, manipulation of trigger areas in muscles and other tissues, or treatment of acupuncture meridian therapy points associated with muscle in question. This type of testing is used for general screening. If weakness is found on clinician started (eccentric) testing, then the muscle is subsequently checked with the other two test procedures.

Type II inhibition patterns are clinically associated with a variety of problems that are presumably suprasegmental. These include chemical imbalances such as nutritional needs, hypothalamic monitored activities such as electrolyte imbalances, general autonomic imbalances, vestibular irregularities, temporomandibular dysfunctions, and other mechanical lesions which are suprasegmental in their origin including lesions of the craniosacral respiratory mechanism. (See previous discussion.) The functional neurological effects of these problems modulate GMNs and AMNs function via various descending suprasegmental pathways such as the reticulospinal tracts, the vestibulospinal tracts, or the rubrospinal tracts.

Type III inhibition patterns are related to withdrawal reflexes following injury (flexor reflex afferents), allergy and hypersensitivity type reactions, systemic functional endocrine imbalances, and visual motor problems such as functional problems with accommodation reflexes.

Oral Nutrient Testing

In 1968, Goodheart introduced testing nutritional substances by monitoring muscle testing responses associated with gustatory stimulation by nutritional substances. Placing substances on the tongue, such as nutrients in which the patient is lacking, were associated with a conditional facilitation of otherwise inhibited muscles. Placing offensive substances on the tongue, such as toxic substances, overdosed medications, and food allergens, were found to be associated with a conditional inhibition of otherwise facilitated muscles.

Afferents from the taste bud receptors of cranial nerves VII, IX, and X synapse in the nucleus of the tractus solitarius with ongoing projections to the thalamus, hypothalamus and cortex. Changes in muscle testing outcomes following taste bud receptor stimulation is hypothesized to be associated with changes in the CIS in the hypothalamus, cortex, or both.

This effect is commonly observed, for example, with gustatory receptor stimulation using syrup of ipecac, which induces an immediate and violent motor response which induces the patient to vomit.

Oral nutrient testing is widely used in AK practice to aid the clinician in making the best choice of nutritional substances, medications, herbs, and other substances when there are numerous possibilities from which to chose. It is also widely employed as a screening test to identify which laboratory evaluation may be best suited to a patient. For example, a patient who shows a strengthening response to insalivation of an anti-histamine would be considered a candidate for allergy testing, regardless of what symptoms are displayed. In this manner, the clinician may efficiently identify dysfunctional physiological processes at the root of patients' symptoms, rather than merely give the symptoms a named diagnosis.

A single blinded, uncontrolled pilot study of AK and allergy testing was performed by Schmitt and Leisman (1998). In this study, 19 of 21 foods associated with muscle weakness on oral challenge showed a subsequent elevation of serum IgE, IgG, and/or IgG immune complexes.

Later anecdotal observations indicate two emerging patterns: 1) strengthening on oral anti-histamine is a useful screening test for allergies, and 2) muscle testing with oral food challenges yields false negatives often enough so as to not be reliable to rule out a food allergy. AK identification of any food allergy reaction is a useful indication to perform allergy laboratory testing in a patient who is not responding adequately to other treatment.

Therapy Localization as a Form of Sensory Challenge

Goodheart (1974) observed that a change in the outcomes of muscle tests could be elicited by having the patient apply their own persistent touching contact to an area of the body in need of treatment. This may be the first recorded recognition that the neurologically mediated effects of touch are diagnostic, as well as therapeutic. The effect became known as "therapy localization" (TL).

Changes observed to occur with TL are hypothesized to be a consequence of alterations in MR afferents from the tissues being stimulated by patient contact. Touching an area of the body increases afferent stimulation from the area, which increases the extent to which that area is represented in brain stem, cerebellum, and cortex. These changes in central representation are reflected as changes in the CIS of neurons in descending motor pathways, affecting the CIS of AMNs.

Therapy localization is extremely valuable in the AK assessment process. Therapy localization allows the clinician to stimulate areas of afferent input to identify those which impact muscle testing

outcomes. The appropriate therapy, designed for the receptors whose stimulation alters motor function in a clinically relevant manner, has been found clinically to return the patient's motor system to a predictable pattern. Following treatment, touching the previously corrected area will have no effect on muscle testing outcomes. This tool helps to make AK assessment quick and precise.

Role of the Parabrachial Nucleus in Disseminated Treatment Effects

The observation of therapeutic effects far afield from a locally applied treatment is common to all complementary and alternative therapies. Structural treatment is often associated with improvements in patient chemistry or emotional states. Biochemical treatment is often associated with resolution of structural or emotional problems. Treatment of emotional problems is often associated with improvement in structural or biochemical symptoms. The treatment of problems in all of these areas is observed clinically to be associated with changes in motor function observable through manual muscle testing. The following discussion proposes the suprasegmental pathways via which these changes in motor function may take place.

In the parabrachial nucleus (PB) in the mesencephalic reticular formation are located groups of neurons which are central pattern generators (CPGs). These CPGs, when stimulated, send efferent signals to groups of cells caudally which cause specific patterns of movement to take place. These stereotyped movements include turning about the midline, flexion-extension, and gait type patterns. These whole body patterned movements are activated when the CPG cells fire (Willis, 1985).

The PB CPGs cause these patterned movements by sending descending efferent signals to neurons in the pontine and the medullary reticular formations which give rise to the descending reticulospinal tracts (RSTs). The RSTs descend to all levels of the brainstem and the spinal cord and, through interneurons, affect AMNs and GMNs which affect muscle function. The RSTs also synapse on IML neurons which are the primary autonomic efferent neurons. Accordingly, activation of PB CPG neurons have facilitation and inhibition effects on muscles throughout the body as well as autonomic function throughout the body.

The PB CPGs receive direct afferent input from several areas which represent structural, chemical, and mental influences on the CIS of the CPGs, including the cerebellum, hypothalamus, and basal ganglia. These changes in CIS yield changes in the impact of CPG efferents on neuromuscular patterns, yielding changes in patterns of conditional facilitation and inhibition observed in the manual testing of muscles.

The cerebellum receives inputs from MRs throughout the body as well as from the cerebral cortex. Inputs from MRs in somatic tissue will change with changes in the status of structural problems. Inputs from the cerebral cortex will change with changes in mental and emotional status. The basal ganglia also impact the CPG in the PB area, giving rise to the synthesis of inputs this area receives, both mental and structural. The hypothalamus, the major neurochemical transducer of the body, is the basis for chemical inputs affecting muscular and autonomic function. The hypothalamus is also impacted by emotional activity which it transmits onward as muscular and autonomic outflow.

CONCLUSIONS

Muscle testing has been observed clinically to be related to many phenomena. But under all circumstances, muscles will only change strength when the CIS of AMNs is either inhibited (weakness) or excited (strength) during the performance of the test.

Muscle testing, applied with the precision used by AK practitioners, serves as a method of functional neurological assessment. Muscle testing has always been an integral part of the neurological exam. With AK techniques, the ability to assess predictable neurological responses to sensory receptor-based challenges of known value has been amplified. The use of AK can assist

the clinician in identifying and treating sources of improper neurologic function in an efficient and accurate manner.

Muscle testing responses to sensory receptor challenges are compared to the predicted normals. These responses guide the clinician in the diagnostic and therapeutic process. Specific types of therapeutic receptor stimulation have two parallel affects in the nervous system. Properly designed receptor activation will normalize afferentation. An additional benefit of normalizing receptor afferentation is the restoration of normal muscle function which increases movement and hence increases joint and other MR activity further increasing afferentation.

Functional disorders can be expressed as many different complaints, ranging from pain to visceral disorders to mental depression. Visceral disorders such as hypochlorhydria, irritable bowel syndrome, and asthma, may be an expression of altered autonomic tone.

Sensory receptor based diagnostic challenges, including nociceptors, mechanoreceptors, visual, and gustatory receptors, may be used to evaluate the functional status of autonomic activity. With these testing procedures, it is no longer necessary to merely describe an organ as dysfunctioning or to guess at its status. Interpretations may be made regarding the SYM or PS imbalance.

The gradual loss of functional integrity of the organism yields a progressive loss of the ability to maintain homeostasis, which may progress to degenerative tissue changes and pathology. This makes the early detection and treatment afforded by AK potentially important. In addition, the diagnostic advantages provided by data derived from the functional neurologic assessment could help the clinician guide therapeutic measures and improve the potential for favorable outcomes.

The significant benefit which these methods appear to provide, along with the favorable outcomes of well designed initial studies, warrants further exploration. The validity of future studies of these methods rests with a proper understanding of their neurophysiologic basis.

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